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Case Presentation

Purpura fulminans secondary to toxic shock syndrome induced by Group A Streptococcal bacteremia in an adult patient

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Abstract

Purpura fulminans is a rare life-threatening presentation of disseminated intravascular congestion that presents with widespread purpura and skin necrosis secondary to considerable tissue thrombosis. It usually occurs in pediatric patients and can have various causes with acute infection being the most common. Common infectious triggers include *Neisseria* and *Streptococcus pneumoniae* as well as varicella. Our report describes a generally healthy adult patient who developed purpura fulminans in the setting of Group A *Streptococcus* bacteremia. Group A *Streptococcus* is a rare cause of purpura fulminans in adult patients with only a few documented cases reported in the literature.

Introduction

Purpura fulminans (PF) is a serious skin manifestation of disseminated intravascular congestion characterized by retiform purpuric patches and plaques that progress to areas of hemorrhagic necrosis with eventual eschar formation. On occasion, secondary lesions including vesicles and bullae may develop. Purpura fulminans has a very significant mortality rate ranging from 20% to 60%.¹ There are three types of PF: 1) neonatal, 2) idiopathic, and 3) acute infectious—with acute infectious being the most common.² Multiple infectious triggers have been described including *Meningococcus*, *Streptococcus pneumoniae*, *Streptococcus pyogenes*, *Staphylococci*, *Enterococci*, *Klebsiella*, and varicella, among others.^{1,3} Pediatric patients are at the highest risk of developing PF, with bimodal peaks at ages one to three and 16 to 18 years old.⁴ It is less common in adult patients, but when it does occur in adults, it often affects the elderly. Although *S. pyogenes* has been described as a rare trigger of PF, most

cases in the literature have involved either a pediatric patient or an elderly patient.

Case Synopsis

A 39-year-old man with a past medical history of gout, eczema, and psoriasis originally presented to an urgent care facility with a one-day history of body aches, fever, and fatigue and was sent home with plans for supportive care. Only 10 hours later, he presented to the emergency department with similar symptoms in addition to altered mental status. He had no rash at the time of presentation. In the emergency department the patient was diagnosed with an acute kidney injury, acute encephalopathy, acute hypoxic respiratory failure requiring intubation, and severe sepsis. He was admitted to the intensive care unit and required four vasopressors owing to severe hypotension. Initial lab work was significant for an elevated international normalized ratio of 1.7 and slightly decreased platelets of 140,000. Two sets of blood cultures were drawn and would later both grow *S. pyogenes*.

On day one of admission, the patient developed diffuse mottling of the skin with violaceous patches most notably on the lower extremities. On day three of admission, a dermatology consult was requested because of progressive rash. Initial exam revealed nonpalpable violaceous macular purpura of the anterior lower extremities which extended onto the abdomen, chest, and upper extremities in a retiform pattern. Scattered flaccid bullae and erosions were present on the lower extremities as well. Nonpalpable purpura of the tip of nose, cheeks, and bilateral helices of the ears were also noted as well as a positive Nikolsky sign on the lower extremities. The patient's skin and distal extremities were cool to the touch suggesting poor peripheral perfusion. This presentation was consistent with purpura fulminans ([Figures 1A-C](#)).

Over the next several days, the patient developed further bullae which progressed to sloughing and erosion. The purpuric patches on the chest and abdomen began

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Figure 1. **A)** Diffuse bluish-purple purpuric patches involving nearly the entire left lower extremity with focal areas of flaccid bullae progressing to erosion. **B)** Purpuric patches in retiform pattern involving large areas of the abdomen, mons pubis, and proximal thighs. **C)** Purpuric patches in a retiform pattern involving the chest and left upper extremity. Also note the dusky purpuric patch involving the left ear.

to fade but the patient continued to have a cyanotic and dusky appearance of his fingers, palms, feet, and scrotum. A plastic surgery consult was requested on day four of admission to aid in the decisions regarding possible debridement and amputation. The patient went on to develop dry gangrene of the upper and lower extremities and it was determined that amputation was necessary. The patient initially underwent bilateral below-knee amputation and bilateral upper extremity transradial amputation. Later in his hospital stay, he required bilateral above-knee amputation.

