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Case Report

Acute Intestinal Involvement in Disseminated *Staphylococcus aureus* Infection in a 9-Month-Old Infant: A Rare and Fatal Complication

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Abstract

Gastrointestinal involvement associated with invasive *Staphylococcus aureus* infection is uncommon and remains poorly characterized in infants. We report the case of a 9-month-old infant admitted to the pediatric intensive care unit with severe disseminated *S. aureus* infection. Chest CT demonstrated multiple bilateral pulmonary nodules, several showing central cavitation and a predominantly peripheral distribution, consistent with septic pulmonary emboli, associated with multifocal parenchymal consolidations and a small pleural effusion. A staphylococcal abscess of the gluteal region provided an additional infectious focus. The clinical course was complicated by septic shock with persistent hypotension requiring norepinephrine and acute kidney injury. *Staphylococcus aureus* was also isolated from stool samples. The infant subsequently developed progressive abdominal distension. Because of renal impairment, abdominopelvic CT was performed without intravenous contrast and demonstrated marked bowel distension with segmental circumferential wall thickening and spontaneous mural hyperattenuation, suggesting a possible hemorrhagic component. No definite pneumatosis intestinalis or pneumoperitoneum was identified. Abdominal distension persisted and serum lactate levels progressively increased. Although the clinical, microbiological, and imaging findings supported intestinal involvement occurring during disseminated *S. aureus* infection, concomitant hypoperfusion-related bowel injury was strongly suspected because of sustained hypotension and vasopressor requirement. Intestinal ischemia could not be definitively confirmed in the absence of contrast-enhanced imaging, angiography, surgery, or histopathology. Despite intensive care management, the infant developed progressive multiorgan failure and died. This case highlights an unusual gastrointestinal manifestation of severe invasive staphylococcal infection and the potential coexistence of infectious and hypoperfusion-related mechanisms of intestinal injury.

Keywords: *Staphylococcus aureus; Disseminated Infection; Intestinal Involvement; Septic Pulmonary Emboli; Septic Shock; Bowel Ischemia; Infant; Computed Tomography*

Introduction

Staphylococcus aureus is a major cause of severe bacterial infections in children and may be responsible for invasive and life-threatening disease, including pneumonia, bacteremia, soft-tissue infection, and septic shock. Severe infections may involve multiple anatomical sites and result in rapid clinical deterioration. Although pulmonary and soft-tissue manifestations are well recognized, gastrointestinal involvement associated with *S. aureus* infection is uncommon and remains poorly described in infants, particularly in the setting of disseminated infection.

In critically ill patients, the interpretation of acute intestinal abnormalities may be particularly challenging because infection-related intestinal injury may coexist with bowel injury secondary to systemic hypoperfusion. Septic shock, sustained hypotension, and vasopressor requirement may compromise splanchnic perfusion and contribute to intestinal ischemic injury.

We report a 9-month-old infant with severe disseminated *S. aureus* infection involving the lungs and gluteal soft tissues, with additional isolation of *S. aureus* from stool samples, who subsequently developed acute intestinal abnormalities on CT. This case highlights the potential gastrointestinal involvement of severe staphylococcal infection and the diagnostic challenge of distinguishing infection-associated intestinal injury from concomitant hypoperfusion-related bowel ischemia.

Case Presentation

A 9-month-old infant was admitted to the pediatric intensive care unit with severe *Staphylococcus aureus* infection. Thoracic CT (Figure 1) demonstrated multiple bilateral pulmonary nodules, several of which showed central cavitation and a predominantly peripheral distribution, consistent with septic pulmonary emboli. These lesions were associated with multifocal areas of parenchymal consolidation and a small-volume pleural effusion. An apical pulmonary lesion, initially presenting as parenchymal consolidation, subsequently underwent cavitation, supporting the necrotizing nature of the pulmonary involvement.

