Published Online: 2025 November 9

**Case Report**

# Management of Severe Fever with Thrombocytopenia Syndrome-Associated Encephalopathy with Mixed Pulmonary Mold Infections: A Case Report

Chenyi Wang [1], Lian Tan [1], Qi Zhang [1], Yao Yuan [1], Lishen Liu [1], Zhengfeng Wen [1], Yahong Qu [1], Xiongxiong Wang [1], Zhongliang Ye [1], Shibo Li [2], Dongjun Hu [1,*]

[1] Yinzhou NO.2 Hospital, Ningbo, China
[2] Zhoushan Hospital, Wenzhou Medical University, Zhejiang, China

[*] ***Corresponding Author:*** Yinzhou NO.2 Hospital, Ningbo, China. Email: dongjunhu40612@163.com

**Received:** 30 September, 2025; **Revised:** 2 November, 2025; **Accepted:** 2 November, 2025

## Abstract

**Introduction:** Severe fever with thrombocytopenia syndrome (SFTS) is an emerging infectious disease of global concern. This case report describes a patient with SFTS-associated encephalopathy complicated by mixed pulmonary infections with *Aspergillus fumigatus* and *Rhizopus microsporus* in Ningbo, China — a combination that has not been previously reported.

**Case Presentation:** A forty-five-year-old man with confirmed severe fever with thrombocytopenia syndrome virus (SFTSV) infection developed pulmonary co-infection by *Aspergillus* and *Rhizopus*, complicated by massive hemoptysis. He received combined antifungal therapy with liposomal amphotericin B and isavuconazole, as well as bronchoscopy and bronchial artery embolization (BAE). Due to recurrent hemoptysis, a left lower lobectomy was performed, resulting in complete clinical and radiological recovery.

**Conclusions:** This case highlights the importance of early recognition and dynamic reassessment of fungal infections in SFTS patients. Prompt initiation of broad-spectrum antifungal therapy and timely surgical intervention are key to improving survival. Nevertheless, as a single case, the findings should be interpreted cautiously; further studies are needed to determine whether similar dual fungal infections occur more widely in SFTS patients or under specific immunosuppressive conditions.

*Keywords:* Severe Fever with Thrombocytopenia Syndrome, SFTS, Mixed Mold Infection, *Aspergillus*, *Rhizopus microsporus*, Liposomal Amphotericin B

## 1. Introduction

Severe fever with thrombocytopenia syndrome (SFTS) is currently classified within the *Phenuiviridae* family, *Bandavirus* genus, and possesses a tripartite genome comprising L, M, and S segments. It is a spherical, enveloped, and segmented negative-strand RNA virus (1). The incubation period of severe fever with thrombocytopenia syndrome virus (SFTSV) is generally five to fourteen days, and transmission occurs primarily through arthropods such as ticks, with a reported mortality rate of up to 30% (2). Patients with SFTSV infection accompanied by central nervous system involvement often experience rapid symptom progression, including headaches, nausea, vomiting, limb tremors, and disturbances of cognition and consciousness. Studies indicate that SFTSV infection increases the risk of opportunistic fungal infections, particularly *Aspergillus*, which can lead to multi-organ system failure and higher mortality rates (3).

Among opportunistic fungi, both *Aspergillus* and *Rhizopus* are notable for causing extensive vascular invasion, resulting in vascular thrombosis and significant tissue infarction. Pulmonary infections caused by *Rhizopus* are frequently associated with respiratory distress and hemoptysis, with a mortality rate of 70% to 100% if left untreated (4).



**How to Cite:** Wang C, Tan L, Zhang Q, Yuan Y, Liu L, et al. Management of Severe Fever with Thrombocytopenia Syndrome-Associated Encephalopathy with Mixed Pulmonary Mold Infections: A Case Report. Jundishapur J Microbiol. 2025; 18 (11): e166706. https://doi.org/10.5812/jjm-166706.

