BMC Infectious Diseases

CASE REPORT

Open Access

# Early plasma exchange for severe fever with thrombocytopenia syndrome (SFTS)-associated encephalopathy and multi-organ dysfunction syndrome (MODS): a case report

Xuebin Wen[1†], Ran Ji[2†] and Keqi Dong[1*]

## Abstract

**Background** Severe fever with thrombocytopenia syndrome (SFTS) is a tick-borne viral disease caused by Dabie bandavirus, associated with high morbidity and mortality. Neurological complications such as encephalopathy are increasingly recognized and are linked to poor prognosis. No specific antiviral treatment is available, and management is mainly supportive.

**Case presentation** We report a rare case of SFTS in a 72-year-old woman who developed acute encephalopathy and multi-organ dysfunction shortly after a wasp sting. The patient presented with thrombocytopenia, elevated liver enzymes, and central nervous system involvement, including seizures and coma. Next-generation sequencing and serological testing confirmed SFTS. Despite standard antiviral and supportive therapies, rapid deterioration prompted therapeutic plasma exchange (TPE) initiation. After two sessions of TPE, the patient showed significant clinical improvement, including recovery of consciousness, normalization of laboratory markers, and resolution of neurological symptoms.

**Conclusion** This case highlights the potential utility of early TPE in managing fulminant SFTS with central nervous system involvement. TPE may attenuate cytokine-mediated inflammation and improve outcomes in patients with severe Bunyavirus-associated encephalopathy. Further studies are warranted to standardize its use in this context.

**Clinical trial** Not applicable.

**Keywords** Severe fever with thrombocytopenia syndrome, Plasma exchange, Sepsis-associated encephalopathy, Multi-organ dysfunction

†Xuebin Wen and Ran Ji contributed equally to this work.

*Correspondence:
Keqi Dong
dongkeqi2008@163.com
[1]Intensive Care Unit, Zhoushan Hospital, Zhoushan, China
[2]Intensive Care Unit, The Second Affiliated Hospital of Zhejiang University, School of Medicine, Hangzhou, China

## Introduction

Severe fever with SFTS is an emerging tick-borne infectious disease caused by the Dabie bandavirus (formerly SFTS virus, SFTSV), a member of the Bandavirus genus in the Phenuiviridae family [1]. First identified in China in 2010, SFTS has since been reported across East Asia, with a case fatality rate ranging from 6% to 30%, depending on disease severity and comorbidities [2]. The disease typically presents with fever, thrombocytopenia, leukopenia, and gastrointestinal symptoms, but severe cases may progress to multi-organ dysfunction syndrome (MODS), including acute encephalopathy, respiratory failure, and circulatory collapse [3] .

Neurological complications, though increasingly recognized, remain poorly understood. Recent studies suggest that 10–30% of SFTS patients develop central nervous system (CNS) manifestations, including seizures, altered mental status, and coma, which are associated with higher mortality [4, 5]. The pathogenesis of SFTS-associated encephalopathy is multifactorial, involving direct viral neuroinvasion, cytokine-mediated neuroinflammation, endothelial injury, and metabolic disturbances. Despite advances in understanding the disease, no specific antiviral therapy exists, and management remains largely supportive.

Therapeutic plasma exchange (TPE) has emerged as a potential adjunctive treatment for severe SFTS, particularly in cases with hyperinflammation and neurological involvement. By removing circulating inflammatory mediators and viral particles, TPE may mitigate cytokine storm and improve outcomes [6, 7]. However, clinical evidence remains limited, and optimal treatment protocols are yet to be established.

Here, we present a rare case of SFTS following a wasp sting, complicated by rapid-onset encephalopathy and multi-organ failure. The patient exhibited dramatic clinical improvement following early initiation of TPE, suggesting its potential role in managing severe SFTS with CNS involvement. This case highlights the importance of early recognition and aggressive immunomodulatory therapy in improving outcomes for this life-threatening infection.

