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Clinical Practice and Cases in Emergency Medicine

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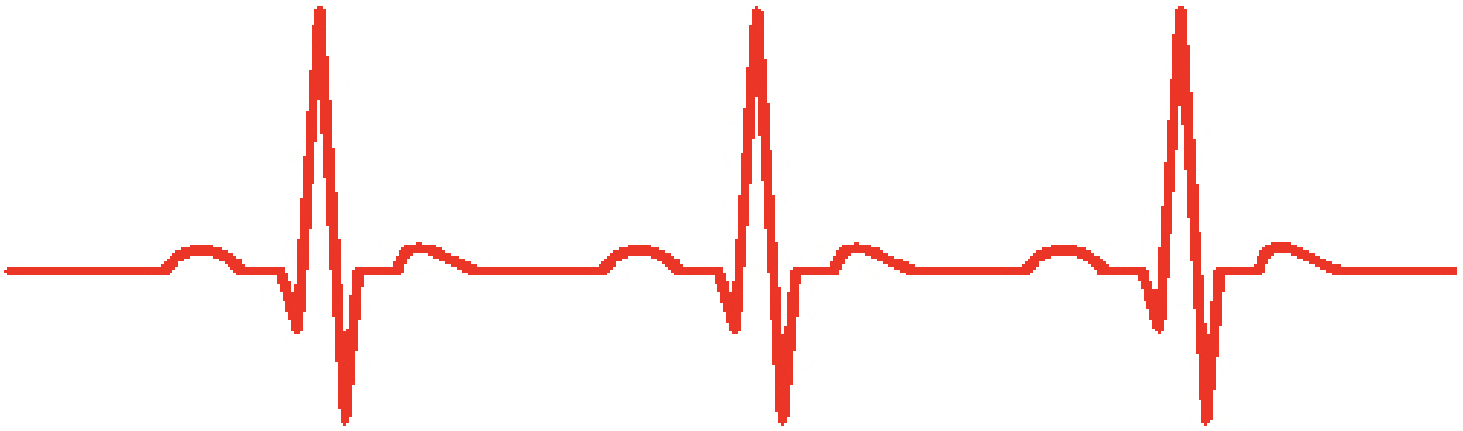

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CPCEM Clinical Practice & Cases

Establishing a Novel Group-based Litigation Peer Support Program to Promote Wellness for Physicians Involved in Medical Malpractice Lawsuits

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Introduction: Being named as a defendant in a malpractice lawsuit is known to be a particularly high-stress and vulnerable time for a physician. Medical malpractice stress syndrome (MMSS) is a consequence of being named as a physician defendant in a malpractice lawsuit. Symptoms include depression, anxiety, and insomnia, which may lead to burnout, loss of confidence in clinical decision-making, substance abuse, strain on personal and professional relationships, and suicidal ideation. Although the legal process requires strict confidentiality regarding the specific details of the legal case, discussing the emotional impact of the case is not prohibited. Given that physicians often do not choose formalized therapy with a licensed professional, there is a recognized need to provide physicians with options to support their wellness during a lawsuit.

Methods: The peer support model is a promising option to address the negative impacts to wellness that physician defendants face during medical malpractice lawsuits. We developed and implemented a peer support program to provide a safe, protected space for discussion of the personal impact of a lawsuit and to normalize this experience among peer physicians.

Results: Physicians were receptive to joining a peer support group to discuss the personal impacts of being named in a medical malpractice lawsuit. Participants in this novel group-based program found it helpful and would unanimously recommend it to others who are being sued.

Conclusion: To our knowledge, this pilot study is the first to implement and assess a facilitated, group-based peer support model for emergency physicians who are named as defendants in malpractice lawsuits. While group discussions demonstrated that symptoms of acute distress and MMSS were prevalent among physicians who were being sued, in this study physicians were receptive to and felt better after peer support sessions. Despite increasing burnout in the specialty of emergency medicine (EM) during the study time frame, burnout did not worsen in participants. Extrapolating from this pilot program, we hypothesize that formal peer support offered by EM groups can be an effective option to normalize the experience of being sued, promote wellness, and benefit physicians who endure the often long and stressful process of a medical malpractice lawsuit. [Clin Pract Cases Emerg Med. 2023;7(4)205–209.]

Keywords: *medical malpractice; peer support; medical malpractice stress syndrome; lawsuit; physician wellness.*

INTRODUCTION

Physicians are at higher risk of occupational burnout, depression, and suicidal ideation compared to the general population. In high-risk specialties, up to 99% of physicians will be sued for medical malpractice by age 65.[1] The additional stress from a lawsuit can increase negative impacts to physician mental health.[2–4]

Medical malpractice stress syndrome (MMSS) is a constellation of physical and mental symptoms and sequelae that physicians may experience during a medical malpractice lawsuit.[5,6] Examples include anxiety, depression, insomnia, stress on personal/professional relationships, and practicing defensive medicine. Physicians can be reluctant to seek help to address stress or improve their mental well-being. In a recent survey of emergency physicians, almost half endorsed a reluctance to seek mental health resources despite 87% of respondents reporting increased stress and 72% suffering from increased burnout since the beginning of the COVID-19 pandemic.[7]

Findings from a survey of physicians seeking mental health services found that physician colleagues were the preferred source of support (88%) compared to employee assistance programs (29%) and mental health professionals (48%).[8] In addition, physicians may be reluctant to engage with others at the outset of a lawsuit due to fear of discoverability and advice from their defense counsel to not discuss the legal case details with others. Study participants were told that they could not discuss any details about their actual cases because any conversations other than those with their lawyers or spouses are potentially discoverable in court. However, they were able to discuss their feelings, coping strategies, and general legal processes.

To our knowledge, there is no published literature regarding the use of group-based peer support for physicians who are named in a medical malpractice lawsuit. This feasibility study describes the development and implementation of a novel litigation peer support program for emergency physicians who are defendants in medical malpractice lawsuits and measures physician receptivity to the program. Further, measures of physical and mental symptoms were piloted on a small scale.

METHODS

Development and Implementation of Litigation Peer Support Program

The study population was recruited voluntarily from a department of emergency medicine (EM) comprised of more than 200 physicians and non-physician practitioners (NPP) who staff three academic and seven community emergency departments. After department chair approval, a team of key personnel was assembled to develop the program, including the vice-chair for clinical affairs, who was informed of all active lawsuits involving emergency physicians and NPPs; the vice-chair of faculty development, who had experience with the peer support model; an attorney from our institution's risk retention group (RRG), and emergency physicians with a breadth of expertise in clinical operations, research, and law. The protocol for the litigation peer support program was reviewed and approved by the institutional review board and the RRG.

Emergency physicians and NPPs who were identified as defendants in active lawsuits were contacted confidentially by the vice-chair for clinical affairs. They were invited and consented to participate in a series of voluntary, virtual, one-hour, peer support sessions. Facilitators for the groups were volunteer emergency physician peers who had previously been sued. Facilitator training was created and delivered based upon American Medical Association guidelines for peer facilitators and resources provided by the National Alliance on Mental Illness.[9–12] Facilitator training included both individual preparations lasting approximately three hours and an online group session lasting 90 minutes. New facilitators "shadowed" for one 60-minute peer support session prior to leading a session.

Although the risk of a mental health emergency during a group session is very low, there is potential to trigger traumatic events. A safety plan was developed in case a mental health emergency was identified during a session. A mental health emergency was defined as active suicidal ideation, homicidal ideation, or acute psychosis.

Based on advice from RRG attorneys, physicians involved in the same lawsuit were separated into different cohorts. Although two NPPs were invited to participate in this study, none enrolled. Thus, our study only included physician participants. The program began with two cohorts of 8–10 physicians and two co-facilitators who met monthly during a three-month pilot study.

Litigation Peer Support Session Structure

The structure of each session was adapted from a model that used group-based peer support for physicians during the COVID-19 pandemic.[11] Physicians were asked to participate in the sessions from a location that ensured privacy. Facilitators opened with a brief overview of and guidelines for the sessions. Participants agreed to confidentiality and avoidance of any discussion of an active lawsuit. They were reminded that peer support is not formal therapy and that individuals who perceived a need for additional mental health assistance could be referred to appropriate services. Study participants were told that they could not discuss any details about their actual cases because any conversations other than those with their lawyers or spouses are potentially discoverable in court. However, they were able to discuss their feelings, coping strategies, and general legal processes.

The purpose of the sessions was to allow participants a space to process their experiences and use group wisdom to

Figure 1. Anatomy of a virtual session for litigation peer support.

apply principles of support.[11] To accomplish this, the basic structure of the support group model included three activity components (Figure 1). Each session began with a 2–3 minute "check-in" by each participant. Physicians introduced themselves and briefly summarized their current state of mind related to their lawsuit and/or what led them to join the group. Next, facilitators used supportive communication to transition into an organic discussion that stemmed from topics mentioned by one or more of the participants during check-in. Finally, facilitators transitioned to closing each session positively by inviting each participant to take 1–2 minutes to highlight a helpful takeaway (Figure 1).

Program Assessment and Data Analysis

To determine the feasibility and receptivity of the program, we used enrollment and attendance rates in addition to a global change rating (GCR) and a net promoter score (NPS) after sessions.[11] Using customized and validated tools we measured changes in acute distress symptoms and burnout before and after sessions and analyzed the changes using dependent *t*-tests.[13–15] Data was collected via voluntary Qualtrics surveys (Qualtrics International Inc, Provo, UT). A customized checklist tool tracked MMSS symptoms and sequelae discussed during each session.[6]

RESULTS

A total of 28 physicians and two NPPs with active lawsuits from three academic and seven community EDs were invited to participate in the pilot study. Of the 28 invited, 18 physicians (64%) enrolled, with 17 (61%) attending one to three peer support sessions. Two NPPs and 10 physicians declined participation for variety of reasons (Figure 2). Of the attendees, all were physicians, 41% identified as female, and 24% as non-White.

Receptivity among physicians participating in the program was high. Physicians were willing to discuss 77% of MMSS symptoms and 86% of MMSS sequelae during the sessions. At the end of the sessions, 96% of participants felt better, with the remainder reporting no change on the GCR. At the end of program participation per the NPS, 100% of physician participants would recommend peer support to colleagues being sued. Of the 18 physicians who participated,

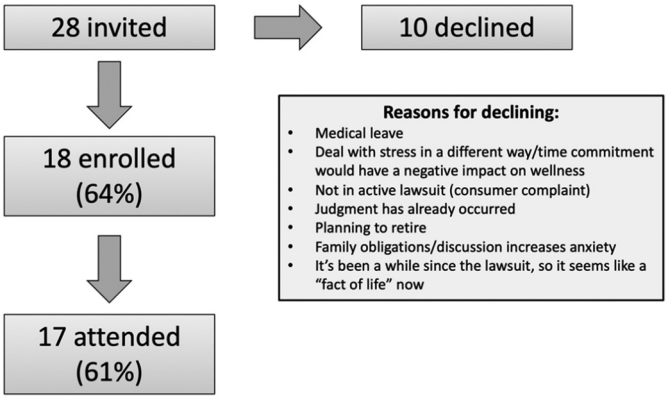

Figure 2. Pilot program feasibility.

15 wanted additional sessions, which subsequently continued quarterly. Several participants expressed interest in becoming facilitators for future groups.

Physician burnout at baseline on the single-item Maslach Burnout Inventory was a mean of 2.93 (scale 0–6), indicating "a few times a month" and remained stable after peer support. At baseline, 73% of physicians reported at least seven of eight acute distress symptoms, with fatigue, anxiety, and insomnia most prevalent. The positive effect size suggested modest improvement in insomnia and depression after peer support but did not reach significance ($P > 0.1$) (Table).

DISCUSSION

We were successful in adapting the peer support model, training facilitators, recruiting participants, and providing a safe space to discuss the personal impact of a lawsuit with peers. The groups adhered to the pre-established guidelines during the sessions and no details of active lawsuits were discussed during the sessions. While group discussions demonstrated that symptoms of acute distress and MMSS were prevalent among physicians who were being sued, physicians were receptive to talking about them and felt better after peer support sessions. Despite increasing burnout in the specialty of EM, burnout did not worsen in participants during the study time frame.

This program has the potential of being discoverable during a lawsuit, even if no specific case information is

Table. Physician burnout and acute distress symptoms pre- and post-peer support sessions (N = 17).

Symptoms	Score range	Mean score pre-intervention (SD)	Mean score post-intervention (SD)	95% CI of the difference	*P*-value[3]	Effect size correlation[4]
Burnout[1]	(0–6)	2.93 (1.62)	3.0 (1.49)	−1.26 to 1.40	0.91	−0.04
Fatigue (tiredness)[2]	(0–10)	4.87 (2.61)	5.30 (2.31)	−1.68 to 2.55	0.68	−0.18
Trouble sleeping (insomnia)[2]	(0–10)	3.0 (2.04)	2.50 (1.84)	−2.16 to 1.16	0.54	**0.26**
Anxiety (nervousness)[2]	(0–10)	3.47 (2.62)	3.30 (1.89)	−2.14 to 1.84	0.88	0.06
Low mood (feeling down)[2]	(0–10)	3.20 (2.31)	3.20 (2.78)	−2.11 to 2.11	1.0	0
Difficulty concentrating[2]	(0–10)	2.87 (2.26)	2.80 (1.87)	−1.86 to 1.72	0.94	0.03
Anger[2]	(0–10)	1.47 (1.46)	1.80 (1.62)	−0.95 to 1.62	0.60	−0.22
Depression (helplessness)[2]	(0–10)	2.60 (2.87)	2.20 (2.66)	−2.76 to 1.96	0.73	**0.15**
Guilt[2]	(0–10)	2.60 (2.44)	3.10 (2.23)	−1.50 to 2.50	0.61	−0.22

[1]Single-item Maslach Burnout Inventory.
[2]SPADE (sleep, pain, anxiety, depression, and low energy/fatigue) and PROMIS (Patient-Reported Outcomes Measurement Information System) measures.
[3]*P*-value <0.05 indicates significance.
[4]Positive effect size correlation represents improvement.
CI, confidence interval; *SD*, standard deviation.

mentioned during a session. Peer support meetings are not legally protected from discovery in the way that privileged conversations such as those between an attorney and client or between spouses are protected. Avoiding mention of case specifics minimizes risk to the defendant. In addition, our attorneys advised that in the unlikely event that a plaintiff attorney inquired about the peer support process, they felt that participation in this program had an extremely unlikely chance of negatively impacting a defendant's case. In fact, the act of seeking mental health support may positively impact the defendant as it humanizes them, which plaintiff attorneys generally attempt to avoid.

Lessons Learned

We faced several challenges in implementing this program. More time than anticipated was required to obtain approval. There was some difficulty in gathering an accurate list of defendants named in active lawsuits from the RRG. The logistics of inviting and scheduling participants required significant time from the vice chair for clinical affairs to maintain confidentiality.

Although we did not perform a formal qualitative analysis of the session due to privacy concerns, facilitators did report that younger physicians and those enduring their first lawsuit seemed to be disproportionately impacted when they were named in a lawsuit. Topics of discussion varied between the sessions and included specific physical and mental health symptoms, general legal processes, and the impact of cases on current clinical practice, teaching, or interpersonal relationships. Often the discussions sparked sharing experiences and ideas for coping strategies. Humor was a common addition to the meetings. In the observed group meetings, guilt, anger, frustration, and self-doubt were some of the emotions that were commonly discussed. One participant described their feelings when they learned that a co-defendant had committed suicide. "Suffering in silence," "gut-punched," and "shattered confidence" were phrases that physicians used during the sessions to describe their experiences of being sued.

Three of the 10 hospitals represented in this program are academic teaching sites. Anecdotally, we discovered that several physicians from those hospitals who participated in this program felt more comfortable discussing their experiences of being sued with learners. Introducing the uncomfortable topic of the personal impact of being sued may have benefits for medical students, residents, and fellows.

LIMITATIONS

There are some limitations to this study. Participants included physicians from one organization and two specialties (EM and pediatric EM). While participation was voluntary, it was encouraged by departmental leadership through faculty meetings and newsletters, which may have influenced receptivity. No NPPs chose to participate. While the physicians and NPPs who declined participation provided a variety of reasons, it is possible that some may

have been hesitant due to concerns regarding anonymity or discussion of topics that they viewed as stressful or triggering. Although there may be varied opinions regarding future inclusion of NPP defendants who seek support during a medical malpractice lawsuit, our department made a thoughtful decision to offer this program to our NPPs. This decision may be institution-specific and vary depending upon employment models and culture. Finally, the number of participants was relatively small, limiting the power to detect changes in distress or burnout. In addition, the number of sessions offered was limited and may not have been of sufficient strength to anticipate change in these outcomes.

In the future we will offer quarterly sessions, expand the sample size, and track longer term changes in distress, burnout, and wellness factors. In addition, we plan to evaluate barriers to participation, develop train-the-trainer materials, and examine generalizability to other specialties.

CONCLUSION

Based on this study, formal peer support offered by EM groups is feasible and well received by physicians. Although results of preliminary effectiveness show promise, larger studies need to be conducted to establish that peer support groups can be an effective option to normalize the experience of being sued, promote wellness, and benefit physicians who are defendants in medical malpractice lawsuits.

This study was approved by the Indiana University Institutional Review Board (#11751).

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Mpox in the Emergency Department: A Case Series

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Introduction: We sought to describe the demographic characteristics, clinical features, and outcomes of a cohort of patients who presented to our emergency departments with mpox (formerly known as monkeypox) infection between May 1–August 1, 2022.

Case Series: We identified 145 patients tested for mpox, of whom 79 were positive. All positive cases were among cisgender men, and the majority (92%) were among men who have sex with men. A large number of patients (39%) were human immunodeficiency virus (HIV) positive. There was wide variation in emergency department (ED) length of stay (range 2–16 hours, median 4 hours) and test turnaround time (range 1–11 days, median 4 days). Most patients (95%) were discharged, although a substantial proportion (22%) had a return visit within 30 days, and 28% ultimately received tecrovirimat.

Conclusion: Patients who presented to our ED with mpox had similar demographic characteristics and clinical features as those described in other clinical settings during the 2022 outbreak. While there were operational challenges to the evaluation and management of these patients, demonstrated by variable lengths of stay and frequent return visits, most were able to be discharged. [Clin Pract Cases Emerg Med. 2023;7(4)210–214.]

Keywords: *mpox; monkeypox; case series; operations; pandemic response.*

INTRODUCTION

Mpox is a viral illness endemic to parts of West and Central Africa that causes fever, lymphadenopathy, and a rash that typically evolves over the course of weeks.[1] Transmission often occurs via contact with an infected animal, although human-to-human transmission has been documented through several mechanisms.[1] Structurally, the mpox virus is closely related to variola virus, which causes smallpox.[2] Vaccination with live (ACAM2000) or attenuated (JYNNEOS) vaccinia virus, which are also used to protect against smallpox, is thought to confer protection against mpox.[2] Management of mpox is mainly supportive, although tecovirimat (TPOXX) is an emerging antiviral treatment.[2]

In 2022, an outbreak of mpox occurred internationally among patients who had never traveled to endemic regions.[1] The first cases of mpox in the United States were noted in May 2022, with average daily case counts reaching a peak of approximately 450 nationally and 70 in New York City (NYC) in early August 2022, and have since declined.[3] The reasons for this decline remain to be fully elucidated but may be due in part to vaccination efforts[4] and behavioral changes within at-risk populations.[5]

During the early months of the outbreak, there were anecdotal reports of clinicians turning away patients with suspected mpox.[6] Without alternative options, emergency departments (ED) became one of the primary sites of care for patients with suspected mpox.[7] Here, we describe a case series of all patients who tested positive for mpox between May 1–August 1, 2022 at two urban, high-volume EDs with annual visits of greater than 100,000 patients per year. We created a deidentified, structured, case-series spreadsheet based on variables of interest (Appendix). These variables

were derived from previous studies and our clinical experience.[1] We then reviewed patient charts retrospectively and entered data into this spreadsheet. Descriptive statistics were derived from this review.

CASE SERIES

During this period, 145 patients were identified as persons under investigation (PUI) and tested for mpox. All samples were collected in the ED and submitted to the NYC Public Health Laboratory. There were 94 PUIs at the Bellevue Hospital Center (BHC) ED and 61 at the New York University Langone Medical Center (NYULMC) ED. A total of 79 (54%) PUIs had a positive mpox test, 46 (32%) had a negative mpox test, and 20 (14%) had missing or inconclusive tests. Demographic characteristics of positive mpox cases are described in Table 1.

All mpox cases in our series were among cisgender men, 92% of which were among men who have sex with men (MSM). In 30 (39%) of the mpox cases, patients reported being human immunodeficiency virus (HIV) positive. Of these cases, 19 were well controlled (defined as a cluster of differentiation 4 [CD4] count greater than 200 cells per microliter (μL) and an undetectable HIV RNA; three were poorly controlled (defined as a CD4 count less than 200 cells/μL and/or a detectable HIV RNA); and eight did not have a recent CD4 count documented. Of mpox cases reported, 14% received at least one dose of the JYNNEOS vaccine. Of note, JYNNEOS vaccination for high-risk individuals without known exposure to mpox did not become available in NYC until late June 2022.

Demographic characteristics of positive mpox cases are described in Table 2. Skin lesions were noted in all mpox cases. Of the positive cases, 72% were found to have genital lesions, and 77% were found to have at least one systemic symptom. Systemic symptoms included fever (58%), chills (22%), lymphadenopathy (35%), myalgias (33%), and sore throat (13%). The most common complications were proctitis (23%), rectal bleeding (10%), cellulitis (10%), phimosis (6%), and eyelid involvement (3%).

Management and outcomes of positive mpox cases are described in Table 3. In addition to symptomatic management, some mpox cases were empirically treated for gonorrhea and chlamydia (16%), syphilis (6%), herpes simplex virus (HSV) (4%), bacterial pharyngitis (6%), and cellulitis (10%). Among patients with mpox who were tested for co-infection with sexually transmitted infections (STI), 9% had chlamydia, 13% had gonorrhea, and 12% had syphilis. None who were tested for herpes simplex virus were found to be positive. There was one new HIV diagnosis.

Among mpox cases, the median length of stay (LOS) in the ED was four hours, and 95% were discharged from the ED. Among the four who were admitted, three were HIV positive, and two had CD4 counts less than 200 cells/μL. The reasons for admission included inability to isolate, psychiatric illness requiring admission, and severe mpox symptoms. Median length of hospital admission was 4.5 days. Twenty-two percent of mpox cases had a return visit to our ED for a reason related to mpox infection. The median time from ED visit to receiving a test result was four days. Ultimately, 28% received TPOXX from our institutions.

CPC-EM Capsule

What do we already know about this clinical entity?
Mpox is a zoonotic disease endemic to parts of Africa. In 2022, an outbreak of mpox occurred internationally.

What makes this presentation of disease reportable?
This case series highlights the demographic and clinical characteristics of 79 mpox cases in an urban emergency department in the United States.

What is the major learning point?
Patients with mpox in this case series were primarily men who have sex with men; most cases were managed in the outpatient setting with the help of telemedicine.

How might this improve emergency medicine practice?
Response to future infectious disease outbreaks might benefit from establishing protocols to evaluate, manage, and follow up with patients.

DISCUSSION

The recent mpox epidemic disproportionally affected men, MSM, and people who live with HIV.[1] Patients with mpox in the recent outbreak often had co-infection with STIs.[1] This data has been reflected in reports from the United Kingdom,[8] Spain,[9] and the US.[10] Data from our case series is consistent with those of previous studies but is unique in its focus on the ED as the site of care.

We noted a wide range in the ED LOS. In our clinical experience, one major contributor to LOS was obtaining approval for mpox testing from the NYC Public Health Laboratory. During most of the period of this case series, this was the only way to obtain a test. The process could take anywhere from a few minutes up to several hours. Other potential contributors to LOS included the following:

Table 1. Demographic characteristics of mpox cases.

Measure	Mpox cases (N = 79)
Median age (range, SD) – years	34 (21–61, 8. 9)
Gender – no. (%)	
Male	79 (100%)
Female	0 (0%)
Transgender	0 (0%)
Race – no. (%)	
White	34 (43%)
Black	17 (22%)
Asian	2 (3%)
Native American or Pacific Islander	1 (1%)
Other	20 (25%)
Unknown	5 (6%)
Ethnicity – no. (%)	
Hispanic	23 (29%)
Non-Hispanic	55 (70%)
Unknown	1 (1%)
Sexual orientation – no. (%)	
MSM	73 (92%)
Non-MSM	3 (4%)
Unknown	3 (4%)
HIV status – no. (%)	
Positive	30 (38%)
Negative	40 (51%)
Unknown	9 (11%)
Mpox vaccine status – no. (%)	
Vaccinated	11 (14%)
Unvaccinated	36 (46%)
Unknown	32 (41%)

No, number; *MSM*, men who have sex with men.

Table 2. Clinical characteristics of mpox cases.

Measure	Mpox cases (N = 79)
Skin lesions – no. (%)	79 (100%)
Non-genital lesions	63 (80%)
Genital lesions	57 (72%)
Penile lesions	38 (48%)
Rectal lesions	25 (32%)
Oral lesions	12 (15%)
Systemic symptoms – no. (%)	61 (77%)
Fever	46 (58%)
Chills	17 (22%)
Lymphadenopathy	28 (35%)
Myalgias	26 (33%)
Sore throat	10 (13%)
Complications – no. (%)	
Penile edema/phimosis	5 (6%)
Rectal pain/proctitis	18 (23%)
Rectal bleeding	8 (10%)
Bacterial superinfection	8 (10%)
Ocular involvement (eyelid)*	2 (3%)
Chlamydia co-infection	
Tested – no. (%)	23 (29%)
Positive – no. (% tested)	2 (9%)
Gonorrhea co-infection	
Tested – no. (%)	23 (29%)
Positive – no. (% tested)	3 (13%)
Syphilis co-infection	
Tested – no. (%)	26 (33%)
Positive – no. (% tested)	3 (12%)
Herpes simplex virus co-infection	
Tested – no. (%)	14 (18%)
Positive – no. (% tested)	0 (0%)
HIV co-infection	
Tested – no. (% without known HIV-positive status)	24 (49%)
Positive – no. (% without known HIV-positive status tested)	1 (4%)

*None of these patients had intraocular lesions or vision changes noted.
No, number; *HIV*, human immunodeficiency virus.

variable familiarity of hospital staff with the mpox testing process leading to delays in collecting the test; stigma associated with mpox resulting in reticence of staff members to enter rooms; and the time required to prepare and terminally clean each isolation room. Additionally, at BHC there was a special pathogens team that was consulted to evaluate and perform testing of patients under investigation (PUIs). The addition of a consulting service may have contributed to ED LOS.

We also noted that more than one in five mpox cases had a second visit to the ED during the study period. Reasons for return visits included worsening symptoms, not having been tested for mpox during their first visit, request for TPOXX prescription, and work clearance. Although some revisits could potentially have been prevented by better recognition and management of symptoms during the initial visit, others were the natural consequence of the ED being the primary site of care for these patients. Although the ED is often a patient's primary site of care, return visits could potentially be avoided by improvements in arranging follow-up care.

Admissions were rare and tended to occur in patients with complex medical, psychiatric, and social histories.

Table 3. Management and outcomes of mpox cases.

