รายงานผู้ป่วย (Case Report)

โรคเกล็ดเลือดต่ำจากลิ่มเลือดอุดตันในหลอดเลือดขนาดเล็กภายหลังจากการติดเชื้องูสวัดในหญิงชาวไทย : กรณีศึกษา

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บทคัดย่อ

บทนำ โรคเกล็ดเลือดต่ำจากลิ่มเลือดอุดตันในหลอดเลือดขนาดเล็ก พบได้ไม่บ่อยนัก แต่มีอัตราการทุพพลภาพและเสียชีวิตสูงมาก ภาวะนี้ผู้ป่วยมักมีอาการแสดงที่ไม่จำเพาะ การวินิจฉัยจึงต้องอาศัยประสบการณ์และความเชี่ยวชาญในการวินิจฉัยโรค

วัตถุประสงค์ เพื่อรายงานกรณีศึกษาผู้ป่วยโรคเกล็ดเลือดต่ำจากลิ่มเลือดอุดตันในหลอดเลือดขนาดเล็ก อาการแสดงของโรค ขั้นตอนการวินิจฉัยแยกโรค และการรักษาผู้ป่วย

วิธีการศึกษา ศึกษาข้อมูลย้อนหลังจากประวัติ การดำเนินโรค อาการ อาการแสดงของผู้ป่วยจากการซักประวัติ ตรวจร่างกาย ภาพถ่ายอาการแสดงของผู้ป่วย ผลการตรวจทางห้องปฏิบัติการและการติดตามการดำเนินโรคจากเวชระเบียนผู้ป่วย

ผลการศึกษา หญิงอายุ 72 ปี เป็นโรคไตวายเรื้อรังระยะสุดท้ายที่บำบัดทดแทนไตด้วยวิธีฟอกเลือดด้วยเครื่องไตเทียมอายุ มาโรงพยาบาลด้วยอาการซึมลง สงสัยว่ามีการติดเชื้องูสวัดในระบบประสาท ได้รับการรักษาด้วยยาต้านไวรัส ภายหลังพบว่าเม็ดเลือดแดงแตกร่วมกับเกล็ดเลือดต่ำ จึงได้รับการรักษาด้วยการเปลี่ยนถ่ายน้ำเหลืองและยาคอร์ติโคสเตียรอยด์ อาการของผู้ป่วยดีขึ้นหลังได้รับการรักษา แต่ไม่สามารถลดยาคอร์ติโคสเตียรอยด์ได้ และได้พบว่ามีวัณโรคช่องท้องแอบแฝง สันนิษฐานว่าเป็นเหตุกระตุ้นร่วมกับการติดเชื้องูสวัดกระตุ้นซ้ำให้เกิดโรค

สรุป กรณีศึกษาโรคเกล็ดเลือดต่ำจากลิ่มเลือดอุดตันในหลอดเลือดขนาดเล็กซึ่งเกิดตามหลังการติดเชื้องูสวัดและวัณโรคเยื่อบุช่องท้องแอบแฝง การตระหนักถึงสาเหตุและตัวโรคสามารถนำไปสู่การส่งตรวจวินิจฉัยอย่างเหมาะสม และการรักษาอย่างทันท่วงทีเพื่อช่วยลดอัตราการเสียชีวิตให้แก่ผู้ป่วย

คำสำคัญ เกล็ดเลือดต่ำจากลิ่มเลือดอุดตันในหลอดเลือดขนาดเล็ก การเปลี่ยนถ่ายน้ำเหลือง โรคไตวายเรื้อรัง โรคงูสวัด วัณโรคเยื่อบุช่องท้อง

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Thrombotic thrombocytopenic purpura following a cutaneous herpes zoster infection in a Thai female: Case report

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Abstract

Introduction: Thrombotic thrombocytopenic purpura (TTP) is a rare but fatal disease. Clinical presentations are nonspecific and usually require a skilled physician to detect. Rapid diagnosis and treatment is imperative in clinical practice.

Objective: To report a case of thrombotic thrombocytopenic purpura in a patient with end stage renal disease.

Methods: Retrospective study that presented the patient's history, physical examinations, laboratory investigations, imaging, clinical course and treatment. The data is derived from the hospital medical records.

