Journal of
Medical Case Reports

CASE REPORT Open Access

# Acute infectious purpura fulminant due to *Enterococcus faecium* infection: a case report

Raghad G. Tanbour[1*], Riad Amer[1], Abdelrahman N. Matar[2] and Banan M. Aiesh[3]

## Abstract

**Background** Purpura fulminant is a rare life-threatening disorder characterized by a dysregulated response that is often associated with poor prognosis and lethal outcomes. It is often associated with disseminated intravascular coagulation, skin necrosis, and protein C deficiency leading to thrombotic occlusion of skin arterioles, causing palpable purpuric lesions, spreading ecchymosis, and gangrene, along with hypotension and fever. Purpura fulminant is classified into three distinct categories according to the trigger mechanisms, including neonatal, idiopathic, and acute infectious. Acute infectious purpura fulminant is the most common manifestation, which occurs after bacterial infections caused by acquired protein C deficiency. Although traditionally occurring in *Neisseria meningitidis* infection and *Streptococcus* infection, acquired protein C deficiency causing purpura fulminant due to *Enterococcus faecium* has not been described in the literature. This case report highlights the fact that purpura fulminans can also be a rare presentation of *Entercoccus faecium* infection.

**Case presentation** A 61-year-old Arab man who is immunocompetent presented with sepsis, which later escalated into septic shock due to *Enterococcus faecium* bacteremia. The patient's hospitalization rapidly developed into multiorgan dysfunction, disseminated intravascular coagulation, and purpura fulminans. Aggressive interventions were initiated, involving the administration of broad-spectrum antibiotics, multiple vasopressors, and mechanical ventilation. Despite these intensive measures, the patient ultimately succumbed to the complications of multiorgan failure and death.

**Conclusion** This case illustrates the devastating outcomes that can present from purpura fulminant. However, physicians should consider purpura fulminant caused by *Enterococcus faecium* infection in the workup of patients presenting with purpuric rash and fever.

**Keywords** Acute infectious purpura fulminant, *Enterococcus faecium*, Disseminated intravascular coagulation

## Background

Purpura fulminant (PF) is a hematological emergency syndrome characterized by the development of an immunological reaction pronounced by rapid, widespread purpuric rash and lesions [1], microvascular thrombosis, fever, disseminated intravascular coagulation (DIC), and cutaneous necrosis, which are the hallmarks of the condition, in addition to hemorrhagic skin and soft tissue destruction, ecchymosis, and hypotension [2]. The classic presentation of this condition is diffused retiform purpura or angular purpuric lesions [3]. If left untreated, this condition can quickly worsen and lead to thrombotic

*Correspondence:
Raghad G. Tanbour
raghadtanbour@yahoo.com
[1] Hematology and Oncology Department, An-Najah National University Hospital, Nablus 44839, Palestine
[2] Internal Medicine Department, An-Najah National University Hospital, Nablus 44839, Palestine
[3] Infection Prevention and Control Department, An-Najah National University Hospital, Nablus 44839, Palestine

blockage of tiny and medium-sized blood arteries. This can cause acute circulatory collapse, leading to multiorgan failure and exacerbating severe sepsis [4].

Despite the severe clinical course associated with this hemostatic infection outcome, the mechanism of PF is yet unknown [5]. There are several mechanisms that negatively regulate coagulation, including severe acquired protein C depletion, which has anticoagulant and anti-inflammatory properties, disruption of the protein C–thrombomodulin pathway, and others [6]. Aggressive treatment with antibiotics, volume expansion, inotropic medications, and protein C replacement is recommended due to the high mortality and morbidity rate associated with this disorder [7, 8].

PF can occur during infancy/childhood or adulthood [9], and it frequently affects young people with no previous comorbidities. The most prevalent form of PF is idiopathic/infectious, which is typically associated with infections caused by *Streptococcus pneumonia* or *Neisseria meningitides* [7, 10]. With a reported death rate as high as 41–50%, it is extremely dangerous and puts survivors at risk of limb necrosis and amputation [7, 11, 12]

Accurate identification of the underlying cause is crucial for treating PF [4]. The early symptoms of PF are nonspecific and similar to those of flu, which makes it difficult to diagnose the condition before the appearance of the purplish-red rash [1]. To diagnose the condition, it is necessary to perform skin biopsies and blood cultures for microbiological analysis [1]. The presence of a distinct skin rash is a critical diagnostic sign of PF and should trigger immediate treatment. Waiting for the results of a skin biopsy could delay diagnosis and lead to severe complications and death [9].

