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Clinical Management Strategies for Severe Fever with Thrombocytopenia Syndrome Induced by Tick Bites: A Case Report and Literature Review

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Patient: Male, 79-year-old
Final Diagnosis: Severe fever with thrombocytopenia syndrome
Symptoms: Severe fever with thrombocytopenia syndrome
Clinical Procedure: —
Specialty: Infectious Diseases

Objective: Rare disease

Background: Severe fever with thrombocytopenia syndrome (SFTS) has a high mortality rate, and diagnosis and treatment options for SFTS-associated encephalopathy/encephalitis(SFTSAE) are limited.

Case Report: A 79-year-old male farmer developed progressive dyspnea and productive cough 72 hours after tick exposure, accompanied by persistent high-grade fever (38.9°C). Following failed empirical antimicrobial interventions, he was transferred to our institution. At the same time, the local disease control center diagnosed him with fever with thrombocytopenia syndrome (SFTS). Initial evaluations at peripheral hospitals revealed thrombocytopenia (platelet 25×10^9/L) and leukopenia (WBC 1.24×10^9/L). Despite aggressive therapy, including intravenous immunoglobulin (IVIG), favipiravir, doxycycline, meropenem, and platelet transfusions, progressive multiorgan failure manifested as metabolic acidosis (pH 7.24, lactate 6.57 mmol/L), hepatorenal and kidney dysfunction (ALT 665.2 U/L, AST 7764 U/L, creatinine 191 umol/L), hypoxemic respiratory failure (PaO_2/FiO_2 160), and neurological decline (GCS 10). Despite 72 hours of aggressive critical care interventions, the patient died due to refractory multiorgan failure secondary to viral sepsis.

Conclusions: This case is a stark reminder of the rapid progression and severe nature of fever with thrombocytopenia syndrome associated with tick bites, emphasizing the critical need for individualized treatment strategies tailored to the clinical classification of the illness, particularly in severe cases that might involve neurological complications.

Keywords: Encephalitis • Fever • Thrombocytopenia

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Introdcution

Ticks are widely distributed across the globe, thriving predominantly in temperate and tropical regions where their populations flourish. Ticks are hematophagic ectoparasites that feed exclusively on mammalian, avian, and occasionally reptilian and amphibian blood, with their body divided into an anterior capitulum (head and mouthparts) and a posterior idiosoma (dorsal scutum, legs, and internal digestive and reproductive organs), as shown in **Figure 1** [1]. These resilient creatures exhibit remarkable ecological adaptability, allowing them to survive in a variety of environments, and they are known to carry numerous viruses and pathogens. According to species and genera, Bunyaviridae is divided into Orthobunyavirus, Phlebovirus, Nairovirus, Hantavirus. The representative viruses are California encephalitis virus [2], severe fever with thrombocytopenia syndrome virus (SFTSV) [3], Crimean Congo hemorrhagic fever virus (CCHFV) [4], and hantavirus [5]. SFTSV occurs mainly in China, Japan, and South Korea, CCHFV is mainly in Africa, the Middle East, Central Asia, and Northwest China, and hantavirus is mainly in East Asia, northern Europe, and South America. SFTSV (a novel bunyavirus, also known as Dabie Banda virus), an intriguing tick-borne RNA virus that has garnered attention due to its unique characteristics, is classified as a novel member of the genus Phlebovirus, which is part of the larger Bunyaviridae family. This virus was initially identified in the regions of Central and North-Eastern China in 2009, a discovery that not only expanded our understanding of tick-borne diseases but also raised awareness about the potential health risks associated with this emerging viral threat [6].

Severe fever with thrombocytopenia syndrome (SFTS) caused by tick bite is a complex clinical condition characterized by the simultaneous presence of fever, which can manifest as a persistent elevation in body temperature, and a significant decrease in platelet count, a condition known as thrombocytopenia, which can lead to various health complications [7]. The clinical manifestations of SFTS vary widely among patients, ranging from mild symptoms such as low-grade fever and fatigue to severe complications that can include multiorgan dysfunction and potentially life-threatening hemorrhagic events, which can significantly impact patient outcomes [8]. The diagnosis of SFTS was often based on a combination of clinical presentation, laboratory findings, and careful exclusion of other potential causes of fever and thrombocytopenia, ensuring that the correct underlying cause is identified [9]. At present, the treatment of new Bunia virus infection remains primarily supportive in nature, largely due to the absence of a licensed vaccine, with ongoing research still in the discovery stage [10]. Emerging vaccine platforms against SFTSV encompass 6 principal modalities: live-attenuated platforms, DNA-based vectors, whole inactivated virions, viral-vectored antigen delivery systems, protein subunit/VLP formulations, and