Results: A 72-year-old Thai female with end stage renal disease on regular hemodialysis, presented with alteration of consciousness and microangiopathic hemolytic anemia. Recent history of herpes zoster skin infection raised a clinical suspicion for herpes meningoencephalitis. However, after adequate treatment, her symptoms deteriorated. Hematology was consulted and a provisional diagnosis of thrombotic thrombocytopenic purpura was made. Plasma exchange was initiated and the patient's clinical improved after five cycles of plasma exchange with adjunctive corticosteroids. Immunosuppression was prolonged due to refractoriness of TTP. An occult infection of abdominal tuberculosis was later identified as a potential TTP trigger and treated.

Conclusion: A case study of Thrombotic thrombocytopenic purpura following a herpes zoster infection. Recognizing the condition facilitates appropriate diagnostic testing and early treatment, thereby helping to reduce patient mortality.

Keyword: Thrombotic thrombocytopenic purpura, Plasma exchange, Chronic kidney disease, Herpes zoster, Tuberculous peritonitis

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Introduction

Thrombotic thrombocytopenic purpura (TTP) is a rare but fatal disease, with a mortality rate up to 80% without treatment and 20% despite treatment[1]. TTP is characterized by ADAMTS13 insufficiency or dysfunction resulting in the accumulation of von Willebrand factor causing thrombotic microangiopathy. Clinical presentations of TTP include a combination of thrombocytopenia, microangiopathic hemolytic anemia, neurologic symptoms, renal dysfunction, and skin purpura. Neurologic symptoms may include confusion, seizures, stroke, or, in severe cases, coma[1-3]. Diagnosing TTP in dialysis patients is highly complex, being confounded by baseline abnormal renal function and anemia of chronic disease; due to its high mortality rate, a rapid diagnosis and treatment are imperative.

We report a case of Thrombotic thrombocytopenic purpura in an elderly Thai female with end stage renal disease, preceding herpes zoster encephalitis following a cutaneous herpes zoster infection, and an occult abdominal tuberculosis infection, presenting with an acute confusional state.

Case Presentation

A 72-year-old Thai female with a previous medical history of hypertension and end stage renal disease on regular hemodialysis every Wednesday and Saturday presented at the emergency department (ED) with acute onset confusion, dysarthria and vomiting for four hours. Four days prior, she was diagnosed at another hospital with a cutaneous herpes zoster infection involving the T9-T10 dermatome and received oral acyclovir 800 mg bid for treatment. One day prior to the ED visit, she complained of dizziness and fatigue, whilst her daughter reported confusion in the patient's speech but denied any dysarthria. On the day of admission, the patient developed dysarthria, which was accompanied by multiple vomiting episodes, with such worsening symptoms.

At the emergency department, the patient was confused, not oriented to time, place and person. She had mild dysarthria and hypersomnolence without any other focal neurological deficits detected. Her initial vital signs showed an increased blood pressure of 201 mmHg of systolic blood pressure and 88 mmHg of diastolic blood pressure. She had normal body temperature and no fever was reported in her present illness. The previously diagnosed herpes zoster skin infection at the abdomen had improved. An initial evaluation was conducted, and a differential diagnosis consisted of acute ischemic stroke, uremic encephalopathy, septic encephalopathy, meningoencephalitis especially of varicella zoster origin due to a recent skin infection. Laboratory investigations such as blood chemistry, complete blood count, blood urea nitrogen levels and serum creatinine were evaluated, furthermore, with acute ischemic stroke as a differential diagnosis, a computed tomography scan of the brain was ordered. Lacunar infarction lesions were identified but

did not fully explain the patient's symptoms. Magnetic resonance imaging of the brain was unremarkable.

Initial laboratory investigations showed a markedly increased level of blood urea nitrogen of 73 mg/dL with serum creatinine 12.4 mg/dL supporting uremic encephalopathy. Normocytic anemia with thrombocytopenia was reported (Table 1). Emergency hemodialysis was prescribed, and the patient was admitted to observe further neurological signs. After hemodialysis, blood urea nitrogen levels decreased to 29 mg/dL (Table 2) and the patient had a mildly improved level of consciousness, however, despite improvement in blood urea nitrogen levels the patient was still confused, not oriented to time, place and person. Lumbar puncture was done after a transfusion of pooled platelets. The CSF was clear and colorless; 13 white blood cells were identified, with 9% of polymorphonuclear lymphocytes and 91% of monocytes. Normal glucose and protein levels were reported, supporting a partially treated viral or aseptic meningitis CSF profile. The CSF gram stain was negative. CSF bacterial culture and herpes polymerase chain reaction (PCR) were pending. Acyclovir in intravenous form along with ceftriaxone for empirical treatment for herpes meningoencephalitis and other bacterial infection were initiated. A further evaluation for anemia with thrombocytopenia was conducted on the following day.