There is a lack of information about infectious PF in adult patients. Our objective was to record the clinical features and outcomes of adult patients hospitalized in the intensive care unit (ICU) with infectious PF, including the risk factors for mortality. Upon reviewing the literature, we found that this is the first reported case of *E. faecium* causing PF.

## Case presentation

A 61-year-old Arab man, a nonsmoker with no significant medical history, presented to the emergency department with a 3-day fever, green-colored diarrhea, and a noticeable blue discoloration of his face. Prior to being admitted to our healthcare facility, the patient had received intravenous ceftriaxone 1 g as part of the antibiotic treatment during a 1-day stay at a local hospital. Upon arrival at the emergency department, the patient reported no relevant medical history related to his family. The patient reported no previous injuries, exposure to pets, or insect bites. He also denied any history of substance abuse or steroid use. Upon examination, the patient's vital signs were recorded as follows: pulse rate of 125 beats per minute, respiration rate of 35 breaths per minute, blood pressure of 105/57 mmHg, and a pulse oximetry reading indicating 95% oxygen saturation. Initial laboratory results revealed the following: hemoglobin 12.9 g/dL (normal range 13.5–17.5 g/dL), white blood cell count $13.5\times10^9$/L (normal range 4.5–$11\times10^9$/L), platelets $110\times10^9$/L (normal range 150–$450\times10^9$/L), international normalized ratio (INR) 1.7 (normal range 0.8–1.1), prothrombin time (PT) of 45 seconds (normal range 11–13.5 seconds), fibrinogen 1 g/L (reference range 2–4.5 g/L), and serum creatinine 4.2 mg/dL (normal range 0.6–1.2 mg/dL).

His liver function tests on admission were as follows: Alanine aminotransferase (ALT) 44 U/L (normal range 7–33 U/L), Aspartate transaminase (AST) 92 U/L (normal range 10–35 U/L), albumin 4 g/dL (normal range 3.5–5 g/dL), total bilirubin 0.4 mg/dL (normal range 0.1–1.2 mg/dL), and direct bilirubin 0.2 mg/dL (normal range 0–0.3 mg/dL).

Toxicology screening and urinalysis yielded normal results, and all viral screening panels, including Human Immunodeficiency Virus (HIV), Hepatitis C Virus (HCV), Hepatitis B Virus (HBV), Cytomegalovirus (CMV), and Epstein-Barr Virus (EBV), returned negative results. After conducting a thorough physical examination, the patient was found to have a Glasgow coma scale of 15. There were no signs of meningitis, such as headache, photophobia, or neck stiffness. Lung and abdominal examinations were unremarkable, and no heart murmur was auscultated. During the dermatological assessment, some notable clinical findings were observed. These included bluish discoloration on the face (Fig. 1) and bilateral purplish lesions on the gluteal area. In addition, there were ecchymosis and purplish lesions on both arms (Fig. 2).

The initial chest X-ray did not show any abnormalities, and the color Doppler ultrasound examination did not detect any deep venous thrombosis. However, the ultrasound image of the abdomen revealed thickening of the large bowel wall. Due to technical issues, a computed tomography (CT) scan could not be performed. The transthoracic echocardiogram (TTE) did not show any valvular lesions or any other abnormal findings. Upon admission to the ICU, the patient's vital signs were recorded as follows: central body temperature was 39 °C, heart rate was 125 beats per minute, arterial blood pressure was 90/40 mmHg, respiratory rate was 50 breaths per minute, and SpO2 was 98% on room air. The patient was alert and oriented.