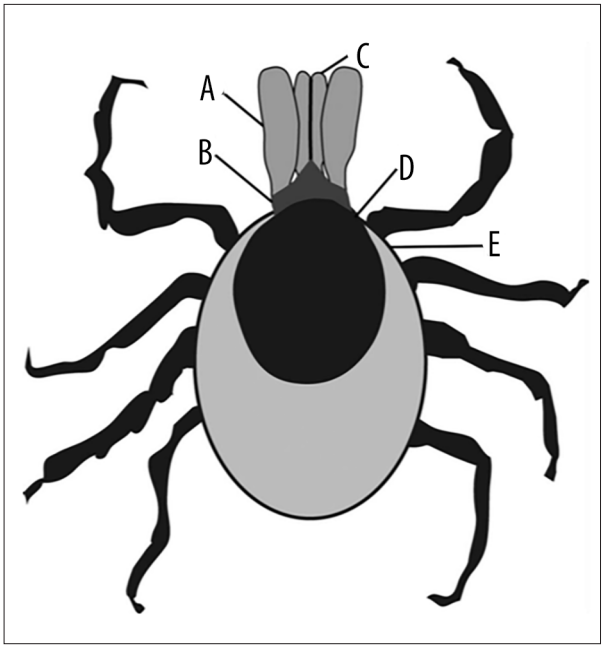

Figure 1. Generic diagram of a hard tick (Ixodes spp., dorsal view). A – palp; B – basis capitulum; C – hypostome; D – scutum; and E – idiosoma. *Used with permission from Wolters Kluwer Health [1].*

nucleoside-modified mRNA constructs encoding viral glycoproteins [11]. These approaches collectively target humoral and cellular immune activation through diverse antigen presentation mechanisms, reflecting global efforts to address SFTSV's expanding epidemiological burden through prophylactic intervention.

In this case report, we introduced a 79-year-old male farmer who developed fever and respiratory symptoms following a tick bite. The final diagnosis was SFTS associated with novel bunyavirus infection. The rapid progression of the disease emphasizes the urgency of timely intervention and personalized treatment strategies in managing this complex syndrome.

Case Report

Patient Information

The patient was a 79-years-old male farmer from the area around Jinhua City, Zhejiang Province, in China, with no other chronic diseases except for a history of cholecystectomy, and had no history of taking drugs. The patient presented to a rural clinic on 26 June following a tick bite. Within 72 hours of arthropod exposure, he developed a febrile illness (peak temperature 38.9°C) accompanied by progressive dyspnea and cough. Initial management included oral levofloxacin (500 mg daily) as empirical antimicrobial therapy. The patient's manual

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extraction of the arthropod compromised tick integrity, precluding definitive exclusion of retained mouthparts through gross visual inspection. Therefore, in the initial clinical evaluation, no serological tests were performed on tick-borne pathogens (eg, Borrelia burgdorferi, Anaplasma phagocytophilum) or doxycycline preventive drugs recommended by the Centers for Disease Prevention and Control (CDC). Persistent febrile recurrences (temperature fluctuations >38°C over 4 days) ultimately necessitated referral to a higher-level care facility for comprehensive infectious disease workup.

On June 30, 2024, just 4 days after his initial visit, he sought treatment at the local county hospital, where medical professionals took a more proactive approach. The surgical exploration of the bite site (left scapular area) unfortunately found residual tick components, and the residual cephalosome was removed using tweezers under sterile conditions. Concurrently, tissue specimens were collected to CDC for multiplex PCR analysis targeting SFTSV, CCHFV, and other related Bunyavirales pathogens. Following this procedure, he gradually became weak and was recommended to transfer to a municipal hospital for further assessment and specialist care, as his situation warranted a more comprehensive assessment to determine the underlying cause of his persistent health issues.