Here we report a rare case of co-infection with SFTSV and mold fungi in eastern China. To the best of our knowledge, no previous reports have documented simultaneous pulmonary infections caused by both *Aspergillus* and *Rhizopus* in the context of SFTS. Therefore, this case represents the first documented instance of dual fungal co-infection associated with SFTS, highlighting a novel and clinically important manifestation of this emerging viral disease. Based on the detection results of metagenomic next-generation sequencing (mNGS), we rapidly identified pathogenic evidence, thereby facilitating prompt and precise treatment for the patient.

## 2. Case Presentation

The patient consented to research authorization for record review, and the study received approval from the institutional review board. A forty-five-year-old male, resident of Xiangshan County, Zhejiang province — a hilly region in southeast China — was bitten by ticks on the inner side of his right ankle while mowing grass on April 6, 2024. He developed generalized malaise, including chills, fever, and diarrhea, and presented to a local hospital on April 18. He was initially prescribed levofloxacin and returned home. On April 23, due to worsening symptoms, he was admitted to the Infection Department of Ningbo Yinzhou No. 2 Hospital.

The patient was previously healthy, with no history of hypertension, diabetes, or hematologic/oncologic disease. Upon admission, routine microbiological and virological examinations were performed. Results for common respiratory pathogens, including influenza viruses A and B, parainfluenza virus, respiratory syncytial virus, adenovirus, cytomegalovirus, Epstein-Barr virus, and bacteria such as *Mycobacterium tuberculosis*, were all negative. Blood and sputum cultures showed no evidence of bacterial or fungal growth, and serologic tests for parasites were negative.

On admission, the patient was alert with a body temperature of 37.9°C (ear temperature), heart rate of 80 beats per minute, respiratory rate of eighteen breaths per minute, blood pressure of 117/66 mmHg, and oxygen saturation ($SpO^2$) of 99% on room air. A dark red, round papular scab was observed on the medial aspect of the right ankle (Figure 1). The remainder of the skin was intact, and the neurologic examination was unremarkable.

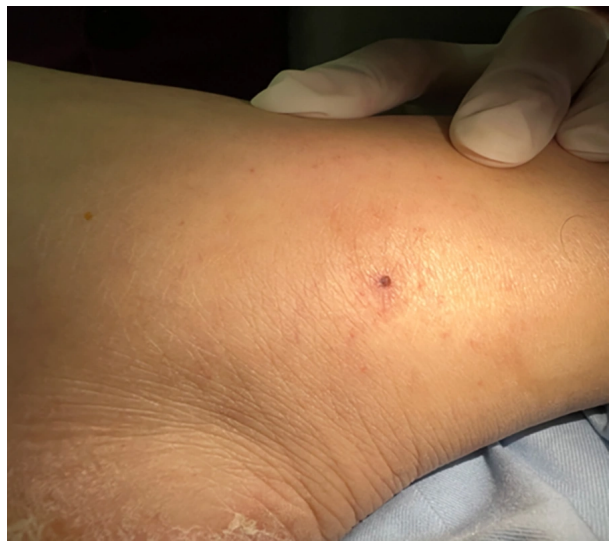

**Figure 1.** Bite mark on the medial ankle

Initial laboratory studies revealed leukocytopenia (white blood cell count: 2,700 cells/$mm^3$), thrombocytopenia (platelet count: 82,000 cells/$mm^3$), elevated C-reactive protein (CRP) level (11.2 mg/L), elevated aspartate aminotransferase (AST) level (407 U/L), elevated alanine aminotransferase (ALT) level (136 U/L), elevated creatine kinase (CK) level (14,897 U/L), elevated lactate dehydrogenase (LDH) level (1,168 U/L), acute renal insufficiency (serum creatinine: 192 µmol/L), elevated cardiac troponin I (0.072 ng/mL), and significantly increased ferritin (> 1,500 ng/mL). Results for the Widal test, cytomegalovirus-DNA, Epstein-Barr virus-DNA, *Mycoplasma pneumoniae*-DNA, influenza A-RNA, influenza B-RNA, respiratory syncytial virus-RNA, rhinovirus-RNA, adenovirus-DNA, and COVID-19-RNA were negative. Hemorrhagic fever with renal syndrome antibodies and dengue fever antibodies were also negative. No *Plasmodium* was found. Chest computed tomography (CT) revealed flocculent high-density shadows in both lungs (Figure 2). Electrocardiogram and echocardiographic findings were normal.