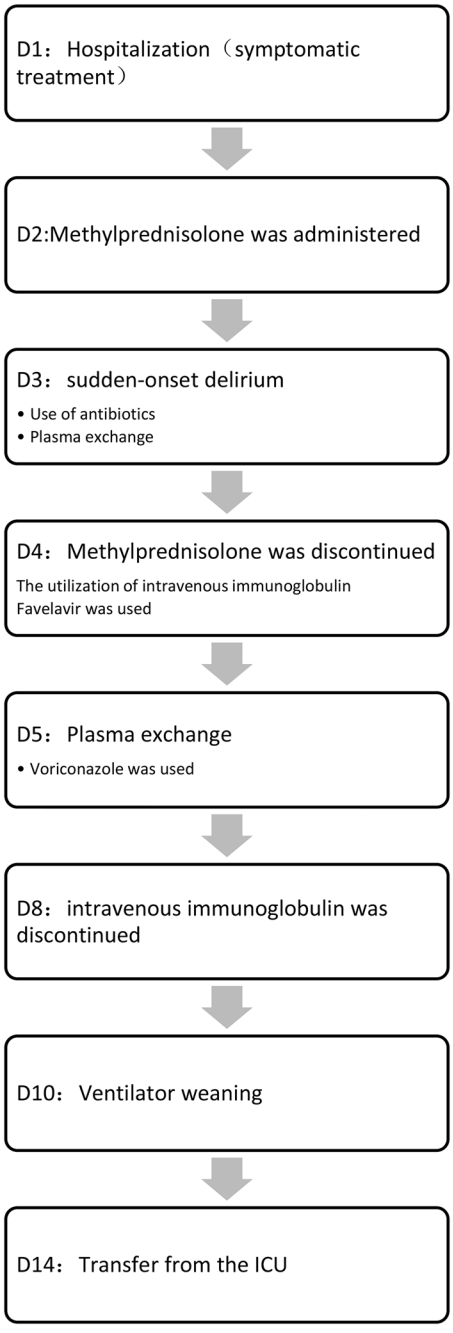



**Fig. 1** A timeline of the different administered treatment

## Case presentation

A 72-year-old female patient with a known history of duodenal malignancy presented to the hospital with complaints of dizziness, fatigue, nausea, and vomiting that had persisted for one day (Fig. 1). According to her family member, these symptoms developed shortly after she had been stung by wasps. Upon admission, the patient was alert and oriented.

The patient was admitted to the hematology department for supportive care, including anti-infective therapy, liver protection, and platelet-enhancing treatment. A bone marrow biopsy was performed and showed no abnormalities.

On the third day of hospitalization, the patient experienced sudden-onset delirium accompanied by limb convulsions and urinary/fecal incontinence. A cranial CT scan was negative. The patient was intubated for airway protection and transferred to the intensive care unit (ICU) for further treatments.

Upon ICU admission, her APACHE II score was 16, and SOFA score was 12, indicative of severe illness with multi-organ dysfunction (MODS), as follows:

1. **Neurological involvement**: The patient had a Glasgow Coma Scale (GCS) score of 5, exhibited limb convulsions and increased muscle tone. Ambulatory EEG demonstrated severe abnormalities, with dominant theta wave activity in the frontal region (Fig. 2).

2. **Respiratory system involvement**: The oxygenation index was 140 mmHg with a respiratory rate of 25 breaths per minute. Chest CT imaging showed bilateral atelectasis (Fig. 3).

3. **Hematological involvement**: Persistent thrombocytopenia with platelet count at $27 \times 10^9$/L. PCT 1.23ng/L, Blood cultures suggested human staphylococcal infection.
4. **Hepatic and renal dysfunction**: AST 890 U/L, ALT 226 U/L, γ-GT 133 U/L, and serum creatinine 104 μmol/L.
5. **Cardiovascular involvement**: The heart rate was 147 beats per minute.Troponin was elevated at 0.144 ng/mL, and myoglobin at 934 ng/mL. ECG findings included atrial flutter and ST-T abnormalities. Echocardiography showed diffuse hypokinesia of the left ventricular wall with impaired coordination and reduced ejection fraction.
6. **Muscle enzyme abnormalities**: CK 704 U/L and LDH 1027 U/L, indicating possible rhabdomyolysis or severe systemic inflammation.

In summary, following admission, the patient presented with a normal body temperature, a respiratory rate of 25 breaths per minute, and a heart rate of 147 beats per minute. exhibited persistent thrombocytopenia, transient leukopenia, and elevated procalcitonin (PCT) levels, which, in conjunction with blood culture results, strongly suggest an underlying infection. Concurrently, the patient developed neurological manifestations including altered mental status and epileptic seizures; respiratory abnormalities such as a decreased oxygenation index and atelectasis; hepatic and renal dysfunction; and cardiovascular complications characterized by reduced myocardial contractility, electrocardiographic changes, and elevated cardiac enzyme levels. Collectively, these clinical findings meet the criteria for multiple organ dysfunction syndrome (MODS).