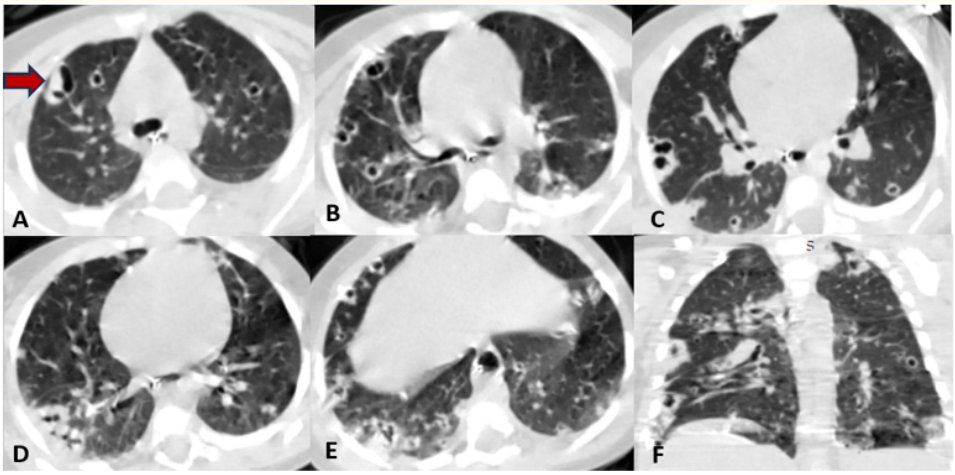

Figure 1: *Chest CT findings in a 9-month-old infant with severe Staphylococcus aureus infection. Axial lung-window images (A-E) and coronal reconstruction (F) demonstrate multiple bilateral pulmonary nodules, several showing central cavitation and predominantly peripheral distribution, consistent with septic pulmonary emboli, associated with multifocal areas of parenchymal consolidation and a small-volume pleural effusion. The apical pulmonary lesion, initially presenting as a focal consolidation, subsequently underwent cavitation, reflecting the necrotizing nature of the pulmonary involvement (Red arrow).*

The infectious assessment additionally demonstrated a staphylococcal abscess involving the gluteal region, supporting multifocal invasive *S. aureus* infection.

The clinical course was complicated by sepsis with marked hemodynamic instability and persistent hypotension requiring norepinephrine support. Laboratory investigations revealed leukocytosis and elevated C-reactive protein levels, while the platelet count remained within the normal range. Renal function progressively deteriorated, consistent with acute kidney injury.

Microbiological examination of stool samples also yielded *Staphylococcus aureus*. The infant subsequently developed progressive abdominal distension, raising concern for gastrointestinal involvement. Because of the acute kidney injury, abdominopelvic CT was performed without intravenous contrast administration.

Unenhanced abdominal CT (Figure 2) demonstrated marked bowel distension associated with segmental circumferential bowel wall thickening. The involved bowel wall showed spontaneous mural hyperattenuation, raising the possibility of a hemorrhagic component. Diffuse edematous changes of the abdominopelvic soft tissues were also present. No definite pneumatosis intestinalis or pneumoperitoneum was identified. Because intravenous contrast could not be administered, bowel wall enhancement and mesenteric vascular perfusion could not be reliably assessed.