Measure	Mpox cases (N = 79)
Empiric treatment	
Gonorrhea and chlamydia – no. (%)	13 (16%)
Syphilis – no. (%)	5 (6%)
Herpes simplex virus – no. (%)	3 (4%)
Strep pharyngitis – no. (%)	5 (6%)
Cellulitis – no. (%)	8 (10%)
Disposition – no. (%)	
Discharged	75 (95%)
Admitted	4 (5%)
Median inpatient admission length of stay (range, SD) – days	4.5 (4–9, 2.4)
Median emergency department length of stay (range, SD) – hours[†]	4 (2–16, 3.0)
Median test turnaround time (range, SD) – days	4 (1–11.2)
TPOXX prescribed – no. (%)	22 (28%)
Revisits related to mpox within 30 days – no. (%)	17 (22%)

[†]For patients with multiple visits, length of stay was determined from the emergency department visit during which they were tested.
No, number; *SD*, standard deviation; *TPOXX*, tecovirimat.

The majority of patients could potentially have been managed outside the ED, underscoring the lack of sufficient outpatient services for people with mpox during this time. This was true in both public and private healthcare environments. Ultimately, 28% of mpox cases received TPOXX. We attribute this success to institutional protocols at each of our sites, which involved designating a member of the special pathogens team (at BHC) or ED follow-up center (at NYULMC) to arrange follow up with PUIs who tested positive for mpox. The majority of these follow-up visits occurred via telehealth, underscoring the importance of creating a multidisciplinary team across various clinical environments for managing public health emergencies.

LIMITATIONS

While our findings suggest a pattern of demographic and clinical characteristics that should raise suspicion for mpox infection, we were unable to demonstrate statistical correlation using our study design. Additionally, while our data is consistent with that of other studies from the 2022 outbreak, the current epidemiological landscape may be different, particularly with the widespread vaccination effort among at-risk populations. Finally, although our study did take place at multiple sites, it suffered from local bias and may not be reflective of the experience outside NYC.

CONCLUSION

Overall, our findings demonstrate that patients who presented to the ED with mpox infection were similar to those who presented in other clinical settings with regard to demographics, clinical features, and co-infections. While there were significant operational challenges to the management of these patients in the ED—demonstrated by variable lengths of stay and frequent return visits—potential solutions were identified along the way, most notably the use of telemedicine to arrange follow up. Most patients were ultimately able to be discharged.

Approval for this case series was provided by the institutional review board of NYU Grossman School of Medicine and New York City Health and Hospitals (Protocol i22-01047).

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Spontaneous Coronary Artery Dissection: A Case Series Reviewing Typical Presentations of an Atypical Pathology

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Introduction: Spontaneous coronary artery dissection (SCAD) is a rare cause of myocardial infarction. Patients suffering SCAD are often young women without typical risk factors for atherosclerotic heart disease. Clinicians should maintain a high index of suspicion for SCAD.

Case series: We report three cases of patients with SCAD from a single physician in a six-month period. Each case is unique and highlights the varied presentations and epidemiological risk factors associated with this condition.

Discussion: We believe these cases are unique in that they provide insights into the variable presentations and conditions frequently associated with SCAD and will help clinicians maintain a high index of suspicion for this difficult to diagnose and rare cause of type 2 myocardial infarction. We discuss differences in interventional techniques and medical management. [Clin Pract Cases Emerg Med. 2023;7(4)215–220.]

Keywords: *case report; myocardial infarction; spontaneous coronary artery dissection; ECG.*

INTRODUCTION

Spontaneous coronary artery dissection (SCAD) is an uncommon cause of myocardial infarction (MI) without underlying acute coronary atherothrombosis, often occurring in individuals with few atherosclerotic risk factors.[1] Spontaneous coronary artery dissection occurs when an epicardial coronary artery dissects without a clear precipitant as in penetrating atherosclerotic plaque or trauma. Vessel dissection may result in coronary artery obstruction caused by formation of an intramural hematoma or intimal disruption at the site of dissection—resulting in myocardial ischemic injury.[1]

Knowledge of the epidemiology and risk factors can heighten a clinician's suspicion for SCAD in the appropriate patient population, leading to early diagnosis and possibly improved prognosis. In this case series, we discuss three patients who presented with SCAD to a single physician over a six-month period, each illustrating key points related to the natural history of the disease. This case series is unique as it presents a spectrum of risk factors and patient presentations that emergency physicians may encounter; we also discuss management strategies. All patients provided written informed consent.

CASE SERIES

Case 1

A 43-year-old female with a history of migraines presented to an urban, university-affiliated emergency department (ED) after she developed unrelenting chest pain and shortness of breath. The pain began following her normal exercise routine, and she reported that the pain radiated to her back and down her left arm. She was hypertensive on

arrival in the ED, including blood pressure of 149/90 millimeters of mercury (mm Hg) and a heart rate of 92 beats per minute. She was noted to be uncomfortable and in moderate distress.

Based on her chief complaint, an electrocardiogram (ECG) was performed on arrival that demonstrated prominent precordial t-waves. The differential diagnosis for those findings included hyperacute T-waves, benign early repolarization, and pericarditis (Image 1). Serial ECGs remained unchanged. Laboratory work-up revealed elevated cardiac enzymes: her initial troponin-I resulted at 0.12 nanograms per milliliter (ng/ml) (normal troponin-I < 0.03 ng/ml). Despite having no conventional risk factors, the patient's unremitting chest pain and elevated cardiac biomarkers were concerning for ongoing acute coronary syndrome (ACS).While her ECGs did not evolve during her diagnostic work-up, her troponin-I continued to rise, peaking at 20.8 ng/ml. She was taken for urgent angiography, which demonstrated SCAD of the left anterior descending artery (LAD), originating just after the origin of the first diagonal artery and continuing to the apex.

Based on the catheterization findings, the decision was made to continue medical management: 72 hours of anticoagulation followed by a year of dual antiplatelet therapy (DAPT). Echocardiography showed normal left ventricular function with a normal ejection fraction (EF); no apical wall motion abnormalities were identified. Her chest pain resolved, and she was discharged in stable condition on DAPT and beta-blockers.

CPC-EM Capsule

What do we already know about this clinical entity?
Spontaneous coronary artery dissection (SCAD) occurs when an epicardial coronary artery dissects without a clear precipitant.

What makes this presentation of disease reportable?
Each case in this series highlights the varied presentations and epidemiological risk factors associated with SCAD.

What is the major learning point?
Spontaneous coronary artery dissection is a rare cause of myocardial infarction, most common in young women without typical risk factors for heart disease.

How might this improve emergency medicine practice?
Clinicians should maintain a high index of suspicion for this pathology. We discuss differences in interventional techniques and medical management.

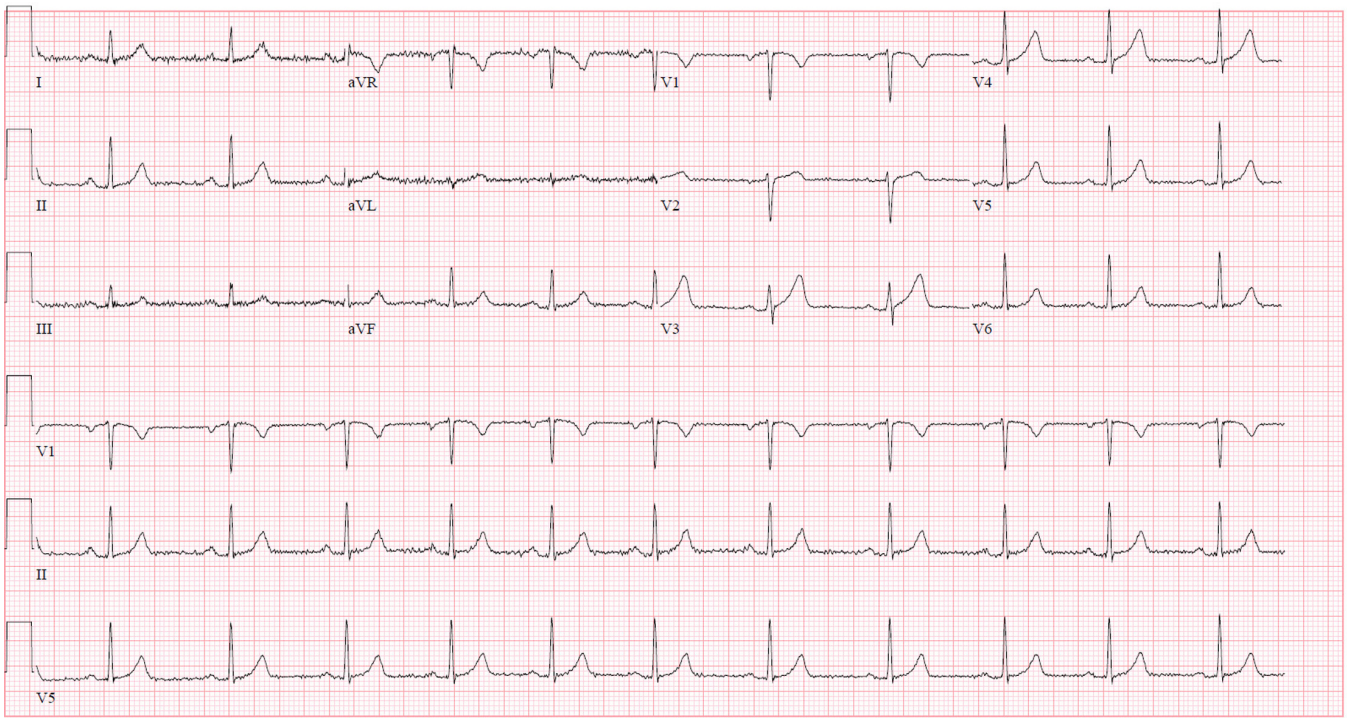

Image 1. Sinus rhythm with prominent T-waves in leads V3–V4.

Case 2

A 45-year-old female with a history of hypertension, fibromuscular dysplasia, and cerebral aneurysm with previous clipping presented to the ED after severe non-radiating chest pain awakened her from sleep. On arrival, the patient was hypertensive (150/91 mm Hg), but the remainder of her vital signs were normal. While in the ED, she began experiencing dyspnea and nausea. She was noted to be uncomfortable, diaphoretic, and pale, but no cardiopulmonary abnormalities were identified on physical exam.

Her ECG on arrival demonstrated ST-segment depression in leads V2–V6 and subtle ST-segment depression/T-wave inversions in the inferior leads. She continued to have unrelenting chest pain, and her repeat ECG showed ST-segment elevation in leads I and aVL concerning for a high-lateral ST-segment elevation myocardial infarction (STEMI) (Image 2). Nitroglycerin administration provided little improvement of her symptoms; she received aspirin, ticagrelor, and anticoagulation with heparin infusion.

She was taken for emergent left heart catheterization, which demonstrated SCAD of a small diagonal branch of the mid-LAD. There were no lesions amenable to percutaneous coronary intervention (PCI). After catheterization, her chest pain resolved, and her troponin-I peaked at 23 ng/ml. Echocardiography showed normal ventricular function without significant wall motion abnormality. She was discharged on an aggressive regimen of blood pressure control that included rate-control agents to prevent propagation of the dissected area.

Case 3

A 33-year-old female, who was two weeks postpartum after cesarean section for preterm premature rupture of membranes, was brought to the ED by emergency medical services (EMS) for crushing chest pain. The initial ECG provided by EMS showed a sinus tachycardia with no signs of ischemia or ST-segment changes. On arrival to the ED, the patient had an episode of seizure-like activity and became unresponsive.Advanced Cardiac Life Support was initiated after the patient was found to be pulseless with agonal respirations. During the first rhythm check, the patient's ECG demonstrated ventricular fibrillation. After two attempts at defibrillation, return of spontaneous circulation was obtained. Her post-arrest ECG showed anterolateral and inferior ST segment elevation (Image 3).

During this ongoing resuscitation, the case was discussed with the multidisciplinary Heart Attack Team at our institution, and the cardiothoracic surgery service was alerted for possible extracorporeal membrane oxygenation cannulation. The interventional cardiology service agreed to take the patient for emergent PCI but maintained a high suspicion for SCAD.The patient was found to have SCAD from the ostium of the LAD to the apex, involving nearly all the diagonal branches. The LAD appeared to be 80% stenosed from intrusion of the ostium of the LAD into the

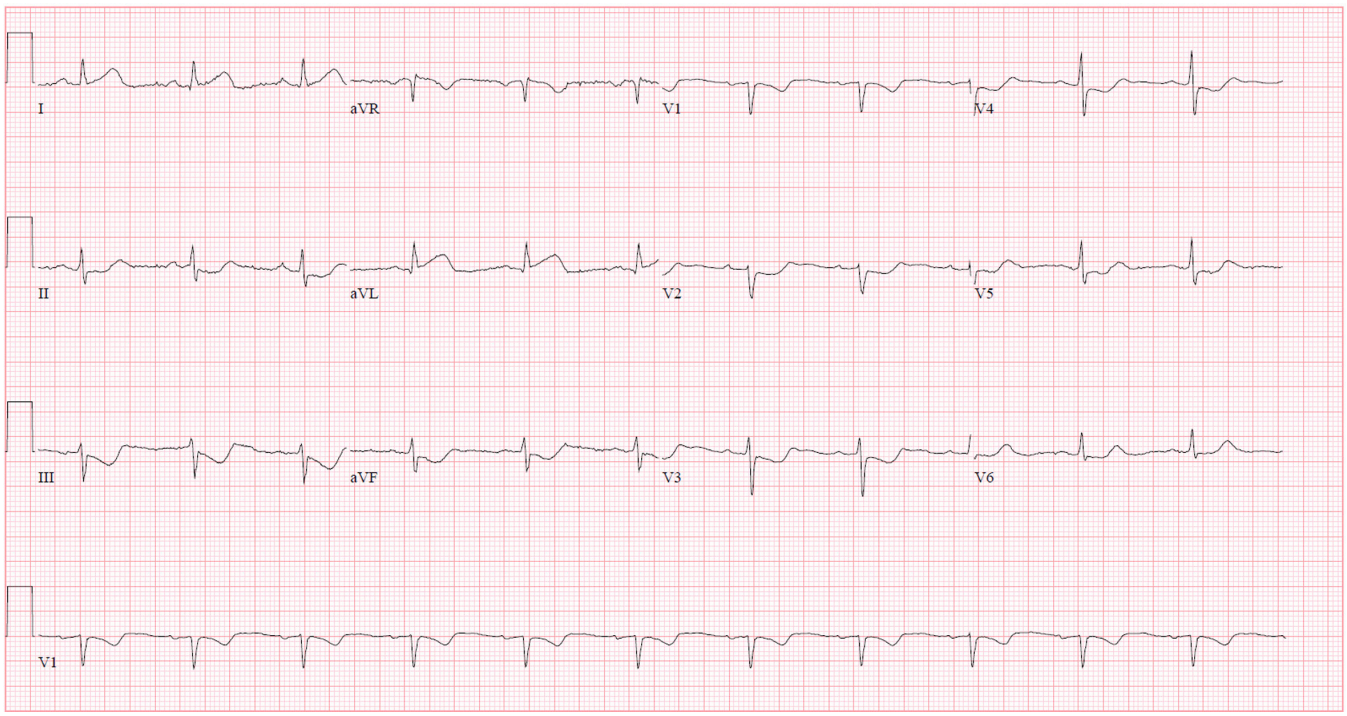

Image 2. Sinus rhythm with ST-segment elevation in leads I and aVL and ST segment depression in leads II, III, aVF, and V2-V5. This electrocardiogram is concerning for high-lateral infarction involving the left anterior descending or left circumflex artery.

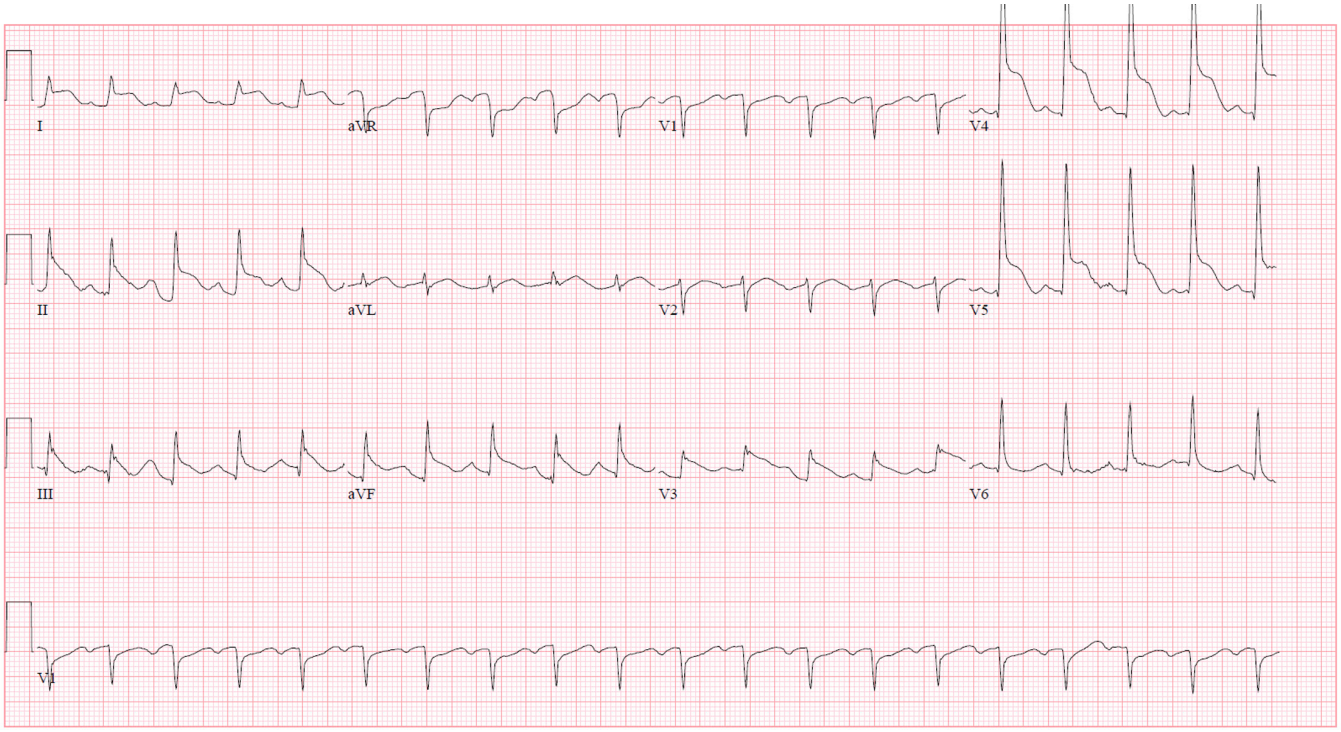

Image 3. Sinus rhythm with ST-segment elevations in leads I, II, III, aVF, and V3-V5 as well as ST-segment depression in aVR and V1. This electrocardiogram is concerning for a large territory. left anterior descending artery infarction with probable left circumflex artery involvement.

true lumen of the vessel. Percutaneous coronary intervention was avoided in the setting of demonstrated thrombolysis in myocardial infarction (TIMI)-3 flow and the increased risk that revascularization posed of propagating the dissection. The patient's care team elected for medical management in the cardiac intensive care unit (CCU).

In the CCU, her ST-segment elevation resolved, and the patient became free of chest pain. Echocardiography demonstrated reduced left ventricular function (EF 25–30%) with apical ballooning. Four days later the patient developed recurrent chest pain, anterior ST-segment elevation and an elevated troponin-I, which peaked at 60.92 ng/ml. Repeat left heart catheterization did not show any changes; so aggressive medical optimization continued. Nine days after presentation, the patient was discharged from the hospital free of chest pain and neurologically intact. She was discharged on goal-directed medical therapy for heart failure with reduced EF. A repeat echocardiogram 40 days after discharge demonstrated improvement in ventricular wall motion, and one year later her EF was stable at 50–55%.

DISCUSSION

Spontaneous coronary artery dissection is a rare condition; however, because diagnosis requires coronary angiography, it is likely under-reported. This condition occurs predominantly in young women who often have no risk factors for atherosclerotic heart disease, and they can present with varied symptoms and ECG patterns that span from normal to ST-segment elevation to ventricular dysrhythmias.[1] Myocardial infarction related to SCAD is considered type 2 as it leads to an imbalance in myocardial oxygen supply and demand without the presence of atherosclerotic plaque rupture.[2]

Prevalence has been reported between 0.3–4% among patients undergoing routine and urgent angiography, respectively.[3] Spontaneous coronary artery dissection is the underlying cause of chest pain in up to 10% of women less than 50 years of age, and SCAD is seen in up to 35% of cases with elevated cardiac biomarkers in this cohort.[1,4] Furthermore, SCAD is the most common cause of MI in pregnant women, making up 43% of these cases.[5] As demonstrated by these findings, it is important to have a high index of suspicion for SCAD in patients presenting with symptoms suggestive of ACS who lack the typical risk factors. Each of the above cases demonstrates a risk factor and potential presentation for this rare condition. Demographic factors of SCAD can be found in Table 1.

The first patient presented with non-specific ECG changes and an elevated troponin-I. Normal or non-specific ECG findings occur in 46% of observed SCAD patients, and troponin elevations occur in approximately 80% of cases.[6] Additionally, this patient suffered from migraine headaches, which is more prevalent in patients with SCAD than those without.[7] Migraine headaches are more commonly associated with SCAD in the setting of pregnancy.[8] It remains unclear whether migraine is an independent risk

Table 1. Demographic factors of patients with spontaneous coronary artery dissection (SCAD). This data is based on data queried from the Mayo Clinic "Virtual" Multicenter SCAD Registry composed of 1,196 patients.[6]

Age, median (IQR)	54 (47–61)
Gender, %	
Female	95.6
Male	3.9
BMI, median (IQR)	25.0 (21.8–29.2)
Race, %	
White	92.3
Black	2.3
Hispanic/Hispanic-White	2.2
Other	3.2
Comorbidities, %	
Classic ACS risk factors	
Hypertension	32.2
Hyperlipidemia	33.0
Diabetes mellitus	2.9
Previous tobacco use, *n* (%)	26.4

IQR, interquartile range; *BMI*, body mass index; *ACS*, acute coronary syndrome.

factor for SCAD, but the epidemiology of patients who suffer from both migraine and SCAD is unique. Further study is needed to definitively elucidate the relationship between migraine and vascular conditions such as SCAD.[7] Despite this association, this patient was still assumed to have atherothrombosis and managed accordingly. Her diagnosis, made on urgent angiography after admission, highlights the diagnostic uncertainty that emergency physicians encounter when managing a patient with ACS.

The second patient presented with an ECG that met STEMI criteria. In patients with SCAD, 39–49% had ECGs diagnostic of STEMI.[3,6] The precordial leads (V1–V4) most frequently demonstrate ST-segment elevation, corresponding to the LAD territory.[3] However, SCAD can involve multiple vessels and, as a result, multiple ECG territories.[1] This patient had a history of fibromuscular dysplasia (FMD). Fibromuscular dysplasia is a vascular disease that affects medium-sized arteries: most commonly affecting the renal, carotid, and vertebral arteries, but it has been known to affect the coronary arteries.[9] A build-up of fibrous tissue and webs in the arterial walls causes arterial stenosis, tortuous arteries, aneurysms, and dissections.[9]

Fibromuscular dysplasia is the most common nonatherosclerotic phenomenon found to cause SCAD.[1] A 2013 prospective and retrospective study in Vancouver screened 86% of patients identified with nonatherosclerotic SCAD for FMD, and found 72% of these patients to have FMD.[10] Other studies have found the association to be weaker: a study using the Mayo Clinic's registry of patients with confirmed SCAD showed that approximately 39% were confirmed positive for FMD; however, 31% of patients included in the study were not screened at all.[6]

The final patient presented with chest pain, followed by cardiac arrest, and was ultimately found to have an ECG that met STEMI criteria. Chest pain is the presenting symptom in approximately 80–90% of SCAD cases.[1,6,11] Ventricular arrythmias, which this patient presented with, were noted in 3–16% of cases.[1,6,11] Of the cases with ventricular arrythmias, 48% were diagnosed with STEMI after defibrillation.[12] While acute atherothrombosis is uncommon in pregnant and postpartum women, SCAD shows a well-defined correlation with late pregnancy and the postpartum period.[3] Clinicians should be aware of this potential diagnosis in pregnant and postpartum patients who present with chest pain.

Overall, initial patient management is similar to management of atherosclerotic ACS: focus should be on revascularization of ischemic myocardial territories, at least until the definitive diagnosis of SCAD is made.[13,14] Compared with the revascularization approach in atherosclerotic ACS, restoration of TIMI 3 flow with as few interventional measures as possible is the goal in management of SCAD—even if coronary architecture is not normalized.[14]

Medical management of patients suffering from SCAD differs from management of atherosclerotic ACS or MI. While there is limited evidence, current guidelines indicate that systemic anticoagulation is unnecessary and may propagate the intramuscular hematoma; therefore, it should not be implemented or discontinued in the absence of other indications for systemic anticoagulation if SCAD is suspected.[1,14] Similarly, there is limited evidence to guide the use of anti-platelet agents such as glycoprotein IIb/IIIa inhibitors in the emergency management of SCAD.[1,14] Theoretical concerns about the extension of dissection and additional bleeding risk limit the use of antiplatelet agents if SCAD is suspected in the ED.[1,14]

Beta-blocker therapy to reduce shear stress is supported by one retrospective study[14] that showed an association with lower rates of SCAD recurrence, but these results have not been replicated.[15] Decisions regarding initiation of systemic rate-control agents to reduce shear stress, anticoagulation, and/or DAPT in the emergency setting should be made in conjunction with an interventional cardiologist when available if concern for SCAD is high. While nuanced management of SCAD is better reserved for subspecialty discussion, it is important for the emergency physician to understand the potential harms of antiplatelet agents, anticoagulation, and revascularization interventions in this population.

While there are other cases of SCAD in the literature highlighting risk factors for this increasingly recognized entity,[16] we believe the cases presented here illustrate

the varied presentations and important risk factors associated with this uncommon and difficult-to-diagnose condition. Clinicians should be aware of the disease's natural history, as early diagnosis and intervention may lead to improved outcomes.[15,16]

CONCLUSION

Spontaneous coronary artery dissection is a rare cause of type 2 myocardial infarction that affects primarily young women who differ in risk profile when compared with patients who suffer type 1 MI. Associated factors include migraine, connective tissue disorders, and pregnancy. Presentations are varied, and clinicians must maintain a high suspicion for this uncommon condition.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

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Costoclavicular Brachial Plexus Block Facilitates Painless Upper Extremity Reduction: A Case Report

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Introduction: The costoclavicular brachial plexus block (CCBPB) has emerged as a more effective approach to regional anesthesia of the upper extremity. The costoclavicular space is the anterior portion of the superior thoracic aperture, located between the clavicle and first rib. The brachial plexus cords traverse this space clustered together in a superficial location lateral to the axillary artery and share a consistent topographical relationship to one another. By targeting the brachial plexus at this specific anatomical location, the CCBPB offers a powerful, single-shot, sensorimotor block of the upper extremity below the shoulder. We present a novel application of the CCBPB to facilitate emergency department (ED) analgesia and closed reduction of an upper extremity fracture.

Case Report: A 25-year-old male presented to the ED with a traumatic Colles fracture sustained during a high-speed motor vehicle collision. Despite multimodal analgesia, the patient reported intractable severe pain with intolerance of radial manipulation. An ultrasound-guided CCBPB was performed to augment pain control and avoid procedural sedation, resulting in dense, surgical anesthesia of the upper extremity, and painless fracture reduction.