Initial management included temporary mechanical ventilation, antibiotics, antiepileptic therapy, liver support, and other supportive care measures. Serological testing returned positive for Newbrynia virus antibody, and the patient was diagnosed with Severe Fever with Thrombocytopenia Syndrome (SFTS). Antiviral therapy was initiated with favipiravir in combination with ribavirin, along with ulinastatin for anti-inflammatory support. Immunomodulatory therapy included methylprednisolone (40 mg,ivgtt, qd, D3-D5) and intravenous immunoglobulin (IVIG) (15 g,ivgtt, qd, D4-D8). Plasma exchange was performed on the 3rd and 5th days of admission.

Following two sessions of plasma exchange, there was a marked improvement in liver enzyme levels (AST, ALT, LDH) and gradual recovery of platelet counts (Fig. 4). On day 5, the patient's blood samples underwent Next-Generation Sequencing (NGS) analysis, which detected RNA sequences of Severe Fever with SFTSV as well as DNA sequences of Aspergillus fumigatus, suggesting a possible co-infection involving both viral and fungal pathogens (Table 1), prompting the addition of voriconazole for antifungal therapy.

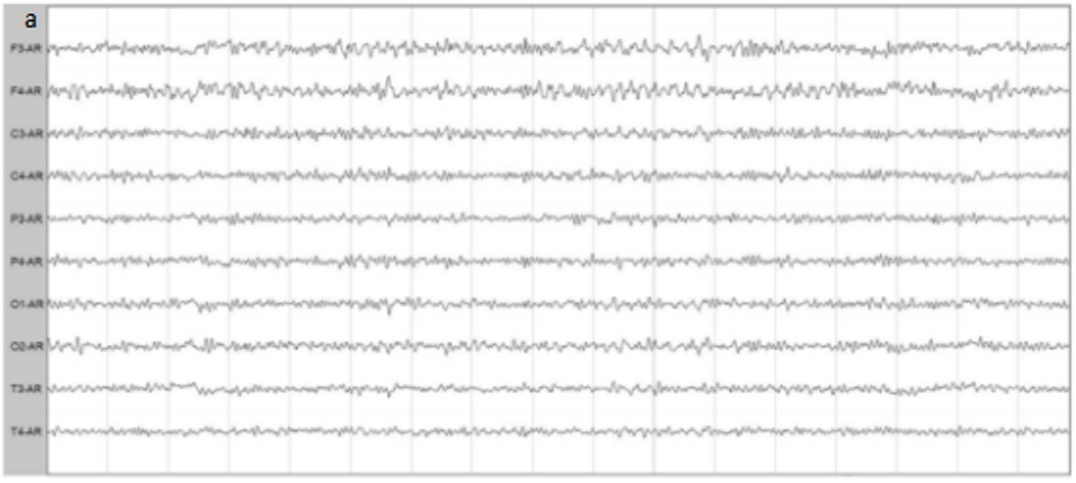
On day 9, a lumbar puncture was performed (Table 2), and cerebrospinal fluid analysis revealed no significant abnormalities. The patient's neurological status gradually improved. She regained full consciousness, was successfully weaned from the ventilator, and was extubated on day 12. Repeat chest CT imaging on day 12 showed near-complete resolution of bilateral pulmonary infiltrates (Fig. 3). Follow-up EEG on day 24 demonstrated significant improvement compared to baseline (Fig. 2).

The patient continued to improve clinically and was discharged on day 28. At discharge, she was fully oriented, able to care for herself independently, and had returned to her normal daily activities.

## Discussion

This case highlights a rare and severe manifestation of *Bunyavirus* infection—specifically Severe Fever with Thrombocytopenia Syndrome (SFTS)—involving both extensive multi-organ dysfunction and acute central nervous system (CNS) impairment. The patient developed a rapid progression of neurological symptoms, including delirium, seizures, and loss of consciousness, indicative of sepsis-associated encephalopathy (SAE). Remarkably, her condition improved significantly after the initiation of therapeutic plasma exchange (TPE), suggesting a potentially critical role for this modality in the management of fulminant SFTS.