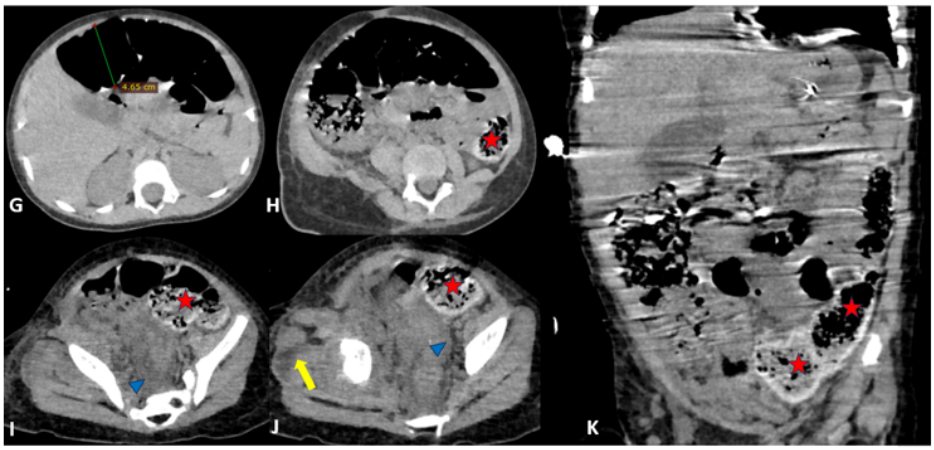

Figure 2: *Abdominal CT findings in a 9-month-old infant with disseminated Staphylococcus aureus infection. Unenhanced axial CT images (G-J) and coronal reconstruction (K) demonstrate marked bowel distension (G), associated with segmental circumferential bowel wall thickening and spontaneous mural hyperattenuation (red stars), suggesting a possible hemorrhagic component. Associated diffuse edematous changes of the abdominopelvic soft tissues are also present (blue arrowheads), along with the known gluteal soft-tissue abscess (yellow arrow). No definite pneumatosis intestinalis or pneumoperitoneum is identified. Intravenous contrast was not administered because of acute kidney injury, precluding assessment of bowel wall enhancement and mesenteric perfusion.*

Despite intensive supportive management, abdominal distension persisted and the patient's condition progressively deteriorated. Serial laboratory investigations demonstrated progressively increasing serum lactate levels, raising concern for worsening systemic and splanchnic hypoperfusion.

In the context of septic shock, sustained hypotension, and norepinephrine requirement, concomitant hypoperfusion-related intestinal injury was strongly suspected, including the possibility of non-occlusive mesenteric ischemia. However, an ischemic mechanism could not be definitively established because contrast-enhanced CT, angiographic assessment, surgical exploration, and histopathological confirmation were unavailable.

Overall, the findings were considered to represent acute intestinal involvement occurring during severe disseminated *S. aureus* infection, potentially aggravated by a superimposed hypoperfusion-related ischemic mechanism. Despite intensive care management, the infant developed progressive multiorgan failure and subsequently died.

Discussion

Staphylococcus aureus is a major cause of severe invasive bacterial infections in children and may be responsible for rapidly progressive and life-threatening disease. Although staphylococcal pneumonia accounts for a relatively small proportion of pediatric pneumonia, its clinical course may be particularly severe. A systematic review and meta-analysis including more than 20,000 pneumonia-related hospitalizations in children younger than 5 years estimated that *S. aureus* was responsible for approximately 3% of hospitalized pneumonia cases, with estimates reaching 6% in studies using higher-quality microbiological methods [1]. Severe pulmonary staphylococcal infection may be complicated by necrotizing pneumonia, pulmonary abscesses, pleural involvement, pneumatoceles, and septic pulmonary emboli.

Septic pulmonary embolism results from hematogenous dissemination of infected thrombotic material into the pulmonary circulation. CT typically demonstrates multiple bilateral pulmonary nodules with a predominantly peripheral distribution, frequently associated with cavitation, wedge-shaped peripheral opacities, and areas of consolidation [2]. In our patient, CT showed multiple bilateral predominantly peripheral pulmonary nodules, several of which were cavitated, associated with multifocal parenchymal consolidations and a small pleural effusion. The evolution of an initially consolidative apical lesion toward cavitation further supported the necrotizing nature of the pulmonary involvement. The concomitant presence of a staphylococcal abscess in the gluteal region supported the diagnosis of a multifocal invasive *S. aureus* infection.

Gastrointestinal involvement associated with *S. aureus* is considerably less common and remains poorly characterized, particularly in children. Staphylococcal enterocolitis has historically been reported in hospitalized and vulnerable patients, especially following antibiotic exposure, gastrointestinal surgery, or severe underlying illness [3]. A systematic review confirmed the existence of antibiotic-associated enterocolitis caused by methicillin-resistant *S. aureus*, while emphasizing the diagnostic difficulties surrounding this entity [3]. Pediatric cases are exceptionally reported. Loerke., *et al*. described MRSA enterocolitis in a 16-month-old boy, illustrating that clinically significant staphylococcal intestinal disease can occur even in very young children [4].