Conclusion: Regional anesthesia is an effective component of multimodal pain management and another tool in the emergency physician's analgesic armamentarium. In acute orthopedic traumas necessitating emergent reduction, regional blocks serve as rescue pain control and can obviate the need for procedural sedation. In terms of targeted upper extremity analgesia, the CCBPB offers effective, single-shot, sensorimotor blockade below the shoulder, mitigating use of opioids and their deleterious side effects, while simultaneously avoiding incomplete blockade or phrenic nerve palsy associated with other approaches to brachial plexus blockade. [Clin Pract Cases Emerg Med. 2023;7(4)221–226.]

Keywords: *costoclavicular brachial plexus block; ultrasound-guided nerve blocks; regional anesthesia; upper extremity; case report.*

INTRODUCTION

Nonfatal traumatic injuries account for approximately 30 million emergency department (ED) visits annually in the United States, with acute pain being the most common symptom reported by over 90% of trauma patients.[1,2] Historically, oligoanalgesia has been a concern in emergency pain management.[3] Given the prevalence and patient burden of post-traumatic pain, emergency physicians should use a multimodal analgesia approach to effectively combat pain and prevent oligoanalgesia. Regional anesthesia is a vital component of multimodal analgesia and is most commonly used in the ED for fracture pain management.[4] It has been shown to provide excellent pain control, reduce opioid intake and opioid-related side effects (sedation, delirium, pruritus, nausea/vomiting, hypotension), improve post-surgical functional outcomes, shorten hospital length of stay, and possibly reduce the incidence and severity of chronic post-traumatic pain syndrome.[5]

For regional anesthesia of the upper extremity, brachial plexus blockade is the gold standard.[6,7] The ultrasound-guided costoclavicular brachial plexus block (CCBPB) is a novel infraclavicular technique that targets the brachial plexus cords at a superficial, compact, and topographically consistent location, resulting in rapid and reliable upper extremity anesthesia below the shoulder.[8] The CCBPB provides a complete distribution of anesthesia for the upper extremity, aside from the skin overlying the medial upper arm, which is innervated by the intercostobrachial nerve. In comparison to other approaches to brachial plexus blockade, the CCBPB is a single-shot, low-volume (15–20 milliliters) technique that provides faster onset of sensory blockade compared to the classic lateral sagittal infraclavicular approach, and it results in a lower incidence of hemidiaphragmatic paralysis compared to the supraclavicular approach.[9,10]

Although use of the ultrasound-guided brachial plexus blockade in the ED remains a relatively uncommon practice, the CCBPB has the potential for widespread adoption given its ease of performance, rapid onset of analgesia, and low risk of complications.[4] We present a case report of a patient with intractable upper extremity pain precluding manipulation of a Colles fracture, who underwent ultrasound-guided CCBPB with complete resolution of pain and successful fracture reduction.

CPC-EM Capsule

What do we already know about this clinical entity?
The costoclavicular brachial plexus block (CCBPB) is a novel infraclavicular approach used for perioperative analgesia of the upper extremity.

What makes this presentation reportable?
Brachial plexus blockade is rarely used in the emergency department (ED) due to feared complications. The CCBPB is a safe and potent analgesic modality for upper extremity trauma.

What is the major learning point?
The CCBPB provides near-complete anesthesia of the upper extremity, mitigating opioid use while avoiding complications associated with other approaches.

How might this improve emergency medicine practice?
Regional anesthesia provides a safe, non-euphorigenic, and effective alternative to opioids and procedural sedation for acute pain management in the ED.

CASE REPORT

A 25-year-old male with no pertinent past medical history presented to the ED by ambulance with obvious right wrist deformity following a high-speed motor vehicle collision as a restrained driver. The patient had braced his outstretched hand against the steering wheel during vehicular impact, resulting in traumatic injury to the wrist. His vital signs on arrival were within normal limits, but he was in significant distress, rating his pain as a 10/10. Physical examination revealed a grossly deformed right wrist with dorsal swelling and severe tenderness to palpation, but no evidence of neurovascular compromise. Radiographs of his right wrist revealed a transverse fracture of the distal radius with dorsal angulation of the distal fragment (Colles fracture), as well as an ulnar styloid fracture.

Despite receiving opioid and nonsteroidal anti-inflammatory analgesia, the patient still reported intractable, severe pain with intolerance of radial manipulation. To avoid procedural sedation, an ultrasound-guided CCBPB was performed to augment patient analgesia and facilitate reduction (Images 1–3, Video).

Informed consent for CCBPB performance was obtained, and the patient was placed on a cardiac monitor with intravenous access established. The patient was positioned supine with the right arm abducted 90 degrees to stretch the pectoralis muscles and bring the costoclavicular brachial plexus more superficial toward the skin surface. The patient was prepped and draped in the standard manner, and sterility was maintained for the duration of the procedure. A high-frequency linear ultrasound probe was oriented transversely just inferior to the midpoint of the right clavicle in the infraclavicular fossa. The probe beam was angled slightly cephalad to visualize the costoclavicular space posterior to the clavicle. The costoclavicular brachial plexus cords were identified just lateral to the axillary artery and between the subclavius and serratus anterior (upper slips) muscles. An in-plane, lateral-to-medial approach was used to guide a 22-gauge, 50-millimeter echogenic block needle between the lateral and posterior cords. Twenty milliliters of 0.5% ropivacaine were injected perineurally around the brachial plexus cords. The procedure was performed without complications.

Fifteen minutes post-block performance, the patient had dense sensorimotor blockade of the right upper extremity and reported his pain at 0/10. Despite aggressive manipulation, the patient reported no pain, and successful reduction of the distal radius fracture was achieved. A sugar-tong forearm splint was applied, and the patient was discharged from the ED with outpatient

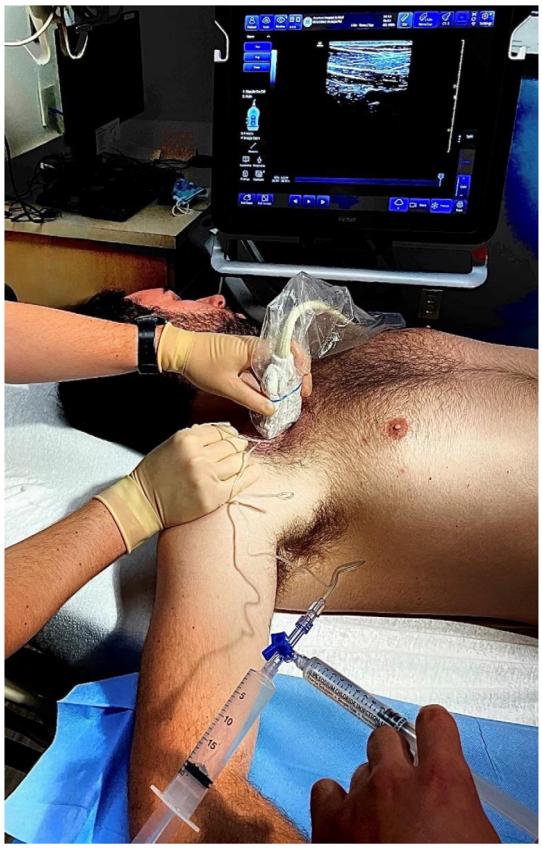

Image 1. Right-sided costoclavicular brachial plexus block performance. The patient is positioned supine with ipsilateral arm abducted 90 degrees. The high-frequency linear array probe is positioned transversely just below the midpoint of the clavicle in the infraclavicular fossa. The probe beam is angled slightly cephalad to visualize the costoclavicular space posterior to the clavicle. The block needle is inserted lateral-to-medial using a standard in-plane technique. A two-person block technique is used, whereby one clinician performs dynamic needle guidance under ultrasound while the other administers local anesthetic.

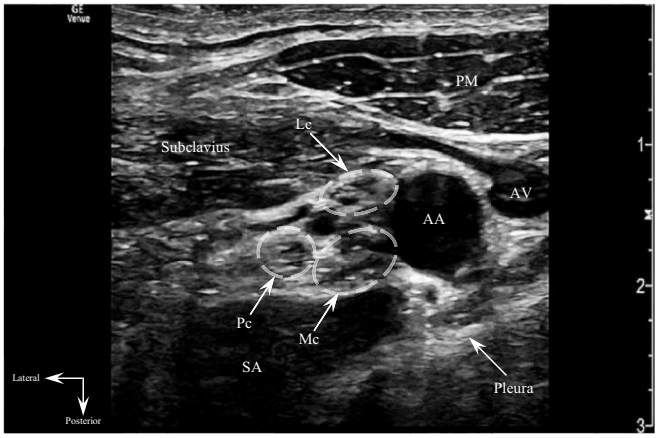

Image 2. Transverse sonogram of the costoclavicular space depicting the relevant sonoanatomy for costoclavicular brachial plexus block performance. The brachial plexus cords (dashed ovals) are shown lying lateral to the axillary artery and between the intermuscular plane composed of the subclavius and serratus anterior (upper slips) muscles. Note the cords are tightly clustered in a relatively superficial location lateral to the axillary vessels, making the costoclavicular space an ideal target site for brachial plexus blockade.
AA, axillary artery; *AV*, axillary vein; *Lc*, lateral cord; *Mc*, medial cord; *Pc*, posterior cord; *PM*, pectoralis major; *SA*, serratus anterior.

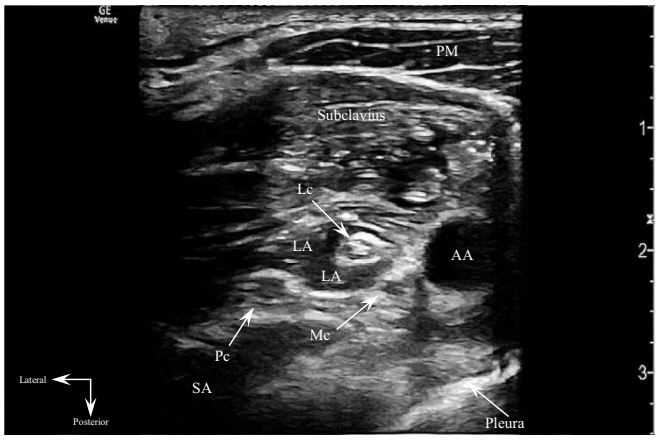

Image 3. Transverse sonogram post-costoclavicular brachial plexus block performance. Anechoic local anesthetic injectate is visualized enveloping and spreading apart the various costoclavicular brachial plexus cords.
AA, axillary artery; *LA*, local anesthetic; *Lc*, lateral cord; *Mc*, medial cord; *Pc*, posterior cord; *PM*, pectoralis major; *SA*, serratus anterior.

orthopedic surgery follow-up. On telephone inquiry the next day, the patient reported no numbness, tingling, or residual sensorimotor deficit.

DISCUSSION

Trauma patients present to the ED at the peak of their pain severity on the trauma care continuum, necessitating analgesic expertise on the part of emergency physicians to effectively treat acute pain. In the wake of the opioid epidemic, there is a dire need for effective yet non-euphorigenic pain management modalities. Regional anesthesia is an ideal peri-traumatic analgesic as it offers targeted, superior pain relief by inhibiting nociceptive signaling of C and Aδ fibers of peripheral nerves, without disruptions in hemodynamics or respiratory status.[11] Furthermore, it mitigates opioid use, preserves mental status, reduces pain from distracting injuries to enable further assessment of concomitant injuries, and may improve tissue viability by augmenting blood flow to injured tissue via regional sympathectomy.[5] Long-term effects of early peripheral nerve blockade include improved post-surgical outcomes, greater patient participation in aggressive physical therapy, and possible reduction in incidence or severity of chronic pain syndromes such as complex regional pain syndrome or phantom limb pain.[5]

Regional anesthesia of the upper extremity has historically centered around brachial plexus blockade.[12] Traditionally, a paracoracoid (lateral sagittal) approach is performed for upper extremity anesthesia in the lateral infraclavicular fossa. However, the brachial plexus cords at this location are deep, diverge from one another, exhibit vast variation in respective location relative to the axillary artery, and are rarely all visualized in one sonographic cross-section.[13,14] At the costoclavicular space (CCS), the brachial plexus cords are more superficial, and clustered together in a consistent anatomical topography lateral to the axillary artery.[15]

The advantageous properties of the costoclavicular brachial plexus allow for shallower needle angles and enhanced needle visualization under ultrasound, with low risk of inadvertent neurovascular or pleural puncture.[16] Furthermore, faster onset of complete sensory blockade is achieved using lower volumes of local anesthetic compared to the paracoracoid approach, likely due to shorter intercordal diffusion distances at the CCS.[9] A costoclavicular block dynamics study in which 20 milliliters of 0.5% ropivacaine was used showed median onset time for sensorimotor blockade of five minutes, with complete blockade development within 30 minutes, consistent with our findings.[17] The rapid onset of analgesia afforded by the CCBPB ultimately obviated the need for procedural sedation, a time-consuming, resource-intensive procedure with rare but significant risk of serious adverse events.

In this case, a single-injection block between the three cords was performed in accordance with the original block description by Karmakar et al.[8] However, Monzo et al performed a clinical and microanatomical study demonstrating the existence of a reliable intraplexal fascial septum separating the costoclavicular brachial plexus into a superficial compartment comprised of the lateral cord and a deep compartment containing the medial and posterior cords.[18] They recommend performing a second injection after piercing this intraplexal septum to ensure adequate spread of local anesthetic to the medial and posterior cords, thereby reducing the occurrence of incomplete upper extremity blockade.[18] Layera et al compared single- vs double-injection CCBPBs, with the double-injection technique resulting in shorter onset and longer duration of anesthesia, although these results were not likely clinically significant.[19]

Our patient exhibited complete pain relief with performance of a single-injection CCBPB. It remains unclear what impact an intraplexal septum plays in local anesthetic diffusion, block onset time, and block success. Further studies are needed to elucidate the optimal CCBPB injection location, clinical significance of intraplexal septa, and the potential need for a multi-injection approach to prevent impedance of local anesthetic spread and subsequent block failure.

Procedural performance time for an ultrasound-guided CCBPB varies depending on multiple factors, including emergency physician proficiency in regional anesthesia, efficiency in patient and procedural setup, and availability of regional block materials. The CCBPB in this case was performed in 15–20 minutes using a two-clinician technique, whereby one performed dynamic needle guidance under ultrasound while the other administered local anesthetic through an attached catheter. Time of performance can be significantly mitigated by having pre-arranged "block bags" or nerve block procedural trays with all materials needed for performance readily available. While a two-clinician technique is not mandatory, it is preferred because it allows for initial tissue hydrodissection with normal saline to confirm needle tip position and open the target injection space prior to deposition of local anesthetic.

Twenty milliliters of 0.5% ropivacaine were used in block performance. In a dose-finding study by Wong et al, this represented the minimum effective volume of ropivacaine in 90% of patients to produce surgical anesthesia after undergoing ultrasound-guided CCBPB.[20] While this block regimen produced potent analgesia facilitating fracture reduction, the patient's recovery was delayed post-procedurally given the long-acting properties of ropivacaine. Rapid onset, short-acting local anesthetic (eg, 2% chloroprocaine) may be a more appropriate selection for trauma patients who must undergo painful procedural performance before planned discharge disposition, to ensure restoration of sensorimotor function prior to discharge. While complete restoration of upper extremity sensorimotor function is not a necessity prior to discharge, all patients who undergo brachial plexus blockade should be placed in a shoulder sling and given instructions detailing expected block duration, specialty follow-up, and return precautions in the rare case of persistent sensorimotor deficit.

Performance of the CCBPB is safe, with procedural complications being rare.[16] The traditional adverse effects of more proximal brachial plexus blockade (Horner syndrome, hemidiaphragmatic paralysis, neuraxial blockade, pneumothorax, hoarseness) are exceedingly uncommon.[9,10] A cadaveric study using methylene blue injections demonstrated consistent sparing of the phrenic nerve, highlighting the preservation of respiratory function post-block performance.[21] Another cadaveric study analyzing critical structures encountered in the needle trajectory demonstrated no occurrence of vascular puncture, although those authors observed consistent contact of the block needle with the lateral cord.[16] Use of echogenic block needles, nerve stimulation, and pressure-injection monitoring, as well as block performance on alert patients who can report occurrence of paresthesia, are all safety measures that reduce the incidence of inadvertent neurovascular puncture or injection.[22] Lastly, emergency physicians should be cognizant of local anesthetic systemic toxicity and adhere to

maximum, weight-based, local anesthetic dosing to minimize the occurrence of this potentially fatal clinical entity.

Despite its safety and effectiveness, there are a few limitations of CCBPB performance. Lack of emergency physician training and experience in ultrasound-guided regional anesthesia is a significant limiting factor, as is lack of access to the materials for proper regional block performance. In addition, CCBPB performance may be precluded in certain patient populations, such as the morbidly obese and those with prior history of clavicular trauma, breast surgery, radiotherapy, or mastectomy, due to poor visualization or distortion of the costoclavicular space.

CONCLUSION

Regional block performance is becoming progressively more ubiquitous in emergency medicine practice. Use of ultrasound-guided nerve blocks by emergency physicians greatly facilitates post-traumatic analgesia and improves long-term patient outcomes. Concerning targeted upper extremity analgesia, the costoclavicular brachial plexus block offers rapid and effective sensorimotor blockade below the level of the shoulder, while avoiding the potential complications associated with opioid utilization or other regional approaches to brachial plexus blockade. Future research is warranted comparing the CCBPB to other upper extremity regional blocks, particularly as it pertains to ED performance in trauma patients.

Video. Sonoclip demonstrating costoclavicular brachial plexus block performance. An echogenic block needle is positioned at the injection target site, between the posterior and lateral cords. Anechoic local anesthetic injectate is visualized spreading apart the costoclavicular brachial plexus cords. The lateral cord is completely enveloped in this sonoclip, illustrating the characteristic "donut sign" of regional anesthesia blocks. *AA*, axillary artery; *LA*, local anesthetic; *Lc*, lateral cord; *Mc*, medial cord; *Pc*, posterior cord; *PM*, pectoralis major; *SA*, serratus anterior.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

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A Case Report of Anesthesia-induced Diffuse Alveolar Hemorrhage Presenting to the Emergency Department

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Introduction: The inhaled anesthetic sevoflurane is an uncommon etiology of diffuse alveolar hemorrhage (DAH). As DAH typically presents in the inpatient, postoperative setting, it has been infrequently reported in the anesthesiology literature and, to our knowledge, has not been reported in the emergency medicine literature to date.

Case Report: We describe the presentation of a young, healthy male in respiratory distress to a busy urban emergency department (ED) after an outpatient surgical procedure. We highlight the etiology of post-anesthesia DAH and the acute management of this rare diagnosis in the ED.

Conclusion: With outpatient surgical centers becoming an increasingly popular option for lower risk procedures, emergency physicians would benefit from understanding this presentation and its pathophysiology. [Clin Pract Cases Emerg Med. 2023;7(4)227–229.]

Keywords: *diffuse alveolar hemorrhage; respiratory distress; anesthesia; critical care.*

INTRODUCTION

Diffuse alveolar hemorrhage (DAH) secondary to the inhaled, volatile anesthetic sevoflurane is an uncommon etiology with few case reports in the anesthesia literature and none in the emergency medicine (EM) literature to date. This readily available and frequently used general anesthetic has a long safety record with few significant side effects. Many patients undergoing general anesthesia receive sevoflurane in both inpatient and outpatient surgical centers.

CASE REPORT

We report the case of a 20-year-old healthy male who was transported by emergency medical services (EMS) to the emergency department (ED) from an outpatient surgery center with a chief complaint of shortness of breath and hemoptysis. The patient reported that upon awakening from anesthesia in the post-anesthesia care unit (PACU), he was coughing up blood clots. He was in the PACU after undergoing general endotracheal anesthesia for an outpatient shoulder tendon repair procedure. Of note, the patient received inhaled sevoflurane for anesthesia and an inter-scalene nerve block for postoperative pain control. The patient reported no significant past medical history and no smoking or drug use history. Family history was significant for a postoperative deep venous thrombosis (DVT) after surgery in the patient's mother and systemic lupus erythematous in his maternal aunt. The patient was of athletic build, participated in track and field, and had no prior history of receiving general anesthesia. Brief discussion with the attending anesthesiologist from the outpatient

surgery center revealed no further details. He reported no difficulty with induction, intubation, or extubation.

On arrival to the ED, the patient was found to be tachypneic with a respiratory rate of 24 breaths per minute. The PACU staff had reported hypoxemia to the EMS personnel, and he was transported on four liters of oxygen via nasal cannula with an ED oxygen saturation of 96%. He was normotensive without tachycardia. The patient had hemoptysis in the ED with bilateral rales on auscultation of his lungs. A portable chest radiograph (Image 1) was obtained, which showed diffuse multifocal patchy alveolar opacities in the bilateral lungs concerning for DAH. Chest computed tomography angiogram (Image 2) further supported the diagnosis of DAH, without any findings of pulmonary embolism, pneumothorax, or other significant chest pathology. Initial laboratory work-up revealed a lactic acid level of 6.8 millimoles per liter (mmol/L) (reference range 0.5–2.2 mmol/L), a hemoglobin level of 14.8 grams per deciliter (g/dL) (14.0–18.0 g/dL), a platelet count of 227,000 per microliter (μL) (150,000–450,000/μL), an international normalized ratio of 0.99 (0.85–1.17), a partial thromboplastin time of 23.2 seconds (22.9–35.8 seconds), and a creatinine level of 1.49 milligrams (mg) per dL (0.50–1.40 mg/dL).

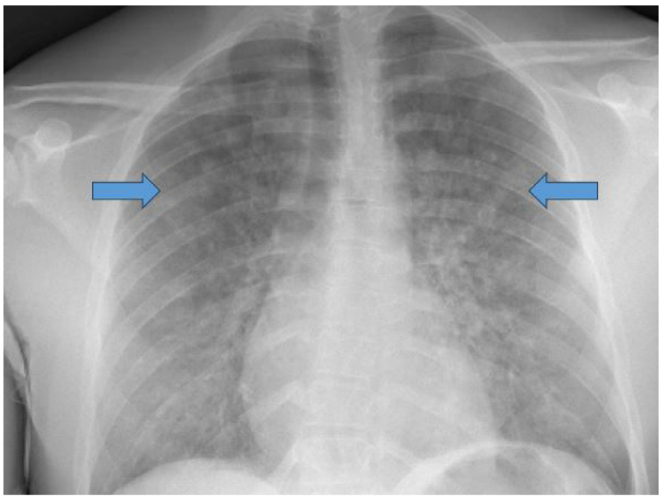

Image 1. Upright anterior-posterior chest radiograph in a patient with diffuse alveolar hemorrhage, demonstrating patchy alveolar opacities (arrows).

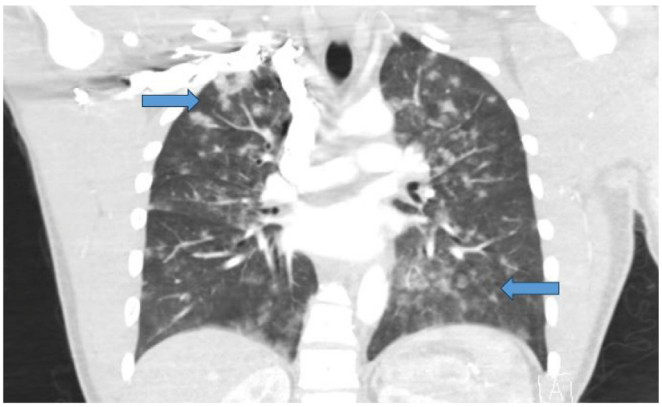

Image 2. Coronal view of computed tomography angiogram of the chest demonstrating diffuse alveolar hemorrhage (arrows).

During the patient's ED stay, he was administered one liter of intravenous crystalloid solution for his lactic acidosis and acute kidney injury. His tachypnea and hemoptysis improved, and he was gradually weaned from supplemental oxygen. After pulmonology consult in the ED, the patient was admitted to inpatient medicine for overnight observation and further work-up of his hemoptysis. He underwent bronchoscopy with bronchoalveolar lavage the next day with results consistent with DAH. He also received lab work-up for autoimmune processes including antinuclear antibody, complement component 3, complement component 4, rheumatoid factor, anti-double stranded DNA antibody, and anti-neutrophil cytoplasmic antibody, all of which were within normal limits. The patient remained clinically stable and experienced resolution of his lactic acidosis (1.3 mmol/L) and acute kidney injury (creatinine 1.10 mg/dL) and was discharged the day after admission with outpatient follow-up.

DISCUSSION

Diffuse alveolar hemorrhage is a rare but serious complication during the perioperative period. One mechanism that has been proposed to cause DAH during the perioperative period is laryngospasm. Laryngospasm is the sustained closure of the vocal cords that results in the loss of a patent airway; its incidence during anesthesia has been reported to be just under 1%.[1] Patients who undergo endotracheal intubation are most likely to experience laryngospasm after extubation during the emergence period, likely due to decreased conscious control of the laryngeal closure reflex.[2] Factors associated with higher incidence of laryngospasm include use of volatile, inhaled anesthetics, younger patients, reactive airway disease, smoking, and airway procedures. Young athletic males are the subjects in most case reports and case series regarding episodes of alveolar hemorrhage and edema related to anesthesia.[3–8] It is hypothesized that when experiencing episodes of laryngospasm, young athletic males generate significant negative inspiratory force that can cause injury to the tracheobronchial vasculature.

Another mechanism that has been proposed as a cause of perioperative DAH is the use of the inhaled anesthetic sevoflurane. Sevoflurane is a volatile, halogenated gas used as an inhaled anesthetic for induction and maintenance of anesthesia. Well known side effects of volatile anesthetics include nausea, vomiting, hypotension, arrhythmias, coughing, breath-holding, respiratory depression, and laryngospasm. Case reports describing episodes of

pulmonary alveolar hemorrhage after sevoflurane use are sparse and generally only described in the anesthesia literature. These case reports describe patients who were young healthy males undergoing routine surgical procedures with general anesthesia.[3,9–12] Volatile anesthetics are known to be lipid soluble and activate the arachidonic cascade within the cell membrane, which may increase alveolar permeability and oxidative stress.[13] Sevoflurane specifically has been shown in vitro to inhibit platelet aggregation.[14] These properties may increase the risk of DAH in patients who are susceptible.

CONCLUSION

This case supports previously published reports of sevoflurane-induced diffuse alveolar hemorrhage. Interestingly, other than naloxone-mediated acute flash pulmonary edema, there are no reports of any intravenous anesthetics or reversal agents resulting in DAH. Hence, the most likely causative agent in this case was the volatile, inhaled anesthetic sevoflurane, as there was no report of laryngospasm at the time of extubation. This represents the first reported case to our knowledge in the EM literature, likely due to the rarity of this diagnosis and the fact that most cases occur in the PACU. With outpatient surgical centers becoming an increasingly popular option for lower risk procedures, emergency physicians would benefit from understanding this presentation and its pathophysiology.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report.

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Cerebellar Infarction from a Vertebral Artery Dissection after Blunt Chest Injury: A Case Report

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Introduction: Traumatic vertebral artery dissections resulting in stroke are relatively rare occurrences, especially in the absence of classic physical examination findings.

Case Report: We present the case of a 30-year-old male with chest pain following a car axle falling onto his chest while trying to change a tire. He was discharged from the emergency department after having a negative workup for thoracic injury. Six hours later, the patient returned with headache and was found to have a cerebellar stroke secondary to vertebral artery dissection. After hospitalization, the patient was discharged home without any neurological deficits.