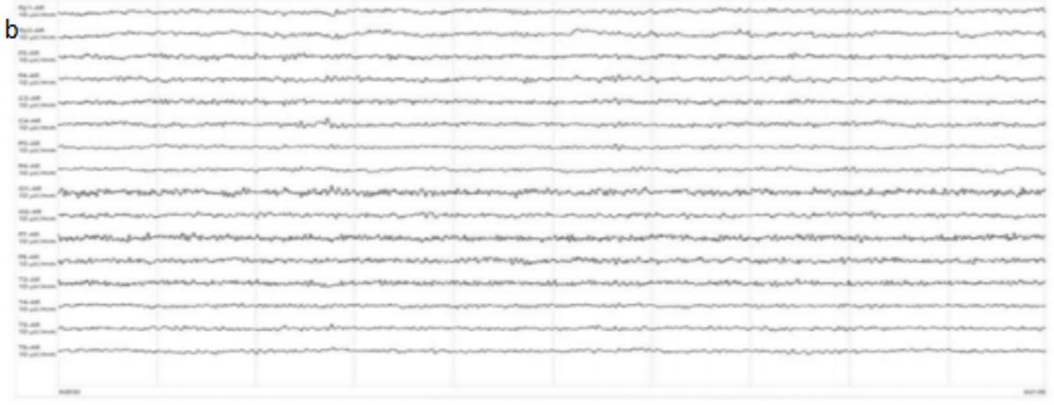
Neurological involvement in SFTS has been increasingly reported in the literature and is considered an indicator of poor prognosis [8–10]. The pathophysiology behind CNS injury in *Bunyavirus*-related sepsis is multifactorial. Proposed mechanisms include direct viral neuroinvasion [11], endothelial dysfunction of the blood-brain barrier [12], and excessive systemic cytokine release contributing




Examination Report:

24-hour dynamic EEG recording:

Background brain activity recorded during drowsiness showed bilateral hemispheric widespread low-to-medium amplitude 4-7 Hz θ activity as the main pattern, with a small amount of medium-to-high amplitude 2-3 Hz δ activity scattered or in segments, with left and right sides roughly symmetrical. The frontal area showed a large amount of low-to-medium amplitude 4-5 Hz θ activity intermittently in a non-rhythmic short-long range burst; significant in the right frontal area; compound background fast waves; occipital area showed disappearance of α rhythm. Sleep spindles and sleep structure were present in the recording. No significant epileptiform discharges were seen during this recording.




Examination Report:

Recorded Findings:

Waveform: No α rhythm observed.

Fast Waves: A large amount of low amplitude fast activity widely distributed.

Slow Waves: A small amount of low-to-medium amplitude 4-7 Hz θ activity and 2-3 Hz δ activity scattered or in segments.

Rhythmic Disorder: None

Specific Waves: None

Hyperventilation: Cooperation is good, and hyperventilation is roughly the same as before.

**Fig. 2** **a**: EEG on the 10th day of hospitalization **b**: EEG on the 24th day of hospitalization

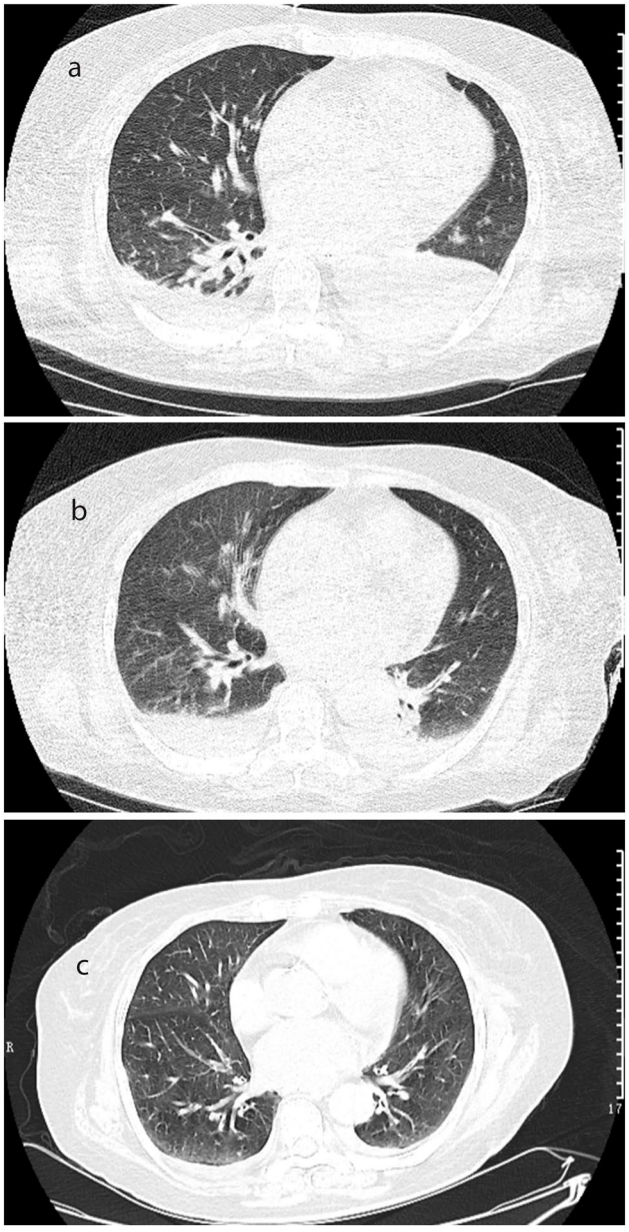



**Fig. 3** **a**: CT scan on the 5th day of hospitalization **b**: CT scan on the 10th day of hospitalization **c**: CT scan on the 17th day of hospitalization