Clinical manifestations of staphylococcal intestinal involvement are variable and may include diarrhea, vomiting, abdominal pain, abdominal distension, fever, and systemic deterioration. Severe cases may progress to intestinal necrosis, septic shock, and multiorgan failure. Nevertheless, the identification of *S. aureus* in stool samples must be interpreted cautiously. Intestinal carriage of *S. aureus* is well documented, and a systematic review and meta-analysis demonstrated that gastrointestinal colonization occurs in both healthy individuals and hospitalized patients, including children [5]. Therefore, a positive stool culture alone cannot establish staphylococcal enterocolitis. The diagnosis should rely on the combination of microbiological findings, compatible gastrointestinal manifestations, exclusion of alternative causes when possible, and supportive imaging or histopathological findings.

In our patient, the microbiological context was particularly relevant because *S. aureus* was identified in stool samples in an infant with already documented multifocal invasive staphylococcal infection. The subsequent development of progressive abdominal distension and bowel abnormalities on CT strengthened the suspicion of gastrointestinal involvement associated with the ongoing infectious process. However, in the absence of surgical or histopathological confirmation, direct bacterial invasion of the intestinal wall could not be demonstrated, and the term intestinal involvement associated with *S. aureus* infection appears more appropriate than definitively diagnosing staphylococcal enterocolitis.

Radiological findings of staphylococcal enterocolitis are nonspecific. Reported CT abnormalities include bowel dilatation, circumferential mural thickening, bowel wall edema, surrounding inflammatory changes and, in severe necrotizing forms, pneumatosis

intestinalis [4,6]. Severe MRSA enterocolitis with bowel wall edema, dilatation, and pneumatosis has been reported, with subsequent pathological examination demonstrating intestinal necrosis and hemorrhagic changes [6]. In our patient, unenhanced CT demonstrated marked bowel distension associated with segmental circumferential mural thickening and spontaneous hyperattenuation of the affected bowel wall. Spontaneous mural hyperattenuation on an unenhanced examination may reflect an associated hemorrhagic component. No definite pneumatosis intestinalis or pneumoperitoneum was identified.

An important issue in interpreting these intestinal findings is the severe hemodynamic compromise experienced by our patient. Septic shock was associated with sustained hypotension requiring norepinephrine support, followed by persistent abdominal distension and progressively increasing serum lactate levels. This clinical setting raises strong concern for concomitant hypoperfusion-related bowel injury. Non-occlusive mesenteric ischemia (NOMI) results from a critical reduction in mesenteric blood flow in the absence of a primary arterial or venous occlusion and is associated with profound circulatory failure and persistent splanchnic vasoconstriction [7]. Although NOMI is predominantly described in adults, pediatric cases have been reported and remain exceptionally rare [8].

Critically ill patients with septic shock are particularly vulnerable to splanchnic hypoperfusion. Systemic hypotension reduces mesenteric perfusion, while compensatory redistribution of blood flow and intense splanchnic vasoconstriction may further compromise the intestinal circulation. Vasopressor therapy is often essential to maintain systemic perfusion in septic shock but may coexist with or potentially aggravate mesenteric vasoconstriction in susceptible patients [7,9]. Norepinephrine administration in our patient should therefore not be considered the primary cause of intestinal injury; rather, it represents a marker of the severity of the underlying circulatory failure and a possible contributing factor in an already profoundly compromised splanchnic circulation.

The progressive elevation of serum lactate levels further increased concern for ongoing tissue hypoperfusion. However, lactate elevation is not specific for mesenteric ischemia and may also occur as part of severe septic shock and multiorgan dysfunction. Similarly, the intestinal CT findings observed in our patient cannot distinguish with certainty between infectious, inflammatory, hemorrhagic, and ischemic mechanisms.

The major imaging limitation in our case was the inability to administer intravenous contrast because of acute kidney injury. Consequently, bowel wall enhancement, mesenteric arterial caliber, vascular patency, and mesenteric perfusion could not be adequately evaluated. Furthermore, neither angiographic assessment, surgical exploration, nor histopathological examination was available. A diagnosis of NOMI therefore cannot be established in this patient and should remain a strongly suspected concomitant mechanism rather than a confirmed diagnosis.