Table 1 Initial Laboratory Investigations.

Parameters	Laboratory results
CBC	
Hemoglobin	7.2 g/dL
Hematocrit	23%
MCV	84.9 fL
White blood cell	7,860 cells/mm^3 (Neutrophil 77%, Lymphocyte 14%, Monocyte 4%)
Platelet Count	48,000 cells/mm^3
Prothrombin time	11.5 seconds (Normal 11.0-13.3 seconds)
Partial thrombin time	24.9 seconds (Normal 20.1-28.7 seconds)
BUN	73 mg/dL
Creatinine	12.4 mg/dL
Sodium	130 mmol/L
Potassium	5.1 mmol/L
Chloride	91 mmol/L
HCO_3	21 mmol/L
LDH	303 U/L (normal135-214 U/L)

Note: CBC = Complete blood count, Hb = Hemoglobin, Hct = Hematocrit, WBC = White blood cell count, MCV = Mean corpuscular volume, BUN = Blood urea nitrogen, Cr = Serum creatinine, Na = Sodium, K = Potassium, Cl = Chloride, HCO_3 = Bicarbonate, LDH = Lactate dehydrogenase

Table 2 Laboratory investigations after hemodialysis (no heparin during dialysis).

Parameters	Laboratory results
BUN	29 mg/dL
Creatinine	5.98 mg/dL
Sodium	136 mmol/L
Potassium	4.6 mmol/L
Chloride	95 mmol/L
HCO3	26 mmol/L

Microangiopathic hemolytic anemia (MAHA) was the leading differential diagnosis of anemia and thrombocytopenia in this patient. The peripheral blood smear was examined the next day and demonstrated a decrease of platelets, schistocytes with microspherocytes suggestive of microangiopathic hemolytic anemia. A normal coagulogram and a high lactate dehydrogenase level (LDH) was demonstrated. Hematology and Nephrology were consulted due to high suspicion for thrombotic thrombocytopenic purpura with a PLASMIC score of 6. Plasma exchange was scheduled for the next day. ADAMTS-13 level, and ADAMTS-13 antibody level were pending. Unfortunately, during the diagnostic process, the patient's consciousness deteriorated showing a stuporous level of consciousness indicating a need for intubation. The patient was intubated and transferred to the intensive care unit. An electroencephalogram was conducted and displayed generalized slow waves at bilateral cerebral hemispheres and no periodic sharp waves were seen. On the subsequent day, the plasma exchange 1.5 times of plasma volume was initiated with adjunctive intravenous dexamethasone. After three cycles of plasma exchange, the patient started having spontaneous eye opening but could not follow commands, increasing of platelet levels and decreasing LDH were demonstrated, showing a response to treatment.

