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CASE REPORT

Thrombotic Thrombocytopenic Purpura During Anti-Tuberculosis Therapy: A Case Report and Literature Review

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Introduction: Thrombotic thrombocytopenic purpura (TTP) is a rare, life-threatening thrombotic microangiopathy and a high mortality rate if untreated. While TTP can be primary or secondary to factors like drugs, reports linking it to anti-tuberculosis (anti-TB) therapy are scarce. This case highlights the diagnostic challenges and need for vigilance in TB patients receiving standard regimens.

Case Presentation: A 76-year-old male on anti-TB therapy (isoniazid, rifampicin, pyrazinamide, ethambutol) presented with acute neurological symptoms, thrombocytopenia (platelets 9×10^9/L), microangiopathic hemolysis (schistocytes), and severely reduced ADAMTS13 activity (<5%). Imaging revealed multiple cerebral infarctions. Despite plasma exchange and steroids, the patient deteriorated and died after family-requested care withdrawal.

Conclusion: This case highlights TTP as a rare but serious complication of anti-TB therapy. Clinical vigilance is essential, including platelet monitoring during initial treatment and a low threshold for ADAMTS13 testing in cases of unexplained thrombocytopenia. Future multicenter studies are needed to investigate immune mechanisms and assess therapies such as rituximab, with the aim of optimizing management strategies for rare adverse drug events and improving patient outcomes.

Keywords: anti-tuberculosis therapy, adverse drug reaction, thrombotic thrombocytopenic purpura, ADAMTS13, case report

Introduction

Thrombotic thrombocytopenic purpura (TTP) is a rare but potentially fatal disorder caused by severe deficiency of ADAMTS13, a metalloprotease responsible for cleaving von Willebrand factor multimers. This deficiency leads to uncontrolled platelet aggregation, microvascular thrombosis, and subsequent end-organ damage. TTP can occur as a primary condition or secondary to factors such as infections, autoimmune disorders, malignancies, or certain drugs. The estimated incidence is 2–6 cases per million people annually, with a mortality rate of up to 90% without timely intervention. Early diagnosis and prompt initiation of plasma exchange can reduce mortality to 10–20%.[1,2]

Drug-induced TTP represents an important clinical subset. Among antimicrobials, anti-tuberculosis (anti-TB) agents, particularly rifampicin, have been sporadically implicated in case reports as potential triggers.[3–5] The proposed mechanisms for anti-TB associated TMA include hapten-induced immune responses leading to ADAMTS13-inhibiting antibodies or direct, dose-dependent endothelial toxicity.[3,6] Previous literature on anti-TB associated TTP has primarily consisted of isolated case reports, often focusing on establishing the temporal association and the role of rifampicin based on recurrence upon rechallenge or the Naranjo probability scale.[4,5,7] However, a comprehensive analysis that integrates detailed clinical timelines, laboratory trends, systematic causality assessment, and a thorough discussion of alternative etiologies within the context of active tuberculosis is often limited in these reports.

Herein, we report a case of fatal TTP occurring in a patient receiving standard first-line anti-TB therapy. This case contributes to the existing body of knowledge by providing a meticulous longitudinal account of the patient's clinical and

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paraclinical data, from the diagnosis of tuberculosis through the development of TTP. This report aims to enhance clinical awareness and provide a structured approach to the diagnosis and management of this critical complication in TB patients.

Case Presentation

A 76-year-old male was urgently admitted to the hospital following a 24-hour period characterized by the acute onset of progressive generalized weakness rendering him unable to walk, alongside expressive aphasia. His family corroborated this history, reporting concomitant new-onset urinary incontinence, intermittent fever, and confusion over the same duration. This presentation occurred approximately one month after the initial diagnosis of pulmonary tuberculosis.

The patient's first hospitalization (the first admission) for tuberculosis was necessitated by his severe clinical condition at the time of diagnosis. As shown in Table 1, laboratory findings during the first admission revealed not only active inflammation (elevated C-reactive protein at 106.23 mg/L and erythrocyte sedimentation rate at 37 mm/h) but also evidence of acute organ dysfunction. This included acute kidney injury (creatinine 140 μmol/L, urea nitrogen 9.2 mmol/L) and a significantly elevated myoglobin level (140.6 μg/L), suggesting possible rhabdomyolysis as a cause for his weakness and renal impairment. Additionally, he had severe hypoalbuminemia (27.7 g/L). Due to these complexities, he was hospitalized to initiate first-line anti-tuberculosis therapy [isoniazid (300 mg/day), rifampicin (450 mg/day), pyrazinamide (1.5 g/day), and ethambutol (750 mg/day)] under close monitoring. His condition stabilized sufficiently for discharge, and he continued medication orally. There was no medical history suggesting an immunocompromised state, such as HIV infection, malignancy, or chronic use of immunosuppressive medications.