Admission to Our Hospital

On 30 June 2024, he was transferred to our tertiary care center following consultation with physicians at the primary county hospital. Initial laboratory investigations following Emergency Department admission revealed marked reductions in white blood cells, lymphocytes, neutrophils, and platelets (PLT 25×10^9/L), indicating elevated bleeding risk. Mild abnormalities in inflammatory markers, hepatic/renal function, cardiac enzymes, and coagulation parameters suggested systemic involvement necessitating comprehensive evaluation. Subsequent notification from local CDC confirmed SFTSV-RNA positivity, highlighting outbreak potential. Imaging studies revealed bilateral pleural effusion with mild interstitial pulmonary edema, suggesting viral-associated respiratory compromise. The diagnosis of SFTS was established based on comprehensive hematological, imaging, and etiological tests. The patient was subsequently transferred to the isolation ward for specialized management. Initial physical examination documented elevated temperature, altered mental status, and mild nuchal rigidity with preserved orientation, suggesting potential involvement of the central nervous system (CNS). To stabilize the patient's clinical status, a comprehensive therapeutic regimen was implemented: 1) platelet transfusion was immediately initiated to mitigate severe thrombocytopenia; 2) intravenous immunoglobulin (IVIG, 20 g/daily) was administered to modulate immune response; 3) doxycycline therapy was commenced with an initial loading dose of 200 mg followed by 100 mg twice daily to cover potential rickettsial co-infections; 4) favipiravir (loading dose 1600 mg on day 1, followed by 600 mg daily from days 2-5) was prescribed for its inhibitory activity against SFTS. Supportive care measures were concurrently implemented, including fluid resuscitation and hemodynamic monitoring, focusing on the change in mental state.

Follow-Up and Progression

On 1 July 2024, repeat laboratory tests at 24 hours after admission demonstrated markedly elevated lactate levels (Lac 6.57 mmol/L) and low pH levels (pH 7.242), as well as base excess (BE -8.3 mmol/L), suggesting metabolic acidosis, progressive thrombocytopenia with coagulopathy(PLT 9×10^9/L), and elevated inflammatory markers (PCT 28.52 ng/mL, IL-6 4870 pg/mL). Concurrent hepatic/renal dysfunction and myocardial injury were evidenced by elevated transaminases (ALT 665.2 U/L, AST 7764 U/L), creatinine (191 umol/L), and troponin-I (1.88 ng/mL). Positive β-D-glucan testing (389.80 pg/ml) and hyperferritinemia (5075.95 ng/ml) further supported systemic hyperinflammation. Serial examinations documented persistent intermittent fever (peak 39.5°C) with neurological manifestations including disorientation, incoherent speech, and nuchal rigidity. According to the results of the appeal inspection, the patient fulfilled diagnostic criteria for SFTS complicated by multiorgan dysfunction: metabolic acidosis, hepatic impairment, acute kidney injury, myocardial injury, and encephalopathy (GCS 10), consistent with MODS progression. The treatment regimen was expanded from the initial IVIG, doxycycline, and favipiravir regimens to include meropenem (2 g every 8h), broad-spectrum β-lactam antibiotics, and voriconazole (200 mg every 12 h) antifungal therapy to enhance empirical coverage of potential bacterial and fungal co-infections (including intracranial).

Despite aggressive interventions, clinical deterioration ensued. On the evening of the second day of admission (01-07-2024), the patient suddenly developed coma with unequal pupil size and hypoxemia (SpO_2 82%), suggesting further deterioration of the condition, manifested as acute neurological dysfunction and acute respiratory failure. Under mechanical ventilation via endotracheal intubation, cranial computed tomography (CT) imaging was performed to rule out intracranial hemorrhage. Given the patient's clinical progression, neuroinvasive SFTSV infection was strongly suspected as a potential etiology for encephalopathy/encephalitis, which is the most severe neurotropic complication associated with SFTSV infection. In the Intensive Care Unit (ICU), multimodal organ support therapy was implemented, comprising therapeutic plasma exchange (TPE), continuous renal replacement therapy (CRRT), and advanced cardiopulmonary monitoring. Despite 72 hours of aggressive critical care interventions, the patient died due refractory multiorgan failure secondary to viral sepsis.