On April 25, the patient developed hand tremor, followed by a generalized tonic-clonic seizure and trismus. He was transferred to the intensive care unit (ICU) after endotracheal intubation. Due to these clinical manifestations, serum samples were sent to the Infectious Disease Diagnosis Laboratory of Ningbo City Center for Disease Control and Prevention for SFTSV analysis. Polymerase chain reaction (PCR) testing of

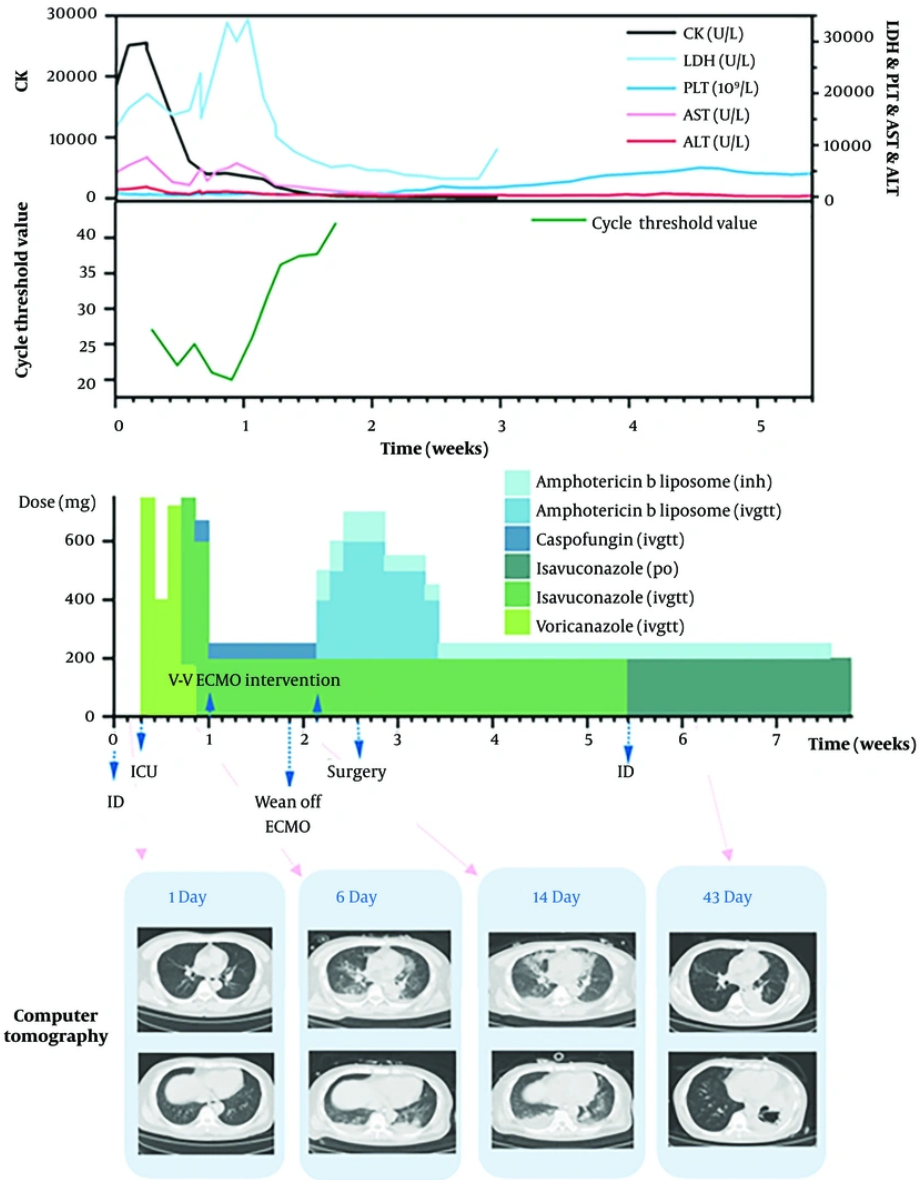


**Figure 2.** Antifungal treatment regimen administered during the patient's intensive care unit (ICU) admission

cerebrospinal fluid (CSF) for SFTSV was weakly positive, and serum reverse transcription Polymerase chain reaction (RT-PCR) for SFTSV was positive, with a cycle threshold (Ct) value of 27 (Figure 2). The CSF examination revealed CSF pressure of 18 cm $H_2O$, leukocyte count of 0/$mm^3$, erythrocyte count of 0/$mm^3$, protein concentration of 351 mg/L, LDH of 15 U/L, and glucose concentration of 4.82 mmol/L. Serum mNGS detected 3,244 RNA sequence reads with a relative abundance of 58.61% corresponding to SFTSV. The patient was thus diagnosed with SFTSV-associated encephalopathy.

Management included antiepileptic drugs, methylprednisolone (40 mg/day for three days),

immunoglobulin (400 mg/kg/day for six days, totaling 180 g) to modulate the immune response, favipiravir as antiviral therapy, and doxycycline to address potential co-infection (given the possibility of *Rickettsia* and *Borrelia burgdorferi* sensu lato from ticks) (5). Additionally, three sessions of therapeutic plasma exchange were performed. Platelets, LDH, CK, Ct values, AST, and ALT were monitored throughout the course and are depicted as curves in Figure 2. Notably, LDH, AST, and ALT levels fluctuated on April 25, 26, and 27, coinciding with plasma exchange procedures.

On day three of admission, fiberoptic bronchoscopy revealed multiple white patches in the main bronchus, congested and edematous bronchial mucosa, and a small amount of adherent white mucous sputum. Sputum culture smear showed septate hyphae, suggestive of *Aspergillus*. The patient was started on voriconazole antifungal therapy.

On day seven of admission, the patient's oxygenation and circulation worsened and septic shock was suspected, accompanied by further respiratory decline. Veno-venous extracorporeal membrane oxygenation (V-V ECMO) was initiated. The same day, mNGS from serum and bronchoalveolar lavage fluid (BALF) confirmed *Aspergillus fumigatus* infection. The antifungal regimen was adjusted to caspofungin in combination with isavuconazole. After six days, the patient was successfully weaned from V-V ECMO, with a decline in plasma viral load to undetectable levels (Figure 2).