In the initial management phase, the patient was given empirical antibiotic therapy with intravenous vancomycin 1 g daily for 3 days and intravenous meropenem

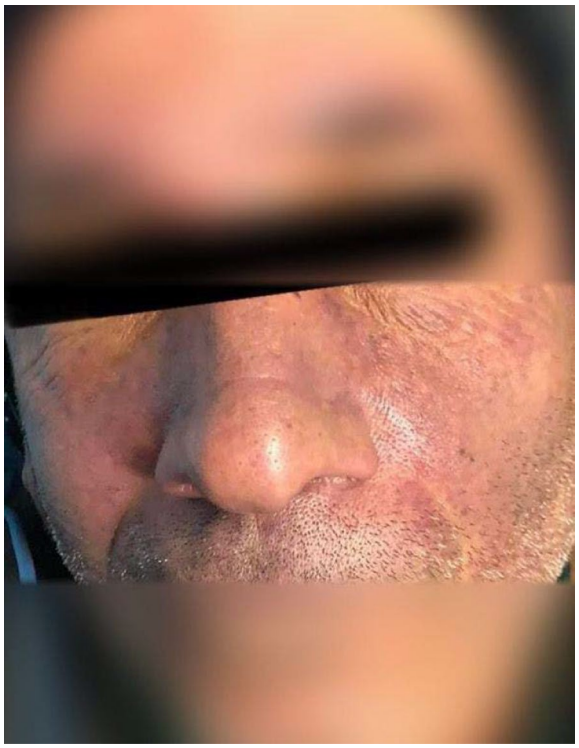

**Fig. 1** Painful, purple, non-blanching, confluent purpura on the cheeks and nose on day 1

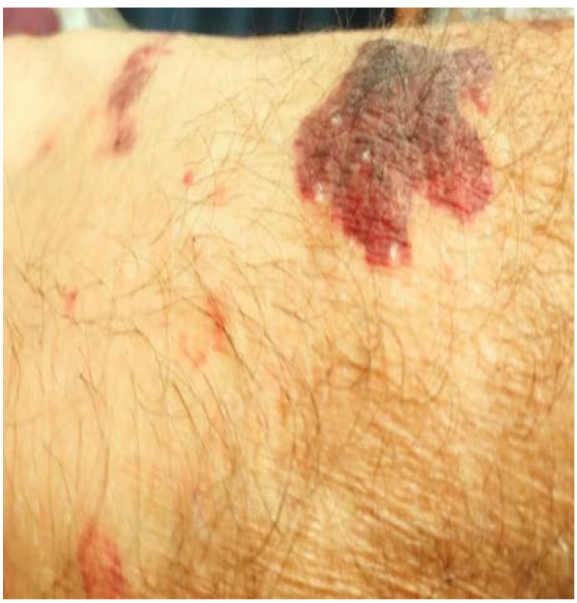

**Fig. 2** Ecchymosis and purpuric lesions on the lower limb

500 mg twice daily for 5 days (up until the day of death). This treatment was initiated within 1 hour of arrival and selected on the basis of the antibiogram and facility-specific guidelines as part of the management for septic shock. The dosing adjustments were made due to the patient's acute kidney injury (AKI). Adequate fluid resuscitation was initiated, along with replacement therapy to address anuria with hyperkalemia. The patient's respiratory status deteriorated due to lactic acidosis, necessitating endotracheal intubation. However, despite aggressive interventions and support, the patient's condition remained critical, marked by hypotension even with inotropic support and hydrocortisone administration. Numerous potential differential diagnoses, including thrombotic thrombocytopenic purpura, toxic shock syndrome, calciphylaxis, necrotizing fasciitis, and meningococcemia, were ruled out. A total of 2 days later, a decline in platelet count to $50\times10^9$/L, elevation in white blood cell count to $25\times10^9$/L, and an increase in INR to 3 were observed. At the same time, new ischemic lesions manifested on the patient's toes and fingertips. Despite receiving intravenous heparin at a rate of 200 units per hour, which was escalated to 500 units per hour, for 4 days from the time of diagnosis until the time of death, the patient continued to deteriorate, with no improvement observed in the skin condition.

The skin manifestations spread to the trunk, and the patient's blood clotting and kidney function began to deteriorate. On the basis of the progression of the skin condition and the laboratory results showing DIC and septic shock, physicians made a tentative diagnosis of sepsis-induced PF. After considering other possibilities, the patient received an infusion of fresh frozen plasma (FFP), platelets, and packed red blood cells (PRBCs), totaling 60 units of FFP and 5 units of PRBCs over a period of 4 days.

