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## Case Report

# Septic shock with multiple organ dysfunction syndrome, disseminated intravascular coagulation and mucormycosis following unsafe abortion: a case of acute infectious purpura fulminans

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## ABSTRACT

A woman in her early thirties presented to the emergency department in the 2<sup>nd</sup> week of her illness. She had a history of self-MTP kit intake at 12 weeks gestation followed by dilatation and curettage by the local practitioner for incomplete abortion. She started having a fever and developed blisters all over her body. On presentation, thorough history and examination confirmed sepsis and septic shock. Management continued with the sepsis campaign recommendation 2021 in the critical care unit. In the sixth week of her stay, she developed methicillin-resistant *Staphylococcus aureus* (MRSA) and mucormycosis further complicating her management. The patient succumbed to her illness after 6 weeks of aggressive treatment including daily wound debridement, amputation, and colostomy. This case emphasizes the importance of contraception use and the prevention of unwanted pregnancy. Further to reduce maternal morbidity and mortality, safe abortion practices should be opted for.

**Keywords:** Septic shock, Purpura fulminans, Gangrene, Mucormycosis, Toxic shock syndrome

## INTRODUCTION

Unsafe abortion is a major global health issue and continues to be a leading cause of maternal morbidity and mortality, especially in low-resource settings. The world health organization (WHO) estimates that each year, 25 million unsafe abortions occur globally. In high-resource settings, approximately 30 women die for every 100,000 unsafe abortions.<sup>1</sup> Mortality rates are considerably higher in low-resource settings, reaching 220 deaths per 100,000 unsafe abortions, and are even more pronounced in certain regions, such as Sub-Saharan Africa, where the rate is 520 deaths per 100,000 unsafe abortions. Underreporting of severe puerperal sepsis cases, particularly those following unsafe abortions, contributes to the underestimation of its impact on maternal mortality. In this report, we present a case involving septic shock, multiple organ dysfunction

syndrome (MODS), disseminated intravascular coagulation (DIC), and purpura fulminans following an unsafe abortion. We emphasize the challenges encountered in managing severe multisystem involvement.

## CASE REPORT

We present the case of a woman in her early thirties (P2L2A4) who was admitted to the emergency department in critical condition. She was intubated, on ventilatory support (VEC mode, PEEP 6, FiO<sub>2</sub> 100%), and required double-strength noradrenaline infusion (4 ml/hour) to maintain her blood pressure at 112/70 mmHg.

Her pulse was 48/min, and her Glasgow coma scale (GCS) score was E1VTM1. Blood gas analysis showed normal lactate level and pH value.

On physical examination, she had active bleeding from the nose and vagina, along with widespread ecchymotic patches and blisters (Figure 1). A femoral hemodialysis catheter was in place. Her systemic examination was otherwise unremarkable.



**Figure 1 (a and b): Ecchymosis patches and blisters all over the body.**

The patient had taken an off-label abortifacient at 12 weeks of gestation, followed by a dilatation and curettage (D and C) procedure performed by a non-medical practitioner. Two days later, she developed a fever, a rash, and vaginal bleeding. Initially treated conservatively at another hospital, she was later diagnosed with septic shock, MODS, DIC, and metabolic acidosis. Despite inotropic support and platelet transfusions, her condition worsened, and she was transferred to a tertiary care center.

Upon arrival at the tertiary care hospital, she was admitted to the ICU due to worsening renal function, for which serial hemodialysis was initiated. Dermatology consultation was sought due to purpuric rashes, and a biopsy confirmed purpura fulminans. Blood transfusions, including 6 units of PRBC, 10 units of cryoprecipitate, and 4 units of FFP, were administered to address her deranged hemogram and coagulation profile.

On day 4 post-D and C, she underwent hysteroscopic evacuation of retained products, as a transvaginal ultrasound showed echogenic material with air foci in the endometrial cavity. Despite these interventions, she was intubated on day 8 due to worsening respiratory distress. A

bronchoscopy was performed, and a mucus plug was removed.