to neurotoxicity. Elevations in pro-inflammatory cytokines such as IL-6, TNF-α, and interferon-γ are commonly observed in SFTS patients with encephalopathy and correlate with disease severity [13]. Additionally, metabolic derangements [14], thrombocytopenia-related microhemorrhages [15], and hypoxic injury secondary to systemic shock may exacerbate neural damage [16]. Current treatment strategies for SFTS remain supportive, with no widely accepted antiviral therapy [17]. Favipiravir and ribavirin have shown limited efficacy in clinical studies and are often used off-label [18]. For severe cases, particularly those with CNS involvement, immune modulation and cytokine suppression are critical. Therapeutic plasma exchange (TPE) has emerged as a promising adjunct therapy in this context. The principle of TPE is based on the removal of circulating inflammatory mediators, pathogenic antibodies, and toxins, and the replacement with fresh frozen plasma or albumin to restore immune homeostasis [6, 19–21].

In the case presented, the patient received two sessions of plasma exchange on days 3 and 5 of hospitalization. Following TPE, there was a significant improvement in liver enzyme levels, platelet counts, and most notably, neurological function—reflected by improvements in Glasgow Coma Scale (GCS) scores and EEG findings. This clinical trajectory is consistent with prior reports that suggest TPE may attenuate cytokine storms and prevent further neurological deterioration in viral sepsis.

Recent studies have begun to explore the role of plasma exchange in severe viral infections, including SFTS. A retrospective analysis of SFTS patients in China indicated that early use of TPE was associated with lower mortality in patients with CNS involvement or high viral load [22]. In experimental models, TPE has been shown to rapidly decrease levels of circulating virus, pro-inflammatory cytokines, and tissue injury biomarkers. Moreover, combining TPE with immunomodulatory therapies such as corticosteroids and intravenous immunoglobulin may have synergistic effects in rebalancing the immune response.

Sepsis is characterized by dynamic immune dysregulation, involving an initial hyperinflammatory phase that may be followed by immune paralysis, increasing the risk of secondary infections and late mortality [23]. In recent years, the clinical management of sepsis has shifted from non-specific anti-inflammatory strategies towards individualized immune modulation. Glucocorticoids and intravenous immunoglobulin (IVIG) have been shown to improve outcomes in selected septic patients through broad immunomodulatory effects [24, 25]. Notably, therapeutic plasma exchange (TPE), as a physical clearance modality, demonstrates high efficacy in removing pathogenic antibodies, a mechanism that underscores its potential utility in sepsis, a condition characterized by elevated levels of circulating inflammatory mediators. The combination of intravenous immunoglobulin (IVIG) and TPE may constitute a sequential therapeutic strategy—"clearance first, regulation later"—in which