The patient also required various antibiotics while hospitalized. He initially started on vancomycin and ceftriaxone in the emergency department for empiric treatment of presumed bacteremia. He also received single doses of doxycycline, acyclovir, and daptomycin in the first 24 hours of admission. He was then transitioned to clindamycin and meropenem; the latter was transitioned to ampicillin-sulbactam on admission day 15 until day 28. Vancomycin was restarted on day 6 and discontinued on day 21. A rapid *streptococcus* PCR collected via throat swab, sputum culture, urine culture, and lumbar puncture all performed in the first 24 hours were negative. The source of the patient's *S. pyogenes* bacteremia was never determined, although it was noted that the patient's wife was actively being treated for *strep throat* at the time of initial presentation. Additionally, heparin was initiated on admission day one and the patient also completed three days of IVIG which was started on admission day four. Eventually the patient was stabilized and transitioned to the general floor, and after nearly three months of hospitalization, he was transferred to an intensive inpatient rehabilitation facility for further recovery.

Case Discussion

In the acute infectious form of PF, bacterial endotoxin release results in endothelial injury and a proinflammatory state.³ Endothelial injury and coagulation activation can

result in decreased activity of protein C and other anticoagulants, further enhancing a prothrombotic environment.^{1,3} Tissue necrosis, multiorgan failure, and death may occur.¹

Our case describes a unique presentation of PF secondary to *S. pyogenes* bacteremia in a previously healthy middle-aged adult man. He developed classic cutaneous findings of purpura fulminans as described above. He also developed superficial bullae, which were potentially secondary to erythrogenic toxins produced by Group A *Streptococcus*, as bullae secondary to skin necrosis would likely be less superficial and would take more time to develop. There are few documented cases of *S. pyogenes*-induced PF in an adult. One reported case describing a 36-year-old man who developed PF secondary to *S. pyogenes* attributes innate immune suppression secondary to NSAID therapy.⁵ Other cases have been described in older adult patients including a 61-year-old woman and a 72-year-old woman.^{6,7} There are multiple case reports of children affected by *S. pyogenes*-induced PF.⁸⁻¹²

Purpura fulminans is a clinical diagnosis and early recognition is paramount owing to the high associated mortality. Conditions in the differential diagnosis include necrotizing fasciitis, meningococcemia, thrombotic thrombocytopenic purpura, toxic shock syndrome, vasculitis, and others.² There can also be overlap among these disease processes. Evaluation to identify the underlying cause is imperative to begin treatment. Laboratory testing to be considered include complete blood count, comprehensive metabolic panel, prothrombin time, partial thromboplastin time, d-dimer, fibrinogen level, protein C level, and any appropriate infectious workup.²

Treatment consists of supportive care along with antibiotic and anticoagulant therapy.⁸ Initial antibiotic therapy should be broad-spectrum, but this can be tailored once an infectious source is identified. Commonly used antibiotics include carbapenem, vancomycin, beta lactam-beta lactamase inhibitors, and clindamycin.² Anticoagulant therapy often consists of heparin, but it is important to weight the prothrombotic state against the high risk of bleeding and close monitoring is necessary. Some reports recommend platelet counts remain greater than 100,000 whereas others state greater than 50,000.³ Early use of recombinant tissue plasminogen activator to aid in tissue perfusion has been described. However, there was significant associated bleeding risk.³ IVIG has been used to neutralize antibodies to the many toxins produced by the causative bacteria.² Finally, protein C concentrate can be given to aid in delaying disease progression and restoring coagulation balance.² Other proposed therapies include hyperbaric oxygen, fresh frozen plasma, and plasma exchange, but there is lack of sufficient data on the efficacy of these options.¹

Conclusion

We present a unique case of PF in a previously healthy adult with *S. pyogenes* as the causative bacteria. The

source of our patient's bacteremia remains unknown. Our patient was treated with various antibiotics, heparin, IVIG, and surgical intervention including bilateral below-knee-amputation (and later above-knee-amputation) and bilateral transradial amputation. The patient is now at home after months of inpatient treatment followed by transfer to intensive inpatient rehabilitation.

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Potential conflicts of interest

The authors declare no conflicts of interest.

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