MRSA sepsis complicated by splenic abscess and nephrotic syndrome in a young diabetic patient: A case report

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Summary

Background: Methicillin-resistant *Staphylococcus aureus* (MRSA) is a significant cause of invasive infections, but splenic abscess and nephrotic syndrome are rare complications, especially in young diabetics. *Case Presentation:* We describe a 27-year-old Vietnamese woman with type 1 diabetes who developed fever, scalp and thigh abscesses, edema, MRSA sepsis, nephrotic-range proteinuria (8.9 g/24h), hypoalbuminemia, pleural effusions, and a splenic abscess. CT also showed an incidental adrenal mass. Cultures from blood and abscesses grew MRSA. She improved with IV vancomycin, meropenem, amikacin, and supportive care (albumin, insulin, antihypertensives). *Conclusion:* This case demonstrates a rare and complex form of MRSA sepsis with nephrotic syndrome and splenic abscess, illustrating the need for prompt recognition and multidisciplinary treatment.

Keywords: Methicillin-resistant Staphylococcus aureus (MRSA), sepsis, splenic abscess, nephrotic syndrome, type 1 diabetes, case report.

I. BACKGROUND

Methicillin-resistant Staphylococcus aureus (MRSA) is a significant pathogen responsible for diverse infections, from minor skin issues to serious bloodstream infections with distant complications[1]. The rise of community-acquired MRSA has blurred distinctions between hospital and community strains, leading to more severe cases in healthy individuals[2]. Disseminated MRSA sepsis with blood infection and metastatic organ involvement remains rare but highly morbid, while splenic abscesses caused by S. aureus are particularly uncommon in less than 15% of cases and often diagnosed late due to vague symptoms[3,4,5].

The emergence of nephrotic syndrome during active infection complicates diagnosis and treatment. While diabetes is the main cause of nephrotic-range proteinuria, sudden onset with severe infection suggests possible secondary glomerular damage, potentially related to MRSA-induced immune responses; these cases are rare and not well understood[6,7].

We report a case of a young woman with type 1 diabetes who developed disseminated MRSA sepsis, splenic abscess, and new nephrotic syndrome a rare clinical triad. This highlights the importance of considering complex presentations, multidisciplinary care, and careful interpretation of immunologic findings in infectious disease management.

II. CASE PRESENTATION

A 27-year-old Vietnamese woman with type 1 diabetes mellitus was admitted to the infectious disease service at 108 Central Military Hospital, Hanoi. She had experienced 10 days of high fever, fatigue, and painful soft-tissue swellings, worsening over time.

***Received**: 2/10/2025, **Accepted**: 28/10/2025*

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Her illness began with a scalp pustule that ruptured after applying a herbal remedy, followed by a left thigh abscess three days later. She also developed increasing periorbital and lower limb edema.

On admission, her symptoms escalated to chills, nausea, shortness of breath, and severe thigh pain. Antipyretics at a local center were ineffective, leading to her referral for specialized care. Examination showed she was alert but ill, with a temperature of 39.1°C, tachycardia (125bpm), and fluctuating blood pressure (110/70 - 144/86mmHg). She had eyelid and leg edema, a left thigh abscess, and crusted scalp lesion, but no neurological deficits.

Lab tests revealed normocytic anemia (hemoglobin 92 g/L), elevated white blood cell count (12.4 $\times 10^9$/L, 75.5% neutrophils), and high procalcitonin (204.8ng/mL). Serum creatinine was 1.2mg/dL (eGFR 64mL/min/1.73m^2), with low protein (56g/L) and albumin (21g/L). Urine protein loss was 8.9g/24h, but liver and coagulation tests were normal.

CT scans showed bilateral pleural effusions, moderate ascites, and a 56 × 62mm hypodense splenic lesion, consistent with abscess. An incidental right adrenal mass was also noted. Aspiration of the splenic abscess yielded chocolate-colored pus that grew MRSA (Figure 1, 2), and blood cultures confirmed MRSA, establishing disseminated MRSA sepsis.

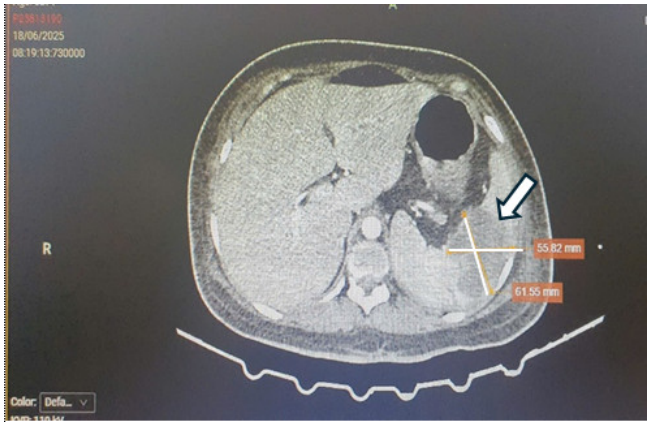

Figure 1. Contrast-enhanced abdominal CT showing a well-defined hypodense lesion in the spleen measuring approximately 56 × 62 mm (white arrow), consistent with splenic abscess.