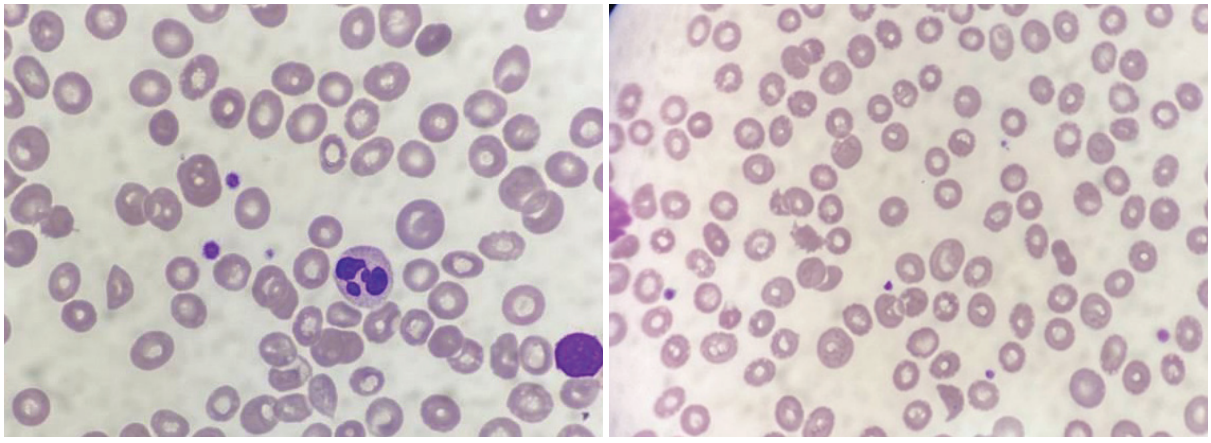


Figure 1 **Peripheral Blood Smear**: The peripheral blood smear showed schistocytes with microspherocytes and a decreased amounts of platelets without clumping suggestive of microangiopathic hemolytic anemia

On the fifth plasma exchange cycle, the patient was fully awake, oriented to time, place and person. She was able to follow simple commands. Extubation was performed without any complications. Despite clinical recovery, laboratory investigations demonstrated a new onset of thrombocytopenia. Drug-induced thrombocytopenia, heparin-induced thrombocytopenia and refractory TTP were suspected. Meropenem was discontinued due to no evidence of infection and a high probability for being the main culprit of drug-induced thrombocytopenia. Further hemodialysis sessions refrained from using heparin and anti-PF4 were investigated. Refractory TTP was listed as a differential diagnosis but was highly unlikely due to the clinical improvement. Five days after cessation of meropenem, platelet levels started to increase to normal levels. Acyclovir was continued for up to 10 days to treat herpes meningoencephalitis. Anti-PF4 levels were reported to be negative. ANA was conducted as a laboratory work-up for secondary causes of TTP and was reported to be negative. Approximately one week later, the ADAMTS level and antibody levels were found to be less than 10% and normal, respectively, confirming the diagnosis of thrombotic thrombocytopenic purpura (Table 3). Cyclosporin A was started with a dose of 25 mg per day and dexamethasone was switched to prednisolone 40 mg per day.

Table 3 ADAMTS13 Laboratory Investigations.

Parameters	Laboratory results
ADAMTS13 Antibody	3.91 U/mL (Normal < 12.00 U/mL)
ADAMTS13 level	3.6 U/dL (73.0-164.0)

She was discharged after 24 days of admission, with prednisolone 35 mg per day and Cyclosporin A 25 mg per day. A scheduled follow-up visits at 2 weeks found a stable clinical response and platelet levels. Cyclosporin was increased to 50 mg per day as a steroid-sparing choice and prednisolone was gradually tapered to 15 mg per day, 10 mg per day, 5 mg and tapered off at 1 month, 2 months, 3 months and 6 months from TTP diagnosis, respectively. However, the patient had a decrease of platelets after discontinuation of prednisolone and developed exogenous adrenal insufficiency, so prednisolone 5 mg every alternate day dosage was maintained.

Table 4 Hemoglobin and platelet values and management.

Date and Time	Hct (%)	Hb (g/dL)	Platelet (cells/mm3)	Serum LDH (U/L)	Management (IV solutions and Oral Medication)
1 Aug 2023	23	7.2	48000	303	Emergency hemodialysis Acyclovir IV, Ceftriaxone IV CXR: no infiltration Blood and platelet transfusion
2 Aug 2023	31	10.2	54000 to 104000*	-	Lumbar puncture, Hematologic evaluation, Platelet transfusion pre-procedure
4 Aug 2023	30	9.6	74000	-	Scheduled for plasma exchange Endotracheal intubation Meropenem IV
5 Aug 2023	27	8.7	102000	-	Plasma exchange cycle 1st
7 Aug 2023	21	6.5	40000	-	Plasma exchange cycle 2nd Dexamethasone 4 mg IV q12h Blood transfusion
8 Aug 2023	21	7.0	51000	217	Plasma exchange cycle 3th Dexamethasone 4 mg IV q12h Blood transfusion Electroencephalogram
9 Aug 2023	27	8.7	63000	214	Plasma exchange cycle 4th Dexamethasone 4 mg IV q12h
10 Aug 2023	26	8.2	38000	211	Plasma exchange cycle 5th Dexamethasone 4 mg IV q12h Meropenem discontinued
11 Aug 2023	26	8.4	77000	-	Dexamethasone 4 mg IV q12h Cyclosporin A 25 mg/day
12 Aug 2023	28	9.3	114000	-	Prednisolone 40 mg/day Cyclosporin A 25 mg/day
14 Aug 2023	27	9.0	181000	-	Prednisolone 40 mg/day Cyclosporin A 25 mg/day

