International Journal of Recent Advances in Multidisciplinary Topics
Volume 7, Issue 9, September 2026
www.ijramt.com | E-ISSN: 2582-7839 | RESAIM Publishing | www.resaim.com

Thrombotic Thrombocytopenic Purpura Presenting as a Transient Ischemic Attack Mimic: A Case Report

Hisham Eltayeb Ahmed Elhassan[1], Tarig Merghani[2], Fathelrahman Ahmed[3*], Syed Majid Hamid[3], Azza Alawad[4]

[1]*Department of Medicine, Gargash Hospital, Dubai, United Arab Emirates*
[2]*Department of Physiology, RAK Medical and Health Sciences University, Ras Al-Khaimah, United Arab Emirates*
[3]*Department of Endocrinology and Diabetes, Ibrahim Bin Hamad Obeidallah Hospital, Ras Al-Khaimah, United Arab Emirates*
[4]*Department of Physiology, Al-Neelain University, Khartoum, Sudan*

***Abstract*: Background: Thrombotic thrombocytopenic purpura (TTP) is a rare, life-threatening thrombotic microangiopathy characterized by microangiopathic hemolytic anemia (MAHA), severe thrombocytopenia, and microvascular ischemia. Neurological involvement is common and may be transient, so TTP can be mistaken for a transient ischemic attack (TIA), delaying diagnosis. Prompt recognition and early treatment are critical to reduce mortality. Case Presentation: A 66-year-old woman presented with sudden aphasia and left-sided weakness that re-solved spontaneously within approximately 30 minutes; she was initially admitted as a suspected TIA. Examination revealed pallor and multiple ecchymoses, with no residual neurological deficit. Brain magnetic resonance imaging showed no ischemic changes. Laboratory evaluation revealed anemia (hemoglobin 7.8 g/dL), severe thrombocytopenia (platelets 15,000/µL), elevated lactate dehydrogenase (LDH) and bilirubin, reticulocytosis, and schistocytes on the peripheral blood smear, with a normal coagulation pro-file and preserved renal function. The PLASMIC score was 6, indicating a high probability of severe ADAMTS13 deficiency. Therapeutic plasma exchange and corticosteroids were started promptly without awaiting ADAMTS13 testing. She made a full neurological recovery and was discharged after two weeks with a hemoglobin of 11.2 g/dL and a platelet count of 194,000/µL. Conclusion: TTP should be considered in patients presenting with transient focal neurological deficits, particularly when accompanied by anemia and thrombocytopenia. Careful review of the full blood count in suspected TIA can uncover this diagnosis, and a high PLASMIC score should prompt immediate plasma exchange without waiting for ADAMTS13 results.**

***Keywords*: Thrombocytopenic purpura, Thrombocytopenia, Hemolytic Anemia, Microangiopathic, ADAMTS13 Protein.**

1. Introduction

Thrombotic thrombocytopenic purpura (TTP) is a rare hematologic emergency with annual incidence of 6 cases or less per million (Pishko et al., 2025). It is characterized by thrombosis, thrombocytopenia and microangiopathic hemolytic anemia (MAHA), often accompanied by neurological, renal, and systemic manifestations. The condition is primarily caused by a severe deficiency of ADAMTS13 (a disintegrin and metalloprotease with thrombospondin type 1 motif, member 13), a protease responsible for cleaving ultra-large von Willebrand factor (vWF) multimers (Joly et al., 2017). Severe ADAMTS13 deficiency (<10% activity) leads to accumulation of large vWF multimers, resulting in widespread platelet aggregation and microvascular thrombosis (Zheng et al., 2020). TTP can be congenital (genetic mutation causing reduced production of ADAMTS13) or acquired (autoantibodies causing inhibition of ADAMTS13), with the autoimmune inhibition being the most common cause.

Clinically, TTP may present with a wide range of symptoms, including fatigue, dyspnea, petechiae, neurological deficits, and organ dysfunction. The classical five features (fever, MAHA, thrombocytopenia, renal dysfunction, and neurological symptoms) are rarely seen in full, and diagnosis often relies on laboratory findings (George & Nester, 2014). The PLASMIC score showed satisfactory results in estimating the likelihood of severe ADAMTS13 deficiency and guiding early treatment decisions (Oliveira et al., 2018; Bendapudi et al., 2017).