The particular interest of our observation lies in the development of acute intestinal abnormalities during severe multifocal invasive *S. aureus* infection in a 9-month-old infant, characterized by cavitated septic pulmonary emboli, multifocal pulmonary consolidations, pleural involvement, a gluteal staphylococcal abscess, and isolation of *S. aureus* from stool samples. The subsequent development of progressive abdominal distension and bowel wall abnormalities supports gastrointestinal involvement occurring during the infectious process. Nevertheless, profound hemodynamic instability, sustained hypotension, norepinephrine requirement, and progressive lactate elevation strongly suggest that intestinal hypoperfusion may have simultaneously contributed to the bowel injury.

Thus, infectious and hypoperfusion-related mechanisms should not necessarily be considered mutually exclusive. In our patient, intestinal involvement associated with disseminated staphylococcal infection may have been aggravated by secondary splanchnic hypoperfusion during septic shock, resulting in a severe and potentially hemorrhagic intestinal injury. This case emphasizes the importance of considering gastrointestinal involvement when new abdominal manifestations develop during invasive *S. aureus* infection and illustrates the particular diagnostic challenge encountered in critically ill infants when contrast-enhanced imaging cannot be performed.

Conclusion

Acute gastrointestinal involvement during invasive *Staphylococcus aureus* infection is uncommon and may be particularly difficult to recognize in critically ill infants. The development of abdominal distension and bowel wall abnormalities in the setting of documented multifocal staphylococcal infection should prompt consideration of gastrointestinal involvement. However, septic shock, sustained hypotension, vasopressor requirement, and increasing lactate levels should simultaneously raise concern for superimposed hypoperfusion-related intestinal injury.

This case illustrates the potential coexistence of infectious and ischemic mechanisms of bowel injury and emphasizes the limitations of unenhanced CT when renal impairment precludes intravenous contrast administration. Early recognition of gastrointestinal deterioration in severe pediatric sepsis is essential, as intestinal involvement may be associated with rapid progression toward multiorgan failure and an unfavorable outcome.

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Conflict of Interest

The authors declare that they have no conflicts of interest.

Patient Consent

Written informed consent for publication of this case and the accompanying images was obtained from the patient's legal guardian.

Author Contributions

All authors contributed to the conception of the work, analysis and interpretation of the imaging findings, drafting and critical revision of the manuscript. All authors read and approved the final manuscript.

Bibliography

1. Kulkarni D., *et al*. "The global burden of hospitalisation due to pneumonia caused by *Staphylococcus aureus* in the under-5 years children: a systematic review and meta-analysis". *EClinicalMedicine* 44 (2022): 101267.
2. Kuhlman JE., *et al*. "Pulmonary septic emboli: diagnosis with CT". *Radiology* 174.1 (1990): 211-213.
3. Iwata K., *et al*. "A systematic review for pursuing the presence of antibiotic associated enterocolitis caused by methicillin resistant *Staphylococcus aureus*". *BMC Infectious Diseases* 14 (2014): 247.
4. Loerke C., *et al*. "Methicillin-resistant *Staphylococcus aureus* enterocolitis in a 16-month-old boy: a case report". *Journal of Medical Case Reports* 16.1 (2022): 155.
5. Gagnaire J., *et al*. "Epidemiology and clinical relevance of *Staphylococcus aureus* intestinal carriage: a systematic review and meta-analysis". *Expert Review of Anti-infective Therapy* 15.8 (2017): 767-785.

6. Gururangan K and Holubar MK. "A case of postoperative methicillin-resistant *Staphylococcus aureus* enterocolitis in an 81-year-old man and review of the literature". *American Journal of Case Reports* 21 (2020): e922521.

7. Al-Diery H., *et al*. "The pathogenesis of nonocclusive mesenteric ischemia: implications for research and clinical practice". *Journal of Intensive Care Medicine* 34.10 (2019): 771-781.

8. Mitsuyoshi A., *et al*. "Nonocclusive mesenteric ischemia rescued by immediate surgical exploration in a boy with severe neurodevelopmental disability". *Case Reports in Pediatrics* (2019): 5354074.

9. Yu H., *et al*. "Non-occlusive mesenteric ischemia in critically ill patients". *PLoS One* 17.12 (2022): e0279196.

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