Following modification of the antifungal regimen, the patient's respiratory status initially improved, with reduced oxygen requirements and gradual resolution of pulmonary infiltrates on follow-up CT performed on May 2. Serum inflammatory markers, including CRP and procalcitonin, declined, and both platelet and leukocyte counts normalized. However, around May 7, clinical deterioration recurred with renewed fever (38.5°C), increased oxygen demand, and rising CRP and LDH levels. Repeat imaging revealed new consolidations and cavitation in the left lower lobe, inconsistent with isolated aspergillosis. This prompted re-evaluation of the antifungal strategy and repeat mNGS, which subsequently identified *Rhizopus microsporus*.

Bronchoscopy was repeated, and on May 8, mNGS of BALF identified both *A. fumigatus* and *R. microsporus* (sequence count: 4,151; relative abundance: 3.07%). Initial bronchoscopic specimens and smears had shown only septate hyphae consistent with *Aspergillus* species; direct microscopy and culture for *Mucorales* were not initially performed due to predominance of *Aspergillus* on mNGS and culture, and rapid clinical deterioration. After detection of *R. microsporus* by mNGS, repeated bronchoscopy and BALF culture targeting *Mucorales* were performed, resulting in isolation and confirmation of *R. microsporus*, consistent with mNGS findings.

Later that evening, the patient developed a dark bloody clot in the airway after choking. Bedside bronchoscopy showed active bleeding in the basal segment of the left lower lobe and cast thrombosis of the main bronchial airway. Bronchial angiography revealed hyperplasia and tortuosity of the left lower lung bronchial artery, with a few abnormal staining foci. After bronchoscopic hemostasis failed, bilateral bronchial artery embolization (BAE) was performed. Liposomal amphotericin B was administered at 3 mg/kg/day, increasing to 5 mg/kg/day (Figure 2). On May 11, emergency left lower lobectomy was performed for severe hemoptysis (Figure 1 in Supplementary File). Simultaneously, *R. microsporus* was detected in BALF culture (Figure 2 in Supplementary File), confirming previous mNGS results.