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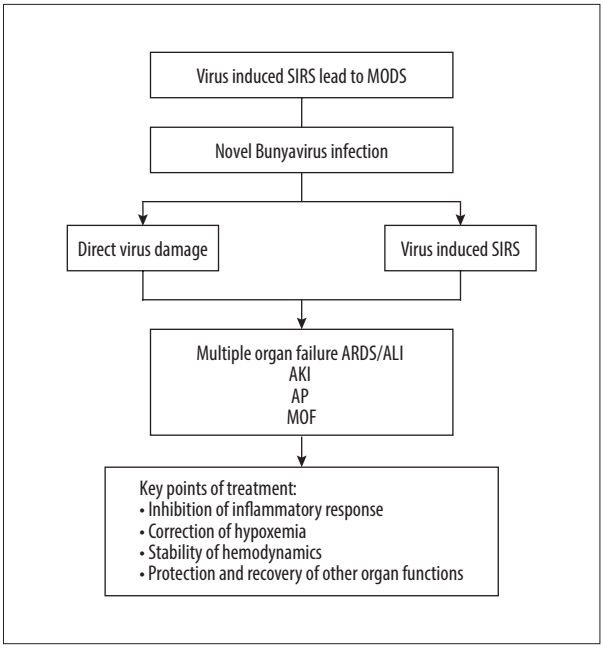

Figure 2. Virus-induced SIRS leads to MODS. After infection with the novel Bunyavirus, the virus exerts a direct and damaging effect on local tissues, which can subsequently induce a systemic inflammatory response syndrome (SIRS). If the condition progresses without appropriate intervention, it can ultimately lead to a severe and life-threatening scenario characterized by multiple organ dysfunction syndrome (MODS). This catastrophic progression may manifest as acute respiratory distress syndrome (ARDS), acute lung injury (ALI), acute kidney injury (AKI), acute pancreatic damage (AP), and the overarching complication of multiple organ failure (MOF). At this critical stage, the key points of treatment focus on several essential objectives: suppressing the inflammatory response to mitigate further tissue damage, correcting hypoxemia to ensure adequate oxygenation of vital organs, stabilizing hemodynamics to maintain proper blood circulation, and protecting and restoring the function of other affected organs to enhance the patient's chances of recovery and survival.

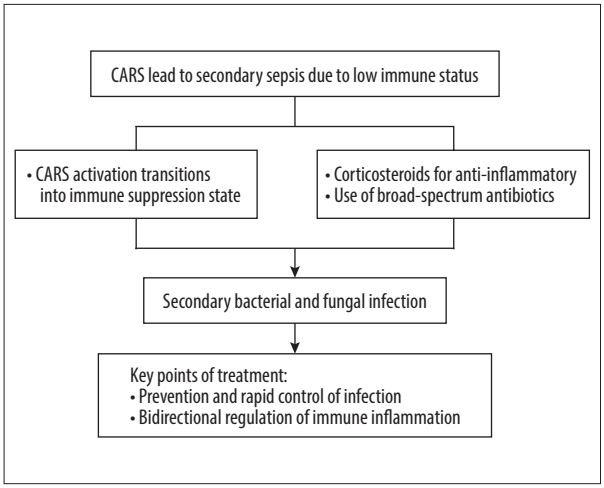

Figure 3. CARS led to secondary sepsis due to low immune status. After compensatory anti-inflammatory response syndrome (CARS) activation, the body switched to the state of immune suppression and exhaustion, which significantly impairs the ability to respond to external threats and maintain optimal functionality. The use of glucocorticoids, which are powerful anti-inflammatory agents, along with broad-spectrum antibiotics during the course of the disease can inadvertently lead to secondary bacterial and fungal infections, complicating the overall health status. Therefore, the primary focus of treatment during this critical period is not only on the prevention and rapid control of these infections but also on the careful and strategic bidirectional regulation of immune inflammation, ensuring that the immune system can recover and regain its balance while effectively combating any potential pathogens that may arise.

Discussion

This case shows SFTS with rapid progression and poor prognosis, lacking specific treatments; management was focused on supportive care. Based on the literature, we summarized the classification, staging, and management of febrile thrombocytopenia syndrome, which can be mild to critical based on clinical presentation and epidemiology, and categorized by organ involvement into types like cardiopulmonary and SFTSAE, the latter being the most severe, with high mortality and neurological symptoms [12-14].

