

Severe necrotizing methicillin-sensitive *Staphylococcus aureus* pneumonia, septic shock, and multiorgan failure in a young man with classical dengue fever: A case report

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ABSTRACT

Dengue fever is one of the most common mosquito-borne viral illnesses worldwide. Although most cases are self-limiting, concomitant bacterial infections may result in severe complications and increased mortality. A 32-year-old previously healthy male presented with a 7-day history of rash over the right side of the neck extending to the occipital region, 4 days of fever, vomiting, malaise, headache, neck swelling with restricted neck movements, and 1 day of cough and shortness of breath. Evaluation revealed neck cellulitis, bilateral pneumonia, thrombocytopenia, acute kidney injury, myocarditis, hepatic dysfunction, and methicillin-sensitive *Staphylococcus aureus* (MSSA) bacteremia. Dengue immunoglobulin M was positive. Despite initial broad-spectrum antibiotic therapy, the patient developed progressive necrotizing pneumonia requiring mechanical ventilation. Following escalation to targeted antimicrobial therapy together with adjunctive therapy, significant clinical improvement was observed, and the patient was discharged in stable condition. Severe bacterial coinfection should be considered in dengue patients with atypical clinical deterioration. Early recognition and prompt treatment of MSSA-associated necrotizing pneumonia may be life-saving.

Key words: Coinfection, Dengue fever, Methicillin-sensitive *Staphylococcus aureus*, Multiorgan failure, Necrotizing pneumonia, Septic shock

Dengue represents the most common arboviral illness in humans. It is transmitted by the mosquitoes of the genus *Aedes*, and the arthropod vector is the *Aedes aegypti* which is widely distributed in the tropical and subtropical areas of the world. Recently, the incidence of dengue has increased, it is estimated that approximately 40–50% of the world's population is at risk [1,2]. Clinical manifestations range from asymptomatic infection and mild illness to severe dengue with plasma leakage, hemorrhage, shock, and multiorgan dysfunction [3]. Although dengue has a relatively low mortality when appropriately managed, studies have shown that the concomitant severe bacterial infection can lead to fatalities. The World Health Organization (WHO) 2009 dengue classification categorizes the disease into dengue without warning signs, dengue with warning signs, and severe dengue [3]. Although dengue infection is primarily viral in origin, concurrent bacterial infections have increasingly been recognized as an important cause of morbidity and mortality in affected patients [4,5]. Bacterial coinfections have been reported in hospitalized dengue

patients and are associated with prolonged hospitalization, septic shock, multiorgan failure, and poor clinical outcomes [4-6]. Methicillin-sensitive *Staphylococcus aureus* is an uncommon but potentially life-threatening bacterial coinfection in dengue fever. Certain strains producing Panton–Valentine leukocidin (PVL) toxin have been implicated in severe skin and soft tissue infections, bacteremia, and rapidly progressive necrotizing pneumonia in otherwise healthy individuals [7,8].

PVL-producing strains account for fewer than 2% of clinical isolates. This specific strain is known to cause widespread disease in previously healthy individuals in the community, as well as in hospitalized patients and healthcare workers. PVL-positive *S. aureus* strains predominantly cause skin and soft tissue infections but can also cause invasive infections. The most severe manifestation is necrotizing hemorrhagic pneumonia, with high mortality, and it may affect otherwise healthy young people in the community. The early diagnosis and the judicious management of the complications can help in a better outcome.

This case report highlights the importance of varying manifestations in dengue fever and the need to investigate

Access this article online	
Received - 18 April 2026 Initial Review - 05 May 2026 Accepted - 04 July 2026	Quick Response code 
DOI: ***	

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the possible fatal complications due to associated bacterial infections in patients with certain varying clinical manifestations. The understanding of the immune mechanisms of dengue illness and bacterial co-infection is important as it can negatively impact the disease outcome. Dengue-associated immune dysregulation may predispose patients to secondary bacterial infections, which have been associated with increased morbidity, prolonged hospitalization, septic shock, and mortality [4-6]. This case report details the case of a young adult with classical dengue fever complicated with methicillin-sensitive *S. aureus* co-infection leading to sepsis, necrotizing pneumonia, and multiple organ system involvement.

CASE PRESENTATION

A 32-year-old male was admitted through the Emergency department with a 1-week history of skin rashes over the right side of the neck extending to the occipital region, a 4-day history of fever, vomiting, malaise, headache, and associated with right-sided neck swelling and restricted neck movements, and a 1-day history of cough and shortness of breath. There was no history of any sick contact or travel outside.

Clinical evaluation revealed of the right neck, bilateral pneumonia, sepsis, acute kidney injury, myocarditis, hepatic dysfunction, thrombocytopenia, and dyselectrolytemia, which necessitated an urgent Intensive Care Unit admission.

The dengue NS1 antigen test was negative, but the dengue immunoglobulin M was positive. Other laboratory parameters showed markedly elevated inflammatory markers, including a C-reactive protein of 274 mg/L and a procalcitonin level >50 ng/mL (more than 50), elevated cardiac markers, deranged renal and hepatic profile, and coagulopathy. His initial paired blood cultures reported positive for methicillin-sensitive *S. aureus*.

He was started on broad-spectrum antibiotics from the beginning and was kept on high-flow nasal oxygen therapy. The clinical condition of the patient deteriorated after 48 h of admission, requiring intubation and mechanical ventilation. Despite culture-directed antimicrobial therapy, he was spiking continuously with high running fevers and developed progressive worsening of the pneumonia with abscess formation. Lung biopsy results showed necrotizing pneumonia.

