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Malignant Staphylococcal Infection Complicated by Cerebral Septic Emboli Responsible for Neurological Impairment: A Case Report and Literature Review

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Abstract **Case Report**

Malignant staphylococcal infection is a severe condition driven by the virulence of Staphylococcus aureus, potentially leading to serious systemic complications. Among these, cerebral septic emboli represent a rare but life-threatening entity. We report the case of a 50-year-old man admitted to the intensive care unit for septic shock due to S. aureus, complicated by multiple cerebral ischemic lesions resulting in prolonged coma. Brain MRI and blood cultures allowed rapid diagnosis. Antibiotic therapy tailored to microbiological results and adjusted to the patient's end-stage renal disease, combined with preventive anticoagulation and intensive care support, resulted in significant neurological improvement and successful extubation on day 12. This case highlights the importance of early and aggressive management in severe S. aureus bacteremia.

Keywords: malignant staphylococcal infection; cerebral septic emboli; neurological impairment.

INTRODUCTION

Malignant staphylococcal infection is a rapidly progressing and severe cutaneous–mucosal infection characterized by marked local aggressiveness and a high risk of hematogenous dissemination, particularly when Staphylococcus aureus is involved [1]. Among systemic complications, septic emboli are well-recognized and may involve the lungs, kidneys, and spleen [2]. Cerebral embolization is less common and often difficult to diagnose, as it may occur even in the absence of valvular vegetations, unlike typical emboli observed in infectious endocarditis [3]. Brain MRI is now considered the most sensitive diagnostic tool for detecting these multiple ischemic lesions [4]. Here, we report an illustrative case of malignant staphylococcal infection complicated by cerebral septic emboli causing prolonged consciousness impairment, and we discuss the diagnostic and therapeutic challenges associated with this condition.

CASE REPORT

A 50-year-old man with type 2 diabetes on insulin and known end-stage renal disease on intermittent hemodialysis was initially admitted to a peripheral hospital for severe pneumonia complicated by hyperkalemia and respiratory deterioration. Thoracic CT revealed bilateral alveolar–interstitial infiltrates with moderate pleural effusion. Laboratory tests showed leukocytosis of 20,000/mm³, CRP of 225 mg/L, an initial urea level of 3 g/L, and creatinine of 132 mg/L. Despite emergency dialysis and empirical antibiotics, the clinical course rapidly worsened, with the onset of respiratory and neurological distress requiring emergent intubation and transfer to our ICU.

Upon admission, the patient was intubated, ventilated, sedated with midazolam and fentanyl, and presented with septic shock requiring norepinephrine infusion. Arterial blood gas showed severe metabolic acidosis (pH 7.18) with bicarbonate at 15 mmol/L, significant hypocapnic acidosis, and moderate hyperlactatemia. A second blood gas indicated further deterioration (pH 7.14, HCO_3^- 17 mmol/L). Laboratory workup confirmed severe inflammation (CRP 181 mg/L). Respiratory examination revealed abundant purulent bronchial secretions and bilateral B-profile on ultrasound.

During the first 48 hours, the clinical course was marked by unexplained delayed awakening despite

sedation withdrawal. The patient showed no seizure activity, had a Glasgow score of 3/15, and exhibited semi-dilated reactive pupils. Brain MRI was therefore performed and showed multiple punctate ischemic lesions consistent with septic emboli. Blood cultures returned positive for Staphylococcus aureus, supporting the diagnosis of malignant staphylococcal infection with embolic dissemination. Infective endocarditis was considered, although initial transthoracic echocardiography revealed neither vegetations nor significant valvular abnormalities.

The septic state remained unstable during the first days, with persistent inflammation, purulent pulmonary secretions, and ongoing hemodynamic instability requiring vasopressors. Targeted antibiotic therapy was initiated and doses were adjusted according to the patient's renal failure. Preventive anticoagulation with unfractionated heparin was administered. Lumbar puncture excluded meningitis, with clear and sterile cerebrospinal fluid. Chest CT confirmed extensive alveolo-interstitial involvement with embolic-appearing pulmonary nodules.

Metabolic management was guided by the patient's pre-existing end-stage renal disease, requiring repeated hemodialysis sessions to control acidosis, hyperkalemia, and uremia. Gradually, hemodynamic stabilization allowed progressive weaning from vasopressors.

Neurologically, the patient remained comatose for the first week (GCS 3/15) despite complete sedation withdrawal. Neurological improvement began only on day 9, with progressive recovery of consciousness and appropriate motor responses. By day 12, significant neurological improvement allowed successful extubation.

Thus, this patient presented with malignant staphylococcal infection complicated by cerebral septic emboli, resulting in prolonged coma in the context of severe septic shock. The delayed but real improvement followed infection control, tailored antibiotic therapy, and intensive supportive care.