Conclusion: As they are usually asymptomatic, up to 80% of patients with blunt cerebrovascular injury will have delayed or missed diagnoses. Given the increased awareness of vascular injuries and their high morbidity, physicians should maintain a high index of suspicion for this diagnosis. [Clin Pract Cases Emerg Med. 2023;7(4)230–233.]

Keywords: *cerebrovascular injury; blunt chest trauma; vertebral artery dissection; cerebellar infarction; case report.*

INTRODUCTION

While injuries are a leading cause of emergency department (ED) visits in the United States, blunt cerebrovascular-related trauma is rare.[1] Blunt cerebrovascular injury (BCVI), a physical insult to the carotid or vertebral artery, occurs in less than 1% of blunt trauma patients and carries a high incidence of morbidity of 20–30%.[1] Mechanisms leading to BCVI are exaggerated rotations of the neck with hyperextension and contralateral spin of the head.[2] Furthermore, a direct blow to the carotid and vertebral arteries can lead to tearing of the tunica intima and media. A hematoma may develop intramurally, and neurological damage occurs from thromboembolism. Exposed endothelium stimulates platelet aggregation on arterial walls, causing luminal narrowing and occlusion, resulting in ischemia and brain infarction within days of injury.[3] The high velocity of arterial pulses on irregular tears can cause arterial transection, possibly resulting in hemorrhagic stroke, ischemic stroke, pseudoaneurysm, and death. We report a case of a 30-year-old male who presented to the ED with chest pain after blunt chest trauma and returned six hours later with stroke-like symptoms secondary to vertebral artery dissection.

CASE REPORT

A 30-year-old male with no significant medical history presented to a community ED with chest pain after a car axle fell onto his chest just before arrival. The patient was working beneath a Ford Mustang held up by a hydraulic jack. The jack broke and the axle fell on him. The patient momentarily lost consciousness. He was immediately removed from beneath the car. He was ambulatory and only complained of

right-sided chest pain, rated 10/10, described as pressure-like without any radiation, worse with movement, and improved with rest. Family history was significant for a parent with cerebrovascular accident (CVA) at age 35 from a drug overdose.

Presenting vital signs were normal with pulse 82 beats per minute, respirations 18 breaths per minute, blood pressure 122/76 millimeters of mercury, temperature 98.3° Fahrenheit, and pulse oximetry 100% on room air. On physical examination, the patient had moderate chest wall tenderness along the right distal clavicle with surrounding erythema. He had painful right-shoulder range of motion with normal distal right arm motor, sensation, and pulses. The patient had a Glasgow Coma Scale (GCS) of 15 and no focal neurologic deficits. He was given morphine for pain. Due to the severe mechanism of injury, broad imaging was performed. Non-contrast computed tomography (CT) of the head and cervical spine were negative for acute injury (Image 1a), although the CT incidentally revealed a congenital segmentation anomaly involving the right lateral mass of the first (C1) and second (C2) cervical vertebrae, with incomplete C1 ring and dysplastic odontoid. Computed tomography of the chest, abdomen, and pelvis with intravenous (IV) contrast revealed a right distal clavicular fracture. Upon re-evaluation, the patient remained hemodynamically stable and neurologically intact. Pain improved to a 1/10. He was placed in a sling and was advised to follow up with an orthopedist and primary care physician within 48 hours. He was prescribed oxycodone-acetaminophen for pain and discharged.

Six hours later, the patient returned to the ED after developing a gradual onset occipital headache, nausea, and vomiting. He took oxycodone-acetaminophen without relief of symptoms. On physical examination, the patient was ambulatory and neurologically intact with no focal neurological deficits. Cranial nerves II-XII were intact, there was 5/5 strength in bilateral upper and lower extremities, and there was normal sensation throughout. Repeat non-contrast head CT revealed an acute infarct involving the right cerebellar hemisphere, new from the prior study (Image 1b). A CT angiography (CTA) of the head and neck was then obtained, which revealed a short-segment intimal dissection in the right vertebral artery just above the level of the foramen magnum (Image 2). Again noted was the bony segmentation abnormality at C1-C2. Given the CVA was secondary to traumatic dissection, the patient was transferred to a trauma center.

Upon the patient's arrival to the trauma center, he was in acute distress and actively vomiting. His neurologic examination was significant for a leftward nystagmus but was otherwise unremarkable. He was alert and oriented to person, place, time, and situation, cooperative, with normal and intact visual fields; extremity strength and sensation (although examination to right upper extremity was limited by a sling); cranial nerves II-XII (besides the nystagmus); gait; and deep tendon reflexes. A cerebral arteriogram was performed and revealed a traumatic dissection of the right vertebral artery third division at the skull base with resultant posterior inferior cerebellar artery (PICA) territory infarct with interval reperfusion and mild mass effect. Although the right vertebral artery dissection was not flow-limiting, it remained a risk factor for distal dissection. The neurointerventionalist felt there was a significant risk of hemorrhagic conversion in the PICA infarct, as he was re-perfused, and he was also at risk of needing a suboccipital decompression if the mass effect continued to increase. Therefore, the patient was initially treated with only aspirin 300 milligrams (mg) by rectum daily, later switched to 325 mg by mouth, rather than dual antiplatelet therapy or anticoagulation.

On day two of his hospital course, the patient developed mild hydrocephalus with mass effect on the fourth ventricle and ataxia on clinical examination. The patient was treated with 3% saline IV continuously at 100 milliliters per hour

CPC-EM Capsule

What do we already know about this clinical entity?
Blunt cerebrovascular injury after trauma is rare. Patients with vertebral artery dissections are typically initially asymptomatic and have delayed presentations.

What makes this presentation of disease reportable?
The patient developed neurologic symptoms six hours after blunt chest injury and was found to have a vertebral artery dissection causing cerebellar infarction.

What is the major learning point?
Patients with vertebral artery dissections are usually asymptomatic. Up to 80% will be misdiagnosed or have a delayed diagnosis even with screening tools.

How might this improve emergency medicine practice?
Given the increased awareness of vascular injuries and the high incidence of morbidity, physicians should maintain a high index of suspicion for this diagnosis.

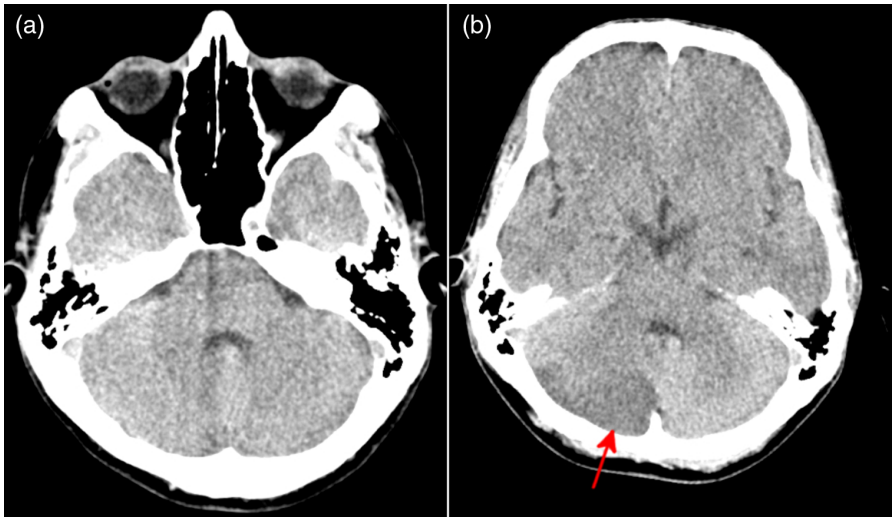

Image 1. Computed tomography of the head on day of injury (a) just after initial injury showing no acute intracranial pathology, and (b) six hours after injury showing acute infarct involving the right cerebellar hemisphere (arrow).

(mL/hr) for the first day, 50 mL/hr subsequently with a goal sodium level of 140–145 millimoles per liter, and later oral sodium chloride tabs 1 gram orally three times per day. He was also treated with dexamethasone 4 mg IV every six hours for the first two days and then switched to an oral taper. Symptoms waxed and waned, but after one week of treatment, symptoms improved and repeat head CT showed decreased mass effect with diminished hydrocephalus. The patient was discharged after a 10-day hospitalization with a completely normal neurological exam. In follow-up two weeks later, repeat head CT revealed ongoing aging of the right cerebellar infarct, which had decreased in size, showing primarily chronic-appearing features, near complete hydrocephalus resolution, and no intracranial hemorrhage midline shift or mass effect.

DISCUSSION

Blunt cerebrovascular injury after blunt trauma is rare, historically reported to be less than 1%, but now upward to about 2% given an increase in awareness.[1] Of all strokes, only 2.6 per 100,000 are caused by vertebral artery dissection.[2] Our patient initially presented with blunt chest injury, with no symptoms concerning for vertebral artery dissection. This presentation is common as patients with vertebral artery dissections are typically asymptomatic immediately after the injury. One study noted an 18-hour delay between traumatic

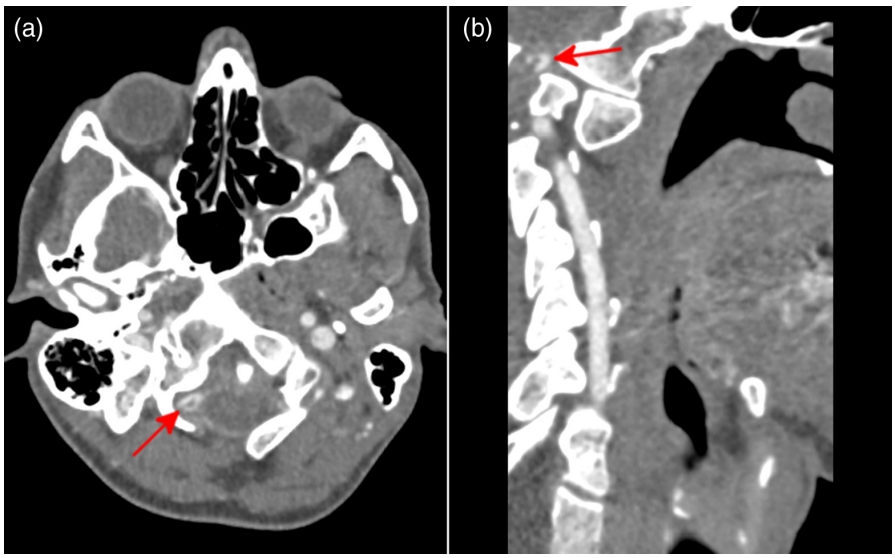

Image 2. Computed tomography angiography of the head and neck showing short segment intimal dissection in the right vertebral artery just above the level of the foramen magnum (arrows) on (a) axial and (b) oblique views.

dissection-causing injury and symptom onset.[4] Often, dissections are not diagnosed until patients present with neurologic deficits secondary to strokes or transient ischemic attacks, as with our patient. Our patient likely had the vertebral artery dissection upon initial presentation, although asymptomatic at the time, perhaps caused by rapid extension and rotational movement of his neck as the vehicle fell on him.

Vertebral artery dissections occur when a tear in the intimal layer of the vessel exposes endothelium, which stimulates platelet aggregation and formation of a thrombus to initiate vessel repair.[5] Stoke symptoms subsequently occur due to vessel occlusion either at the dissection site by the intimal flap or thrombus or, more distally by thrombus embolization into the cerebral circulation.[3] These differing mechanisms explain why symptom presentation onset vary. In our patient, the dissection likely occurred immediately after the accident but went undetected because vessel occlusion had not yet occurred. The vertebral artery supplies the spinal cord, brainstem, cerebellum, and posterior brain. Dissections most commonly occur superior to the C2 vertebra where the artery is mobile and not anchored as it ascends into the foramen magnum,[5,6] which was the location of our patient's dissection. His variant C1-C2 bony anatomy may have predisposed him to the injury, with the vertebrae providing less protection and allowing for more mobility of the vessel.

To help identify who requires a CTA to evaluate for BCVI, the Denver criteria were developed and include the following: arterial hemorrhage; cervical bruit; expanding hematoma; focal neurological deficit; and stroke on CT.[7] Risk factors include high-energy mechanism with cervical spine fracture, LeForte II or III fracture, basilar skull fracture with carotid canal involvement, diffuse axonal injury with GCS less than six, or near-hanging with anoxic brain injury.[7,8] Another study revealed cervical spine, facial, and basilar skull fractures were the strongest predictors of BCVI.[9] Our patient presented with a stroke on secondary CT, prompting further imaging, leading to diagnosis. He was then treated with anti-platelet therapy to decrease the risk of hemorrhagic conversion. Treatment of vertebral artery dissections consists of thrombolytic therapy if presenting within 4.5 hours, anti-platelet therapy, or anticoagulation. Endovascular or open operative repair are usually reserved for high-grade lesions.[8] Despite ongoing cerebellar infarct at two-week follow-up, our patient had a full neurological recovery.

CONCLUSION

Cerebellar infarctions secondary to vertebral artery dissections are rare. As they are usually asymptomatic, up to 80% of patients will be misdiagnosed or have a delayed diagnosis even with screening tools. Our patient, with variant C1-C2 anatomy and blunt upper chest injury, had risk factors for vertebral artery dissection. Given the increased awareness of vascular injuries and their high incidence of morbidity, physicians should maintain a high index of suspicion for this diagnosis.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

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Not All Sacral Wounds Are Sacral Decubitus Ulcers: A Case Report

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Introduction: Sacral wounds are commonly seen in the emergency department and typically get diagnosed as a pressure ulcer of varying stage. However, other disease processes and infections can affect the sacrum.

Case Report: Presented here is the case of an evolving sacral wound in a 70-year-old, immunocompromised woman that was eventually found to be localized herpes zoster and later became disseminated.

Conclusion: This case demonstrates the need for a broad differential diagnosis for sacral wounds that include atypical presentations for herpes zoster or herpes simplex virus. We discuss the guidelines for treatment and the classification of localized vs disseminated herpes zoster. [Clin Pract Cases Emerg Med. 2023;7(4)234–236.]

Keywords: *herpes zoster; sacral wounds; immunocompromised; disseminated; case report.*

INTRODUCTION

Herpes zoster is a viral infection that commonly affects the skin and nervous system and occurs in more than 1.2 million individuals annually.[1] It appears in many areas of the body with the most common dermatomes affected being thoracic (55%), cervical (20%), trigeminal including ophthalmic (15%), and lumbosacral (11%).[2] It commonly presents in one or two adjacent dermatomes and usually does not cross midline; however, there have been limited reports of bilateral herpes zoster.[3] Disseminated zoster is typically described as 20 or more lesions beyond the primary or adjacent dermatome.[2] Common complications of herpes zoster include: ophthalmic involvement (possible vision loss), bacterial superinfection of the lesions (*Staphylococcus aureus* and less commonly *Streptococcus pyogenes*), cranial and peripheral nerve palsies, pneumonitis, hepatitis, or meningoencephalitis, among others.[4] This case report illustrates the need for a broad differential when evaluating a sacral wound to prevent misdiagnosis and initiate prompt treatment and care of the patient.

CASE REPORT

A 70-year-old woman with lupus complicated by shrinking lung syndrome and chronic respiratory failure, chronic anemia, ovarian cancer in remission, type 2 diabetes mellitus, hypertension, and necrotizing fasciitis, currently on hydroxychloroquine and mycophenolate mofetil, presented to the emergency department (ED) for symptomatic anemia. On initial lab work, the patient had a glucose of 139 milligrams per deciliter (mg/dL) (reference range 70–99 mg/dL), hemoglobin of 7.0 grams (g)/dL (13.5–17.5 g/dL), and a white blood cell count of 7.6 thousand per microliter/μL (4.0–11.0 thousand/μL). The patient also reported an evolving "bruise" to her sacral region that had developed over the prior seven days. She described a burning sensation for the first three days, which had resolved. The patient was fully ambulatory, without periods of immobilization. On

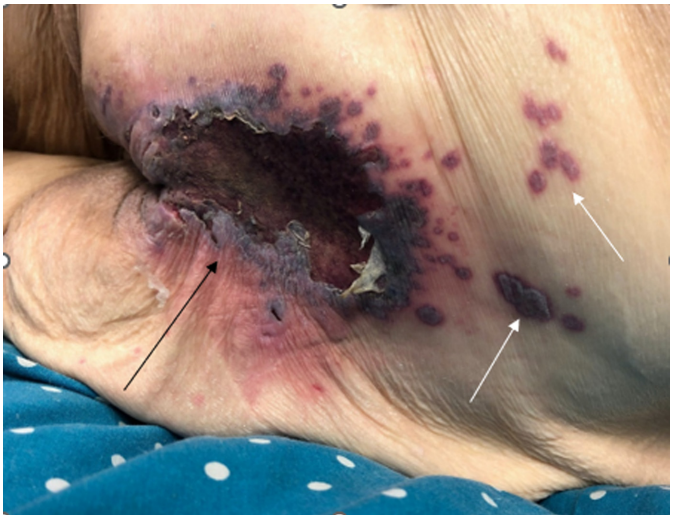

Image. Sacral wound (black arrow) found on the patient, with satellite lesions (white arrows) superior to sacral wound.

exam of her sacral area (Image), there was a 5 centimeter area of shallow ulceration with scalloped gray borders, dark purple erythema, and an erythematous base with satellite lesions extending caudally from the wound. The ulcer was dry and nontender to touch. Dermatology was consulted in the ED due to concern for vasculitis.

The dermatology team obtained further context about the lesion—there was no prodrome prior to the lesion developing, and the patient had a prior history of cold sores, most recently three months prior. The patient also had photographs showing vesicular lesions in the satellite lesions of the wound during the first several days. The dermatology team was concerned for herpes simplex virus (HSV) or herpes zoster virus and sent a swab of the wound for varicella-zoster virus (VZV) and HSV polymerase chain reaction test. This patient's rash was noted to cross midline but was localized to two adjacent dermatomes with only two satellite lesions; thus, the rash was considered localized and not disseminated. The patient was started on valacyclovir 1,000 milligrams three times daily for the next seven days and mupirocin 2% ointment and discharged to home.

The patient's sacral wound swab tested positive for VZV and negative for HSV. She returned four days later with worsening pain at the sacral wound and over 20 new vesiculopustular lesions to her nose, lower back, abdomen, and posterior oropharynx. She was admitted for disseminated herpes zoster virus at that time and was started on intravenous acyclovir. Her hospital course showed improvement in her lesions with crusting, and she was transitioned to oral valacyclovir and discharged to home on hospital day seven to finish her course of oral valacyclovir and to start lifelong prophylactic dosing of valacyclovir.

CPC-EM Capsule

What do we already know about this clinical entity?
Herpes zoster is a viral infection that affects the skin and nervous system in a dermatomal distribution and is frequently diagnosed in the emergency department.

What makes this presentation of disease reportable?
This is an atypical presentation of herpes zoster, which appeared similar to a sacral decubitus ulcer and evolved into disseminated herpes zoster.

What is the major learning point?
Herpes zoster can present atypically, especially in immunocompromised patients. These patients have an increased likelihood of having disseminated herpes zoster.

How might this improve emergency medicine practice?
Keep a wide differential for skin wounds, especially atypical sacral wounds, and send a herpes simplex virus and varicella-zoster virus swab if concerned.

DISCUSSION

In a patient with a sacral wound, the leading differential is a pressure-induced sacral decubitus ulcer of varying stage as these are commonly seen in the ED. However, this patient was fully ambulatory and had no prolonged periods of immobilization, making a pressure-induced wound exceedingly unlikely; ultimately, this led to a dermatology consult. The differential for sacral wounds should include cellulitis, contact dermatitis, ischemic ulcer, venous ulcer, necrotizing fasciitis, hypertensive ulcers, vasculitis, HSV, varicella-zoster virus virus, and candidiasis among others.

Herpes zoster can also present atypically in immunocompromised individuals. One presentation, hemorrhagic herpes zoster, is described as a purpuric or ecchymotic base. It is an atypical presentation and typically occurs in patients who are immunosuppressed, thrombocytopenic, or coagulopathic.[5] There are six types of herpes zoster documented, which can cloud diagnostic capability: bullous; verrucous-crusted; hemorrhagic, ulcerative-necrotic-gangrenous, disseminated (or varicelliform), and double herpes zoster (which involves at least one dermatome on both sides of the body and is not symmetrical in appearance).[6]

In addition, HSV may develop in a similar distribution to herpes zoster and mimic zoster (zosteriform herpes simplex); it frequently occurs in the face or genital/buttock regions. Therefore, it is important to send both HSV and VZV swabs for patients with suspicion of herpes zoster or herpes simplex.

CONCLUSION

This case demonstrates the need for a broad differential diagnosis for sacral wounds that include atypical presentations for herpes zoster and herpes simplex virus, as a missed diagnosis could result in delay to appropriate treatment. In cases of herpes zoster, it is essential that the clinicia focus immediate efforts on determining whether herpes zoster is localized or disseminated and whether the patient is immunocompetent or immunocompromised. Per US Centers for Disease Control and Prevention guidelines, when treating an immunocompetent patient with localized herpes zoster, no airborne or contact precautions are needed.

If the patient is immunocompromised and has localized herpes zoster the infection should be considered airborne, and contact and airborne precautions should be taken until disseminated infection is ruled out.[7]

Herpes zoster is considered disseminated when there are 20 or more vesicles outside the primary or adjacent dermatomes.[2] Disseminated herpes zoster occurs in about 2% of the general population and up to 15–30% in immunocompromised hosts.[8] Finally, if herpes zoster is disseminated in either an immunocompetent or immunocompromised patient, airborne and contact precautions should be taken until the lesions are crusted.[7]

Patient consent has been obtained and filed for the publication of this case report.

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A Case Report of Pneumoretroperitoneum from Blunt Trauma in a Patient with Chronic Obstructive Pulmonary Disease

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Introduction: Pneumomediastinum is a rare complication of blunt traumatic injury and is thought to be due to the Macklin effect, a pathophysiologic process comprised of three steps: alveolar rupture secondary to blunt injury; air dissecting along bronchovascular sheaths; and spread of pulmonary interstitial edema into the mediastinal space. Pneumomediastinum is rarely associated with pneumoretroperitoneum.

Case Report: We present a case of a patient who suffered a cardiac arrest after a fall during a chronic obstructive pulmonary disease exacerbation, leading to pneumoretroperitoneum.

Conclusion: This case highlights the complications that can arise from blunt trauma and how underlying lung pathology can worsen these complications. [Clin Pract Cases Emerg Med. 2023;7(4)237–241.]

Keywords: *case report; pneumoretroperitoneum; pneumomediastinum; blunt trauma.*

INTRODUCTION

Pneumomediastinum is a relatively common injury found after blunt thoracic trauma and portends a higher degree of severe injury with a higher complication risk than those who do not develop pneumomediastinum following blunt thoracic injury.[1] Pneumomediastinum was proposed by Macklin to be caused through a stepwise downstream effect of a sudden increase in intrathoracic pressure that causes alveolar rupture and subsequent emphysematous dissection of air along the bronchovascular sheaths, which spreads into the mediastinum. The first symptom of pneumomediastinum is most commonly chest pain, followed closely by dyspnea as the second most common presenting symptom. It is more common in males and more commonly associated with underlying lung disease such as asthma.[2] After development of pneumomediastinum, air can dissect into the mediastinum along the parietal pleura, leading to pneumothorax. Air can further dissect along the great vessels or the esophagus through the diaphragmatic hiatus and cause pneumoretroperitoneum.[3]

Pneumoretroperitoneum is most often described in the literature as being associated with respiratory tract rupture or rupture of alveoli, infection with gas-forming organisms, or interruption of the barriers between the gastrointestinal tract and the retroperitoneal space, commonly after colonoscopy or surgical manipulation of the gastrointestinal tract.[4] Pneumoretroperitoneum is rarely associated with blunt traumatic injury, such that in one study, only one patient in a cohort of 233 patients with blunt abdominal trauma was noted to have pneumoretroperitoneum.[5] Although it appears that both pneumomediastinum and pneumoretroperitoneum have been found in patients with blunt traumatic injury, they only rarely occur together, and barotrauma from ventilation can subsequently cause extension of underlying injury, resulting in high peak pressures and significant difficulty in adequate ventilation of these patients.[6]

CASE REPORT

A 50-year-old female patient presented to the emergency department (ED) in significant respiratory distress. She had a

known past medical history of tobacco dependence, chronic obstructive pulmonary disease (COPD), gastroesophageal reflux disease, and hypertension. On arrival, the patient was noted to be apneic and was actively being ventilated with a bag-valve-mask ventilation. She rapidly progressed from an irregularly irregular cardiac rhythm to sinus bradycardia, and then into a pulseless electrical activity (PEA) arrest within minutes of arrival. She underwent approximately 30 minutes of cardiopulmonary resuscitation, with endotracheal intubation performed via video laryngoscopy. She was noted to have significant resistance to bagging after intubation, and after return of spontaneous circulation, was noted to have high peak pressures on the ventilator and was persistently hypotensive.

On examination, the patient was noted to have distant, rhonchorous, and wheezy breath sounds bilaterally, and she was treated for her bronchospasm with nebulized albuterol-ipratropium solution, continuous nebulized albuterol, magnesium sulfate, methylprednisolone, subcutaneous terbutaline, and titratable epinephrine (to also assist with her hypotension). She was noted to have continuously high peak pressures; so, an intravenous push of vecuronium was given without improvement in ventilation and with continuously high peak airway pressures noted on the ventilator. She had an electrocardiogram concerning for new-onset atrial fibrillation with rapid ventricular response, left axis deviation, a new right bundle branch block, and a left anterior fascicular block with signs of right heart strain. Given her difficulty with ventilation with high peak pressures and hypotension, she was taken for a computed tomography (CT) with pulmonary embolism protocol as there was concern that the etiology of her arrest may have been a massive pulmonary embolism.

This revealed no evidence of pulmonary embolism; however, it did show evidence of alveolar rupture with pneumomediastinum consistent with the Macklin effect and a small left-sided pneumothorax (Image 1) with associated medial left hemi diaphragmatic rupture (Image 2) with pneumoretroperitoneum tracking along the left upper abdomen and left perinephric space with left-sided nondisplaced rib fractures of the fifth and sixth ribs (Image 3). A CT of the abdomen and pelvis was then performed, which showed a small amount of free air consistent with pneumoretroperitoneum adjacent to the gastric cardia, left kidney, and left adrenal gland, but with no definitively identified intra-abdominal traumatic injury.

Further history from the family, who arrived several hours after the patient's initial presentation, revealed she had been complaining of difficulty breathing earlier in the day, and she subsequently suffered a witnessed fall down a flight of stairs with head trauma and apparent loss of consciousness. The patient's physical examination revealed no external signs of trauma on initial arrival.

CPC-EM Capsule

What do we already know about this clinical entity?
Pneumoretroperitoneum is a rare diagnosis that represents a barrier breakdown between the retroperitoneum and an air-containing space, typically due to disease.

What makes this presentation of disease reportable?
There are very few reports of pneumoretroperitoneum occurring with blunt traumatic injury, and we present a novel combination of cardiac arrest, chronic obstructive pulmonary disease (COPD), and trauma.

What is the major learning point?
Underlying lung pathology, such as COPD, can contribute to worse outcomes in traumatic injury, and we highlight one such complication that can arise in this setting.

How might this improve emergency medicine practice?
This case highlighting a complication from blunt traumatic injury may help others quickly identify and address this complication.