On day 9 post-D and C, she was transferred to our facility. A multidisciplinary approach was employed to manage septic shock with MODS and DIC leading to purpura fulminans. She was started on meropenem (renal-modified doses), teicoplanin, and caspofungin. The serial monitoring of her hemogram, liver function, and coagulation profile showed gradual improvement in the second week and marked improvement in liver function in week 5. Despite initially sterile blood cultures, by the fifth week, blood culture results showed MRSA (*Staphylococcus hemolytic*) sensitive to daptomycin and vancomycin. Endotracheal aspirates and high vaginal swabs revealed persistent bacterial growth in the second and third weeks (Table 1). Antibiotics were adjusted accordingly.

Surgical interventions included debridement of wet gangrene, amputation of all toes on the left foot (Figure 2), and the third toe on the right foot. Due to perianal skin involvement, a trephine colostomy was performed.

Rheumatology evaluation for hypercoagulation studies identified positive SSA Ro and RNP Sm antibodies.



**Figure 2 (a and b): Wet gangrene, amputation of left foot toes.**

In the fifth week, she developed upper gastrointestinal bleeding. Endoscopy revealed gastric erosions and small ulcers. While endoscopy revealed a dark lesion on the hard palate and swelling in the maxilla raising suspicion of mucormycosis, which was confirmed by KOH mount.

Despite aggressive treatment, including antifungal therapy and multiple surgeries, the patient's condition continued to decline, and she succumbed to her illness in the sixth week.

**Table 1: Serial monitoring of blood investigations and body fluid cultures.**

| Weeks                                   | 2 <sup>nd</sup>                               | 3 <sup>rd</sup> | 4 <sup>th</sup>                               | 5 <sup>th</sup> | 6 <sup>th</sup>     |
|-----------------------------------------|-----------------------------------------------|-----------------|-----------------------------------------------|-----------------|---------------------|
| Hemoglobin (gm/dl)                      | 8.9                                           | 6.7             | 7.1                                           | 6.9             | 9.1                 |
| WBC/mm <sup>3</sup>                     | 29.33                                         | 20.68           | 15.6                                          | 16.02           | 27.6                |
| Platelet count /mm <sup>3</sup>         | 87k                                           | 2.6 lac         | 1.08 lac                                      | 95k             | 81k                 |
| Total bilirubin/ direct bilirubin mg/dl | 14.4/9.6                                      | 18.8/11         | 29/15                                         | 6.7/4.2         | 2.6/1.5             |
| SGOT/SGPT U/l                           | 77/107                                        | 84/33           | 177/92                                        | 105/92          | 34/42               |
| INR/APTT                                | 1.3/237                                       | 1.5/47          | 1.4/25                                        | 1.2             |                     |
| Fibrinogen/D-dimer                      | 26.5/>5.5                                     | 489/>5.5        | 217                                           |                 |                     |
| Week                                    | 2 <sup>nd</sup>                               |                 | 3 <sup>rd</sup>                               |                 | 4 <sup>th</sup>     |
| Endotracheal aspirate                   | <i>K. pneumoniae</i> and <i>A. baumannii</i>  |                 | <i>E. meningoseptica</i>                      |                 | <i>A. baumannii</i> |
| High vaginal swab                       | <i>K. pneumoniae</i>                          |                 | <i>K. pneumoniae</i> and <i>P. aeruginosa</i> |                 |                     |
| Skin wound C/S                          | <i>K. pneumoniae</i> and <i>P. aeruginosa</i> |                 | <i>K. pneumoniae</i> and <i>P. aeruginosa</i> |                 |                     |

## DISCUSSION

Because the third international consensus definition for sepsis and septic shock (Sepsis-3) excluded pregnant women, the WHO launched a global maternal and neonatal sepsis initiative in 2017 to address maternal and neonatal mortality. This initiative adopted the term maternal peripartum infection based on an existing definition of puerperal sepsis.<sup>2,4</sup> The Society of obstetric medicine of Australia and New Zealand (SOMANZ) recommended that sepsis be considered when two or more obstetrically modified qSOFA criteria are met.<sup>5</sup>

Pregnant and postpartum women have a 50% higher risk of infection than non-pregnant young adults.<sup>6</sup> Both obstetric and non-obstetric factors can increase the risk of sepsis and septic shock in pregnant women. The bacteria involved in septic abortions are often polymicrobial, originating from the normal flora of the vagina and cervix or sexually transmitted pathogens. Gram-negative bacteria produce endotoxins and are common culprits in septic shock cases. Some women may develop methicillin-resistant *Staphylococcus aureus* infections during prolonged stays in critical care units. Life-threatening infections, such as toxic shock syndrome (TSS) and necrotizing soft tissue infections, are most commonly caused by a mix of aerobic and anaerobic bacteria.