The patient's consciousness gradually returned to normal, and a head MRI scan was completed (Figure 3 in Supplementary File). He was subsequently transferred to the Infection Unit for rehabilitation. The patient received five weeks of amphotericin B liposome followed by oral isavuconazole (200 mg once daily) until complete radiological resolution was achieved on follow-up chest CT. The clinical progression and treatments are summarized in Figure 2. Surgical pathology revealed two types of fungal hyphae: Narrow, septate, acute-angle branching hyphae consistent with Aspergillus species, and broad, ribbon-like, sparsely septate hyphae with right-angle branching characteristic of Rhizopus species. These features confirmed coexistence of Aspergillus and Mucorales infection, as annotated in Figure 4 in Supplementary File.

On day ten of admission, despite combined antifungal therapy targeting *Aspergillus* (caspofungin plus isavuconazole), fever persisted and oxygenation worsened. Follow-up CT showed progressive left lower lobe consolidation and new cavitation inconsistent with typical invasive aspergillosis alone. Laboratory assessment revealed rising serum β-D-glucan and persistently elevated inflammatory markers, while galactomannan did not further increase. These atypical findings, in combination with worsening hemoptysis and lack of improvement on anti-*Aspergillus* therapy, raised suspicion for mucormycosis. Consequently,

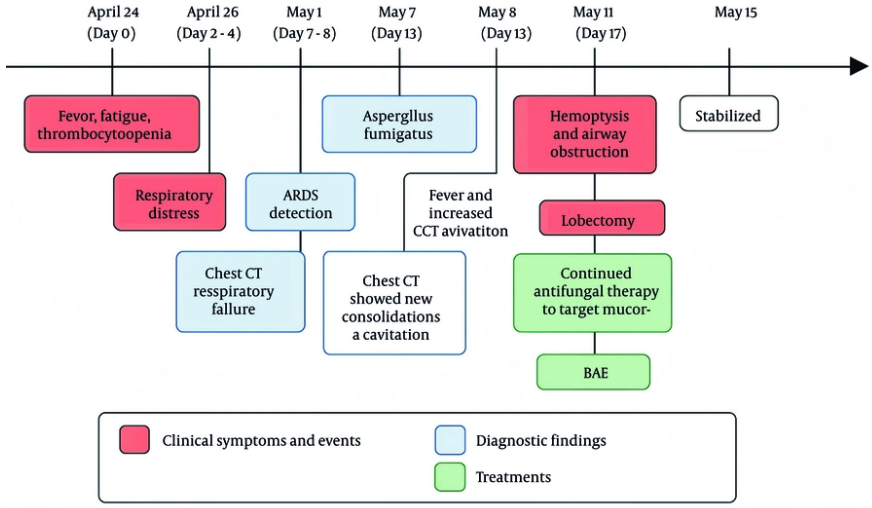


**Figure 3.** Timeline summarizing the patient's clinical course, diagnostic findings, and treatments – the figure illustrates the sequence of key clinical events, including the onset of severe fever with thrombocytopenia syndrome (SFTS), respiratory deterioration requiring extracorporeal membrane oxygenation (ECMO), detection of *Aspergillus fumigatus* and *Rhizopus microsporus*, antifungal therapy adjustments, and surgical interventions [bronchial artery embolization (BAE) and lobectomy].

repeat mNGS of BALF confirmed *R. microsporus* sequences. A detailed timeline of the patient's clinical course, diagnostics, and interventions is presented in Figure 3.