Table 4 Hemoglobin and platelet values and management **(Continued).**

Date and Time	Hct (%)	Hb (g/dL)	Platelet (cells/mm3)	Serum LDH (U/L)	Management (IV solutions and Oral Medication)
21 Aug 2023	28	9.1	139000	-	Prednisolone 35 mg/day Cyclosporin A 25 mg/day
23 Aug 2023	-	-	-	-	Discharged from hospital
13 Sep 2023 OPD Visit	31	10.3	102000	228	Prednisolone 15 mg/day Cyclosporin A 50 mg/day
12 Oct 2023 OPD Visit	29	9.0	130000	174	Prednisolone 10 mg/day Cyclosporin A 50 mg/day
16 Nov 2023 OPD Visit	25	7.8	231000	155	Prednisolone 5 mg/day Cyclosporine A 50 mg/day
30 Jan 2024 OPD Visit	-	-	-	-	Abdominal paracentesis
8 Feb 2024	22	6.8	163000	-	Prednisolone 5 mg/day Cyclosporine A 50 mg/day CXR: no infiltration Start IRZE
7 Mar 2024	26	8.5	190000	-	Cyclosporine A 50 mg/day
4 Apr 2024	31	10.3	153000	177	Cyclosporine A 50 mg/day
19 Jul 2024	26	8.5	95000	-	Cyclosporine A 50 mg/day Prednisolone 5 mg/day
8 Aug 2024	-	-	-	-	Treatment of TB peritonitis was completed
10 Oct 2024	24	7.8	151000	188	Cyclosporine A 50 mg/day Prednisolone 5 mg every alternate day
9 Jan 2025	24	7.8	145000	189	Cyclosporine A 50 mg/day Prednisolone 5 mg every alternate day

The patient had a routinely scheduled annual follow up visit with the urologist due to her having a neurogenic bladder. A bedside ultrasound was done to evaluate vesicoureteral reflux and found new-onset ascites. Abdominal paracentesis showed a low serum ascites albumin gradient and high ascitic protein levels, indicating a peritoneal disease (Serum albumin 3.5 g/dL, Ascitic albumin 2.5 g/dL, Ascitic protein 7.5 g/dL). A mononuclear white blood cell was dominant and had a normal-high range of ascitic adenosine deaminase (ADA). Cancer or abdominal tuberculosis were suspected. Further age-group cancer screening was found to be negative for malignancy. The patient and family denied endoscopic

screening procedures. However, after about 6 weeks, the mycobacterium culture reported back to be positive for *Mycobacterium tuberculosis* complex. There was no radiologic evidence of pulmonary infection. A standard antituberculosis treatment regimen consisting of isoniazid, rifampicin, pyrazinamide and ethambutol (IRZE) was initiated. The regimen was changed to isoniazid, ethambutol, levofloxacin (IEL) due to a suspected drug-induced liver injury from the previous regimen. A total of 6 months treatment was commenced with good clinical response within the first two months showing decreased ascites and resolved ascites at four months of treatment.

Table 5 Ascitic Profile.

Yellow and cloudy appearance, WBC 1201 cells/mm^3, PMN 16%, Monocyte 84%, RBC 3000 cells/mm^3
Albumin 2.5 g/dL, Protein 7.5 g/dL, ADA 32.5 U/L
Gram stain negative, Culture negative, AFB stain negative, PCR for mycobacterium negative
Cytology: Mixed small lymphoid cells, histiocytes and scattering reactive mesothelial cells in clearing background
Culture for mycobacterium: Positive *Mycobacterium tuberculosis* complex

After resolution of abdominal tuberculosis, her platelet levels were stable, prednisolone was maintained at 5 mg every alternative day.