The patient's condition continued to worsen despite undergoing treatments. Eventually, he died due to circulatory failure and multiple organ dysfunction within 5 days of being admitted to the ICU. On the third day of admission, *Enterococcus faecium* was isolated from blood culture. The isolated pathogen was sensitive to vancomycin with an MIC $\leq$ 1 mg/L.

This case report highlights a rare instance of PF caused by *Enterococcus faecium* in a previously healthy patient. The patient developed severe sepsis and ultimately died from the infection despite receiving prompt and efficient antibiotic therapy. It is worth noting that *Enterococcus faecium* has not been previously reported as a pathogen causing PF.

## Discussion and conclusion

We present a case report of a previously healthy patient who developed PF due to *Enterococcus faecium* infection. The patient's condition deteriorated rapidly, progressing to septic shock within 72 hours of exhibiting initial symptoms. Despite receiving initial treatment with

intravenous antibiotics and adequate fluid resuscitation, the patient ultimately succumbed to the condition 5 days after admission to the ICU. The patient exhibited skin lesions typical of PF, and laboratory tests showed DIC. While *Neisseria meningitidis* and *Streptococcus pneumoniae* are widely recognized as the primary causative agents of PF globally [13], this case highlights the potential for *Enterococcus faecium* to also cause the condition [13, 14].

*Enterococci* are Gram-positive cocci that are found in nature but primarily reside in human and animal gastrointestinal tracts as part of the microbiota [9, 15]. Hospital outbreaks of bacteremia, urinary tract infections, and infective endocarditis are often associated with it [16]. *Enterococcus* can cause invasive infections that lead to a high mortality rate of up to 20%. The risk of mortality is significantly influenced by various key factors, including the patient's baseline condition, underlying comorbidities, the presence of complicated bacteremia or infective endocarditis, antimicrobial resistance, and the necessity of ICU admission [17]. On the basis of the patient's presentation, it was reported that he experienced diarrhea as one of his symptoms. A study revealed that a greater amount of the pathobiont *Enterococcus* was associated with a higher risk of diarrhea, which could lead to the translocation of pathogens from the bowel to the blood, causing bacteremia [18]. Infections caused by *Enterococci* typically manifest in elderly patients or those with compromised immune systems, particularly after prior antibiotic usage that disrupts their normal flora [15]. In this case, *Enterococcal* sepsis resulting in septic shock may have been caused by a recent gastrointestinal infection. The patient had diarrhea 1 day before hospital admission but was not immunocompromised. He has a clear past medical history and was previously in good health. There is no record of any medications taken, including immunosuppressants, and initial laboratory results showed no signs of an immune disorder. Additionally, he has no prior history of antibiotic use.

PF is a blood clotting disorder that is well known for causing unique skin symptoms. It can also lead to complications such as shock, DIC, and multiple blood clots in different parts of the body [19]. Diagnosing PF can be challenging, especially in patients who experience rapid clinical deterioration and do not respond to standard treatments [20]. The patient exhibited abrupt-onset fever and hypotension that rapidly progressed into septic shock, accompanied by the emergence of skin lesions and coagulation dysfunction. On the basis of this presentation, it is likely that the patient is suffering from PF complicated by DIC. The characteristics observed in this case are consistent with the rare incidence of infectious PF, which is infrequently associated with *Enterococcus faecium*. Lesions in PF have a uniform appearance regardless of the triggering factor or pathogen. Key symptoms include localized skin bruising and symmetrical gangrene in the extremities, accompanied by coagulation abnormalities that indicate disseminated intravascular coagulation [21, 22].