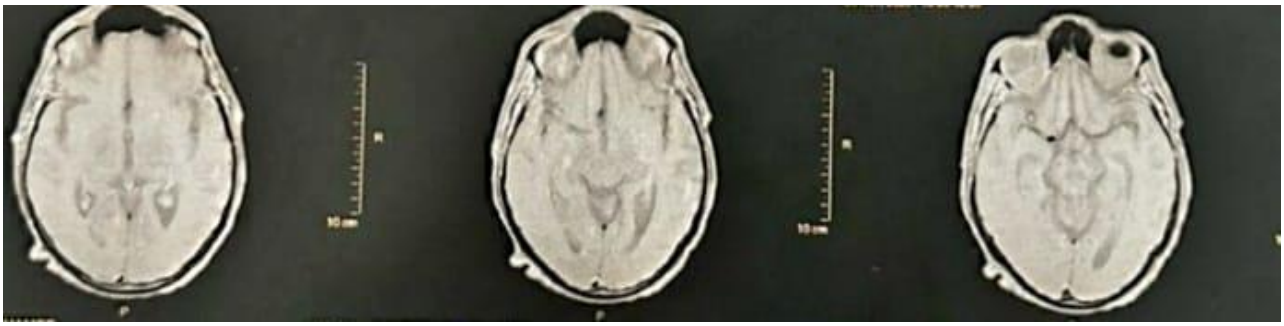

Figure 1: Axial FLAIR MRI sequences showing multiple hyperintense lesions suggestive of septic emboli

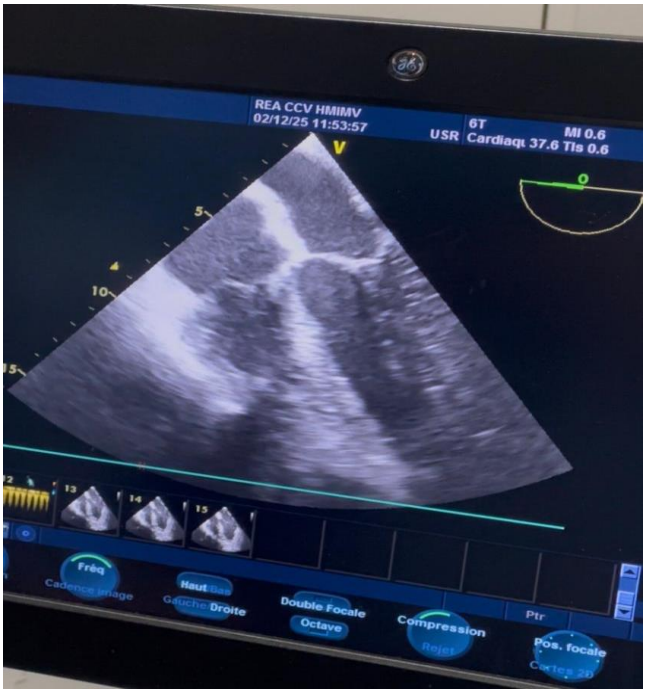

Figure 2: Apical four-chamber transthoracic echocardiography showing no significant abnormalities.

DISCUSSION

Malignant staphylococcal infection is a serious condition with rapid progression, largely driven by the virulence of Staphylococcus aureus, whose potential for inducing bacteremia and spreading to distant organs is well documented [1]. Cerebral septic emboli are rare but particularly severe complications. Their pathophysiology involves the formation of infected microthrombi that migrate through the systemic circulation and lodge in the cerebral parenchyma, causing ischemic lesions or microabscesses visible on MRI [2]. Cerebral involvement may occur even without endocarditis, making diagnosis challenging.

Neurological complications occur in 6–12% of S. aureus bacteremias [3], and MRI is the preferred imaging modality for detecting septic emboli, which are often multiple and punctiform, sometimes undetectable on CT scans [4]. In this case, the characteristic MRI findings combined with positive blood cultures allowed rapid diagnosis.

Several similar clinical cases have been reported. Permyakov et al. described a patient with severe cellulitis complicated by cerebral and pulmonary septic emboli without endocarditis, with MRI findings nearly identical to ours [5]. Neurological recovery occurred only after several days of targeted antibiotic therapy, highlighting the delayed nature of clinical improvement. Another case reported by Sexton et al. involved a patient admitted for S. aureus septicemia complicated by prolonged coma due to multiple septic emboli; neurological improvement began around day 10 of therapy [6]. These cases parallel our patient's course, both in MRI presentation and recovery delay.

Management relies on intensive and prolonged antibiotic therapy tailored to microbiological results, in line with major infectious disease guidelines [7]. Dose adjustment according to renal function is essential in end-stage renal disease. Curative anticoagulation is not indicated in acute cerebral septic emboli due to the high risk of hemorrhagic transformation [8], whereas preventive anticoagulation remains appropriate for immobilized ICU patients, as performed here.

Neurological improvement around day 9 aligns with published reports describing recovery between 7 and 14 days once effective therapy is initiated [6]. This delay reflects the gradual resolution of infected microvascular foci and reduced systemic bacterial load. Our case thus illustrates the rare but typical presentation of cerebral septic emboli complicating malignant staphylococcal infection without endocarditis and emphasizes the need for early MRI-based diagnosis and aggressive management

CONCLUSION

Cerebral septic emboli represent a severe but frequently underdiagnosed complication of invasive S. aureus infections. MRI is the key diagnostic modality, especially when prolonged coma persists after sedation withdrawal. Antibiotic therapy adapted to microbiological findings and comorbidities, combined with early intensive care support, allows neurological improvement even when delayed. This case highlights the need for high clinical suspicion in severe S. aureus bacteremia and the importance of rigorous management to achieve favorable outcomes despite severe initial presentation.

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