## 3. Discussion

This case highlights the diagnostic and therapeutic complexity of managing SFTS-associated encephalopathy complicated by dual pulmonary fungal infections. The patient presented with progressive respiratory failure and septic shock, despite standard antiviral and antibacterial therapy. Early bronchoscopy identified septate hyphae, leading to a diagnosis of invasive aspergillosis and initiation of targeted antifungal therapy (6). One of the main clinical challenges was the atypical disease progression despite adequate anti-*Aspergillus* therapy. Although the patient initially improved, with decreasing oxygen requirements and inflammatory markers, he soon deteriorated, developing new pulmonary cavities and hemoptysis. These findings prompted reconsideration of the original diagnosis and repeat mNGS testing, which revealed *R. microsporus* co-infection. This diagnostic turnaround underscores the importance of maintaining suspicion for mucormycosis in immunocompromised or critically ill patients, even when another fungal pathogen has already been identified (7, 8).

Previous reports from China and South Korea have primarily described SFTS patients with single fungal infections, such as invasive aspergillosis or candidiasis, but not dual mold co-infections (9-12). In those cases, the fungal infection typically occurred during the late phase of SFTS, often associated with severe immunosuppression or corticosteroid exposure. In contrast, our case demonstrates that simultaneous infection with both *Aspergillus* and *Rhizopus* can occur early in the disease course, emphasizing the need for early fungal surveillance and broad diagnostic testing in critically ill SFTS patients.

Another major challenge involved balancing aggressive antifungal treatment with the patient's hepatic and renal dysfunction secondary to SFTS. Initial antifungal therapy with voriconazole was effective against *Aspergillus* but provided limited coverage for *Mucorales*. Therefore, the regimen was switched to liposomal amphotericin B and isavuconazole to broaden antifungal coverage and ensure activity against both *Aspergillus* and *Rhizopus* species, while minimizing toxicity, consistent with current treatment guidelines (13). Therapeutic drug monitoring guided dosing adjustments.

The patient also experienced life-threatening hemoptysis, necessitating timely multidisciplinary intervention — including BAE and eventual left lower lobectomy (14). These steps were crucial for achieving hemostasis and source control. In this case, BAE was performed immediately after failure of bronchoscopic hemostasis, when active bleeding from the left lower lobe resulted in airway obstruction and oxygen desaturation. Although temporary hemostasis was achieved, recurrent massive hemoptysis occurred three days later, and emergency lobectomy was performed. The timing of surgery was based on persistent bleeding, localized lesions observed on imaging, and stabilization of vital signs following antifungal therapy and supportive care. Early surgical intervention after initial hemostatic control, once systemic condition permits, is critical to preventing fatal airway obstruction and achieving complete removal of necrotic foci colonized by *Mucorales*. This experience suggests that prompt but well-timed surgery, guided by clinical stability and lesion localization, can substantially improve outcomes in patients with invasive fungal co-infection.

Similarly, recent multicenter evidence demonstrates that early, multidisciplinary intervention significantly improves survival among patients with invasive mucormycosis. For example, a large retrospective study from Iran involving ninety-seven patients with rhino-orbito-cerebral mucormycosis reported that timely combined antifungal therapy and surgical management markedly reduced mortality rates (15). These findings further support our clinical observation that prompt diagnosis and well-timed surgical resection are critical to improving outcomes in patients with mixed mold infections.

Several risk factors may have contributed to the development of dual fungal infection in this case. The SFTS-related immune dysregulation and corticosteroid therapy can suppress host immunity and facilitate fungal proliferation (16). Specifically, SFTSV infection induces marked endothelial injury and a cytokine storm characterized by elevated levels of interleukin 6, tumor necrosis factor alpha, and interferon gamma, which compromise vascular integrity and tissue oxygenation. Increased vascular permeability and endothelial apoptosis not only contribute to multi-organ failure but also create a microenvironment favorable for fungal invasion. Furthermore, SFTS can transiently increase blood–brain barrier permeability and disrupt innate immune cell function — including neutrophil chemotaxis and macrophage phagocytic activity — thereby facilitating deeper tissue invasion by both viral and fungal pathogens. In addition, iron overload and previous exposure to voriconazole may predispose to mucormycosis by promoting fungal resistance (17-19).