SFTSAE can be classified into 2 subtypes based on pathogenic mechanisms: non-inflammatory storm type and inflammatory storm type [15]. The former resembles typical viral encephalitis, where viruses directly invade neurons, requiring primarily supportive care with minimal anti-inflammatory intervention [16]. The latter involves cytokine storms driven by immune hyperactivation, necessitating aggressive anti-inflammatory therapy to mitigate organ damage and improve outcomes. Mortality rates are significantly higher in the inflammatory storm subtype compared to the non-inflammatory type [17] (**Figures 2 and 3** illustrate this classification). IVIG was administered to modulate cytokine storm-associated SFTSAE in this case, but failed to effectively alleviate disease progression. Mechanistically, IVIG therapy correlates with elevated SFTSV viremia and depletion of lymphocyte subsets (CD4+ T cells, NK cells) [18]. Contemporary evidence suggests optimized IVIG dosing (80 g/day for ≥5 days) achieves 17% mortality reduction, highlighting the critical need for protocol standardization in neurotropic bunyavirus infections [19].

The cytokine storm associated with inflammatory encephalitis can be categorized into 3 distinct pathophysiological phases: the fever phase, the encephalopathy/encephalitis phase, and

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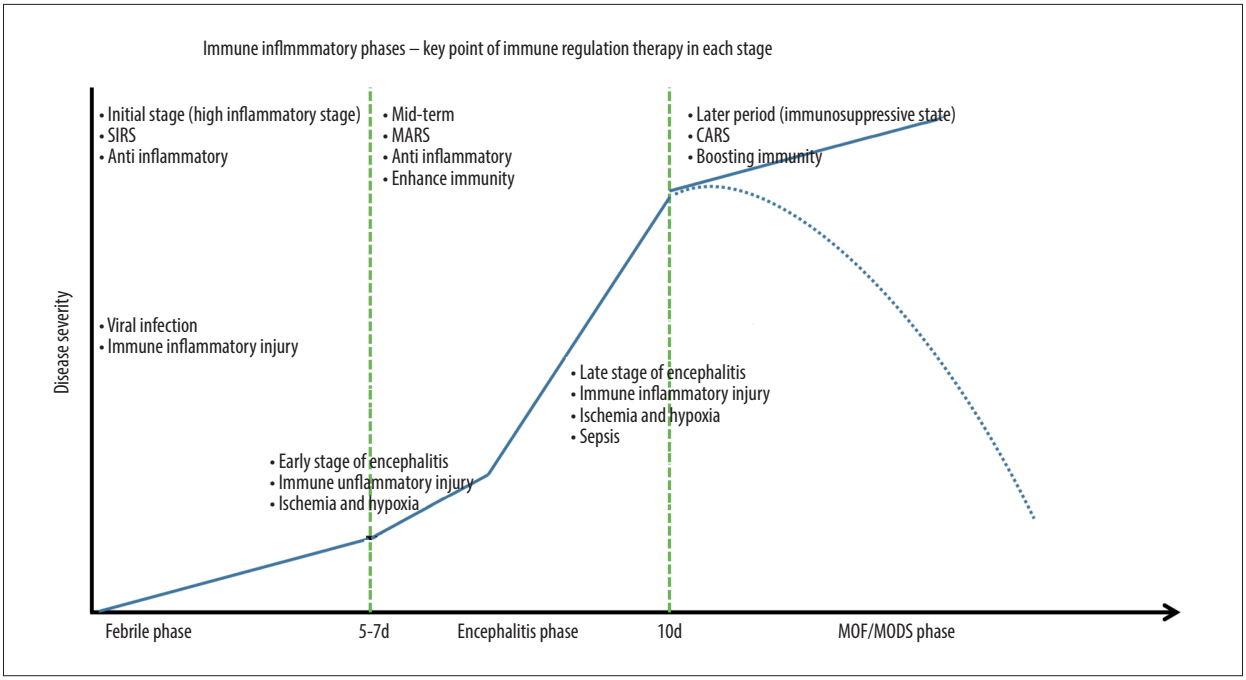

Figure 4. The staging of inflammatory storm encephalitis/encephalopathy, its immune inflammatory phase, and the key points of immune regulation treatment in each stage. Storm encephalitis/encephalopathy can be divided into 3 stages: fever stage, encephalitis stage, and MODS stage. The fever period was generally within 1 week, and after 5-7 days, some patients may experience it at around 2-10 weeks, which is the encephalitis period. Once the condition gets out of control, serious complications can occur. During the immune inflammation phase, immune regulation therapy has several stages. In the early stage, it was in a state of high inflammation, requiring anti-inflammatory therapy. In the later stage, it was a problem of secondary infection caused by immune suppression, and it seemed more important to enhance immunity during this period. In the mid-term, it was a mixed process of SIRS and CARS, known as mixed antagonistic response syndrome (MARS), which requires both anti-inflammatory and immune enhancement.