Figure 2. Aspirated pus from splenic abscess

Empirical intravenous antibiotics were started quickly due to the severity and spread of the infection. The initial treatment included vancomycin (1.5 g daily) and meropenem (1 g every 8 hours). As her condition improved, cultures from blood and splenic abscess confirmed MRSA sensitive to vancomycin. The antibiotics were then streamlined to vancomycin and amikacin, optimizing effectiveness and reducing unnecessary broad-spectrum coverage.

Daily intravenous albumin was given for severe hypoalbuminemia. Diuretics were used cautiously to manage edema, with close monitoring to avoid dehydration. Glycemic control was maintained with subcutaneous insulin, adjusted regularly. Blood pressure management focused on keeping systolic pressure below 130 mmHg to protect kidney function. Due to marked proteinuria and hypoalbuminemia, nephrology was consulted to investigate nephrotic syndrome. Autoimmune glomerulopathies were excluded by negative ANA, anti-dsDNA, ANCA, anti-GBM, and anti-PLA2R IgG tests. Serologies for HBV, HCV, and HIV were also negative, ruling out viral causes.

Renal biopsy was postponed because of ongoing sepsis risks. Immunosuppressive therapy was not started, given the active infection and negative immunologic results. The team concluded the nephrotic syndrome was likely infection-associated, secondary to MRSA-induced glomerulonephritis.

Over two weeks, the patient's condition gradually improved with resolution of fever and regression of the splenic abscess. However, renal function worsened, requiring continued monitoring of kidney function and proteinuria for future management.

III. DISCUSSION

Rare clinical constellation: splenic abscess, nephrotic syndrome, and type 1 diabetes mellitus

This case highlights an exceptionally rare convergence of three distinct clinical entities: splenic abscess due to methicillin-resistant Staphylococcus aureus (MRSA), new-onset nephrotic syndrome, and longstanding type 1 diabetes mellitus (T1DM).[8] While each of these conditions has been described independently in the medical literature, their simultaneous occurrence in a single patient is exceedingly uncommon and, to our knowledge, has not been previously reported.

Unusual combination of events

Splenic abscess is an uncommon complication of bacteremia, with reported incidence rates ranging from 0.2% to 0.7%[4]. The widespread use of antibiotics has further reduced its frequency, and cases attributable to S. aureus represent only a minority. The presence of a splenic abscess often implies hematogenous dissemination, particularly in individuals with underlying immunocompromise or conditions such as endocarditis.

The concurrent emergence of nephrotic syndrome during an active bloodstream infection adds further diagnostic complexity, especially in diabetic patients. In this context, nephrotic-range proteinuria may suggest either diabetic nephropathy or a secondary form of glomerular injury. Notably, splenic involvement in MRSA-specific infections is infrequently documented, though analogous cases have been reported,[8] reinforcing the likelihood of hematogenous seeding as the underlying mechanism[5].

The simultaneous presentation of MRSA sepsis, splenic abscess, and nephrotic syndrome in this patient highlights a unique intersection of pathophysiological processes that merits further investigation and clinical attention.

Pathogenesis: From bacteremia to organ injury

The development of a splenic abscess in this patient can be attributed to hematogenous dissemination during MRSA bacteremia. Evidence from prior case series indicates that septic emboli or microinfarctions within the spleen may compromise the integrity of the splenic parenchyma, thereby facilitating abscess formation[3]. In patients with diabetes, additional factors such as impaired neutrophil chemotaxis and microvascular dysfunction further increase susceptibility to metastatic infections, including splenic abscesses[1].

The development of nephrotic syndrome in this patient, characterized by the sudden appearance of massive proteinuria and hypoalbuminemia, prompts crucial considerations regarding the host immune response to MRSA infection. Both experimental and clinical evidence indicate that bacterial superantigens, such as toxic shock syndrome toxin-1 (TSST-1) and various enterotoxins, have the capacity to activate T lymphocytes. This activation results in widespread stimulation of polyclonal B-cell populations, ultimately driving the formation of immune complexes. These immune complexes, primarily composed of immunoglobulin A (IgA), immunoglobulin G (IgG), and complement component C3, can deposit within the glomerular mesangium and basement membrane. Their deposition triggers an inflammatory cascade that culminates in a form of glomerulonephritis, which is typically self-limited in nature.[9] Notably, clinical observations by Koyama and colleagues, as well as Hashimoto et al., have documented cases of IgA-dominant glomerulonephritis developing in the aftermath of MRSA bacteremia. In these instances, resolution of the glomerular injury was achieved with antibiotic therapy alone, without the need for corticosteroid administration[6,7].