The key lessons from this case include: (1) Early mNGS testing can rapidly detect mixed fungal infections that conventional microscopy and culture may miss; (2) dynamic reassessment of antifungal efficacy is essential when clinical deterioration occurs; and (3) combined medical and surgical management significantly improves outcomes in invasive fungal co-infections. In summary, this case demonstrates that prompt recognition of mixed mold infections and individualized, multidisciplinary management are critical to improving survival in patients with SFTS complicated by invasive fungal disease.

## Supplementary Material

Supplementary material(s) is available here [To read supplementary materials, please refer to the journal website and open PDF/HTML].

## Footnotes

**Authors' Contribution:** Study concept and design: Ch. W. and D. H.; Acquisition of data: Ch. W., L. T., Y. Y., L. L., Zh. W., X. W., Zh. Y., and D. H.; Analysis and interpretation of data: Q. Zh., Y. Q., and Sh. L.; Drafting of the manuscript: Ch. W. and D. H.; Critical revision of the manuscript for important intellectual content: Sh. L. and D. H.; Statistical analysis: Ch. W. and L. T.; Administrative, technical, and material support: D. H.; Study supervision: D. H.

**Conflict of Interests Statement:** The authors declare no conflict of interest.

**Data Availability:** The individual participant data can be requested and accessed by contacting the study management teams of the studies on reasonable request.

**Ethical Approval:** Approval for the study was obtained from the Ethics Committee of Ningbo Yinzhou No. 2 Hospital (No. 2024-008) and the research was conducted in accordance with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards..