2. Case Presentation

A 66-year-old female presented to the emergency department with sudden aphasia and left-sided weakness. Before reaching the hospital, the neurological deficits were resolved spontaneously, with a total duration of approximately 30 minutes. She was initially admitted as a case of transient ischemic attack (TIA). On examination, she was conscious and oriented (Glasgow Coma Scale score of 15/15). Neurological examination was unremarkable, with normal tone, power, reflexes, and intact sensation. However, general examination revealed pallor and multiple ecchymotic patches over the trunk and limbs. Cardiovascular, respiratory, and abdominal examinations were unremarkable.

*Corresponding author: fathi_ahmed90@hotmail.com

3. Laboratory Findings

Initial laboratory investigations (Table 1) showed severe anemia with a hemoglobin level of 7.8 g/dL and significant thrombocytopenia with a platelet count of 15,000/μL on admission, both of which remained low by day 5. The white blood cell count was normal. Renal function and electrolyte levels were also within normal ranges throughout the observation period. C-reactive protein was slightly elevated whereas D-dimer was increased above the reference range and showed a rising trend during follow-up (Table 1).

Table 1
Laboratory findings on admission and follow-up

Parameter	Day 1	Day 5	Reference Range
Hemoglobin (g/dL)	7.8	7.7	12–15
Platelets (/μL)	15,000	14,000	150,000–450,000
WBC (/μL)	4,200	4,200	4,000–11,000
Sodium (mmol/L)	143	144	135–145
Potassium (mmol/L)	4.3	4.0	3.5–5.0
Creatinine (mg/dL)	0.89	0.9	0.6–1.2
CRP (mg/L)	0.69	0.69	<5
D-dimer (ng/mL)	1330	1650	<500

Further laboratory evaluation demonstrated biochemical and hematological features consistent with hemolysis. Total bilirubin was elevated at 2.3 mg/dL, and lactate dehydrogenase (LDH) levels were markedly increased. In addition, the reticulocyte count was elevated, indicating a compensatory bone marrow response. Peripheral blood smear examination revealed the presence of schistocytes, supporting a diagnosis of microangiopathic hemolytic anemia. Importantly, the coagulation profile remained within normal limits, and blood cultures were negative, helping to exclude disseminated intravascular coagulation and infectious etiologies.

Radiological investigations were unremarkable. Magnetic resonance imaging (MRI) of the brain showed no evidence of ischemic changes, while chest X-ray and abdominal ultrasound did not reveal any significant abnormalities.

The PLASMIC score was calculated as 6, corresponding to a high probability of severe ADAMTS13 deficiency. The laboratory findings strongly supported the diagnosis of thrombotic thrombocytopenic purpura.

4. Management and Outcome

Based on the high clinical suspicion of TTP, laboratory findings and PLASMIC score, treatment was initiated with corticosteroids (prednisolone) and therapeutic plasma exchange (plasmapheresis). Treatment was initiated without waiting for ADAMTS13 activity testing. The patient also received one unit of packed red blood cells and a platelet transfusion during management. She demonstrated progressive clinical improvement, with complete resolution of neurological symptoms and a steady rise in platelet count. She was discharged in stable condition after two weeks with hemoglobin 11.2 g/dL and platelet count of 194000/μL. Arrangements were made for close outpatient follow-up.

5. Discussion

Thrombotic thrombocytopenic purpura (TTP) is a medical emergency that requires prompt diagnosis and treatment due to its high mortality rate if treatment is delayed. In most adult cases, this deficiency is acquired and immune-mediated, resulting from autoantibodies directed against ADAMTS13, which explains the predominance of acquired TTP in clinical practice (Joly et al., 2017; Scully et al., 2019). The disease is rare, with an estimated annual incidence of approximately 10 cases per million population, and shows a female predominance, occurring nearly twice in women compared to men (Joly et al., 2017; Scully et al., 2019).

In this case, the patient presented with transient neurological symptoms that were diagnosed as transient ischemic attack (TIA). About 60% of TTP patients may show neurological involvement as the most frequent organ manifestation, ranging from mild confusion and headache to focal deficits, seizures, or coma (George & Nester, 2014; Azoulay et al., 2025). The variable symptoms and signs in presentation cause diagnostic challenges, particularly when symptoms are transient and imaging studies are unremarkable, as observed in this case.