The patient was admitted to the medical intensive care unit in the setting of cardiac arrest with prolonged down time, with improvement in her pneumomediastinum and pneumoretroperitoneum with lung-protective ventilation strategies and paralytics. The patient's treatment for her COPD exacerbation was extensive and included continuous albuterol nebulization of 40 milligrams (mg) over four hours, intermittent scheduled three-milliliter ipratroprium-albuterol nebulizers every six hours, 0.25 mg of budesonide twice daily, 0.5 mg of ipratroprium four times daily, isolated two mg of magnesium sulfate administration daily if worsening wheeze on examination, 80 mg of methylprednisolone daily, a one-time dose of 0.25 mg of subcutaneous terbutaline, and a one-time dose of one microgram (μg) of epinephrine. Sedation was maintained with both ketamine at 1.5 mg per kilogram (kg) per hour and propofol at 20 μg/kg per minute. For the patient's hypotension, she was treated with epinephrine as the first-line vasopressor choice given its underlying beta adrenergic effects in the setting of her profound bronchospasm.

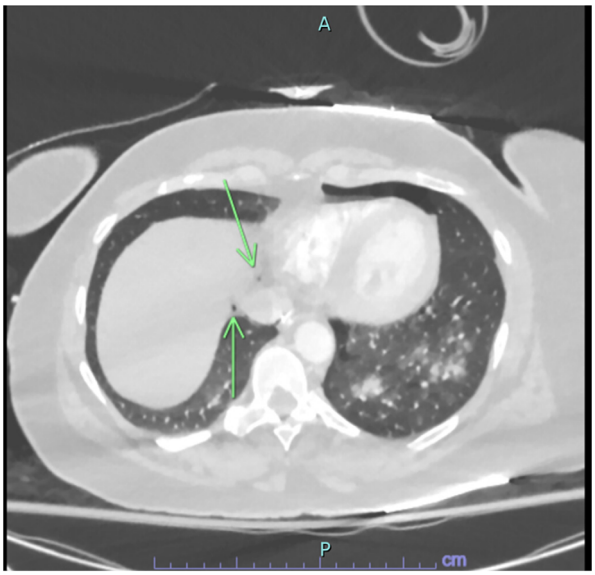

Image 1. Initial computed tomography with pulmonary embolism protocol performed, revealing pneumomediastinum in the lower portion of the mediastinum (green arrows) favored to be secondary to alveolar rupture as well as a left-sided pneumothorax.

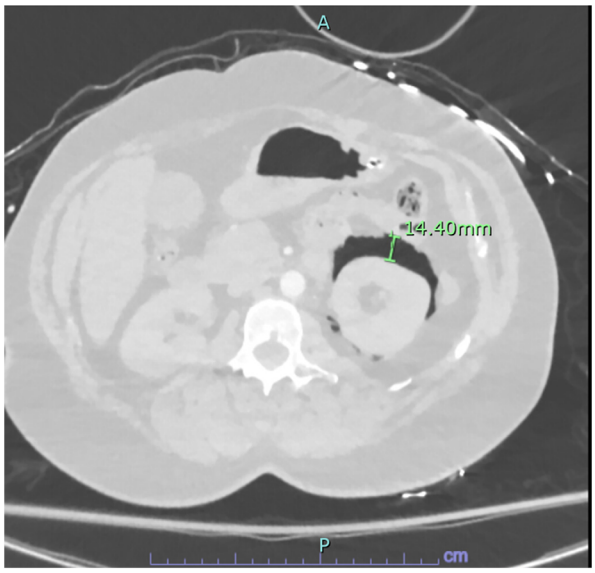

Image 3. Image from patient's computed tomography (CT) with pulmonary embolism protocol performed on the day of presentation revealing air in the left perinephric space, which is likely related to retroperitoneal extension of air from the patient's pneumomediastinum as a CT of the abdomen and pelvis was performed and revealed no other acute traumatic injuries in the abdomen or pelvis that could account for the free air.

However, the patient's cardiac arrest resulted in severe hypoxic brain injury, leading to subsequent diffuse cerebral edema with effacement of the basal cisterns and tonsillar herniation seen on CT of her head. She became increasingly hypertensive and was weaned off vasopressors and started on titratable nicardipine at a maximum of 12.5 mg per hour and was given a hypertonic saline bolus. The patient lost all evidence of brainstem reflexes five days after suffering cardiac arrest but continued to trigger some spontaneous breaths on the ventilator. Multiple family meetings were held regarding patient prognosis, and ultimately the patient was palliatively extubated with subsequent demise 26 days after arrival.

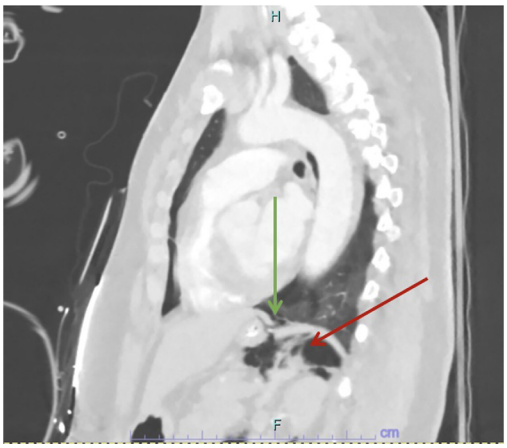

Image 2. The initial computed tomography with pulmonary embolism protocol performed on the day of presentation demonstrated discontinuity of the medial left hemidiaphragm **posteriorly** (green arrow) with air tracking from the lower mediastinum into the left retroperitoneal space posterior to the stomach (red arrow).

DISCUSSION

Our patient presented to the ED unresponsive and in severe respiratory distress and shortly afterwards went into PEA cardiac arrest which was followed by multiple rounds of cardiopulmonary resuscitation. After intubation, the patient was found to have significant resistance to bagging and significantly elevated peak pressures, which was initially thought to be secondary to positive end-expiratory pressure caused by the progressive accumulation of air, breath stacking, and the patient's underlying COPD. A CT with pulmonary embolism protocol was performed, which revealed non-displaced rib fractures along the anterior aspects of the left fifth and sixth ribs with a small, left-sided pneumothorax anteriorly. There was also discontinuity of the medial left hemidiaphragm posteriorly with air tracking from the lower mediastinum into the left retroperitoneal space from the lower mediastinum. While it is unclear whether the patient suffered her rib fractures (and subsequently left anterior pneumothorax) from cardiopulmonary resuscitation or from her fall, the instability of the patient at presentation suggests she suffered these rib fractures prehospital.

The exact etiology of the patient's diaphragmatic rupture and pneumoretroperitoneum is also unclear but could have been directly caused by blunt trauma, iatrogenically during resuscitation, or secondarily caused through extension of her pneumomediastinum, which likely was secondary to the Macklin effect and was further exacerbated by positive pressure ventilation. The fact that the patient's rib fractures were located anteriorly and her diaphragmatic rupture was posterior in nature also suggests the diaphragmatic rupture was not necessarily directly caused by her traumatic injuries. In review of the literature, pneumoretroperitoneum appears to be a rare diagnosis, especially in the setting of blunt trauma with no identifiable abdominal injuries.

Diaphragmatic injury also appears to be somewhat rare in the setting of blunt trauma. In fact, a study performed by Fair et al, published in the *American Journal of Surgery*, analyzed the incidence of diaphragmatic injury secondary to trauma and found that of 3,783 patients diagnosed with a traumatic diaphragmatic injury, only 33% of these patients suffered a diaphragmatic injury secondary to blunt trauma with only 7.6% of these patients suffering diaphragmatic injury secondary to falls.[7] Mortality was also significantly higher in patients with blunt traumatic diaphragmatic injury (19.8%) compared to patients with traumatic diaphragmatic injury secondary to penetrating trauma (8.8%), demonstrating the importance of rapid identification of diaphragmatic injury due to blunt trauma. In patients without a history of trauma, pneumoretroperitoneum is usually secondary to duodenal ulcer perforation, colonic perforation, retrocecal appendicitis, emphysematous cholecystitis, or secondary to iatrogenic causes of duodenal or colonic perforation due to endoscopic retrograde cholangiopancreatography and colonoscopy, respectively.[8]

Pneumoretroperitoneum secondary to pneumomediastinum is also a rare complication of status asthmaticus. While the incidence of pneumomediastinum secondary to asthma exacerbations is unknown, a small study done by Vianello et al revealed that five of 45 patients diagnosed with a severe asthma exacerbation also were diagnosed with pneumomediastinum.[9] None of the patients with pneumomediastinum required needle decompression, surgical intervention, or chest tube placement, and there were no in-hospital deaths; they had similar length of hospital stays as their counterparts without pneumomediastinum. Other studies have found the incidence to be much lower (one in 30,000).[10] The main treatment strategy employed for the patient's pneumomediastinum, pneumoretroperitoneum, and hypercapnic respiratory failure was lung protective ventilation. The patient's ventilator settings were continuously readjusted based on arterial blood gas findings that were performed daily and in response to changes in clinical status. The patient was also intermittently maintained on cisatracurium 0.1 mg/kg per hour to help maintain synchronicity with the vent, as occasionally the patient would hyperventilate resulting in breath stacking, which worsened her underlying respiratory failure. The patient had interval improvement in her pneumoretroperitoneum, pneumomediastinum, and pneumothorax using this closely monitored ventilatory strategy in conjunction with pulmonary critical care-trained intensivists.

When the patient's CT findings revealed evidence of tonsillar herniation, given worsening of her anoxic brain injury, her family elected for palliative extubation, and the patient passed peacefully after palliative extubation with family at bedside and discontinuation of all sedating medications in line with institutional protocols.

CONCLUSION

Whether traumatic or spontaneous in nature secondary to our patient's underlying history of obstructive lung disease, her pneumoretroperitoneum was thought to be secondary to the Macklin effect (causing pneumomediastinum) as well as via the defect in the left hemidiaphragm, given the patient had no abdominal or pelvic traumatic injuries identified. It is entirely possible that the patient's pneumoretroperitoneum was in fact secondary to profound intrathoracic pressures secondary to her COPD, alveolar rupture, pneumothorax, and positive pressure ventilation, which through the Macklin effect caused pneumomediastinum and subsequent pneumoretroperitoneum. The critically ill nature of the patient on presentation to the ED stresses the importance of prompt identification of this pathology to help guide management.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

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Common Iliac Artery Mycotic Pseudoaneurysm Associated with a Prevertebral Infection: A Case Report

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Introduction: Mycotic pseudoaneurysms are rare but severe sequelae of an arterial wall infection. If undiagnosed and untreated they can lead to significant morbidity and mortality through complications such as arterial rupture or dissection.

Case report: This report details the case of a 64-year-old-male who developed a left common iliac artery mycotic pseudoaneurysm from *Proteus mirabilis*, which was associated with a prevertebral abscess. The patient presented with isolated, left lower extremity edema and intermittent fevers. The case is unique in both the pathogen (*P mirabilis*) and in its association with presumed direct arterial wall infection from an adjacent prevertebral abscess.

Conclusion: The obscure presentation highlights the need for a high clinical suspicion of such a diagnosis when a patient presents with a certain constellation of symptoms and the right predisposing risk factors in their history. [Clin Pract Cases Emerg Med. 2023;7(4)242–245.]

Key words: *case report; mycotic pseudoaneurysm; Proteus mirabilis.*

INTRODUCTION

Bacterial and fungal infections of an arterial wall can lead to significant morbidity and mortality through the creation of aneurysms and pseudoaneurysms and the complications they entail.[1] The term "mycotic" is used to encompass both fungal and bacterial causes of infection.[2] These infections can occur in any artery, and case reports describe several locations.[3–5] These aneurysms and pseudoaneurysms are commonly attributed to septic emboli from infective endocarditis or direct injury (iatrogenic and traumatic) to the vessel wall.[4] Infected aneurysms are relatively rare and comprise less than 2% of all aortic aneurysms.[3] Additionally, isolated iliac artery aneurysm/pseudoaneurysms are exceedingly rare with an incidence of 0.03%.[6]

Patient presentations for pseudoaneurysms vary widely as the symptoms depend on size and location of the defect.[4] They are often painful and can cause local erythema.[5] Often, presentations are related to secondary effects of the vascular defect and not the defect itself. Infected aneurysms can present with persistent and recurrent fever without clear etiology.[7] In the modern antibiotic era, the most common organisms are *Salmonella* and *Staphylococcus aureus*; however, a wide range of Gram-positive and Gram-negative organisms has been associated with these infections.[3,7] To date, no case of *Proteus mirabilis* causing an iliac artery pseudoaneurysm associated with direct spread from a prevertebral abscess has been reported.

CASE REPORT

A 64-year-old-male with a history of type II diabetes mellitus and surgical lumbar decompression in 2013 presented to the emergency department (ED) for left leg swelling. His lumbar decompression was complicated by a methicillin resistant *S aureus* infection requiring multiple surgical washouts and intravenous antibiotics. After surgical washouts in 2013, he was transitioned to suppressive doxycycline until 2019. Shortly after stopping the doxycycline the patient developed a new paraspinal abscess

requiring interventional radiology drainage and further antibiotics in 2019. He was placed back on suppressive doxycycline and did well until 2022.

Beginning in late 2022 the patient began to have recurrent fevers and lumbar back pain. He was subsequently diagnosed with a small lumbar paraspinal abscess and was treated with six weeks of vancomycin and gentamicin via a percutaneous indwelling central catheter (PICC). However, his symptoms persisted, and he developed new-onset, isolated left lower extremity edema. This new symptom prompted an ED presentation. This department had no access to the patient's previous imaging or records except for one computed tomography (CT) of the abdomen and pelvis in the picture archiving and communication system. The patient had reported a possible "groin aneurysm," but these images were formally reviewed and showed no evidence of aneurysm.

On presentation, the patient reported subjective dyspnea but appeared comfortable in bed. Vital signs showed mild hypertension but no other abnormalities. Exam was remarkable for isolated, non-pitting edema of the left leg from mid-thigh to foot. The left lower extremity was warm, had normal pulses, and no motor or sensory abnormalities.

Initial work-up for deep venous thrombosis (DVT) with Doppler ultrasound was negative. Labs showed no leukocytosis, mild anemia with a hemoglobin 11.5 grams per deciliter (g/dL) (reference range: 13.5–17.0 g/dL) and a C-reactive protein elevated at 68.44 milligrams per liter (mg/L) (0.00–10.90 mg/L). Computed tomography of the chest, abdomen, and pelvis showed moderate left hydronephrosis with mid-ureter obstruction within a prevertebral fluid collection spanning the third lumbar to first sacral vertebra with possible ureter leak. This fluid collection partially encircled the inferior vena cava (IVC) and bilateral common iliac veins and contacted the posterior wall of the infrarenal aorta. It also extended to contact the posterior wall of the rectum where there was concern for possible fistulous connection (Image 1).

Considering these findings, both urology and the orthopedic spine team were consulted. Subsequent magnetic resonance imaging of the spine did not show evidence of an acute process, and no surgical interventions were recommended. Computed tomography cystogram showed no evidence of ureter leak. The urologist felt the obstruction was likely a partial stricture, and the urology team followed the patient during his hospital stay. The patient did not require any urologic intervention.

Due to the complexity of the patient and imaging findings, the images were reviewed with the attending radiologist on call who noted possible evidence of left common iliac DVT and possible common iliac arterial wall abnormalities. The decision was made to obtain a dedicated CT angiogram (CTA) of the pelvis. The CTA was concerning for a left common iliac artery mycotic pseudoaneurysm (MPA) within a prevertebral lumbosacral abscess (Image 2).

CPC-EM Capsule

What do we already know about this clinical entity?
Mycotic pseudoaneurysms (MPA) are relatively rare and difficult-to-diagnose clinical entities that can lead to severe morbidity and mortality.

What makes this presentation of disease reportable?
The presentation described in this case highlights a unique presentation of a MPA associated with a prevertebral infection.

What is the major learning point?
This case demonstrates the limitations of some initial diagnostic tests and the need to consider further testing if clinical suspicion remains for a diagnosis.

How might this improve emergency medicine practice?
Clinical practice can be improved through this case review by reminding clinicians to consider the benefits and limitations of common diagnostic testing.

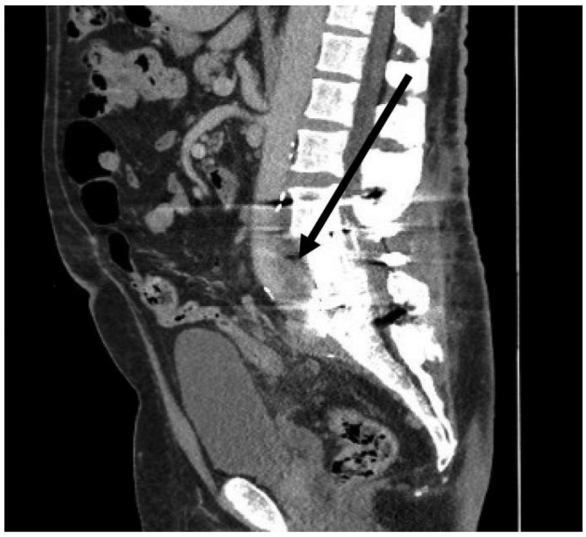

Image 1. Arrow demonstrating prevertebral abscess spanning the third lumbar to first sacral vertebra. Significant artifact present from spinal hardware.

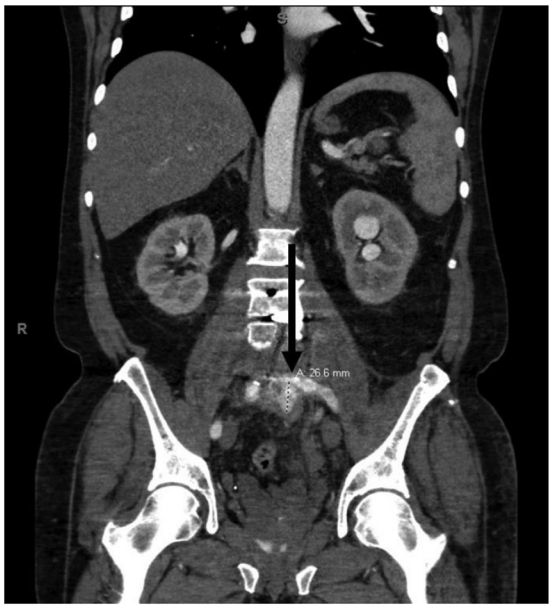

Image 2. Coronal computed tomography slide with marker indicating 26.6-millimeter left common femoral artery mycotic pseudoaneurysm.

The patient was taken to the operating room with vascular surgery where he underwent placement of a left common iliac artery stent as well as left femoral artery cutdown and repair. Computed tomography of the abdomen and pelvis with rectal contrast showed no evidence of fistulous connection to the rectosigmoid wall. Definitive repair was completed four days later with right common femoral artery to left superficial femoral artery bypass, IVC filter placement, sartorius myoplasty, and left common iliac artery embolization.

His blood cultures were positive for *P mirabilis,* which was thought to be the culprit of the MPA. His treatment included a prolonged course of vancomycin and ceftriaxone. The patient had a remote history of *S aureus* infection associated with his spinal hardware, but the prevertebral infection was not directly sampled during this presentation.

He was discharged six weeks after presentation with prescriptions for continued home intravenous (IV) antibiotics via PICC. Review of his three-month follow-up appointment revealed the patient was continuing to do well.

DISCUSSION

Mycotic pseudoaneurysms can occur in multiple different vascular areas and be caused by an array of organisms both bacterial and fungal. They present significant challenges to the clinician and the patients they affect. Diagnosis can be difficult due to their often indolent presentation with non-specific symptoms. Care must be taken when taking a history from patients who may be affected. Identifying possible risk factors for infections (IV drug use, endocarditis, skin and soft tissue infections, vascular grafts, diabetes mellitus) are key to beginning the proper work-up. If they are not diagnosed and treated, they can rapidly lead to significant morbidity and mortality if they rupture.[1] This case presented a unique patient who was diagnosed with a left common iliac MPA due to *P mirabilis,* which appeared to be a direct complication from an extensive retroperitoneal abscess associated with a previous spinal procedure.

Proteus mirabilis is well known for its ability to cause urinary tract infections (UTI) and is often associated with long-term catheterization.[8] It can cause bacteremia, which is typically associated with an existing UTI. There are reports of *P mirabilis* causing vertebral column infections by hematogenous seeding via the Batson plexus.[8] This venous plexus consists of valveless veins that allow blood flow between the deep pelvic veins and the internal vertebral veins.[8] It is much less likely to cause bacteremia from another source, and it is also less likely to be a contaminate in blood cultures, considering it is not skin flora. Literature review did reveal two cases of *P mirabilis*-associated MPA; however, they were both associated with indwelling valves or grafts.[9,10] The patient discussed did have a history of *S aureus* paraspinal infections. Blood cultures were obtained and showed no evidence of other organisms on initial and subsequent testing.

There is sparse data on the occurrence of mycotic aneurysms or pseudoaneurysms. which can be linked to direct bacterial invasion of the vessel wall.[7] There are some case reports describing spinal-associated infections leading to arterial wall infections in the aorta.[7,11] Literature review showed no known cases of a spinal associated abscess leading to pocket expansion and subsequent involvement of a common iliac artery.

Another interesting feature of this case is the relation of the presenting complaint (unilateral leg swelling) with the ultimate diagnosis (mycotic aneurysm due to *P mirabilis)*. Sensitivities of venous ultrasound for DVT have been reported between 62–94%.[12,13] This wide variation depends on the type of ultrasound performed (compression, duplex, and triplex) and the site(s) involved. Indirect CT venography has been found to better visualize proximal DVTs in the large pelvic veins.[14] For patients in whom there is a high suspicion for a proximal DVT and negative DVT ultrasound, indirect CT venography would be a reasonable next step. Determining the best imaging for complicated patients can be difficult. A discussion with a radiologist about the specific concerns surrounding a patient can help select the best modality of CT and assist in accurate contrast timing to delineate the high-yield structures.

Mycotic aneurysms may exert mass effect on or fistulize with surrounding structures, including adjacent deep veins, ureters, and bowel.[15] This patient's initial presentation was due to unilateral leg swelling from the DVT associated with the MPA. The symptoms most associated with mycotic aneurysm are nonspecific and include fever, pain over the

affected vascular site and, rarely, septic shock.[16] This case highlights the importance of maintaining a high index of suspicion in patients who present with unexplained symptoms of mass effect (hydroureter, unilateral leg swelling) and occult infection (historical risk and subacute systemic infectious symptoms), even when initial and routine imaging does not demonstrate causative pathology.

This case involves multiple different imaging modalities and demonstrates the importance of selecting the correct modality for the suspected abnormality. This patient had multiple CT images prior to the angiogram, which was ultimately diagnostic. Discussions with the attending radiologist reviewing the imaging revealed the detail needed to see the MPA was only obtained through the contrast timing associated with a CT angiogram. The routine contrasted scan did not allow for the detail needed to diagnose the patient's MPA.

CONCLUSION

The patient discussed in this case report had a unique and rare presentation of an already rare disease process. Mycotic pseudoaneurysms alone are rare, but this case was associated with an uncommon organism and presentation that was further complicated by lack of access to historical surgical records. While making the diagnosis of a MPA is difficult, missing it can lead to devastating outcomes for patients. This patient's history included important risk factors and symptoms, which indicated the possible underlying etiology. A high clinical suspicion is needed to accurately diagnose these cases. To accurately diagnose this life-threatening condition our patient received multiple imaging modalities and consultations during an extended ED stay. Treating clinicians in the ED must realize that the uncovering of a MPA is a tough diagnostic challenge that is not made quickly. Clinical perseverance will be needed to make an accurate and critical diagnosis.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

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Spontaneous Aortic Rupture: A Case Report

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Introduction: Acute aortic syndrome (AAS) includes the disease processes of aortic dissection, penetrating atherosclerotic ulcer, and intramural hematoma. This case demonstrates an atypical presentation of the disease and offers approaches to potentially prevent missed diagnoses.

Case Report: An 87-year-old female with hypertension and Alzheimer's dementia presented to the emergency department with stable vital signs and a chief complaint of throat pain. Initial work-up was significant for ischemia on electrocardiogram and elevated troponin. Computed tomography of the soft tissue neck revealed evidence of a ruptured aorta.

Conclusion: Aortic rupture is a fatal complication of AAS. In an elderly patient with a history of hypertension, ischemic changes on electrocardiogram, and nonspecific pain, AAS should be on the emergency physician's differential even in the setting of a benign or limited history and exam. [Clin Pract Cases Emerg Med. 2023;7(4)246–249.]

Keywords: *acute aortic syndrome; aortic rupture; throat pain; case report.*

INTRODUCTION

Acute aortic syndrome (AAS) is a medical diagnosis that includes multiple thoracic and abdominal aortic pathologies.[1] Penetrating atherosclerotic ulcer (PAU) results from an atherosclerotic aortic plaque invading through the internal elastic lamina into the aortic media (Figure 1). Intramural hematoma (IMH) results from the rupture of vasa vasorum, small vessels that provide oxygen to the arterial wall, which subsequently causes bleeding into the outer layers of the aortic media.[2] Aortic dissection (AD) includes the pathophysiology identified in IMH with additional disruption of the intima.[3] Current literature suggests PAU and IMH account for 2.3–7.6% and 6–10% of cases of AAS, respectively.[2] Aortic rupture is a fatal complication of all three entities; however, the risk is greatest with PAU with a rate of 42%, followed by IMH at a rate of 35%.[4] Current estimates indicate an incidence of AD between 2.6–3.5 cases per 100,000 person-years with the majority of patients being males in their sixth decade of life according to the International Registry of Acute Aortic Dissection.[5] The sudden onset of severe, sharp pain was the single most common presenting complaint.

In this case, we present a female patient in her ninth decade of life with dementia at baseline, endorsing a chief complaint of throat pain. Given the wide variety of clinical presentations and high mortality rate associated with aortic rupture, diagnostic strategies include contrast-enhanced spiral computed tomography (CT), transesophageal echocardiogram, and magnetic resonance imaging when clinical suspicion is high. However, in cases with history limited by memory impairment and non-specific symptomatology unexplained by physical examination, an algorithmic approach to diagnosis and treatment becomes difficult. The case report presented here details the late identification of a life-threatening aortic rupture in an emergent setting with weak initial evidence to suggest the diagnosis.

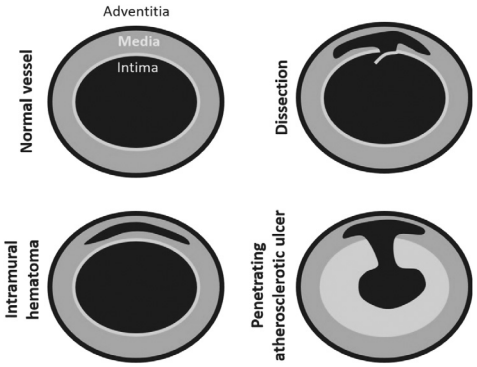

Figure 1. Acute aortic syndrome pathophysiology with illustration of the layers of the aorta.[6]

CPC-EM Capsule

What do we already know about this clinical entity?
Acute aortic syndrome involves multiple pathologies including penetrating atherosclerotic ulcer, which accounts for 42% of fatal cases of aortic rupture.

What is the major learning point?
Diagnosing aortic injury in patients presenting with non-specific symptoms is challenging and requires a blend of clinical gestalt and targeted diagnostic work-up.

How might this improve emergency medicine practice?
Symptomatology may be benign even in life-threatening situations of aortic injury, thereby increasing the importance of maintaining an inclusive differential.