It is crucial to acknowledge the severity of PF and provide personalized treatment plans. The empirical management of PF involves addressing infections, administering early and aggressive fluid resuscitation in cases of septicemia, promptly initiating broad-spectrum antibiotic therapy, using vasopressors to manage shock that does not respond to fluid resuscitation, and providing necessary blood product replacement [20, 22]. Early detection and intervention are crucial in reducing mortality rates for diseases with a rapid onset. However, for emergency providers who lack expertise in hemostasis and thrombosis, this can present significant challenges. Sepsis-associated PF can be reversed with timely therapeutic measures. However, in the case of the patient mentioned here, who had end-organ damage, early and aggressive fluid resuscitation, antibiotic treatment, and blood product transfusion did not yield favorable results. Consequently, clinicians often use therapeutic approaches such as activated protein C therapy, protein C concentrate, or plasma exchange to manage PF on the basis of their clinical observations [20, 23]. However, it is important to note that randomized controlled studies and published research on therapeutic interventions and their outcomes in the context of PF are scarce [23, 24]. Prompt identification and immediate treatment of the underlying cause are essential to achieving hemodynamic stability in patients.

Acute infectious PF usually presents with widespread purpura skin lesions that rapidly progress to extensive ecchymotic areas, often requiring surgical interventions, including amputation. Therefore, early diagnosis and treatment of the underlying cause can be crucial in preventing such drastic measures [15, 25]. However, the patient here, who was in septic shock and suffering from multiple organ failure, was deemed too high-risk for an interventional procedure, leading to the decision not to pursue surgical intervention.

PF is a medical condition that involves an inflammatory response that can cause severe damage to the body, leading to a high risk of morbidity and mortality. This condition is often associated with the loss of limbs, either partially or completely. It is important for medical professionals to be aware of this condition and the possible causes, including rare pathogens such as *Enterococcus faecium*. A comprehensive history of the patient should be taken into account when making a diagnosis, and physical examination plays a crucial role in identifying

skin lesions indicative of PF. This article presents a rare case of acute PF caused by *Enterococcus faecium* bacteremia, a pathogen that is not commonly associated with this condition. It is important to note that PF has a high mortality rate of more than 50% in cases that lead to multiple organ failure [22]. Therefore, timely and appropriate initial treatment and management are crucial factors that can significantly improve patient outcomes.

**Abbreviations**

| | |
|---|---|
| CT | Computed tomography |
| DIC | Disseminated intravascular coagulation |
| ICU | Intensive care unit |
| INR | International normalization ratio |
| IV | Intravenous |
| PF | Purpura fulminant |
| PT | Prothrombin time |
| TTE | Transthoracic echocardiography |
| MIC | Minimum inhibitory concentration |

**Acknowledgements**
The authors thank the patient and the family for accepting case publication and thank An-Najah National University Hospital for providing the needed data.

**Author contributions**
The first draft of the manuscript was written by RT and BART, and AM reviewed the medical records and extracted the needed data. RT, BA, and RA worked on the final version and critically reviewed it. Finally, all authors approved the final manuscript.

**Funding**
No funding was received for this study.

**Availability of data and materials**
Data and materials used in this work are available from the corresponding author upon request.

## Declarations

**Ethics approval and consent to participate**
Not applicable.

**Consent for publication**
Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

**Competing interests**
The authors declare that they have no competing interests.

Received: 19 February 2024 Accepted: 28 February 2025
Published online: 12 May 2025