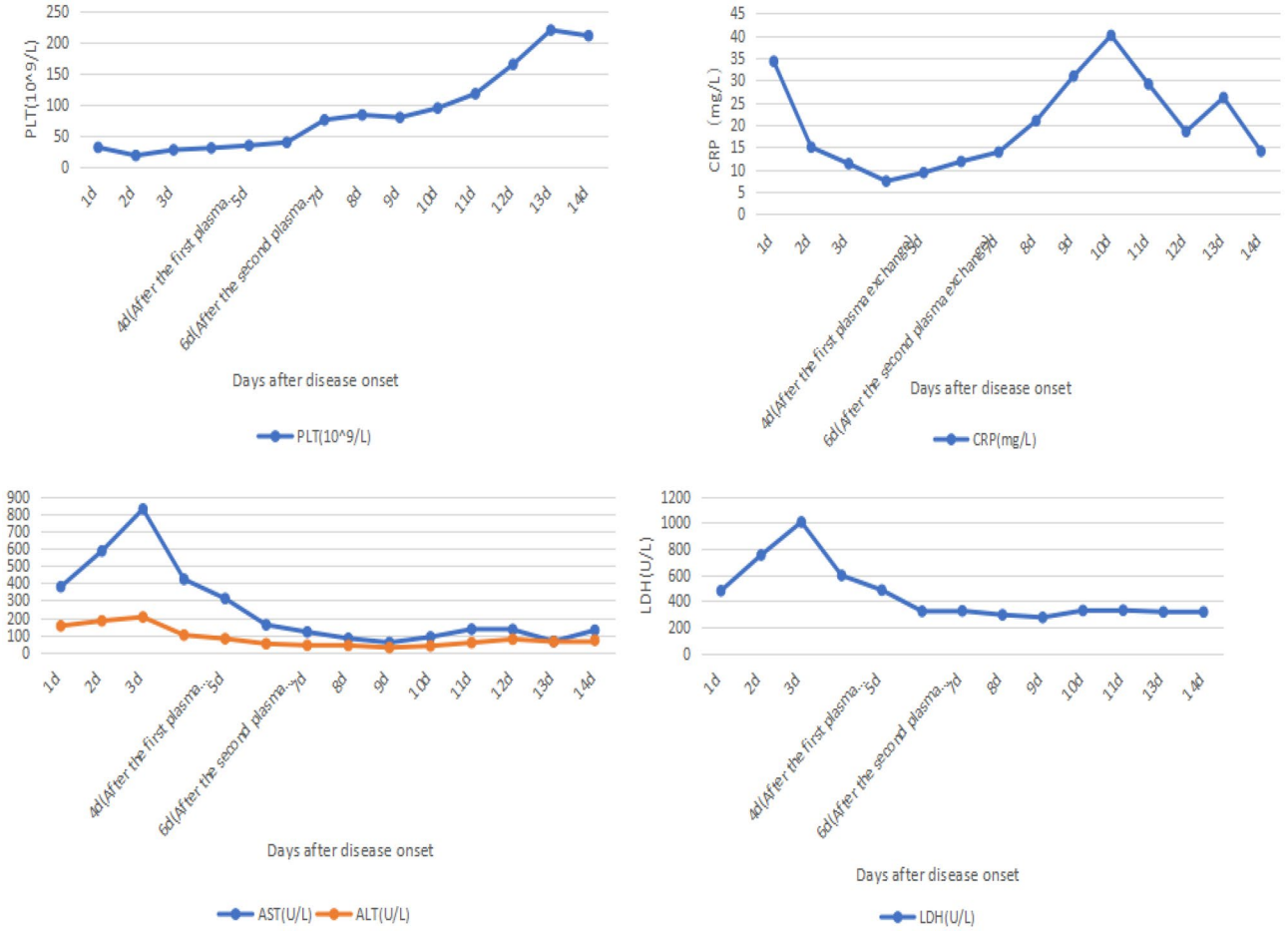



**Fig. 4** Trends of laboratory parameters during the patient's treatment period

**Table 1** Next generation sequencing results in peripheral blood

| **Fungi** | | | | | |
|---|---|---|---|---|---|
| **Genus** | | | **Species** | | |
| **Name** | **Sequence number** | **Relative abundance %** | **Name** | **Sequence number** | **Relative abundance %** |
| Aspergillus | 1 | 6.67 | Aspergillus fumigatus | 1 | 6.67 |
| **RNA viruses** | | | | | |
| **Genus** | | | **Species** | | |
| **Name** | **Sequence number** | **Relative abundance %** | **Name** | **Sequence number** | **Relative abundance %** |
| Bandavirus | 115 | 0.30 | ASevere fever with thrombocytopenia virus | 26 | 0.08 |

TPE rapidly reduces the burden of pathogenic antibodies and inflammatory mediators, followed by IVIG-mediated sustained immune modulation and functional restoration. This approach has shown success in the treatment of refractory heparin-induced thrombocytopenia (HIT) [26], thereby providing a theoretical foundation and strategic direction for investigating its application in complex cases of sepsis.

Despite these promising findings, the optimal timing, frequency, and volume of plasma exchange in SFTS remain uncertain. In this case, early intervention with two TPE sessions appeared sufficient to reverse

**Table 2** Cerebrospinal fluid examination and other laboratory results

| Test items | Results |
|---|---|
| appearance | transparent |
| Pandy test | negative |
| Leukocyte count ($10^6$/L) | 1 |
| bacterial examination | negative |
| Glucose(mmol/L) | 4.68 |
| Chloride (mmol/L) | 138.4 |
| Protein(mg/dL) | 26.4 |
| White blood cell count($10^9$/L) | 2.4 |
| CRP(mg/L) | 30.9 |
| PLT(($10^9$/L) | 79 |
| AST(U/L) | 57 |
| ALT(U/L) | 27 |
| Serum creatinine(umol/L) | 109.3 |
| LDH(U/L) | 276 |
| CK(U/L) | 316 |

life-threatening complications, though additional studies are needed to establish standardized protocols.