Endothelial dysfunction is a key factor in the pathophysiology of sepsis-induced MODS. Inflammation-induced elevated levels of nitric oxide (NO) can result in myocardial contractile dysfunction and septic acute myocardial injury (SAMI). This condition disrupts VE-cadherin, increases pulmonary capillary permeability, and can lead to pulmonary edema. Additionally, high levels of NO can combine with superoxide to form peroxynitrite (OONO-), which in turn causes oxidative damage and increased permeability in various organs and tissues.<sup>7</sup>

DIC is a common complication of severe sepsis, resulting in both bleeding and thrombotic events.<sup>8</sup> Bacterial endotoxins initiate the consumption of proteins C and S, along with antithrombin III, creating a pro-coagulative

state that leads to thrombosis of dermal vessels and is associated with DIC. Meningococcus (10-20% cases) and *Streptococcus pneumoniae* are the most frequent bacterial causes, while varicella is the leading viral trigger. Skin lesions may initially present as petechial rashes, quickly progressing to larger ecchymotic areas. As the condition advances, hemorrhagic bullae may develop, forming the classic hard eschars characteristic of purpura fulminans.<sup>9</sup> In summary, septic patients may develop DIC, which can result in a deficiency of protein C, a natural anticoagulant. Protein C deficiency can contribute to purpura fulminans (PF), potentially necessitating protein C concentrate administration. However, in patients with PF who also experience bleeding due to DIC, using protein C concentrate poses a challenge as it may worsen bleeding.

Protein C concentrate administration did not result in a mortality benefit in the PROWESS-SHOCK and the ADDRESS trials.<sup>10,11</sup>

Thorough history, examination, and laboratory findings, including an elevated white blood cell count, abnormal coagulation, and deranged liver and kidney function tests, it was confirmed that the patient had sepsis.

The severity of organ dysfunction was assessed using the qSOFA score (quick sequential organ failure assessment), the SOFA score, and the NEWS score (National early warning score). A step-by-step treatment approach was initiated, beginning with securing the airway through intubation and ventilation. Tissue perfusion was evaluated, and crystalloid fluids along with norepinephrine were administered to maintain a mean arterial pressure (MAP) above 65 mmHg. Broad-spectrum antibiotics were continued per India's antimicrobial use guidelines.<sup>12</sup> An early hysterectomy should be considered to remove the source of the infection.<sup>13</sup> Prophylaxis for venous thromboembolism, intensive insulin therapy, stress ulcer prevention, and parenteral nutrition were given in line with surviving sepsis campaign 2021 recommendations.<sup>14</sup> Despite a multidisciplinary approach and last-resort antibiotics, she acquired MRSA.



Additionally, the patient developed mucormycosis, a life-threatening fungal infection associated with immune dysfunction or diabetes. Mucormycosis often requires prompt surgical debridement and antifungal therapy due to its high mortality risk, especially when diagnosed late.<sup>15</sup>

In a similar case, Umar F. Bhatti et al. reported a healthy young woman who presented with sepsis and developed erythematous lesions four days after the vaginal delivery of a healthy baby. Despite resuscitation and antibiotic therapy, her condition worsened, leading to DIC and purpura fulminans. Although her sepsis gradually subsided, she suffered severe ischemic injuries to her limbs, necessitating amputation of all four extremities.<sup>16</sup>

## CONCLUSION

This case highlights the severe consequences of unsafe abortion, leading to septic shock, MODS, DIC, and purpura fulminans. The literature underscores the importance of early intervention in septic shock, aggressive management of DIC, and the necessity of a multidisciplinary approach in treating such critically ill patients. Addressing unsafe abortion practices through public health measures is crucial in preventing these life-threatening complications.

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