CASE REPORT

An 87-year-old female with a past medical history significant for hypertension and Alzheimer's dementia presented to the emergency department (ED) with her daughter, endorsing throat pain that began upon awakening in the morning. The patient's daughter reported that her mother had consumed a regular meal without difficulty the night prior. The patient also reportedly had consumed caustic substances in the past including cleaning supplies, confusing them for water/juice. According to the patient and her family, she had not had any upper respiratory infection symptoms such as cough and fever. The patient also specifically denied chest pain, shortness of breath, abdominal pain, and vomiting. She was hemodynamically stable and afebrile on presentation including blood pressure of 138/87 millimeters of mercury and heart rate of 82 beats per minute with an oxygen saturation of 98% on room air. On physical examination, airway was patent with no erythema or swelling of the oropharynx, no pulse deficits in the extremities, lymphadenopathy, or neurological deficits with the exception of baseline orientation to self only.

Laboratory work-up was significant for elevation in serial troponin from 0.048 to 0.086 nanograms/milliliter (ng/mL) (reference range: 0.000–0.034 ng/mL). Initial electrocardiogram (ECG) showed sinus bradycardia at a rate of 43 beats per minute with T-wave inversions in leads I and aVL, more pronounced compared to prior studies. Chest radiograph revealed atherosclerotic calcifications of the aorta and mild blunting of the right costophrenic angle without widening of the mediastinum. There was less than 5 millimeters of separation of the intimal calcification from the outer aortic border, which was similar to findings in a previous radiograph (Image 1).

Given the patient's ECG changes and elevated troponin, admission for acute coronary syndrome work-up was pursued. Additionally, the history of previous caustic ingestions in the setting of severe dementia and presenting complaint of throat pain raised clinical suspicion for corrosive upper gastrointestinal tract injury. A CT soft tissue neck with contrast was obtained to evaluate for edema, stranding, stricture formation, and associated fistulous complications.

During evaluation, the patient became severely bradycardic, apneic, and eventually unresponsive. Resuscitation efforts were initiated and discontinued shortly thereafter, following clarification from the patient's daughter of "Do Not Resuscitate/Do Not Intubate" code status. At

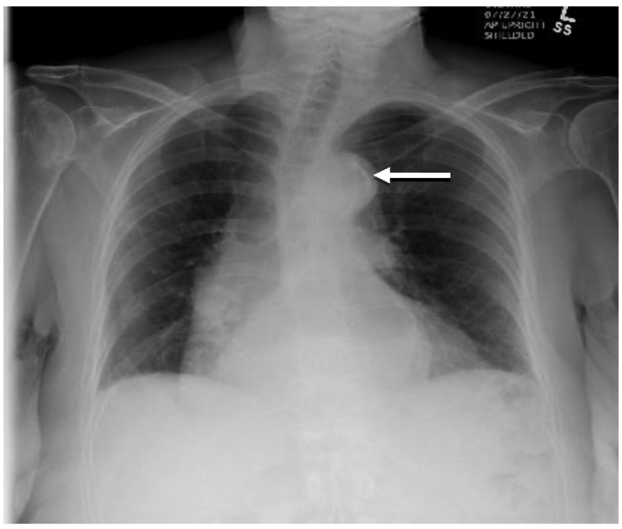

Image 1. Chest radiograph showing mild left costophrenic blunting and atherosclerotic calcification of the aorta (arrow).

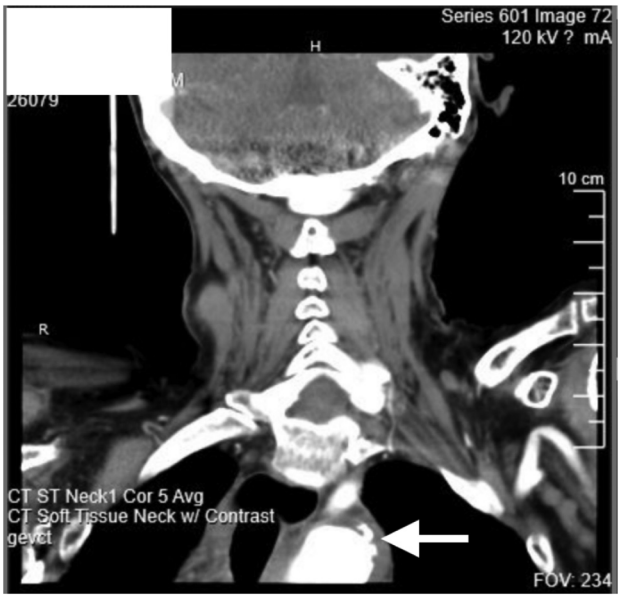

Image 2. Computed tomography soft tissue neck with arrow indicating the contrast extending into the posterior wall of the proximal descending aorta consistent with a penetrating atherosclerotic ulcer in coronal view.

the conclusion of the code, radiology called to inform of fluid in the superior mediastinum suggestive of aortic leak and contrast extending into the posterior wall of the proximal descending thoracic aorta suggestive of penetrating atherosclerotic ulcer (Image 2).

DISCUSSION

As documented in emergency medicine literature, most case reports discuss aortic injuries secondary to traumatic mechanisms. In these instances, routinely obtained contrast-enhanced imaging of the chest and abdomen lead to the prompt identification of pathology even in the absence of hemodynamic instability. The limited case reports of spontaneous aortic rupture recommend that physicians maintain a high clinical suspicion for aortic injury in a bimodal age distribution to include younger patients with connective tissue disease and older males with chronic hypertension and other atherosclerotic risk factors. Greater than 50% of patients describe tearing/ripping pain in the chest radiating to the back with non-specific physical exam findings, which may include pulse discrepancies, neurologic deficits, or new-onset murmurs. The imaging modality of choice for AAS is CT angiogram (CTA).[7] Treatment varies depending on the patient's symptoms, location of injury, and whether the patient has a singular disease process vs multiple manifestations of AAS with options ranging from medical management focused on blood pressure control to surgical intervention with open and endovascular approaches available.

In the case described above, the patient presented hemodynamically stable with an atypical chief complaint of throat pain with a primary risk factor of hypertension. Given her advanced dementia, she was unable to provide reliable chronological and qualitative information regarding her symptoms. Work-up was remarkable for ischemic changes on ECG and elevation in troponin. Physicians reached the ultimate diagnosis of aortic rupture incidentally through imaging initially obtained to further investigate potential caustic ingestion as the etiology of the patient's throat pain. However, at the time of diagnosis, the patient had already become unresponsive and pulseless.

Spontaneous aortic rupture is a diagnosis with high mortality that has great potential to be seen and missed by emergency physicians. Elements of initial work-up in patients with undifferentiated chest/back pain and history of hypertension that can aid in the prompt diagnosis of aortic rupture include calcium sign on chest radiograph (white line of calcium within aortic knob and measure to outer edge of the aortic knob), ischemia on ECG, and widened aortic outflow tract on point-of-care ultrasound.[8] Abnormalities in the aforementioned diagnostic studies may bolster clinical suspicion for aortic injury and serve as indication for further investigation including CTA of the chest and abdomen.

Admittedly, diagnosis of spontaneous aortic rupture without convincing history and physical elements is difficult to reach. Few cases of aortic dissection presenting as throat pain have been reported in previous literature. In a case from 2004, a 53-year-old male presented to the ED with sensation of retained foreign body in his throat in the setting of known history of cigarette smoking. The patient was discharged following a benign oropharyngeal exam including plain films of the neck and laryngoscopy. Ten hours later, the patient returned and was found to be hypotensive with tachycardia. Imaging of the chest and abdomen was significant for ascending aortic dissection with cardiac tamponade. The patient passed away during an emergent attempt at operative repair.[9]

Similarly, in 2010 a 58-year-old male with a history of hypertension presented with intermittent dysphagia with stable vital signs. Laboratory work-up was largely unremarkable and imaging was not obtained, but the patient was referred to an emergency otolaryngology unit for suspected peritonsillar abscess. Shortly thereafter, the patient expired after being found to have a massive ascending aortic dissection on imaging and cardiac tamponade physiology.[10] The case presented here and the reports found through literature review share very important elements including initial presentation characterized by throat pain in a patient with atherosclerotic risk factors and normal oropharyngeal exam. Significant consideration in this patient population should be given to a possible diagnosis of AAS. Ultimately, successful management will depend on a clinically broad

diagnostic approach and flexible index of suspicion with consideration of risk factors and overall epidemiology of aortic rupture.

CONCLUSION

Acute aortic syndrome is an umbrella term to describe pathologies involving aortic injury generally seen in patients with a history of trauma or risk factors for atherosclerosis. While a standard for medical and surgical management of the condition has been agreed upon in literature, reaching the diagnosis in select patients can be difficult, as discussed in the case above. Due to the high risk of mortality in patients presenting with aortic rupture, it is important for physicians to consider it in their differential diagnosis based on presentation, history, ECG, and radiologic findings including radiograph and ultrasound.

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Intentional Overdose on Liquid Clonazolam Reversed with Flumazenil: A Case Report

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Introduction: Clonazolam is a designer benzodiazepine that can be purchased illicitly on the internet. The use of designer benzodiazepines is increasing in both the United States and abroad, and patients may present to the emergency department (ED) after intentional or non-intentional overdose.

Case report: This case report describes a patient who presented to a community ED after an intentional overdose on liquid clonazolam and was successfully treated with flumazenil.

Conclusion: Since the pharmacologic action of clonazolam is similar to benzodiazepines, the sedative-hypnotic effect can be reversed with flumazenil, a benzodiazepine antagonist. [Clin Pract Cases Emerg Med. 2023;7(4)250–252.]

Keywords: *case report; clonazolam; designer benzodiazepine; flumazenil.*

INTRODUCTION

Designer drugs, also known as novel psychoactive substances, are synthetic analogs of a controlled substance that are designed to mimic the effect of the original substance while avoiding regulation and law enforcement.[1] Clonazolam is a designer benzodiazepine that was first synthesized in 1971.[2] Clonazolam toxicity was identified in Europe in 2016 and in the United States in 2017.[3] The psychiatry community sounded the alarm in 2015,[4] and the toxicology community has been reporting and following the trends of designer benzodiazepines toxicity.[1,3,4–6] However, there have been no reports to date of clonazolam toxicity in the emergency medicine literature.

This case report describes a patient who took an intentional overdose of concentrated, liquid clonazolam. He presented to a community ED with a sedative-hypnotic toxidrome and was managed successfully with flumazenil.

CASE REPORT

A 31-year-old male with a history of untreated depression and alcohol abuse was brought by ambulance to the emergency department (ED) for somnolence after an intentional overdose of liquid clonazolam, which he had received from a friend. The patient had sent a suicidal text to his ex-girlfriend shortly before he ingested approximately half of a three-milliliter bottle of liquid clonazolam (Image 1). When she arrived at his house, within approximately 30 minutes of his text, he appeared confused and intoxicated, and she called emergency medical services (EMS). Upon EMS arrival, the patient was somnolent. His fingerstick blood sugar was 108 milligrams per deciliter (mg/dL) (reference range 70–100 mg/dL). When the patient arrived at the community ED, his blood pressure was 115/79 millimeters of mercury, heart rate 89 beats per minute, respiratory rate 22 breaths per minute, and oxygen saturation of 94% on room air. He was somnolent and minimally following commands but was protecting his airway. Pupils were 4 millimeters, equal, round, and reactive to light. He was not diaphoretic. Lungs were clear, and heart was regular rate and rhythm. The abdomen was soft and nontender, and bowel sounds were present. He had normal patellar reflexes without clonus. There were a few old, superficial abrasions to the left forearm. Glascow Coma Score was 12 (minus three for verbal response).

Lab work was significant for a low normal bicarbonate of 22 milliequivalents per liter (mEq/L) (reference range 22–29 mEq/L). The anion gap was normal. Potassium was slightly low at 3.2 millimoles per liter (mmol/L) (3.6–5.2 mmol/L). Salicylate and acetaminophen levels were

Image 1. Bottle of clonazolam ingested by patient.

undetectable. Ethanol was 204 mg/dL. Urine drug screen (UDS) was positive for cocaine. It should be noted that benzodiazepines are not evaluated on the hospital's UDS. An electrocardiogram (EKG) was non-ischemic with normal intervals.

Approximately one hour after arrival in the ED (90 minutes after ingestion), the patient became more somnolent with respiratory depression and oxygen saturations in the mid-80s. Flumazenil 0.2 mg intravenous (IV) was administered with immediate improvement in respiratory effort and oxygenation. One hour later, an additional 0.2 mg dose of IV flumazenil was again administered for respiratory depression and hypoxia. Because there were no intensive care unit beds available, the patient continued to board and be managed in the ED. Within six hours, his mental status normalized, and he was medically cleared for psychiatric evaluation. The next day, the patient recounted taking the clonazolam as a suicide attempt. He recalled drinking alcohol and perhaps using cocaine.

CPC-EM Capsule

What do we already know about this clinical entity?
Clonazolam is a designer benzodiazepine that was first synthesized in 1971. Its toxicity was first identified in 2016.

What makes this presentation of disease reportable?
To date there have been no reports of clonazolam toxicity in the emergency medicine literature nor of its reversal with flumazenil.

What is the major learning point?
Designer benzodiazepines use is increasing, and patients may present to the ED after an overdose. The sedative-hypnotic effect can be reversed with flumazenil.

How might this improve emergency medicine practice?
This case raises the awareness of designer benzodiazepine, its easy availability online, and the ability to reverse its adverse effects with flumazenil.

DISCUSSION

Clonazolam (6-(2-chlorophenyl)-1-methyl-8-nitro 4H-triazolo[4,3-α] benzodiazepine) is an analog of clonazepam.[2] It can be found in tablet, capsule, pellet, blotter, and liquid form, and can be purchased on the internet.[5] Clonazolam is considered a designer benzodiazepine (along with dozens of others)[1] that has no medicinal indication and is not currently regulated by the US Food and Drug Administration (FDA). In December 2022, the FDA published a temporary order to add five synthetic benzodiazepines (clonazolam, etizolam, flualprazolam, flubromazolam, and diclazepam) to Schedule 1 under the Controlled Substances Act.[7]

Since clonazolam behaves similarly to benzodiazepines, it is likely safe to assume that it could be reversed by flumazenil, a benzodiazepine antagonist which "competitively inhibits the activity of benzodiazepine and non-benzodiazepine substances that interact with benzodiazepine receptors site on the gamma-aminobutyric acid (GABA)/benzodiazepine receptor complex. It can also reverse the binding of benzodiazepines to benzodiazepine receptors."[8] Typical onset of action is 1–2 minutes with an 80% response rate within three minutes. Its peak effect is 6–10 minutes with a duration of 19–50 minutes.[8] There is currently a black box warning for flumazenil in the US as there has been a correlation with seizures, especially in patients on benzodiazepines long term, and in those with severe tricyclic antidepressant overdose.[9] Flumazenil is used more liberally in Europe. The package insert from Europe states not to use flumazenil if it is being administered to control a potentially

life-threatening situation such as elevated intracranial pressure or a serious epileptic seizure. It also warns not to use flumazenil in mixed intoxications involving tri- or tetracyclic antidepressants, as the toxicity of the antidepressants can be masked by the protective benzodiazepine effects.[10]

The patient presented here had a co-ingestion of highly concentrated clonazolam (approximately 7.5 mg) along with ethanol and cocaine. There is no regulated dose for clonazolam, but profound sedation is thought to occur at doses of 0.5 mg.[11] The cocaine may have counteracted some of the sedative effects of the clonazolam; however, the patient could not later recall the precise timing of his cocaine use. Since he had no known long-term use of benzodiazepines, the decision to try reversal with flumazenil was chosen as opposed to intubation. The treating emergency physician is licensed in both the US and Europe and, therefore, was comfortable and experienced with its use. This patient responded favorably to the flumazenil within the expected time frame and had no resultant seizure activity.

CONCLUSION

The use of designer benzodiazepines is increasing, and patients may present to the ED after accidental or intentional overdose. The sedative-hypnotic toxidrome is similar to benzodiazepine overdose and, after assessment of risk vs benefit, flumazenil may be helpful in its reversal.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

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Traumatic Anterior Tibial Artery Pseudoaneurysm: A Case Report

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Introduction: Traumatic pseudoaneurysms of the limbs are rare, with few cases described in vascular literature. Treatment is variable and dependent upon presentation and impact on local anatomy affected. Rapid assessment can be performed with ultrasound and assist in treatment decisions. We describe a case of traumatic anterior tibial artery pseudoaneurysm, which was rapidly identified with point-of-care ultrasound leading to definitive surgical management.

Case Report: A 37-year-old female presented to the emergency department for evaluation of right lower extremity pain and swelling following an exercise session with weighted squats and thigh abductor machines. She was found to have an anterior tibial artery pseudoaneurysm on point-of-care ultrasound, later confirmed with formal ultrasound as well as angiography, and was admitted for surgical management.

Conclusion: Traumatic pseudoaneurysms can rapidly be differentiated from other mass lesions and contributors to compartment syndrome using point-of-care ultrasound. [Clin Pract Cases Emerg Med. 2023;7(4)253–256.]

Keywords: *pseudoaneurysm; ultrasound; compartment syndrome; case report.*

INTRODUCTION

Arterial pseudoaneurysm is defined as a vascular wall abnormality resulting in blood collection in adjacent extraluminal space.[1] Traumatic pseudoaneurysms of the limbs are quite rare, and there is a paucity of literature to standardize work-up in the emergency department (ED) setting. There are few case reports on tibial artery pseudoaneurysms with the majority related to prior trauma or infectious etiologies. Spontaneous occurrences are essentially unreported. Multiple studies in the surgical literature have demonstrated various repair techniques. However, there is a paucity of data in the emergency medicine literature regarding identification on acute presentation, potential emergent complications, and options for management.[2,3] Lower extremity pseudoaneurysms are often found in the femoral vasculature with trials of compression therapy favored before surgical intervention based on the patient's clinical presentation.[4,5]

CASE REPORT

A 37-year-old female presented to the ED for evaluation of right lower extremity pain and swelling immediately following an exercise session about 24 hours earlier that involved squat motion and hip abduction with maximum load of 40 pounds. She additionally complained of decreased sensation to the dorsum of her right foot. These symptoms progressed through the next 24 hours, and the patient noticed a worsening expanding mass to the lateral aspect of her proximal right lower leg. Eight months prior to presentation, the patient had been in a motor vehicle collision that resulted in an open right tibial shaft fracture and fibular fracture requiring intramedullary nail fixation of the tibia. Three

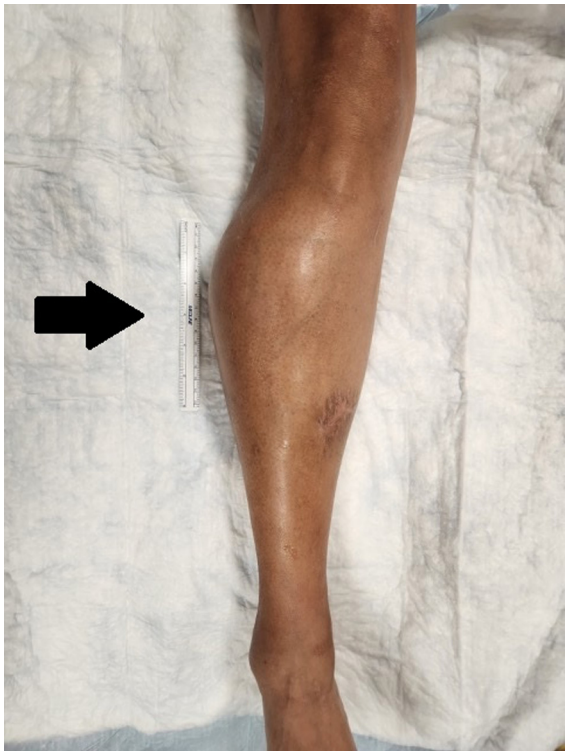

Image 1. Anterior view of patient's right lower leg with black arrow indicating area of tense and painful swelling.

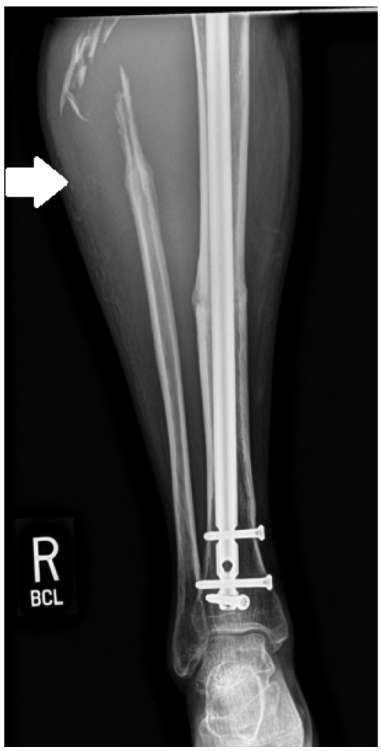

Image 2. Initial anterior-posterior view of tibia/fibula on presentation, demonstrating unrepaired proximal fibular fragments with surrounding soft tissue swelling indicated by white arrow.

CPC-EM Capsule Summary

What do we already know about this clinical entity?
Arterial pseudaneurysms of the limbs are rare and are usually the result of trauma. They are potentially limb-threatening.

What makes this presentation of disease reportable?
Point-of-care ultrasound rapidly identified pulsatile pseudoaneurysm, avoiding bedside compartment pressure measurement that could have caused hemorrhage.

What is the major learning point?
Point-of-care ultrasound (POCUS) can rapidly characterize vascular sources of limb swelling, including pseudaneurysm.

How might this improve emergency medicine practice?
Continued POCUS practice for emergency clinicians will enable them to feel comfortable making limb- saving decisions.

months later, she sustained a mechanical fall causing further displacement of the right fibula, which was not repaired and resulted in chronic nonunion. She successfully completed a physical rehabilitation program and had been doing well until the time of presentation to the ED. The patient had been participating in a rehabilitation program for narcotic dependence that arose after her previous orthopedic surgery. She was otherwise healthy.

Examination was remarkable for marked swelling of the anterolateral portion of the right lower extremity just distal to the knee (Image 1). The right lower leg compartments were firm, mildly compressible, tender, and nonpulsatile. Her right dorsalis pedis and posterior tibialis artery pulses were intact, and she had some mild numbness and paresthesias in the distribution of the superficial peroneal nerve distally. She did not have pain with passive ankle dorsiflexion or plantarflexion, and there were no overlying skin changes.

Radiographs demonstrated prior traumatic injury with hardware in place and marked soft tissue swelling in the lateral proximal calf (Image 2). Point-of-care ultrasound (POCUS) revealed pulsatile flow at the right anterolateral lower leg, suggestive of a pseudoaneurysm (Image 3). Formal ultrasound confirmed the presence of a three-centimeter pseudoaneurysm arising from the anterior tibial artery along the margin of an 11-centimeter hematoma in the upper,

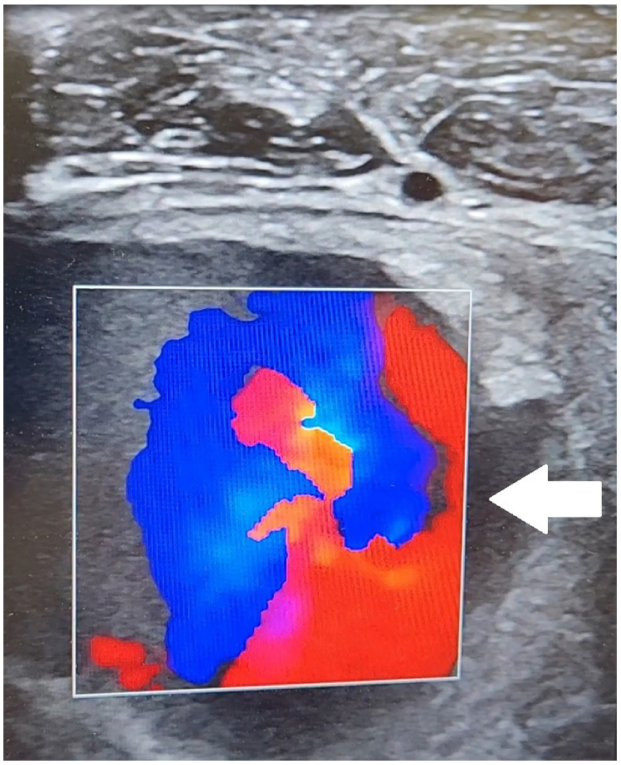

Image 3. Point-of-care ultrasound demonstrating a 4 × 3 centimeter anechoic collection arising from the anterior tibial artery with to-and-fro color flow seen within the collection, consistent with a pseudoaneurysm as indicated by white arrow.

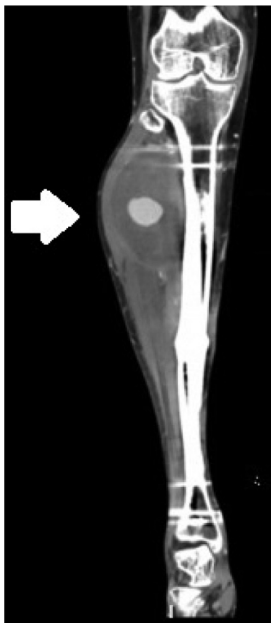

Image 4. Coronal cut of computed tomography angiogram of the right lower extremity demonstrating a heterogeneous collection in the lateral, upper right calf, consistent with a hematoma. On the arterial phase, there is contained ballooning of contrast arising from the anterior tibial artery, consistent with a pseudoaneurysm in the area indicated by white arrow.

lateral right calf. This finding was also confirmed on computed tomography angiogram (Image 4).

Vascular surgery was consulted, and the patient underwent an open repair of the right anterior tibial artery pseudoaneurysm with evacuation of the hematoma and a four-compartment fasciotomy. Intraoperatively, the patient was noted to have jagged bone fragments from her fibular fracture in the pseudoaneurysm cavity, which were removed. Fasciotomy was also performed at that time because the patient had clinical signs of compartment syndrome, which included sensory deficit in addition to tense and swollen compartment. Her numbness resolved, and she was discharged home on postoperative day two.

DISCUSSION

We describe the case of a patient presenting with a presumed post-traumatic pseudoaneurysm in the setting of chronic right fibular fracture with nonunion. The initial presentation of lower extremity pain and swelling with sensory deficit was concerning for compartment syndrome. The patient's workout activity 24 hours prior to presentation was suspected to have caused right anterior tibial artery damage from the adjacent right fibular bone fragments. This ultimately resulted in pseudoaneurysm formation. Traumatic pseudoaneurysm has been previously described only rarely in case reports.[2,3] Because compartment syndrome and pseudoaneurysm can present similarly, it is essential to discern vascular involvement. Fortunately, POCUS was performed and rapidly revealed pseudoaneurysm, which was confirmed upon formal ultrasound and computed tomography. Point-of-care ultrasound has become a mainstay of ED evaluations and in this case prevented catastrophe. Rapid identification of anechoic mass with definitive color demonstration of pulsatile component was achieved prior to decompression of the area. This facilitated appropriate vascular surgery consultation and controlled operative management. The insertion of a needle for measurement of compartment pressures in this patient with a swollen, tense extremity could have resulted in hemorrhage. We advocate for the use of POCUS to rapidly assess patients with similar presentations to rule out underlying vascular pathology such as psuedoaneurysms before proceeding with compartment pressure measurements.

CONCLUSION

Traumatic pseudoaneurysms of the limbs are rare and should be included in the differential of limb swelling. Emergency physicians must be able to rapidly identify and initiate treatment for this potentially limb- threatening condition. This can easily be accomplished with point-of-care ultrasound, which has become an essential tool in emergency medicine. In this case, we were able to use POCUS to direct operative intervention preventing

premature compartment pressure measurement, which could have led to massive hemorrhage.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

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An Uncommon Diagnosis of Necrotizing Mastoiditis Presenting as Bell's Palsy: A Case Report

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Introduction: The benign nature of Bell's palsy has led to a lack of a standardized work-up, and dangerous underlying mimics are at risk of being missed.