Discussion

Thrombotic thrombocytopenic purpura (TTP) is rare but is associated with a high mortality rate of up to 80% without treatment and 20% despite treatment[3]. Clinical presentation of TTP is usually described in literature as a pentad comprising of fever, neurologic manifestations, renal manifestations, thrombocytopenia and microangiopathic hemolytic anemia[2,4,5] with only 10% presenting with these symptoms[6]. The main pathophysiology is due to ADAMTS13 severe deficiency leading to von-Willebrand factor dysregulation causing abnormal platelet adhesion and resulting microangiopathy[2,5,6]. TTP is diagnosed if serum ADAMTS13 levels are < 10%[1,2], however, most constitutions in Thailand usually outsource due to the unavailability of serum ADAMTS13 level laboratories, delaying the process for diagnosis, as was done in this case.

The PLASMIC scoring system, used to identify risk for severe ADAMTS13 deficiency in patients with thrombocytopenia and microangiopathic hemolytic anemia, is proven to be valuable in situations where ADAMTS13 levels cannot be assessed in a timely fashion, with high sensitivity (90%) and specificity

(92%), interpreted by a score of 0-4, 5 and 6-7 suggesting a low risk, intermediate risk and high risk for severe ADAMTS13 deficiency, respectively[7].

Current clinical practice guidelines recommend plasma exchange within 4-8 hours from a suspected diagnosis of TTP[2,3]. Corticosteroid is also recommended as an adjunct to plasma exchange in immune-mediated TTP[2,3]. Both mainstays of treatment were implemented in this case and displayed a positive clinical response following treatment. Response to treatment is monitored by presenting clinical symptoms and platelet levels, with clinical remission defined as a normal platelet count (> 1.5 x 10^9 /L).[3]

TTP can be classified into two major groups: congenital and acquired**.** Congenital TTP typically presents with a bimodal distribution in early childhood and early adulthood[1,6,8]. In contrast, TTP in the elderly should generally be considered acquired. The primary pathophysiology of acquired TTP involves an autoimmune process driven by anti-ADAMTS13 antibodies; however, in rare instances, no antibodies are detected, and the condition is classified as TTP of unknown etiology[1,6]. Numerous associations of herpes zoster, CMV infections, and tuberculosis complicating TTP have been reported (Table 6).

In this case report, our patient was diagnosed with acquired TTP secondary to herpes zoster. A week after infection, she developed a high-grade fever and an altered mental status, and her peripheral blood smear demonstrated a MAHA pattern. An ADAMTS13 activity level of less than 10% confirmed the diagnosis. Her clinical recovery and platelet count improved after she received her fifth cycle of plasma exchange combined with corticosteroids. During outpatient follow-ups, she maintained normal LDH and platelet levels; however, she could not be tapered off immunosuppressive drugs and was subsequently diagnosed with abdominal tuberculosis. Peritoneal tuberculosis in this patient could have resulted from complications of immunosuppressive therapy or a pre-existing occult infection. Ultimately, plasma exchange followed by targeted treatment of the underlying infection was performed; the patient survived and was able to reduce her cyclosporine and prednisolone dosages.

Table 6 Case reports of acquired TTP associated with infection.

Age, Sex	Presentation	TTP Treatment	Clinical outcomes	Associations
32-year-old, Male [9]	Fever, thrombocytopenia, anemia, MAHA, acute kidney injury	Plasma exchange Anti-TB med	Clinical recovery and discharged after 40 days	Pulmonary, pleural, peritoneal tuberculosis
53-year-old, Male [10]	Fever, thrombocytopenia, anemia, MAHA Acute kidney injury	Plasma exchange Platelet transfusion Anti-TB med	Clinical recovery and discharged after 26 days	Pulmonary tuberculosis
49-year-old, Female [11]	Headaches, ecchymoses, Anemia, thrombocytopenia, MAHA	Plasma exchange Steroid Anti-TB med	Clinical recovery and discharged after 8 days	Pulmonary tuberculosis
44-year-old, Female [12]	Altered mental status, seizure, anemia, thrombocytopenia, MAHA	Plasma exchange Steroids Acyclovir	Clinical recovery and discharged after 14 days	Varicella zoster encephalitis
19-year-old, Female [13]	Bleeding, Anemia, Thrombocytopenia, MAHA	Plasma exchange, Steroids Acyclovir	Clinical recovery	Varicella zoster infection
68-year-old, Female[14]	Fever, Altered mental status, Anemia, Thrombocytopenia, MAHA, acute kidney injury	Plasma exchange, Acyclovir	Death at day 25	Varicella zoster infection
35-year-old, Male [15]	Bleeding, headache, Anemia, thrombocytopenia, MAHA, seizure	Plasma exchange, Valganciclovir	Clinical improvement on day 6	CMV infection