While infection-associated glomerulonephritis remains a significant diagnostic consideration in this case, it is important to acknowledge that underlying

diabetic nephropathy cannot be entirely excluded. Diabetic kidney disease (DKD) is the most common cause of nephrotic-range proteinuria in young adults with type 1 diabetes mellitus (T1DM). Notably, DKD may occasionally manifest abruptly, even in the absence of prolonged hypertension or diabetic retinopathy[10,11].

In the present patient, the sudden onset of proteinuria may be explained by a "second-hit" hypothesis. Here, the inflammatory burden imposed by MRSA infection could have exacerbated pre-existing, subclinical glomerular injury resulting from chronic hyperglycemia. Histopathological features of DKD including mesangial expansion, glomerular basement membrane thickening, and podocyte loss cannot be definitively identified without a renal biopsy[12].

Autoimmune serologies (ANA, anti-dsDNA, ANCA, anti-GBM, anti-PLA2R) and viral markers (HBV, HCV, HIV) were negative in this patient, thereby effectively excluding lupus nephritis, post-infectious glomerulonephritis, and virus-associated nephropathy as potential etiologies. Considering these immunological findings alongside the patient's clinical background of T1DM, DKD remains a plausible cause, either precipitated or intensified by the presence of systemic infection.

According to the current KDIGO (Kidney Disease: Improving Global Outcomes) guidelines, a renal biopsy is advised for patients with diabetes who present with atypical features of nephrotic syndrome. This is particularly relevant when there is an absence of diabetic retinopathy or when there is a rapid decline in glomerular filtration rate (GFR), as these findings may suggest an alternative or superimposed glomerular pathology (KDIGO, 2020)[13].

In this case, however, performing a renal biopsy was not feasible due to the presence of active sepsis, which poses a significant procedural risk. As a result, alternative approaches must be utilized to clarify the predominant mechanism underlying the nephrotic syndrome. Serial monitoring of proteinuria and renal function over time, in conjunction with detailed fundoscopic examination, may offer valuable insights. These assessments can help discern whether diabetic nephropathy or an alternative glomerular process is primarily responsible for the patient's renal presentation.

Diagnostic and therapeutic considerations

Autoimmune and viral serologies were negative in this patient, effectively excluding lupus nephritis, HBV- or HCV-related glomerulonephritis, and HIV-associated nephropathy. Although a renal biopsy was not performed due to concerns about active infection, the clinical picture aligns with a post-infectious glomerulopathy triggered by MRSA. Immunosuppressive therapy was judiciously withheld, in accordance with current expert opinion and reports such as that by Hashimoto et al., where such cases improved solely with infection control.

Therapeutically, the patient benefited from a multifaceted approach combining broad-spectrum antibiotics tailored to MRSA sensitivity (vancomycin and amikacin), drainage of the splenic abscess under CT guidance, and supportive management of fluid status and glycemia. The decision to defer immunosuppression likely prevented worsening of sepsis and was validated by clinical improvement.

Clinical outcome and follow-up

After the resolution of sepsis, the patient experienced a decline in renal function, as evidenced by a creatinine level of 125 µmol/L. This outcome highlights the importance of a comprehensive approach that addresses both infection control and renal management in patients with MRSA-related complications.

Given the uncommon nature of this clinical presentation, ongoing nephrology follow-up is essential. Long-term monitoring will allow for timely identification of chronic glomerular complications or recurrence of renal injury, ensuring that appropriate care is provided as the patient's condition evolves.

Broader implications and future directions

This case highlights the critical need for maintaining a high index of suspicion for visceral

abscesses in patients presenting with persistent MRSA bacteremia, especially among those with underlying diabetes or immunocompromised states. Early and targeted imaging should be prioritized to detect hidden metastatic infection sites, as timely identification can significantly impact outcomes.

Additionally, the emergence of new-onset nephrotic syndrome during systemic infection calls for a comprehensive immunologic and microbiologic evaluation prior to considering immunosuppressive therapy.

This approach ensures that underlying causes are appropriately addressed, and unnecessary immunosuppression, which could exacerbate infection, is avoided.

IV. CONCLUSION

There remains a need for further research to clarify the mechanisms linking MRSA infection to glomerular injury. Prospective registries focusing on MRSA-associated nephropathies may be instrumental in enhancing our understanding of these rare but clinically meaningful intersections. Case reports like this one serve as valuable contributions, laying the groundwork for future investigations into the complex interplay between infectious disease processes and immune-mediated renal injury.

Declarations

Ethics approval and Consent to participate: Ethical approval was waived as this report describes a single clinical case without patient identifiers. Institutional review board (IRB) review was not required per local ethical guidelines.

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 editorial office upon request.

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

Funding: This work received no external funding. All treatment and reporting were performed as part of standard clinical care.

Authors' contributions: Ph.D.Tung Nguyen Van contributed to patient care, data collection, and manuscript preparation. Other authors reviewed and approved the final manuscript. All authors read and approved the final manuscript.

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