In conclusion, this case underscores the potential role of plasma exchange as a therapeutic strategy in *Bunyavirus*-induced sepsis with neurological involvement. Given the absence of definitive antiviral treatments, TPE represents a valuable adjunctive approach to mitigate systemic inflammation, protect organ function, and improve outcomes in patients with severe SFTS.

#### Abbreviations

| | |
|---|---|
| SFTS | Severe fever with thrombocytopenia syndrome |
| TPE | Therapeutic plasma exchange |
| SFTSV | Severe fever with thrombocytopenia syndrome virus |
| MODS | Multi-organ dysfunction syndrome |
| CNS | Central nervous system |
| ALT | Alanine aminotransferase |
| AST | Aspartate aminotransferase |
| LDH | Lactate dehydrogenase |
| GCS | Glasgow coma scale |
| γ-GT | Gamma-glutamyl transpeptidase |
| CK | Creatine kinase |
| ECG | Electrocardiogram |
| IVIG | Intravenous immunoglobulin |
| NGS | Next-generation sequencing |
| SAE | Sepsis-associated encephalopathy |
| EEG | Electroencephalogram |
| PCT | Proealcitonin |
| NF-κB | Nuclear factor-κB |
| HIT | Heparin-induced thrombocytopenia |

## Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12879-026-13235-4.

Supplementary Material 1

#### Acknowledgements

We thank all the patients included in this study and Zhoushan Hospital for the provision of the cases and the collaboration in the treatment of patients.

#### Author contributions

Ran Ji and Xuebin Wen contributed equally to this work. RJ： data collection and manuscript write. XW: data collection and manuscript write. All authors reviewed the manuscript.

#### Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

#### Data availability

The raw sequence data reported in this paper have been deposited in the Genome Sequence Archive (Genomics, Proteomics & Bioinformatics 2021) in National Genomics Data Center (Nucleic Acids Res 2022), China National Center for Bioinformation / Beijing Institute of Genomics, Chinese Academy of Sciences (GSA: CRA028576) that are publicly accessible at https://ngdc.cncb.ac.cn/gsa.

## Declarations

#### Ethical approval

The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was approved by the Ethics Review Committee of Zhoushan Hospital (No. 2025-088-001), and written informed consent for this retrospective analysis was waived.

#### Consent for publication

Patients gave written informed consent for their personal or clinical details along with any identifying images to be published in this study.

#### Competing interests

The authors declare no competing interests.

Received: 9 July 2025 / Accepted: 30 March 2026
Published online: 11 April 2026

#### References

1. Silvas JA, Aguilar PV. The emergence of severe fever with thrombocytopenia syndrome virus. Am J Trop Med Hyg. 2017;97(4):992–6.
2. Casel MA, Park SJ, Choi YK. Severe fever with thrombocytopenia syndrome virus: emerging novel phlebovirus and their control strategy. Exp Mol Med. 2021;53(5):713–22.
3. Sanchez-Pinto LN, Stroup EK, Pendergrast T, Pinto N, Luo Y. Derivation and validation of novel phenotypes of multiple organ dysfunction syndrome in critically Ill children. JAMA Netw Open. 2020;3(8):e209271.
4. Fei X, Feng B, Fang K, Ren W. Risk Factors for mortality in severe fever with thrombocytopenia syndrome patients with central nervous system complications. Med Sci Monit. 2023;29:e938427.
5. Wang L, Xu Y, Zhang S, Bibi A, Xu Y, Li T. The AST/ALT ratio (De Ritis Ratio) represents an unfavorable prognosis in patients in early-stage SFTS: an observational cohort study. Front Cell Infect Microbiol. 2022;12:725642.
6. Guironnet-Paquet A, Hamzeh-Cognasse H, Berard F, Cognasse F, Richard JC, Yonis H, Mezidi M, Desebbe O, Delannoy B, Demeret S, et al. Therapeutic plasma exchange accelerates immune cell recovery in severe COVID-19. Front Immunol. 2024;15:1492672.
7. Al-Hashami S, Khamis F, Al-Yahyay M, Al-Dowaiki S, Al-Mashaykhi L, Al-Khalili H, Chandwani J, Al-Salmi I, Al-Zakwani I. Therapeutic plasma exchange: A potential therapeutic modality for critically ill adults with severe acute respiratory syndrome coronavirus 2 infection. J Clin Apher. 2022;37(6):563–72.
8. Xu Y, Shao M, Liu N, Dong D, Tang J, Gu Q. Clinical feature of severe fever with thrombocytopenia syndrome (SFTS)-associated encephalitis/encephalopathy: a retrospective study. BMC Infect Dis. 2021;21(1):904.
9. Liu Z, Zhang R, Liu Y, Ma R, Zhang L, Zhao Z, Ge Z, Ren X, Zhang W, Lin L, et al. Eosinophils and basophils in severe fever with thrombocytopenia syndrome patients: Risk factors for predicting the prognosis on admission. PLoS Negl Trop Dis. 2022;16(12):e0010967.
10. Xiong S, Zhang W, Li M, Xiong Y, Li M, Wang H, Yang D, Peng C, Zheng X. A simple and practical score model for predicting the mortality of severe fever with thrombocytopenia syndrome patients. Med (Baltim). 2016;95(52):e5708.