Conclusion

Thrombotic thrombocytopenic purpura is a rare and fatal disease. Diagnosis is limited due to ADAMTS-13 assay availability. A high clinical suspicion and PLASMIC score correlation can aid in guiding prompt treatment. Awareness of TTP as a post-infectious complication such as zoster and tuberculosis should be heightened, as demonstrated in our case report, to reduce the currently high mortality rate of TTP.

Acknowledgement

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References

1. Joly BS, Coppo P, Veyradier A. Thrombotic thrombocytopenic purpura. Blood. 2017; 129: 2836-46.

2. Scully M, Rayment R, Clark A, Westwood JP, Cranfield T, Gooding R, et al. A British Society for Haematology Guideline: Diagnosis and management of thrombotic thrombocytopenic purpura and thrombotic microangiopathies. Br J Haematol. 2023; 203: 546-63.
3. Zheng XL, Vesely SK, Cataland SR, Coppo P, Geldziler B, Iorio A, et al. ISTH guidelines for treatment of thrombotic thrombocytopenic purpura. J Thromb Haemost. 2020; 18: 2496-502.
4. Adeyemi A, Razakariasa F, Chiorean A, de Passos Sousa R. Epidemiology, treatment patterns, clinical outcomes, and disease burden among patients with immune-mediated thrombotic thrombocytopenic purpura in the United States. Res Pract Thromb Haemost. 2022; 6: e12802.
5. Long B, Bridwell RE, Manchanda S, Gottlieb M. Evaluation and Management of thrombotic thrombocytopenic purpura in the Emergency Department. J Emerg Med. 2021; 61: 674-82.
6. Joly BS, Coppo P, Veyradier A. Thrombotic thrombocytopenic purpura. Blood. 2017; 129: 2836-46.
7. Bendapudi PK, Hurwitz S, Fry A, Marques MB, Waldo SW, Li A, et al. Derivation and external validation of the PLASMIC score for rapid assessment of adults with thrombotic microangiopathies: a cohort study. Lancet Haematol. 2017; 4: e157- 64.
8. Scully M. Congenital TTP: next stop, acuity and therapy. Blood. 2021; 137: 3469-71.
9. Masumoto S, Tada M, Katsuma A, Minami E, Katagiri D, Shibata M, et al. Thrombotic microangiopathy associated with tuberculous infection. Intern Med. 2011; 50: 1825-8.
10. Zheng J, Pan X, Jiang Y. Pulmonary tuberculosis complicated by thrombotic thrombocytopenic purpura: a case report and literature review. Infect Drug Resist. 2024; 17: 3593-8.
11. Contreras K, Amorocho OMC, Giraldo JS. Acquired thrombotic thrombocytopenic purpura as a clinical manifestation of pulmonary tuberculosis: a case report. Germs. 2023; 13: 259-65.
12. Mostel Z, Nguyen BA, Costanzo L, Bankhead S, Ayat P, Taluru H, et al. Thrombotic thrombocytopenic purpura due to varicella-zoster virus meningoencephalitis in an immunocompetent patient. J Glob Infect Dis. 2023; 15: 172-4.
13. Satoh K, Takahashi H, Nagai K, Shibata A. Thrombotic thrombocytopenic purpura and herpes zoster infection. Ann Intern Med. 1988; 108: 154-5.
14. Xu B, Ritchie D. Thrombotic thrombocytopenic purpura associated with disseminated varicella zoster in a multiple myeloma patient. Intern Med J. 2010; 40: e7-8.
15. Boteju M, Weeratunga P, Sivashangar A, Chang T. Cytomegalovirus induced refractory TTP in an immunocompetent individual: a case report. BMC Infect Dis. 2019; 19: 394.