the organ failure due to infections phase. The fever phase lasts about 1 week, followed by encephalopathy/encephalitis, which may extend to 2 weeks in some patients. Severe complications can arise if the illness worsens, including septic and non-septic shock [20,21]. Early stages show heightened inflammation requiring anti-inflammatory treatment, while later stages involve immune suppression and secondary infections, making immunity enhancement vital [22]. The transitional phase exhibits a mix of systemic inflammatory response syndrome (SIRS) and compensatory anti-inflammatory response syndrome (CARS), requiring both anti-inflammatory and immune-boosting therapies, along with preventive anti-infective measures [23,24], as shown in **Figure 4**. Our patient developed rapid clinical deterioration on day 5 of illness onset, manifesting as sepsis, altered mental status, and acute respiratory failure. These complications aligned with characteristic neuroinflammatory pathophysiology of SFTSAE, yet demonstrated an unusually rapid progression exceeding typical disease trajectories.

Inflammatory storm encephalitis/encephalopathy, marked by high mortality [25], requires early aggressive intervention: (1) Anti-inflammatory therapy (eg, methylprednisolone pulse) to counteract viral-induced systemic inflammatory response syndrome (SIRS) and direct viral damage [26]; (2) Severe dehydration management and anti-infection strategies to prevent rapid progression to multiorgan dysfunction; and (3) Blood purification or renal replacement therapy to remove inflammatory mediators [27-29]. Critical goals include rapid SIRS suppression, organ protection, and preventing downstream injury to improve outcomes [26]. In patients with excessive fluid overload and capillary leakage (regardless of encephalitis diagnosis), evidence-based interventions focus on achieving negative fluid balance to mitigate organ edema (especially cerebral/pulmonary) and reduce cardiac strain [30]. This requires dual support: pharmacological (diuretics) and mechanical (continuous renal replacement therapy (CRRT)/blood purification) to maintain organ "dryness" [31]. Concurrently, early anti-infection therapy is critical due to high risks of immune exhaustion-induced bacterial/fungal infections, with vigilance for secondary viral infections that exacerbate immune dysregulation [32,33].

Critically ill patients with fever and thrombocytopenia require heightened vigilance for Aspergillus infection, particularly those with tick-bite exposure, as Aspergillus is a tick-borne pathogen and its onset often precedes bacterial infections [34]. Epidemiological data reveal over 50% of such patients develop

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invasive pulmonary aspergillosis (IPA), linked to high mortality, driven not only by immunosuppression (eg, steroid/antibiotic overuse) but also inherent disease susceptibility [35]. Management demands early broad-spectrum anti-infective strategies: (1) priority antifungal therapy (targeting Aspergillus); and (2) empirical antibacterial coverage for nosocomial pathogens (eg, Enterobacteriaceae, non-fermenting bacteria), especially drug-resistant strains in ICU settings [36]. The dual focus on fungal and bacterial threats is critical to mitigate mortality in this high-risk cohort. In this case, following confirmation of novel bunyavirus infection, the patient received prompt antiviral therapy with favipiravir alongside empirical doxycycline coverage for potential tick-borne co-pathogens (spirochetes and Rickettsia spp.). Upon detection of fungal co-infection, targeted antifungal therapy with voriconazole was initiated. Concurrent antimicrobial management was escalated to carbapenems based on progressive inflammatory markers, ensuring comprehensive anti-infective intervention aligned with evolving microbiological evidence.

Conclusions

This study has notable limitations: despite aggressive resuscitation measures failing to alter the fatal outcome, potential therapeutic gaps in SFTS management require critical evaluation. The absence of longitudinal cytokine profiling (eg, IL-6, IFN-γ) or immunoregulatory cell monitoring (eg, Treg dynamics) limits insights into optimal therapeutic windows. This fatal case underscores the fulminant progression of tick-borne SFTS and highlights the imperative for risk-stratified protocols targeting neurological complications.

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Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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