11. Proenca-Modena JL, Hyde JL, Sesti-Costa R, Lucas T, Pinto AK, Richner JM, Gorman MJ, Lazear HM, Diamond MS. Interferon-Regulatory Factor 5-Dependent Signaling Restricts Orthobunyavirus Dissemination to the Central Nervous System. J Virol. 2016;90(1):189–205.
12. de Vries L, Harding AT. Mechanisms of neuroinvasion and neuropathogenesis by pathologic flaviviruses. Viruses. 2023;15(2).
13. Chen Z, Li G. Immune response and blood-brain barrier dysfunction during viral neuroinvasion. Innate Immun. 2021;27(2):109–17.
14. Slaughter AL, Nunns GR, D'Alessandro A, Banerjee A, Hansen KC, Moore EE, Silliman CC, Nemkov T, Moore HB, Fragoso M, et al. The Metabolopathy of Tissue Injury, Hemorrhagic Shock, and Resuscitation in a Rat Model. Shock. 2018;49(5):580–90.
15. Kockelmann F, Maegele M. Acute haemostatic depletion and failure in patients with traumatic brain injury (TBI): pathophysiological and clinical considerations. J Clin Med. 2023;12(8).
16. Nistor M, Behringer W, Schmidt M, Schiffner R. A systematic review of neuroprotective strategies during hypovolemia and hemorrhagic shock. Int J Mol Sci. 2017;18(11).
17. Li J-C, Zhao J, Li H, Fang L-Q, Liu W. Epidemiology, clinical characteristics, and treatment of severe fever with thrombocytopenia syndrome. Infect Med (Beijing). 2022;1(1):40–9.
18. Yan X, Luo C, Yang J, Wang Z, Shao Y, Wang P, Yang S, Li Y, Dai Q, Li W, et al. Antiviral activity of selective estrogen receptor modulators against severe fever with thrombocytopenia syndrome virus in vitro and in vivo. Viruses. 2024;16(8).
19. Tonev DG, Momchilova AB. Therapeutic plasma exchange in certain immune-mediated neurological disorders: focus on a novel nanomembrane-based technology. Biomedicines. 2023;11(2).
20. Tabibi S, Tabibi T, Conic RRZ, Banisaeed N, Streiff MB. Therapeutic Plasma Exchange: A potential Management Strategy for Critically Ill COVID-19 Patients. J Intensive Care Med. 2020;35(9):827–35.
21. Kuklin V, Sovershaev M, Bjerner J, Keith P, Scott LK, Thomas OMT, Szpirt W, Rock G, Stegmayr B. Influence of therapeutic plasma exchange treatment on short-term mortality of critically ill adult patients with sepsis-induced organ dysfunction: a systematic review and meta-analysis. Crit Care. 2024;28(1):12.
22. Oh WS, Yoo JR, Kwon KT, Kim HI, Lee SJ, Jun JB, Ryu SY, Kim HA, Hur J, Wi YM, et al. Effect of Early Plasma Exchange on Survival in Patients with Severe Fever with Thrombocytopenia Syndrome: A Multicenter Study. Yonsei Med J. 2017;58(4):867–71.
23. Hotchkiss RS, Coopersmith CM, McDunn JE, Ferguson TA. Tilting toward immunosuppression. Nat Med. 2009;15(5):496–7.
24. Zou H, Yi F, Wang Q. Efficacy and safety of glucocorticoid in the treatment of sepsis: a systematic review and meta-analysis. Asian J Surg. 2023;46(7):2912–4.
25. Pan B, Sun P, Pei R, Lin F, Cao H. Efficacy of IVIG therapy for patients with sepsis: a systematic review and meta-analysis. J Transl Med. 2023;21(1):765.
26. Onuoha C, Barton KD, Wong ECC, Raval JS, Rollins-Raval MA, Ipe TS, Kiss JE, Boral LI, Adamksi J, Zantek ND, Onwuemene OA. Therapeutic plasma exchange and intravenous immune globulin in the treatment of heparin-induced thrombocytopenia: a systematic review. Transfusion. 2020;60(11):2714–36.


## Publisher's note