Therapid progression of necrotizing pneumonia despite culture-directed antimicrobial therapy raised suspicion of a toxin-mediated MSSA infection, his antimicrobial regimen was modified to include clindamycin and linezolid while continuing cephalosporin therapy and a short course of steroid was added. Protein synthesis inhibitors such as clindamycin and linezolid suppress the production of staphylococcal exotoxins, including Panton-Valentine leukocidin (PVL), thereby reducing toxin-mediated tissue injury. In addition, linezolid achieves excellent lung tissue penetration, making it a valuable adjunct in severe necrotizing pneumonia. The

significant clinical improvement observed after the addition of clindamycin and linezolid further supports their role as adjunctive therapy in suspected PVL-associated *Staphylococcus aureus* pneumonia. Within 48 hours, there was a significant improvement in his clinical condition, and his fever spikes also subsided. Serial close monitoring showed improvement in his renal, hepatic, and cardiac profile, but he developed anemia, significant leukopenia, and persistent thrombocytopenia, which took a couple of days to return to normal, and he required multiple platelet and packed red blood cell transfusions during this period. Another important event that happened was the spontaneous pneumothorax, which required intercostal drainage. There was a concern for the weaning process initially with a plan of tracheostomy, but he was extubated uneventfully on the 14th day of hospitalization and stepped down to the ward after 48 h of extubation. intercostal chest drain (ICD) for another week and was removed uneventfully and discharged home in stable condition.

DISCUSSION

Dengue is an acute febrile illness caused by one of the four dengue viruses (DENV), which is transmitted by the mosquitoes *Aedes aegypti* or *Aedes albopictus* [1,3]. The clinical manifestations of this viral illness vary from asymptomatic to mild febrile illness to a life-threatening condition like dengue hemorrhagic fever or dengue shock syndrome. Dengue fever typically is a self-limited illness with a mortality rate of <1% if it is detected and treated early, but if it is left untreated, the mortality rate can rise to 20%. If the dengue illness is associated with concomitant severe bacterial infections leading to sepsis, septic shock, and multiorgan failure [3], it can increase mortality to as high as 80% [4-6].

There are four serologically distinct dengue virus serotypes (DENV-1 to DENV-4), which can have a transient cross-protection that disappears over months. Cases have been reported in a small percentage in which an infection with a different serotype happens to a previously infected person; the chance of complications, including bleeding and endothelial leak, is high. The WHO 2009 classified dengue into the categories of dengue without warning signs, dengue with warning signs, and severe dengue. The incubation period of dengue infection is 3–14 days. There are three phases in dengue infection: A febrile phase, a critical phase, and a recovery phase, which are not mandatorily seen in all cases.

Bacterial co-infection with dengue illness is rare, but the concomitant infections can be life-threatening [4-6]. Skin and soft tissue infection developing at the mosquito bite site may provide a portal of entry to enter the bloodstream, producing invasive infections like pneumonia, leading to sepsis, septic shock, and multiorgan failure, as observed in the present patient. Several studies identified *S. aureus* among the pathogens

causing secondary bacteremia in dengue patients [4,5]. The *Staphylococcus* bacteria, either methicillin-resistant or methicillin-sensitive, can cause a life-threatening condition even in young adults without any comorbid conditions, especially if the strains are toxin-producing ones [9]. The pathophysiological mechanisms proposed include immune dysregulation, endothelial dysfunction, transient immunosuppression, and skin barrier disruption at mosquito bite sites, facilitating bacterial invasion [6,8]. PVL is such a toxin that induces leukocyte destruction and is a virulence factor in some strains of *Staphylococcus*. Even though it predominantly causes skin and soft tissue infections, it can also lead to invasive infections like necrotizing hemorrhagic pneumonia with a high mortality rate [10,11]. Clinicians should maintain a high index of suspicion for PVL-associated *Staphylococcus* infection if the patient has a skin and soft tissue infection with associated invasive conditions such as community-acquired necrotizing hemorrhagic pneumonia in an immunocompetent person. The toxic gene profiling can detect its presence. The occurrence of prolonged fever, worsening respiratory symptoms, and elevated inflammatory markers in dengue patients should prompt evaluation for concurrent bacterial infection [9,12]. Any suspected case of toxin-associated *S. aureus* should be recognized early, and prompt diagnosis and early targeted antimicrobial therapy may improve patient outcomes [8,9], and adjunctive therapy with intravenous immunoglobulin (IVIG) should be considered in necrotizing pneumonia.

CONCLUSION

This case report highlights the importance of concomitant severe bacterial infections in dengue fever, in which the mosquito bite site may act as a portal of bacterial entry leading to the invasive infections with a high mortality rate up to 80%. Clinicians should be well aware of the possible complications related to dengue infections, especially concomitant infections with life-threatening bacteria. This case highlights the importance of early recognition of bacterial coinfection in dengue fever and may assist clinicians in making timely diagnostic and therapeutic decisions.

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Funding: Nil; Conflicts of interest: Nil.

How to cite this article: Vijayan L. Severe necrotizing methicillin-sensitive *Staphylococcus aureus* pneumonia, septic shock, and multiorgan failure in a young man with classical dengue fever: A case report. *Indian J Case Reports*. 2026; July 09 [Epub ahead of print].