In this case, the diagnosis of TTP was supported by severe thrombocytopenia, microangiopathic hemolytic anemia (anemia, schistocytes, elevated LDH), a normal coagulation profile, and a high PLASMIC score. However, it is important to note that the classical pentad of TTP (fever, thrombocytopenia, microangiopathic hemolytic anemia, neurological symptoms, and renal dysfunction) is now considered obsolete, as recent studies showed that fewer than 10% of patients present with all these five features, indicating the need for updated diagnostic approaches (Scully et al., 2017). The PLASMIC score is a valuable tool for predicting severe ADAMTS13 deficiency and guiding early treatment decisions (Bendapudi et al., 2017). A score of ≥ 6 indicates high probability and justifies immediate initiation of plasma exchange. In addition to neurological involvement, other organ systems may be affected due to microvascular ischemia. These include cardiac ischemia, mesenteric ischemia and renal involvement (Joly et al., 2017; Azoulay et al., 2025).

Therapeutic plasma exchange is the cornerstone of treatment, as it removes circulating autoantibodies and replaces ADAMTS13 (Rock et al., 1991; Azoulay et al., 2025). Corticosteroids are used to suppress autoantibody production, and rituximab may be considered to prevent clinical relapses (Zheng et al., 2020; Azoulay et al., 2025). Intensive approaches such as twice-daily plasma exchange or additional immunosuppressive agents may be required in refractory cases.

Early initiation of treatment significantly reduces mortality, which historically exceeded 90% but has now decreased to less than 20% with appropriate therapy. Despite these advances, TTP continues to be life-threatening condition with reported mortality rates ranging from 10% to 20% even with optimal management (Joly et al., 2017; Azoulay et al., 2025). It is worth noting that TTP is characterized by a relapsing course, necessitating long-term follow-up for recurrence, associated autoimmune conditions, and possible long-term complications.

6. Conclusion

This case shows that transient ischemic attack (TIA) could

be the initial manifestation of thrombotic thrombocytopenic purpura (TTP), indicating early microvascular cerebral involvement. Therefore, TTP should be considered in patients presenting with transient neurological symptoms in association with hematological abnormalities, particularly anemia and thrombocytopenia. Early diagnosis is based on clinical features and laboratory findings, supported by the PLASMIC score. Prompt initiation of plasma exchange and administration of immunosuppressive therapy are critical for reducing morbidity and mortality and improving overall patient outcome.

References

[1] E. Azoulay *et al.*, "Thrombotic thrombocytopenic purpura: Early diagnosis and effective treatment in 2025," *Intensive Care Med.*, vol. 51, no. 7, pp. 1225–1239, Jul. 2025.

[2] P. K. Bendapudi *et al.*, "Derivation and external validation of the PLASMIC score," *Lancet Haematol.*, vol. 4, no. 4, pp. e157–e164, Apr. 2017.

[3] J. N. George and C. M. Nester, "Syndromes of thrombotic microangiopathy," *N. Engl. J. Med.*, vol. 371, no. 7, pp. 654–666, Aug. 2014.

[4] B. S. Joly, P. Coppo, and A. Veyradier, "Thrombotic thrombocytopenic purpura," *Blood*, vol. 129, no. 21, pp. 2836–2846, May 2017.

[5] D. S. Oliveira, T. G. Lima, F. L. N. Benevides, S. A. T. Barbosa, M. A. Oliveira, N. P. Boris, and H. F. Silva, "PLASMIC score applicability for the diagnosis of thrombotic microangiopathy associated with ADAMTS13-acquired deficiency in a developing country," *Hematol., Transfus. Cell Ther.*, vol. 41, no. 2, pp. 119–124, Apr.–Jun. 2019.

[6] A. M. Pishko, A. Li, and A. Cuker, "Immune thrombotic thrombocytopenic purpura: A review," *JAMA*, vol. 334, no. 6, pp. 517–529, Aug. 2025.

[7] G. A. Rock, K. H. Shumak, N. A. Buskard, V. S. Blanchette, J. G. Kelton, R. C. Nair, and R. A. Spasoff, "Comparison of plasma exchange with plasma infusion in the treatment of thrombotic thrombocytopenic purpura," *N. Engl. J. Med.*, vol. 325, no. 6, pp. 393–397, Aug. 1991.

[8] M. Scully *et al.*, "Caplacizumab treatment for acquired thrombotic thrombocytopenic purpura," *N. Engl. J. Med.*, vol. 380, no. 4, pp. 335–346, Jan. 2019.

[9] X. L. Zheng *et al.*, "ISTH guidelines for treatment of thrombotic thrombocytopenic purpura," *J. Thromb. Haemost.*, vol. 18, no. 10, pp. 2496–2502, Oct. 2020.