**Funding/Support:** This project was funded by Zhejiang Provincial Natural Science Foundation (No. LY21H100002).

**Informed Consent:** Written informed consent was obtained from the participants.

## References


1. Li J, Li S, Yang L, Cao P, Lu J. Severe fever with thrombocytopenia syndrome virus: a highly lethal bunyavirus. *Crit Rev Microbiol*. 2021;**47**(1):112-25. [PubMed ID: 33245676]. https://doi.org/10.1080/1040841X.2020.1847037.
2. Wang L, Wan G, Shen Y, Zhao Z, Lin L, Zhang W, et al. A nomogram to predict mortality in patients with severe fever with thrombocytopenia syndrome at the early stage-A multicenter study in China. *PLoS Negl Trop Dis*. 2019;**13**(11). e0007829. [PubMed ID: 31765414]. [PubMed Central ID: PMC6934327]. https://doi.org/10.1371/journal.pntd.0007829.
3. Xu Y, Shao M, Liu N, Tang J, Gu Q, Dong D. Invasive pulmonary aspergillosis is a frequent complication in patients with severe fever with thrombocytopenia syndrome: A retrospective study. *Int J Infect Dis*. 2021;**105**:646-52. https://doi.org/10.1016/j.ijid.2021.02.088.
4. Larche J, Machouart M, Burton K, Collomb J, Biava MF, Gerard A, et al. Diagnosis of cutaneous mucormycosis due to Rhizopus microsporus by an innovative PCR-restriction fragment-length polymorphism method. *Clin Infect Dis*. 2005;**41**(9):1362-5. [PubMed ID: 16206119]. https://doi.org/10.1086/497078.
5. Zhao B, Hou H, Gao R, Tian B, Deng B. Mononucleosis-like illnesses due to co-infection with severe fever with thrombocytopenia syndrome virus and spotted fever group rickettsia:a case report. *BMC Infect Dis*. 2021;**21**(1). https://doi.org/10.1186/s12879-021-06434-8.
6. Zhang Y, Huang Y, Xu Y. Associated microbiota and treatment of severe fever with thrombocytopenia syndrome complicated with infections. *J Med Virol*. 2022;**94**(12):5916-21. [PubMed ID: 35945160]. https://doi.org/10.1002/jmv.28059.
7. Cui N, Liu R, Lu QB, Wang LY, Qin SL, Yang ZD, et al. Severe fever with thrombocytopenia syndrome bunyavirus-related human encephalitis. *J Infect*. 2015;**70**(1):52-9. [PubMed ID: 25135231]. https://doi.org/10.1016/j.jinf.2014.08.001.
8. Kaneko M, Shikata H, Matsukage S, Maruta M, Shinomiya H, Suzuki T, et al. A patient with severe fever with thrombocytopenia syndrome and hemophagocytic lymphohistiocytosis-associated involvement of the central nervous system. *J Infect Chemo*. 2018;**24**(4):292-7. https://doi.org/10.1016/j.jiac.2017.10.016.
9. Song L, Zhao Y, Wang G, Zou W, Sai L. Investigation of predictors for invasive pulmonary aspergillosis in patients with severe fever with thrombocytopenia syndrome. *Sci Rep*. 2023;**13**(1):1538. [PubMed ID: 36707667]. [PubMed Central ID: PMC9883384]. https://doi.org/10.1038/s41598-023-28851-2.
10. Zhao Y, Wu X, Wang X, Li L. Severe fever with thrombocytopenia syndrome complicated with aspergillus endocarditis and multiple organ infarctions after glucocorticoid treatment in an immunocompetent man: a case report. *BMC Infect Dis*. 2025;**25**(1). https://doi.org/10.1186/s12879-025-10503-7.
11. Hu L, Kong Q, Yue C, Xu X, Xia L, Bian T, et al. Early-warning immune predictors for invasive pulmonary aspergillosis in severe patients with severe fever with thrombocytopenia syndrome. *Front Immunol*. 2021;**12**:576640. [PubMed ID: 34025635]. [PubMed Central ID: PMC8138034]. https://doi.org/10.3389/fimmu.2021.576640.
12. Kim UJ, Kim DM, Kim SE, Kang SJ, Jang HC, Park KH, et al. Case report: detection of the identical virus in a patient presenting with severe fever with thrombocytopenia syndrome encephalopathy and the tick that bit her. *BMC Infect Dis*. 2018;**18**(1). https://doi.org/10.1186/s12879-018-3092-y.
13. Cornely OA, Alastruey-Izquierdo A, Arenz D, Chen SCA, Dannaoui E, Hochhegger B, et al. Global guideline for the diagnosis and management of mucormycosis: an initiative of the European confederation of medical mycology in cooperation with the mycoses study group education and research consortium. *Lancet Infect Dis*. 2019;**19**(12):e405-21. https://doi.org/10.1016/s1473-3099(19)30312-3.
14. Chretien ML, Legouge C, Pagès PB, Lafon I, Ferrant E, Plocque A, et al. Emergency and elective pulmonary surgical resection in haematological patients with invasive fungal infections: a report of 50 cases in a single centre. *Clin Microbiol Infect*. 2016;**22**(9):782-7. https://doi.org/10.1016/j.cmi.2015.12.029.
15. Arian M, Aeen S, Bojdi A, Jabbari Nooghabi M, Tajik A, Baghoveh M, et al. Improving the outcome of patients diagnosed with rhino-orbito-cerebral mucormycosis in the last two decades: 97 patients in two referral centers in the northeast of iran. *Arch Clin Infect Dis*. 2024;**19**(5). https://doi.org/10.5812/archcid-151510.
16. Nakamura S, Azuma M, Maruhashi T, Sogabe K, Sumitani R, Uemura M, et al. Steroid pulse therapy in patients with encephalopathy associated with severe fever with thrombocytopenia syndrome. *J Infect Chemo*. 2018;**24**(5):389-92. https://doi.org/10.1016/j.jiac.2017.11.004.
17. Dadwal SS, Tegtmeier B, Liu X, Frankel P, Ito J, Forman SJ, et al. Impact of pretransplant serum ferritin level on risk of invasive mold infection after allogeneic hematopoietic stem cell transplantation. *Europ J Haematol*. 2014;**94**(3):235-42. https://doi.org/10.1111/ejh.12421.
18. Pongas GN, Lewis RE, Samonis G, Kontoyiannis DP. Voriconazole-associated zygomycosis: a significant consequence of evolving antifungal prophylaxis and immunosuppression practices? *Clin Microbiol Infect*. 2009;**15**:93-7. https://doi.org/10.1111/j.1469-0691.2009.02988.x.
19. Lin E, Moua T, Limper AH. Pulmonary mucormycosis: clinical features and outcomes. *Infect*. 2017;**45**(4):443-8. https://doi.org/10.1007/s15010-017-0991-6.