Case Report: An 84-year-old female with a history of vertigo presented to the emergency department with a left-sided facial droop consistent with Bell's palsy. After further work-up, the patient was diagnosed with bilateral necrotizing mastoiditis.

Conclusion: Unilateral facial weakness involving the forehead and palpebral fissures is often diagnosed as idiopathic Bell's palsy. Various pathologies can present with unilateral facial weakness, and the differential needs to remain broad. [Clin Pract Cases Emerg Med. 2023;7(4)257–261.]

Keywords: *case report; Bell's palsy; necrotizing mastoiditis.*

INTRODUCTION

Bell's palsy is a sudden-onset, unilateral facial weakness primarily affecting the peripheral portion of the seventh cranial nerve.[1] This results in a one-sided facial droop involving the forehead and palpebral fissures. It is the most common cause of unilateral facial weakness (60–70%) with an incidence of approximately 20 per 100,000 worldwide.[1] The etiology is often unknown with most cases classified as idiopathic.[1–3] Approximately 71% of cases resolve spontaneously, and 84% of patients regain near full function including facial muscle and nerve control.[1,4] Due to this lack of a common etiology and the frequency of spontaneous improvement, a standardized treatment protocol for Bell's palsy does not exist other than a possible short course of corticosteroids or antivirals.[1] Given the characteristic physical exam findings in Bell's palsy and the ability to differentiate from stroke using history and exam, most guidelines recommend against routine imaging in acute cases due to cost and lack of impact on the course of therapy.[5]

There is a subset of patients who have Bell's palsy due to a more dangerous underlying pathology. Phenothiazine has been observed to cause a dystonic reaction that presents similarly to Bell's palsy.[6] Adenoid cystic carcinomas of the parotid gland can also be disguised as Bell's palsy for years.[7] These diagnoses may be missed due to the assumption that they fall in the same category as the vast majority of benign Bell's palsy cases. We report a case of an 84-year-old female who presented to the emergency department (ED) with a left-sided facial droop that was consistent with Bell's palsy, but was caused by bilateral bacterial necrotizing mastoiditis.

CASE REPORT

An 84-year-old female with a past medical history of chronic leukopenia, chronic headaches, vertigo, hypertension, and osteoarthritis presented to the ED as a stroke alert from her outpatient otolaryngology appointment for progressive hearing loss and bilateral ear pain. Upon arrival, the patient revealed a four-day history of left-sided facial droop and a four-week history of headaches, describing the pain as originating from both ears radiating to her jaw. The patient also noted bilateral tinnitus over the prior 2–3 weeks with decreased hearing and ear swelling. She denied any significant change in the chronic periodic vertigo,

which she had suffered for years. She also denied any peripheral numbness, tingling, or weakness.

Physical examination found a well-appearing elderly female in no acute distress. Vital signs were concerning for hypertension (184/89 millimeters of mercury) but were otherwise within normal limits. The patient had an obvious left-sided facial droop, with paralysis of the left-sided facial muscles involving the forehead and palpebral fissures. She was unable to raise her left eyebrow but was able to close her eyes bilaterally. Pupils were equal and reactive to light with intact extraocular motions. The remainder of the neurologic examination was reassuring with normal mental status, no strength or sensory deficits in bilateral upper and lower extremities, normal finger-to-nose test, no visual field deficits, and an ability to ambulate with a steady gait. There was significant swelling and tenderness of the external auditory canals and mastoids bilaterally. The tympanic membranes were non-visualized bilaterally due to significant canal edema. All other physical exam findings were unremarkable.

The patient's notable mastoid process tenderness, acute hearing loss, and inflammation of her auricles raised concern for an erosive or infectious process leading to her Bell's palsy. Her facial paralysis involved the ipsilateral forehead, making stroke a less likely cause of her presentation. She had no other signs or symptoms on exam concerning for an acute embolic or hemorrhagic stroke including weakness or numbness in her limbs, ataxia, gait instability, or visual deficits. Due to the high suspicion for mastoiditis or other underlying infectious etiologies, a computed tomography (CT) brain and temporal bone was performed and revealed erosion of the mastoid segments of the greater facial nerve canals on the left side.

CPC-EM Capsule

What do we already know about this clinical entity?
Unilateral facial paralysis involving the forehead is a common presentation in the ED with Bell's palsy at the top of the differential. The work-up is often minimal.

What makes this presentation of disease reportable?
Unilateral facial paralysis involving the forehead due to bilateral necrotizing mastoiditis is an uncommon and life-threatening cause of this condition.

What is the major learning point?
To avoid missing more insidious pathology, clinicians should develop a robust differential when presented with unilateral facial paralysis involving the forehead.

How might this improve emergency medicine practice?
Maintaining a robust differential for unilateral facial weakness with forehead involvement will reduce misdiagnoses and delays in diagnosing serious mimics.

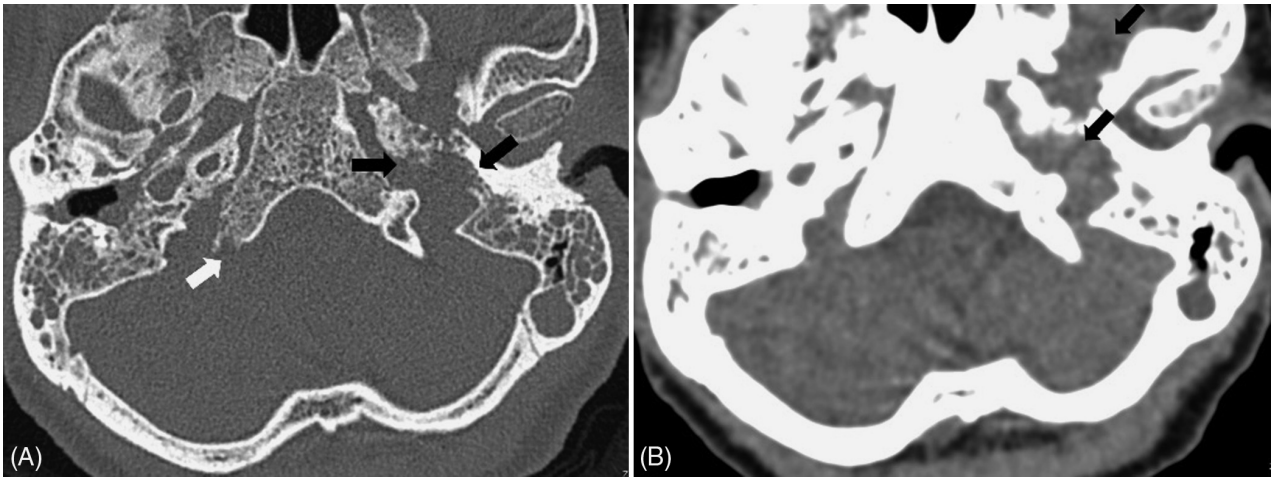

Image 1. Axial computed tomography bone (A) and soft tissue window (B) images demonstrate opacification of the bilateral mastoid air cells and right middle ear cavity, compatible with otomastoiditis. Image A shows erosive changes in the right posterior clivus (white arrow) and left petrous apex and jugular foramen (black arrows). Image B shows abnormal soft tissue seen in the left jugular foramen and masticator space (black arrows).

The CT also demonstrated opacification of the mastoid air cells bilaterally with erosion of the bilateral proximal styloid processes (Image 1). New erosive and destructive changes were also seen in the right occipital condyle and right posterior clivus suspicious for an infectious or neoplastic process.

Given these imaging findings, the patient was empirically treated with intravenous (IV) vancomycin and clindamycin for presumed erosive mastoiditis. Subsequent head and neck magnetic resonance imaging demonstrated widespread skull base osteomyelitis (SBO). There was noted to be involvement of the stylomastoid foramen bilaterally, extending along the left greater than right facial nerves, likely an extension of the infectious process. There was also likely sigmoid sinus thrombosis on the right extending into the jugular vein (Image 2).

Otolaryngology was consulted and placed bilateral ear wicks in the ED for topical ofloxacin administration. The next day, the patient underwent a bilateral myringotomy tube placement, adenoidectomy, and complete bilateral mastoidectomy. She was continued on IV antibiotics with transition to cefepime, vancomycin, and metronidazole for broader coverage while middle ear cultures taken in the operating room were pending. On post-operative day one, the patient had significant pain relief and improvement in her facial nerve function. Cultures from the mucoid discharge of the left middle ear grew *Pseudomonas aeruginosa*, and her antibiotic regimen was changed to oral ciprofloxacin based on culture sensitivities.

During her hospitalization a CT venogram also confirmed a right venous sinus thrombus, and the patient was placed on enoxaparin 80 milligrams four times daily for the next three months. She was discharged with a six-week course of oral ciprofloxacin and outpatient follow-ups with otolaryngology, neurology, infectious disease, and her primary care physician. The patient was seen in clinic three months later and was doing well with resolved facial nerve palsy, resolved headaches, and significantly improved hearing.

DISCUSSION

This case represents a unique presentation of Bell's palsy due to erosive mastoiditis and SBO. The patient presented with bilateral ear pain radiating down to her jaw with associated swelling and tenderness of the bilateral external auditory canals. These concerning symptoms, in addition to her several weeks of bilateral hearing loss, placed an erosive otogenic process high on the differential. As a result, it was decided to defer corticosteroids and pursue imaging. Although the medical team suspected an erosive process, SBO was an unexpected diagnosis.

Skull base osteomyelitis is a rare complication of infectious otogenic processes such as necrotizing mastoiditis. The soft tissue infection spreads from the mastoid process to the base of the skull via fissures and sutures, with resulting facial nerve palsy in up to 60% of cases.[8,9] The most common causative organism in SBO is *P aeruginosa* (75–95%). Although SBO predominantly afflicts patients with

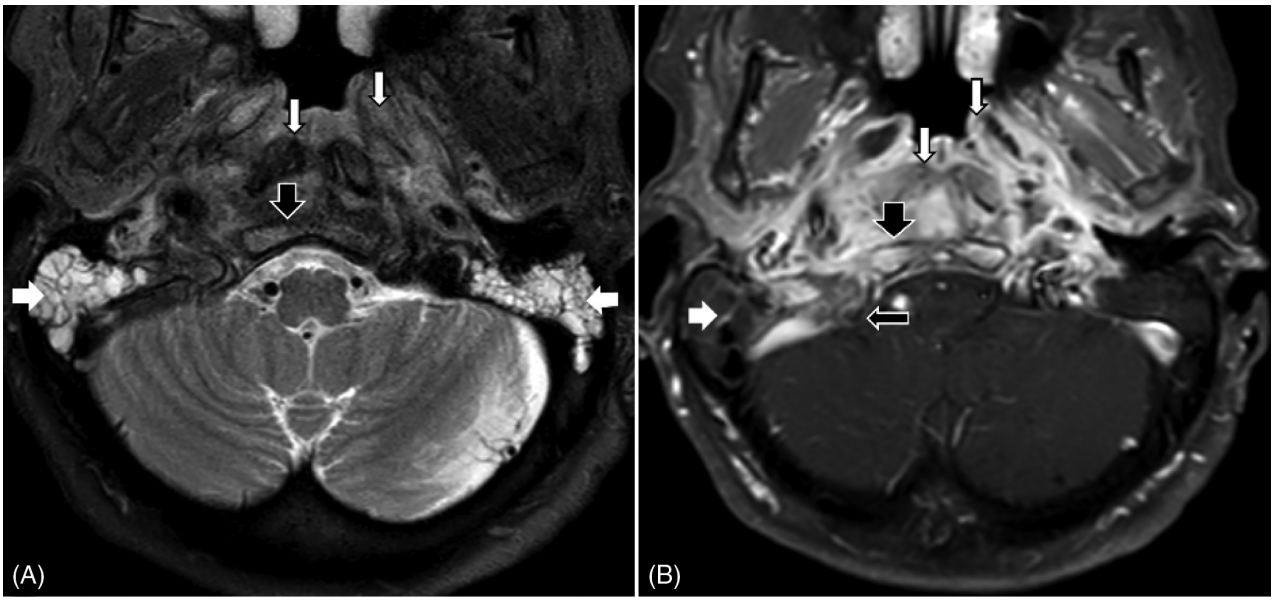

Image 2. Axial T2 fat-suppressed image (A) and T1 fat-suppressed post-contrast image (B) through the nasopharynx, skull base, and mastoid air cells demonstrate opacification of the mastoid air cells bilaterally with enhancement of the septa on the right (wide white arrows) compatible with otomastoiditis. Edema and enhancement in the clivus (wide black arrows) compatible with skull base osteomyelitis. Edema, enhancement, and soft tissue thickening of the nasopharynx and prevetebral muscles (thin white arrows) compatible with extensive inflammation. Filling defect in the right sigmoid sinus and jugular bulb compatible with dural venous sinus thrombosis (thin black arrow).

uncontrolled diabetes,[9] the patient presented in this case report did not have diabetes. Additionally, the clinical presentation is typically delayed and has a mortality of up to 20%.[9] The patient in this case was incredibly well appearing and had no obvious risk factors apart from age and chronic leukopenia. This unique case raises the importance of maintaining a broad differential when approaching often benign pathologies such as Bell's palsy, which are encountered in the ED.

When building this differential, it is important to consider recent research elucidating the multiple etiologies possibly responsible for Bell's palsy.[10] The leading cause currently is herpes simplex virus type 1 (HSV-1). This also correlates with the increased incidence of the palsy as patients age, as seroconversion of HSV-1 follows the same trend.[1,2] Other causative viruses include varicella zoster virus, cytomegalovirus, and Epstein-Barr virus.[11] A subset of Bell's palsy is caused by bacterial pathogens, often following otitis externa, otitis media, or mastoiditis.[12] A sequencing study performed in 2020 found that clinical samples from Bell's palsy patients undergoing surgical decompression grew a variety of organisms. Viral, fungal, and bacterial organisms were all found in various patients' samples with human betaherpesvirus 7, *Malassezia restricta*, and *P aeruginosa* being the most common etiologies, respectively.[11] With the increasing number of causative agents being reported—some associated with serious sequelae—it is important for emergency physicians to apply due diligence when working up Bell's palsy.

The literature shows multiple cases where a diagnosis of idiopathic Bell's palsy was incorrectly made. In a retrospective cohort study of 43,979 patients across California EDs, 358 patients presenting with a unilateral facial paralysis were misdiagnosed with an idiopathic Bell's palsy. The patients would be later diagnosed as having Guillain-Barré syndrome (10.9%), herpes zoster (23.2%), otitis media or mastoiditis (24.0%), or ischemic stroke (27%).[13] Multiple cases show that involvement of the forehead does not rule out stroke entirely. Strokes within the dorsal pons have been reported in the literature to cause ischemia in the motor facial nerve and result in isolated unilateral facial paralysis involving the forehead.[13]

The effects of these misdiagnoses can be devastating, with some patients experiencing delays in care of up to three years for parotid malignancies due to their slow-onset facial paresis similar to Bell's palsy.[14] This data further emphasizes the importance of maintaining a broad differential for unilateral facial paralysis to prevent misdiagnoses and the resulting delays in care that will ultimately harm patients.

To reduce additional iatrogenic harm to these patients, it is critical to consider these less common etiologies prior to administering corticosteroids. Despite their effectiveness in alleviating symptoms in many Bell's palsy patients, corticosteroids can also leave them vulnerable to underlying infection due to recruitment of T helper type 2 cells over type 1.[1,15] In a case-control retrospective study of 2,632 critically ill patients exposed to corticosteroids, it was found that 46% developed secondary infections in the hospital with an increased incidence in mortality compared to 23% of the control group. If the patient in the currently reported case had been given corticosteroids without further evaluation, she could have suffered a significant worsening of her necrotizing mastoiditis. Therefore, it is important to evaluate these patients thoroughly for an underlying bacterial etiology of their facial palsy before initiating corticosteroids.

CONCLUSION

Bell's palsy is a common and often benign condition. The patient presented here had a presentation of Bell's palsy in the setting of serious bilateral necrotizing mastoiditis complicated by skull base osteomyelitis. This underlying process was only caught due to thorough evaluation and special attention to red flags present in her history and physical examination. This case emphasizes the importance of maintaining a broad differential when approaching commonly benign pathologies such as Bell's palsy. Maintaining a high clinical suspicion for the rarer etiologies of unilateral facial paralysis despite initial impressions will lead to more accurate diagnoses and fewer delays in care for those who need it most.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

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Diagnosis of Endophthalmitis and Orbital Abscess by Ultrasound: A Case Report

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Introduction: The diagnosis of ocular pathology by point-of-care ultrasound (POCUS) has been well established for entities such as retinal detachment, vitreous hemorrhage, posterior vitreous detachment, and lens dislocation.[1] However, the use of ultrasound to detect other conditions such as orbital abscess and endophthalmitis in the emergency setting is rarely reported.

Case Report: We present a case in which POCUS was used to confirm the suspected diagnosis of endophthalmitis and orbital abscess. This case report will review the ultrasonographic findings of orbital abscess and endophthalmitis, as well as briefly discuss the literature for the use of ultrasound for these applications.

Conclusion: Point-of-care ultrasound can be used to rapidly diagnose infectious pathology of the eye and orbit, which could potentially decrease time to diagnosis and time to consultation of these vision-threatening pathologies. [Clin Pract Cases Emerg Med. 2023;7(4)262–265.]

Keywords: *case report; ultrasound; endophthalmitis; orbital abscess.*

INTRODUCTION

Infections of the orbit and periorbital region are common ophthalmic emergencies resulting in significant local and systemic morbidity. Loss of vision occurs in approximately 10% of patients, and systemic complications can include meningitis, intracranial abscess, sepsis, and death.[2] Rapid diagnosis of orbital infections is crucial to prevent complications and improve patient outcomes. Clinical examination is not always reliable to distinguish pre-septal pathology from other serious vision-threatening conditions. The utility of point-of-care ultrasound (POCUS) to diagnose entities such as retinal detachment, vitreous hemorrhage, posterior vitreous detachment, and lens dislocation has been well established.[1] However, the use of ultrasound for the diagnosis of other conditions such as orbital abscess and endophthalmitis in the emergency department (ED) setting has been rarely described.[3–5] This case illustrates the ultrasonographic findings of orbital abscess and endophthalmitis, and the role of ultrasound in the evaluation of the patients presenting to the ED with these conditions.

CASE REPORT

A 52-year-old female with past medical history of hypertension, type 2 diabetes mellitus, hypothyroidism, and bilateral cataract surgery was transferred to our ED for further evaluation. She initially presented to the outside ED for seven days of worsening right eye pain, redness, swelling, and worsening vision, which progressed to complete right-sided vision loss five days prior to her initial presentation. She also noted three days of fever but no recent symptoms of upper respiratory infection, dental infection, eye trauma, or recent surgeries. Vital signs were unremarkable. On examination, the right periorbital soft tissues were significantly swollen and erythematous with chemosis of the conjunctiva, cloudy anterior chamber, and exophthalmos of the right eye. Right eye intraocular pressure was 40 millimeters of mercury (mm Hg) (normal 10–21 mm Hg)

with painful extraocular movements. The left eye was normal.

Point-of-care ultrasound performed in the ED showed loculated echogenic material within the vitreous humor, posterior vitreous detachment, and fluid in sub-Tenon's space (potential space between the capsule of the eye and choroid) consistent with endophthalmitis. It also showed soft tissue thickening of the eyelid, edema in the orbital fat, and a hypoechoic fluid collection next to the lateral aspect of the globe consistent with orbital cellulitis and orbital abscess (Image 1, Video 1). An orbital computed tomography (CT) demonstrated a right orbital abscess (18 mm × 5 mm × 12 mm) along the lateral and inferolateral margin of the right orbit with preseptal and postseptal cellulitis without osseous involvement (Image 2).

Ophthalmology was consulted and a bedside intraocular aspiration and intravitreal injection of antibiotics was performed. The patient was admitted to internal medicine for broad-spectrum antibiotics. Her intravitreal aspirate grew coagulase negative staphylococcus, and the inciting event was thought to be syphilitic uveitis and vitritis complicated by staphylococcal infection given her positive syphilis serology. The patient did not have any other physical findings of secondary or tertiary syphilis, such as rash or gummas. She was discharged on hospital day 12 on three weeks of intravenous (IV) ceftriaxone and vancomycin and two weeks of oral metronidazole. She was scheduled for a vitrectomy at another facility.

Population Health Research Capsule

What do we already know about this clinical entity?
Orbital abscess and endophthalmitis are uncommon vision-threatening infections that require timely intervention for good outcomes.

What makes this presentation of disease reportable?
The co-occurrence of orbital abscess and endophthalmitis in the same patient is a rare and complex presentation of these serious eye infections.

What is the major learning point?
Point-of-care ultrasound can rapidly diagnosis both orbital abscess and endophthalmitis, which can lead to earlier IV antibiotics and surgical consultation.

How might this improve emergency medicine practice?
Recognizing the ultrasound findings of orbital abscess and endophthalmitis would improve time to diagnosis and treatment.

DISCUSSION

Rapid diagnosis of endophthalmitis and/or orbital abscess is paramount due to the vision-threatening nature and risk for intracranial extension of infection. The most common ultrasonographic findings of endophthalmitis include a moderate to severe quantity of echogenic debris in the vitreous humor and vitreous membranes with loculations.[6,7] Less common ultrasonographic findings include retinal or choroidal detachments, which are present in 7–20% of cases of endophthalmitis but portend worse visual outcomes.[6,7] Other less common findings include choroidal thickening, macular edema and optic disc edema, posterior vitreous detachment, and fluid in sub-Tenon's space.[6,7] The diagnostic accuracy of ultrasound of these findings is not well established, but small, non-randomized studies suggest ocular ultrasound has a sensitivity of >90% and specificity of >79% for the diagnosis of endophthalmitis.[8,9]

Ultrasonographic findings of orbital cellulitis include hyperechoic inflammatory intraconal fat, heterogenous collection of intraorbital material with mixed echogenicity (hyperechoic and hypoechoic), and extraocular muscle edema and/or displacement.[4,10–12] Orbital abscess typically appears as an anechoic to hypoechoic fluid collection, but as with soft tissue abscesses elsewhere in the body, they can also appear isoechoic and contain hyperechoic debris.[4,10–12]

The diagnostic accuracy of ultrasound of these findings of orbital cellulitis and abscess has not been well established, but small non-randomized studies suggest ocular ultrasound has a potential for high diagnostic accuracy.[13,14] Computed tomography is more sensitive for diagnosing orbital abscess than ultrasound and will assist with operative planning. However, there are several situations in which ultrasound can be particularly helpful in the evaluation of infections of the eye and orbit. Firstly, POCUS can be performed rapidly at bedside, leading to earlier surgical consultation and hospital admission for administration of IV antibiotics, both of which could potentially lead to better outcomes for these vision-threatening conditions. Additionally, ultrasound can help provide important clinical information beyond what can be obtained in the history and physical examination alone, especially in cases without a clear-cut diagnosis, and the clinician is debating the necessity of additional imaging with CT. For example, a patient may have trouble differentiating eye pain from eyelid edema and inflammation versus pain from extraocular movements.

Objective findings of deeper infection on ultrasound would indicate the need for a CT scan in cases that lack obvious physical exam findings of orbital cellulitis, such as

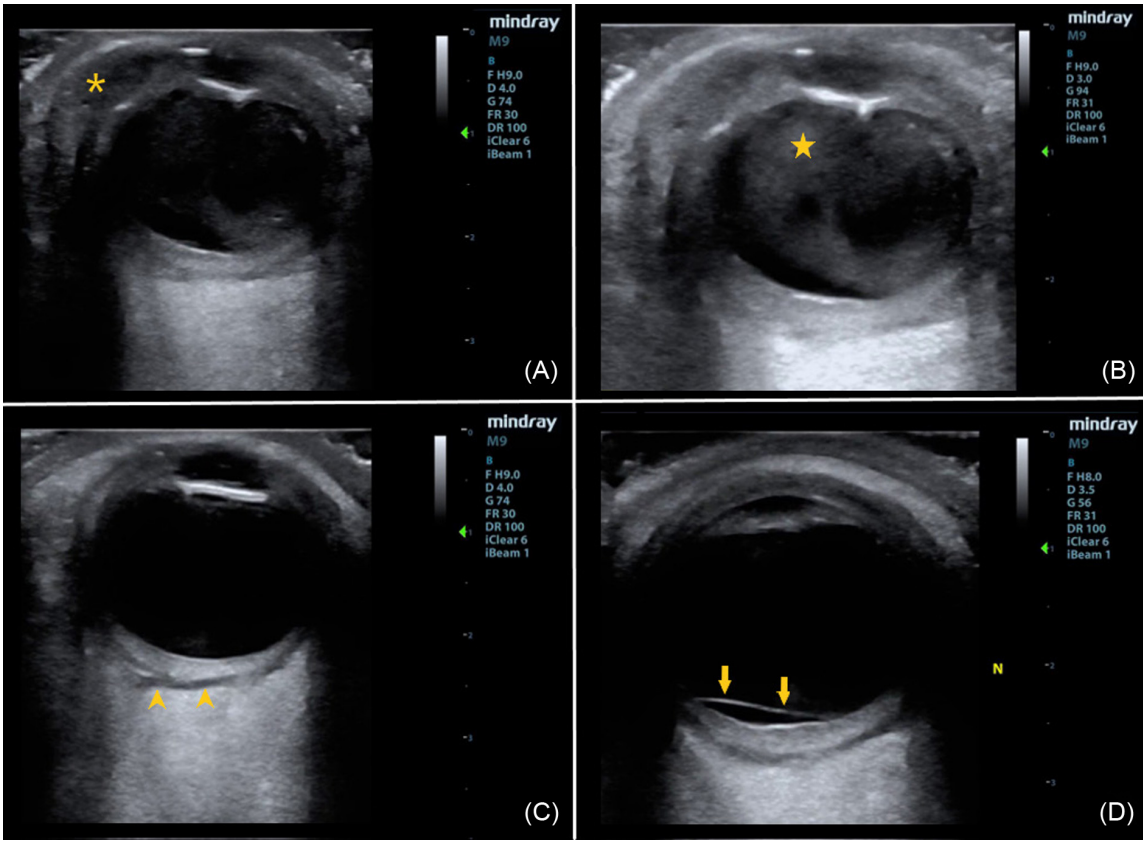

Image 1. (A) The hypoechoic fluid collection (asterisk) in the lateral aspect of the orbit is consistent with orbital abscess. (B) The echogenic purulent debris within the vitreous humor (star) has an irregular and loculated appearance and is the key ultrasonographic finding of endophthalmitis. (C) and (D) The hypoechoic region posterior to retina and choroid (arrowheads) represents fluid in sub-Tenon's space (potential space), and the thin membrane crossing the optic nerve (arrows) represents a posterior vitreous detachment, both of which are less common ultrasonographic findings of endophthalmitis.