**References**

1. Contou D, de Prost N. Purpura fulminans in adult patients. Rev Prat. 2023;73(1):71–8.
2. Spicer TE, Rau JM. Purpura fulminans. Am J Med. 1976;61(4):566–71.
3. White B, Livingstone W, Murphy C, Hodgson A, Rafferty M, Smith OP. An open-label study of the role of adjuvant hemostatic support with protein C replacement therapy in purpura fulminans-associated meningococcemia. Blood. 2000;96(12):3719–24.
4. Chalmers E, Cooper P, Forman K, Grimley C, Khair K, Minford A, Morgan M, Mumford AD. Purpura fulminans: recognition, diagnosis and management. Arch Dis Child. 2011;96(11):1066–71.
5. Chu DZ, Blaisdell FW. Purpura fulminans. Am J Surg. 1982;143(3):356–62.
6. Colling ME, Bendapudi PK. Purpura fulminans: mechanism and management of dysregulated hemostasis. Transfus Med Rev. 2018;32(2):69–76.
7. Contou D, Sonneville R, Canoui-Poitrine F, Colin G, Coudroy R, Pène F, Tadié JM, Cour M, Béduneau G, Marchalot A, *et al*. Clinical spectrum and short-term outcome of adult patients with purpura fulminans: a French multicenter retrospective cohort study. Intensive Care Med. 2018;44(9):1502–11.
8. Famularo G, Pozzessere C, Trinchieri V, De Simone C. Fulminant purpuric rash. Eur J Emerg Med. 2000;7(4):313–5.
9. Nasrullah A, Javed A, Tariq U, Young M, Moeen Z, Balaan M. Purpura fulminans: a rare but fierce presentation of pneumococcal sepsis. Eur J Case Rep Intern Med. 2020;7(1):001373.
10. Guerra M, Marado D. Fulminans purpura due to Morganella morganii. Eur J Case Rep Intern Med. 2022;9(11):003670.
11. Findley T, Patel M, Chapman J, Brown D, Duncan AF. Acquired versus congenital neonatal Purpura fulminans: a case report and literature review. J Pediatr Hematol Oncol. 2018;40(8):625–7.
12. Ghosh SK, Bandyopadhyay D, Dutta A, Jane EP, Biswas SK. A profile of 23 Indian patients with Purpura fulminans: a retrospective, descriptive study. Indian J Dermatol. 2020;65(5):381–7.
13. Irfan Kazi SG, Siddiqui E, Habib I, Tabassum S, Afzal B, Khan IQ. Neonatal Purpura Fulminans, a rare genetic disorder due to protein C deficiency: a case report. J Pak Med Assoc. 2018;68(3):463–5.
14. Kim MC, Patel J. Recognition and management of acute Purpura fulminans: a case report of a complication of Neisseria meningitidis Bacteremia. Cureus. 2021;13(3):e13704.
15. Miller WR, Munita JM, Arias CA. Mechanisms of antibiotic resistance in enterococci. Expert Rev Anti Infect Ther. 2014;12(10):1221–36.
16. Zhang Y, Du M, Chang Y, Chen L-a, Zhang Q. Incidence, clinical characteristics, and outcomes of nosocomial Enterococcus spp. bloodstream infections in a tertiary-care hospital in Beijing, China: a four-year retrospective study. Antimicrob Resist Infect Control. 2017;6(1):73.
17. Rosselli Del Turco E, Bartoletti M, Dahl A, Cervera C, Pericàs JM. How do I manage a patient with enterococcal bacteraemia? Clin Microbiol Infect. 2021;27(3):364–71.
18. Nguyen AD, Andréasson K, McMahan ZH, Bukiri H, Howlett N, Lagishetty V, Lee SM, Jacobs JP, Volkmann ER. Gastrointestinal tract involvement in systemic sclerosis: The roles of diet and the microbiome. Semin Arthritis Rheum. 2023;60:152185.
19. Olivieri M, Huetker S, Kurnik K, Bidlingmaier C, Keil J, Reiter K, Hoffmann F. Purpura fulminans - It's not always sepsis. Klin Padiatr. 2018;230(4):225–6.
20. Betrosian AP, Berlet T, Agarwal B. Purpura fulminans in sepsis. Am J Med Sci. 2006;332(6):339–45.
21. Rintala E, Kauppila M, Seppälä OP, Voipio-Pulkki LM, Pettilä V, Rasi V, Kotilainen P. Protein C substitution in sepsis-associated purpura fulminans. Crit Care Med. 2000;28(7):2373–8.
22. Warner PM, Kagan RJ, Yakuboff KP, Kemalyan N, Palmieri TL, Greenhalgh DG, Sheridan RL, Mozingo DW, Heimbach DM, Gibran NS, *et al*. Current management of purpura fulminans: a multicenter study. J Burn Care Rehabil. 2003;24(3):119–26.
23. Dalugama C, Gawarammana IB. Rare presentation of rickettsial infection as purpura fulminans: a case report. J Med Case Rep. 2018;12(1):145.
24. Klifto KM, Gurno CF, Grzelak MJ, Seal SM, Asif M, Hultman CS, Caffrey JA. Surgical outcomes in adults with purpura fulminans: a systematic review and patient-level meta-synthesis. Burns Trauma. 2019;7:30.
25. Childers BJ, Cobanov B. Acute infectious purpura fulminans: a 15-year retrospective review of 28 consecutive cases. Am Surg. 2003;69(1):86–90.

## Publisher's Note