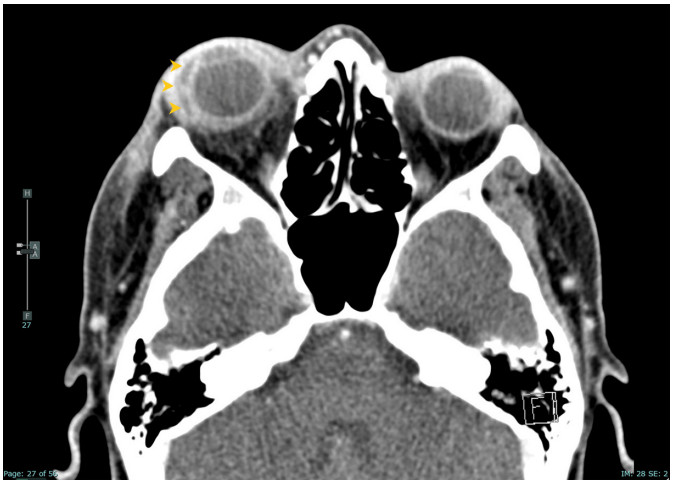

Image 2. Rim-enhancing fluid collection (arrowheads) lateral to the right globe consistent with orbital abscess.

proptosis and ophthalmoplegia. In these indeterminate cases, a point-of-care ultrasound showing only superficial eyelid edema would potentially increase the confidence in the diagnosis of periorbital cellulitis and the appropriateness of outpatient management with oral antibiotics which would lead to decreased cost, decreased ED length of stay, and avoidance of ionizing radiation exposure to the patient from CT. Lastly, in resource-limited settings lacking CT availability, ultrasound could help guide appropriate management toward transfer of the patient to a CT-capable facility.

CONCLUSION

This patient's case of simultaneously diagnosed endophthalmitis and orbital abscess is a rare presentation of ocular/orbital infection. It highlights the potential for POCUS to rapidly diagnose non-traumatic infectious pathology of the eye as well as lead to early ophthalmology consultation and appropriate treatment of these vision-threatening disease processes.

Video. Short cine clip (1/4x speed) demonstrating fluid in sub-Tenon's space, echogenic purulent debris within the vitreous humor, and posterior vitreous detachment, all of which are potential findings of endophthalmitis.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

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Pyolaryngocele Presenting with Acute-onset Stridor

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Case presentation: This case describes the classic imaging findings of pyolaryngocele and highlights the importance of prompt imaging for diagnosis of clinically occult airway lesions. The case also highlights how pyolaryngoceles can become large and present with acute-onset clinical symptoms, including stridor and dyspnea.

Discussion: Pyolaryngoceles represent an uncommon but life-threatening complication of laryngoceles. Laryngoceles are frequently seen as an incidental, abnormal, air-filled dilation of the laryngeal saccule related to various local pathologies of the larynx. They are often asymptomatic. Occasionally they can become secondarily infected, in which case they are called pyolaryngocele, and they can cause rapid-onset, life-threatening airway compromise. [Clin Pract Cases Emerg Med. 2023;7(4)266–267.]

Keywords: *pyolaryngocele; stridor; computed tomography.*

CASE PRESENTATION

A 61-year-old male presented to the emergency department (ED) with difficulty breathing, stridor, and fever (100°Farenheit) that developed over the course of 24 hours. Laboratory studies were significant for borderline leukocytosis with elevated neutrophil count. Contrast-enhanced computed tomography (CT) of the neck demonstrated a peripherally enhancing, lobulated fluid collection with layering debris within the right paraglottic space, with external extension through the right thyrohyoid membrane and severe airway compromise (Image).

Imaging characteristics in conjunction with the clinical findings were consistent with pyolaryngocele. Emergency tracheostomy was performed to relieve the dyspnea, followed by surgical incision and drainage of the fluid collection, which was notable for pus. The patient was discharged in stable condition and was without complication at outpatient follow-up visit.

DISCUSSION

Stridor is a high-pitched breathing sound produced by the abnormal flow of air, most prominently heard during inspiration. Although stridor is more common in children, it can present acutely across different age groups due to several conditions including foreign body aspiration, tracheitis, epiglottitis, and anaphylaxis. Abscesses along the pharyngeal and laryngeal compartments can also present with stridor, however, with a more subacute clinical course. Radiographic evaluation is usually the first line of investigation; however, this can frequently be unrevealing. Moreover, atypical clinical presentation and older age group should prompt the emergency physician toward further imaging work-up in the form of CT. Given the widespread availability of CT scanners in the ED, this is quickly replacing radiographs as the imaging modality of choice for evaluation of acute airway conditions.[1]

A laryngocele is an abnormal, air-filled dilation of the laryngeal saccule that communicates with the lumen of the larynx. It is believed to be secondary to increased laryngeal pressures and traditionally described in trumpet players; however, it is now more commonly seen secondary to excessive coughing and obstructive lesions.[2,3] Laryngoceles can be classified into internal, external, or mixed (most common) subtypes based on whether or not the laryngocele is confined to the larynx or herniates through the thyrohyoid membrane.[4] Laryngoceles are often asymptomatic but can become secondarily infected, in which case they are called

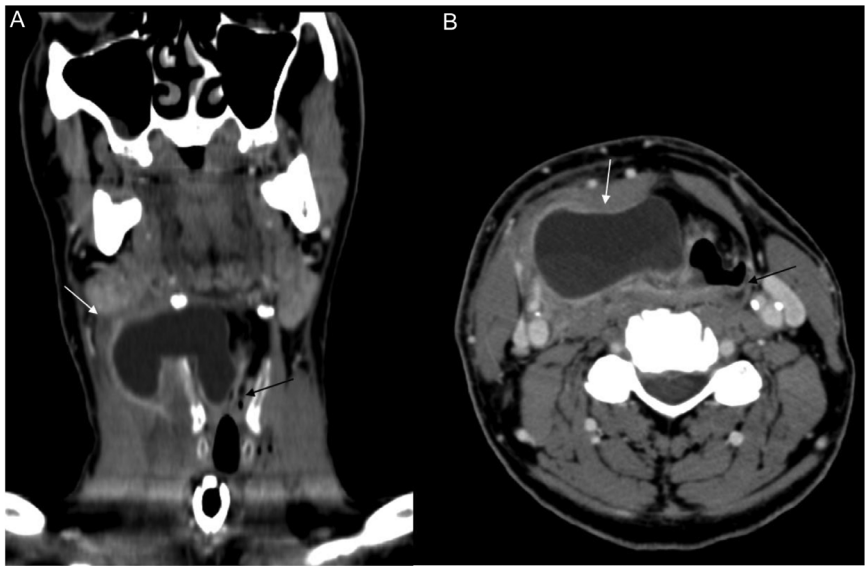

Image. Coronal (A) and axial (B) contrast-enhanced computed tomography of the neck demonstrates a peripherally enhancing fluid collection arising from the right laryngeal vestibule, extending into the right anterior neck through the thyrohyoid membrane (*white arrow*), with narrowing of the laryngeal airway (*black arrow*), consistent with pyolaryngocele. Mild inflammatory changes noted in the surrounding soft tissues.

CPC-EM Capsule

What do we already know about this clinical entity?
Pyolaryngoceles are superinfected laryngoceles (8–10%) that present with muffled voice, odynophagia, and stridor.

What is the major impact of the image(s)?
Images show characteristic appearance of pyolaryngocele with smooth peripheral enhancement and central fluid contents.

How might this improve emergency medicine practice?
Clinicians should be aware of this entity and have a low threshold to recommend contrast-enhanced computed tomography for evaluation.

pyolaryngoceles. These are rare clinical entities, presenting with fever, sore throat, and stridor. Imaging (CT) can identify the pyolaryngocele subtype, location, and extent of involvement of adjacent laryngeal structures, and aid in the treatment approach. Pyolaryngoceles are managed with drainage of the fluid collection and resection of the underlying laryngocele.[5]

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

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Bullous Pemphigoid Causing Successive Emergency Department Visits

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Case Presentation: In this case presentation, an 84-year-old male with Fitzpatrick type IV skin tone experienced blistering due to bullous pemphigoid (BP), first on the distal upper left extremity and then on the distal lower extremities, chest, and back. These symptoms resulted in three visits to the emergency department within a month, as well as an episode of hospitalization. Despite treatment, the blistering did not resolve until future outpatient care with dermatology.

Discussion: Bullous pemphigoid is a rare autoimmune disease where autoantibodies target hemidesmosomal proteins causing basement membrane destruction and tense subepithelial bullae with pruritus. While uncommon, the incidence of BP is increasing. Bullous pemphigoid tends to affect older adults, appearing as a rash prior to bullae formation on the abdomen, extremities, groin, axillae, or mucosa. Bullous pemphigoid may also be drug-related with atypical symptoms. Diagnosis of BP should be based on immunopathology, and initial treatment of BP is through corticosteroid or doxycycline. [Clin Pract Cases Emerg Med. 2023;7(4)268–270.]

Keywords: *bullous pemphigoid; bullae; blister; pruritus.*

CASE PRESENTATION

An 84-year-old male with a history of diabetes mellitus, Alzheimer's disease, Parkinson's disease, and coronary artery disease with previous coronary artery bypass graft presented to the emergency department (ED) with hyperglycemia and blistering on his distal upper and lower extremities at multiple stages of healing (Images 1–3). Four weeks prior, the patient went to the ED for painless pruritic blistering on the left upper extremity that had spread toward the axilla and groin over a period of two months. He was treated for presumptive bullous pemphigoid (BP) and discharged on doxycycline and clobetasol. Symptoms improved until two weeks following ED discharge, and the patient returned to the ED for now painful blistering of his distal lower extremities, as well as blistering of the chest and back. He was admitted to the hospital for three days and seen by dermatology, who entertained the diagnosis of bullous lymphedema versus bullous diabeticorum. A biopsy was not performed due to concern for poor wound healing. Leg compression and elevation was recommended upon discharge.

The patient returned to the ED one week after discharge due to ongoing blisters and extremity swelling. At this visit, vital signs were stable with most recent hemoglobin A1C elevated at 10.0% (normal below 5.7%; prediabetes 5.7%–6.4%; diabetes above 6.5%). Dermatology was consulted and recommended outpatient evaluation. Insulin was modified, and the patient was discharged. Diagnosis of BP was confirmed at later outpatient evaluation through positive tests for BP antibodies. The patient responded to prednisone on an oral treatment regimen of 60 milligrams (mg) daily for 30 days, tapered to 40 mg for three weeks and then 20 mg for three weeks.

DISCUSSION

Bullous pemphigoid is a rare autoimmune disease in which autoantibodies target hemidesmosomal proteins dystonin-e (BP antigen 1 or BP230) and collagen XVII (BP antigen 2 or

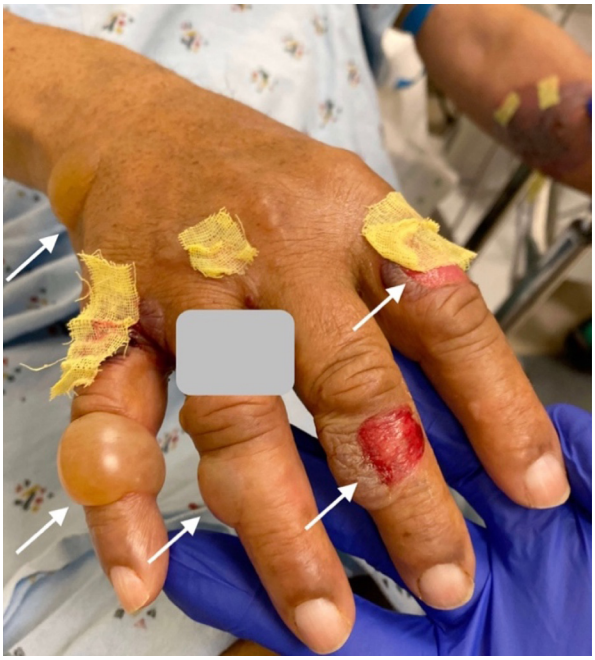

Image 1. Dorsal aspect of hand with bullous pemphigoid blisters, some ruptured; arrows indicate blisters. (Gray box covers patient identifier.)

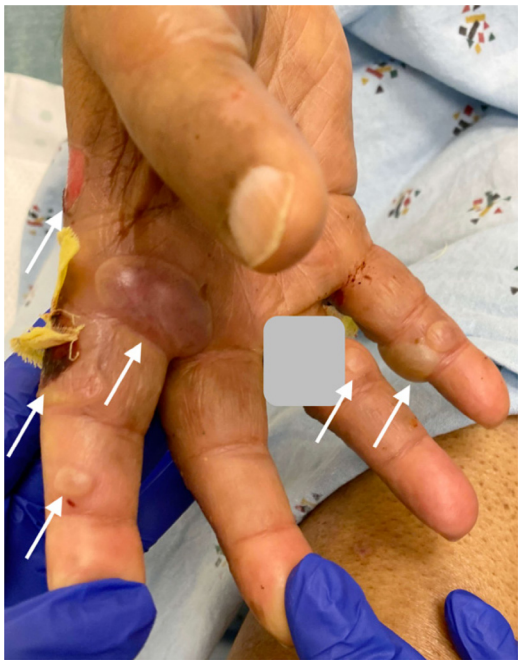

Image 2. Ventral aspect of hand with bullous pemphigoid, some ruptured; arrows indicate blisters. (Gray box covers patient identifier.)

BP180), causing basement membrane destruction and tense subepithelial bullae with pruritus.[1,2] Incidence is increasing with estimates in the United States and European states between 10-43 per one million individuals per year.[2,3] Classically, BP affects older adults, sometimes appearing as a

CPC-EM Capsule

What do we already know about this clinical entity?
In bullous pemphigoid (BP) autoantibodies target hemidesmosomal proteins causing basement membrane destruction and subepithelial bullae with pruritus.

What makes this presentation of disease reportable?
An 84-year-old male with Fitzpatrick type IV skin tone experienced BP that spread across multiple regions and resulted in three ED visits.

What is the major learning point?
This case, showing images of BP on darker skin tone, demonstrates how the severity of the condition may evolve despite treatment.

How might this improve emergency medicine practice?
The case aids in recognition of BP and offers a quick guide to initial treatment as well as insight into the possible persistence of the condition.

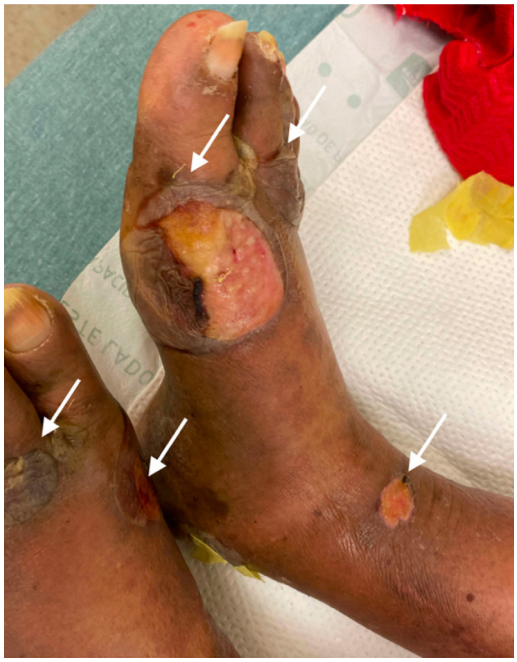

Image 3. Foot and ankle with bullous pemphigoid, some ruptured. Arrows indicate blisters.

rash before bullae of 1–3 centimeters (cm) diameter appear.[1] The images presented here demonstrate BP of moderate severity on light brown skin tone (Fitzpatrick type IV). Distribution is often symmetric and commonly affects the abdomen, extremities, groin, axillae, or mucosa.[1] The disease is usually chronic with exacerbations and remissions.[1]

Bullous pemphigoid is associated with some neurologic disorders, including dementia and Parkinson's disease.[2] Atypical presentations of BP include non-bullous pemphigoid, which accounts for 20% of cases.[2] Additionally, BP has been associated with many classes of drugs, and this may also cause atypical presentations, including younger age of onset or blisters without initial rash.[4] Drug-related BP may resolve fully with cessation of the offending agent.[1] Diagnostic studies used in the evaluation of patients with lesions suspicious of BP include biopsy (for histopathologic examination and direct immunofluorescence microscopy) and serum tests to detect circulating anti-basement membrane zone antibodies.[1,2] The mainstays of initial treatment for this condition include corticosteroids and doxycycline.[1,5]

Patient consent has been obtained and filed for the publication of this case report.

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A Woman with Abdominal Pain After Lap-belt Trauma

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Case presentation: A 24-year-old female presented to the emergency department with diffuse abdominal pain after involvement as a restrained driver in a motor vehicle collision (MVC). Computed tomography of the abdomen revealed a traumatic abdominal wall hernia due to rectus wall rupture with complete bowel herniation.

Discussion: A traumatic abdominal wall hernia is a rare complication of blunt abdominal trauma that is typically associated with injury from a motorcycle handlebar but is more commonly seen after a MVC. It is important to consider this diagnosis when evaluating patients with abdominal pain after blunt abdominal trauma from either of these mechanisms. [Clin Pract Cases Emerg Med. 2023;7(4)271–273.]

Keywords: *traumatic abdominal wall hernia; blunt abdominal trauma; handlebar hernia.*

CASE PRESENTATION

A 24-year-old female presented by ambulance as a trauma alert to the emergency department after a head-on motor vehicle collision (MVC). Emergency medical services reported that she was restrained with only the lap portion of the seatbelt. On arrival, the patient reported loss of consciousness in the field, abdominal pain, and bilateral hip and leg pain. The primary exam was notable for an intact airway, as well as adequate breath sounds and circulation. She was initially tachycardic with a heart rate of 133 beats per minute; her other vital signs were stable. She was afebrile, normoxic at 97% on room air with 20 respirations per minute, and her blood pressure was 128/76 millimeters of mercury.

The secondary exam was notable for an immobilized cervical spine, ecchymosis across the lower abdomen, and diffuse abdominal tenderness. Labs were drawn, and plain radiographs of the chest and pelvis were obtained. The labs were significant for a leukocytosis of 34.98×10^3 cells per cubic millimeter (cmm) (reference range: $4–11 \times 10^3$/cmm). Chest and pelvis radiographs were unremarkable. Computed tomography of the head, cervical spine, chest, abdomen, and pelvis (Image 1 and 2) were subsequently performed.

Computed tomography revealed a rectus wall rupture with complete bowel herniation. After imaging, the patient was taken to the operating room for an exploratory laparotomy with small bowel resection, omentectomy, ileocecectomy, and reduction of the abdominal hernia. The surgical findings included a full-thickness small bowel injury, a bucket handle tear to the mesentery of the small bowel, a devitalized ischemic omentum, and a sigmoid colon serosal injury. Her abdomen was temporarily closed, and postoperative broad-spectrum antibiotics were initiated. The patient had five more abdominal surgeries, and after an 18-day hospital stay she was discharged to inpatient rehabilitation with an abdominal wound vacuum.

DISCUSSION

This case describes a traumatic rectus abdominis muscle rupture with resulting large ventral hernia. This rare injury, sometimes referred to as a "handlebar hernia," [1] is commonly associated with blunt abdominal trauma from a motorcycle handlebar.[1–2] This case demonstrates the under-reported but more common mechanism for this traumatic abdominal wall hernia following an MVC.[3] Three-point seatbelt harnesses are designed to secure over the sternum and hips, distributing deceleration forces in a way that decreases risk of serious injury. Improper use of a seatbelt, including use of the two-point lap belt only, can lead to significant injury[4]

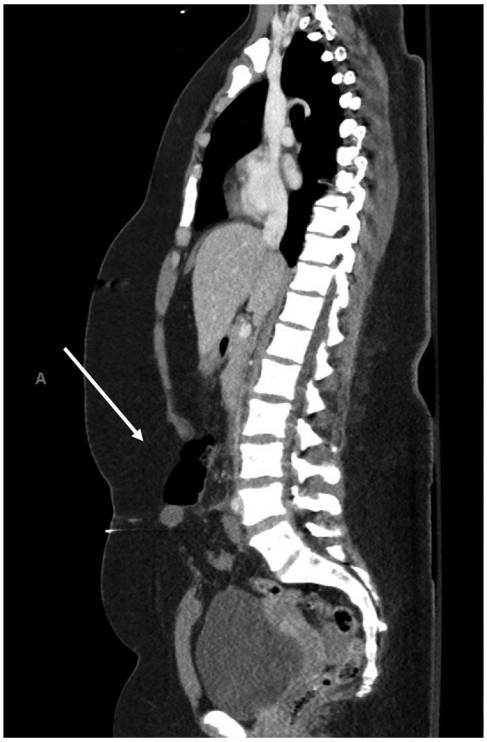

Image 1. Sagittal computed tomography showing rectus muscle disruption with abdominal content herniation (arrow).

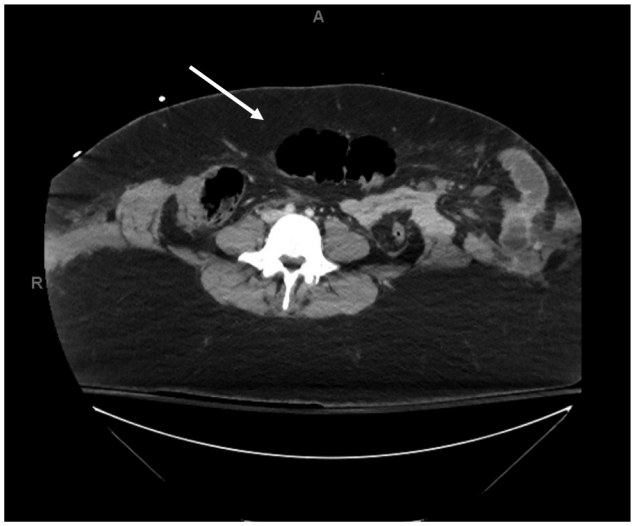

Image 2. Transverse computed tomography showing large traumatic ventral hernia (arrow).

including abdominal wall herniation. The force exerted on the abdomen by the lap belt leads to rupture of the anterior abdominal wall.

Computed tomography is the most sensitive diagnostic tool when evaluating for a rectus wall rupture. This patient's abdominal wall rupture was classified as a grade V abdominal wall injury due to complete rectus abdominis muscle disruption with herniation of abdominal contents.[5]

CPC-EM Capsule

What do we already know about this clinical entity?
A traumatic abdominal wall hernia is a rare complication of blunt abdominal trauma that is typically associated with injury from a motorcycle handlebar.

What is the major impact of the image(s)?
This case describes the under-reported but more common mechanism for a traumatic abdominal wall hernia following a motor vehicle collision.

How might this improve emergency medicine practice?
Emergency physicians should consider this diagnosis when evaluating patients with abdominal pain after blunt abdominal trauma from a motorcycle or vehicle collision.

Known risk factors for this condition include old age, weak abdominal muscles, and preexisting hernias. While rectus wall rupture with herniation is rare, it can be fatal if not treated immediately.[5] The presence of abdominal wall ecchymosis, otherwise known as a "seatbelt sign," after MVC is frequently associated with abdominal injuries.[5]

ACKNOWLEDGMENTS

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The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

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Clarifications on: Pectoralis Blocks Nomenclature and Clinical Applications of Regional Anesthesia Techniques for Breast and Thorax

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To The Editor:

I read with great interest the recently published case series applying pectoralis blocks (Pecs blocks) for infective breast conditions.[1] I greatly commend Brewer et al for employing pecs blocks in the emergency department (ED) and hope many emergency physicians will adopt the interfascial plane blocks introduced in the last decade. I wish to provide a few clarifications regarding them.

Regarding nomenclature, the authors used the term "Pecs I and Pecs II"[1] and attributed it to a "lack of consensus" and cited our article to support that.[2] However, we categorically stated that it is incorrect to use that term (despite weak consensus); hence, I am surprised that Brewer et al used it throughout their article. I reiterate that stating "Pecs II block" (modified pecs block) itself is enough, as it is a combination of the Pecs I block (ie, interpectoral plane (IPP) block) and the pectoserattus plane (PSP) block. To make it simple, we must use the term either pecs II or IPP+PSP blocks. Otherwise, it defeats the very purpose of the suggestion of nomenclatures by the experts.[3]

Regarding the choice of the block, we must pay careful attention to the sensory coverage of each block. For instance, in my view the IPP block is not required for drainage of breast abscess as it provides only relief from myofascial pain due to the disruption of pectoral muscles and does not block the thoracic nerves that are involved in the sensory innervation of the breast.[4] Also, there is a potential possibility of the presence of infection at the needle entry, precluding an IPP block in some cases. As infective conditions of the breast involve mainly the skin and subcutaneous tissues, either PSP block alone or its equivalent in sensory coverage, a serratus anterior plane (SAP) block, would be adequate. Of note, the SAP block is technically easier to perform compared to the PSP block. Also, the site of needle entry would be farther away from the infected tissues. Alternatively, I suggest a modification of the pecs block ("Pecs Zero") introduced by Tulgar et al wherein the needle entry would be above the clavicle.[5]

Recently, the erector spinae plane (ESP) block has gained popularity as a promising technique for pain relief in the ED setting for various conditions.[6] This block requires less expertise and time when compared to other fascial plane blocks. While Pecs blocks and the SAP block can be performed in the supine position, the ESP block requires a position other than supine such as sitting, lateral recumbent, or prone.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this letter to the editor. Documentation on file.

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Response to Clarifications on: Pectoralis Blocks Nomenclature and Applications of Regional Anesthesia Techniques

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To the Editor:

We want to thank the author of the recent letter to the editor regarding our case series.

Regarding the first point, we do utilize the term "Pecs I and Pecs II" throughout our study and did cite the Sethuraman and Narayanan paper, which had the more descriptive nomenclature (interpectoral and pectoserratus blocks, respectively). Because our target audience is emergency physicians who are being introduced to these blocks, we wanted to use the classic terms that have been used by numerous anesthesiologists in online and published educational material (ie, NYSORA, Duke Anesthesiology, etc). We agree that the more descriptive nomenclature should be used in the future as they become more standard. With the goal of introducing this block to the specialty, we recognize that Pecs II does include a Pecs I block. However, since two of our cases are isolated to a Pecs I block we split the terms for simplicity. We do firmly agree that nomenclature established by experts is important and, therefore, we will stick to whatever the most agreed upon nomenclature is at the time of publication moving forward.

With regard to the second point, we do agree that careful attention must be paid to the sensory coverage of each block. While all our cases benefited from a Pecs I, or interpectoral plane (IPP) block, this could have been resultant due to some myofascial pain. We also recognize the limitations of a case series and note that this is an introductory paper on a subject that should be further studied on a larger scale. Furthermore, we do mention that a contraindication to the block will be overlying infection such as cellulitis. This is true of almost all procedures performed within the emergency department, and we agree that an injection into a deeper fascial plane should never be performed through infected tissue. In addition, the "Pecs Zero" block introduced by Tulgar et al has promise and is a very interesting concept that warrants further study. However, their case report only achieved sensory block of the lateral breast, upper outer quadrant, and axillary areas and lacked medial coverage.[3] While this modification could be very useful when the presence of infected tissues exists, we believe that it warrants further research before it can be considered as a replacement as the initial letter suggests.

Finally, while we agree that the erector spinae plane block (ESPB) is an excellent block for many painful complaints, such as rib fractures, we do feel that even though thoracic ESPBs are commonly used by our anesthesiology colleagues, many emergency physicians are uncomfortable with the anatomy near the spine. The pectoralis region is often a simple target with clear anatomy. Also, there is some evidence that the Pecs II block is superior to this block in the postoperative setting for pain control of the breast.[4] Our goal in writing our case series was to introduce a novel technique to emergency physicians that could work synergistically with oral and intravenous analgesics for painful, breast-related complaints.

We do want to thank the author of this letter again for their contribution and healthy discussion. We look forward to continued research and application within the realm of emergency medicine.

The authors attest that their institution requires neither Institutional Review Board approval, nor patient consent for publication of this case report. Documentation on file.

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