Upon the second admission, the patient was critically ill, cachectic, and confused. Vital signs recorded a body temperature of 37.5°C, with blood pressure, heart rate, and respiratory rate within normal limits. Anthropometric measurements indicated a height of 160 cm, weight of 60 kg, and a body mass index (BMI) of approximately 23.4 kg/m^2. General physical examination revealed no skin rash, lymphadenopathy, or organomegaly. A systematic

Table 1 The Laboratory Findings of Patient

Variable	Reference Ranges	First Admission	First Discharge	Second Admission	Day after the Second Admission	Third day of the Second Admission	Seventh Day of the Second Admission
WBC (×10^9/L)	3.97–9.15	4.83	6.68	6.58	8.63	7.37	11.78
PLT (×10^9/L)	85-303	242	173	9	10	16	15
HB (g/L)	120-160	115	116	99	89	65	75
ESR (mm/h)	0-15	37	16	50			
CRP (mg/L)	0-3	106.23	70.79	86.15			
PCT (ng/mL)	0-0.5		0.36	0.6		0.52	
LDH (U/L)	114-240	239		754.4	818.1	434	519
Myoglobin (μg/l)	0-70	140.6		218.3	358.4	462.1	355.4
Cr (μmol/L)	53-115	140	164	208.3	221.2	221	
UN (mmol/L)	2.9–8.2	9.2	7.5	14.25	15.69	17.9	
ALT (U/L)	0-40	15.5	13	28.9	28.9	14.9	
AST (U/L)	0-45	27.5	25	117.4	99.3	33.4	
Serum albumin (g/L)	35-55	27.7	23.6	28.4	26.9	34.1	
PT(s)	10-15	13.4		11.7	13.7	14.5	15
APTT(s)	23-40	42.4		36	42.1	38.2	35.2
Fib (g/L)	2-4	4.3		3.32	2.97	2.2	1.92
D-dimer (ug/mL)	0-1	1.15		2.08	1.99	1.34	1.98
PO2 (mmHg)	80-100			103	98		89
PCO2 (mmHg)	35-45			27	22.3		21.6

Abbreviations: WBC, White blood cell; PLT, Platelets; HB, Hemoglobin; PCO2, Partial pressure of carbon dioxide; PO2, Partial pressure of oxygen; ALT, Alanine aminotransferase; AST, Aspartate aminotransferase; Cr, Creatinine; UN, urea nitrogen; LDH, Lactate dehydrogenase; ESR, Erythrocyte sedimentation rate; PCT, Procalcitonin; CRP, C reactive protein; PT, Prothrombin time; APTT, Activated partial thromboplastin time; Fib, Fibrinogen.

neurological examination confirmed the aphasia and profound weakness, but cranial nerve assessment was normal with bilateral pupils reactive to light. The cardiovascular, respiratory, and abdominal examinations were unremarkable.

Initial laboratory investigations at this admission revealed catastrophic hematological findings: severe thrombocytopenia (9×10^9/L), anemia (hemoglobin 99 g/L), and markedly elevated lactate dehydrogenase (754.4 U/L). A peripheral blood smear confirmed the presence of schistocytes (Figure 1), consistent with microangiopathic hemolytic anemia. Critically, ADAMTS13 activity was measured at less than 5%, confirming the diagnosis of thrombotic thrombocytopenic purpura (TTP). Cerebral magnetic resonance imaging (MRI) identified multiple acute infarctions in the bilateral cerebellar hemispheres and cerebrum (Figure 2), explaining the acute neurological deficit. A concurrent chest CT showed progression of left-sided pulmonary lesions compatible with active tuberculosis (Figure 3).

The patient was managed in the intensive care unit. Supportive care measures were comprehensively instituted, including continuous hemodynamic monitoring, fluid and electrolyte balance management, and seizure prophylaxis. Specific therapy for TTP was initiated without delay on the day of admission, comprising daily therapeutic plasma exchange and high-dose intravenous dexamethasone (10 mg per day).

Despite these aggressive interventions, which represent the standard of care and encompass all recommended first-line therapies for acute TTP, the patient's clinical course was relentlessly progressive. His neurological status deteriorated further, and he developed convulsions and a high fever (38.8°C) on the fourth day of hospitalization. Given the irreversible nature of his extensive neurological injury and the refractory nature of the TTP despite maximal medical therapy, the family, after detailed consultation with the treating team, made the decision to withdraw life-sustaining treatment. The patient died on the fourth hospital day. The cause of death was attributed to refractory TTP with massive cerebral infarction.

The Naranjo scale yielded a score of 6, indicating a probable adverse drug reaction, with rifampicin as the most likely culprit given its known associations (Table 2).

Discussion

This report presents a case of TTP occurring in a patient receiving anti-tuberculosis therapy. It adds to the sparse literature on drug-induced TTP. Below, we expand the discussion by providing a comprehensive literature review, comparing it with similar cases, delving into alternative causes of thrombocytopenia, and analyzing laboratory and treatment outcomes in depth.

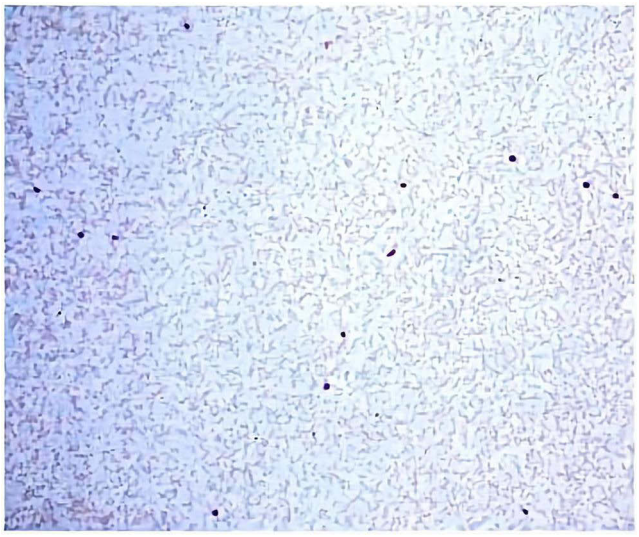

Figure 1 Peripheral blood smear examination.

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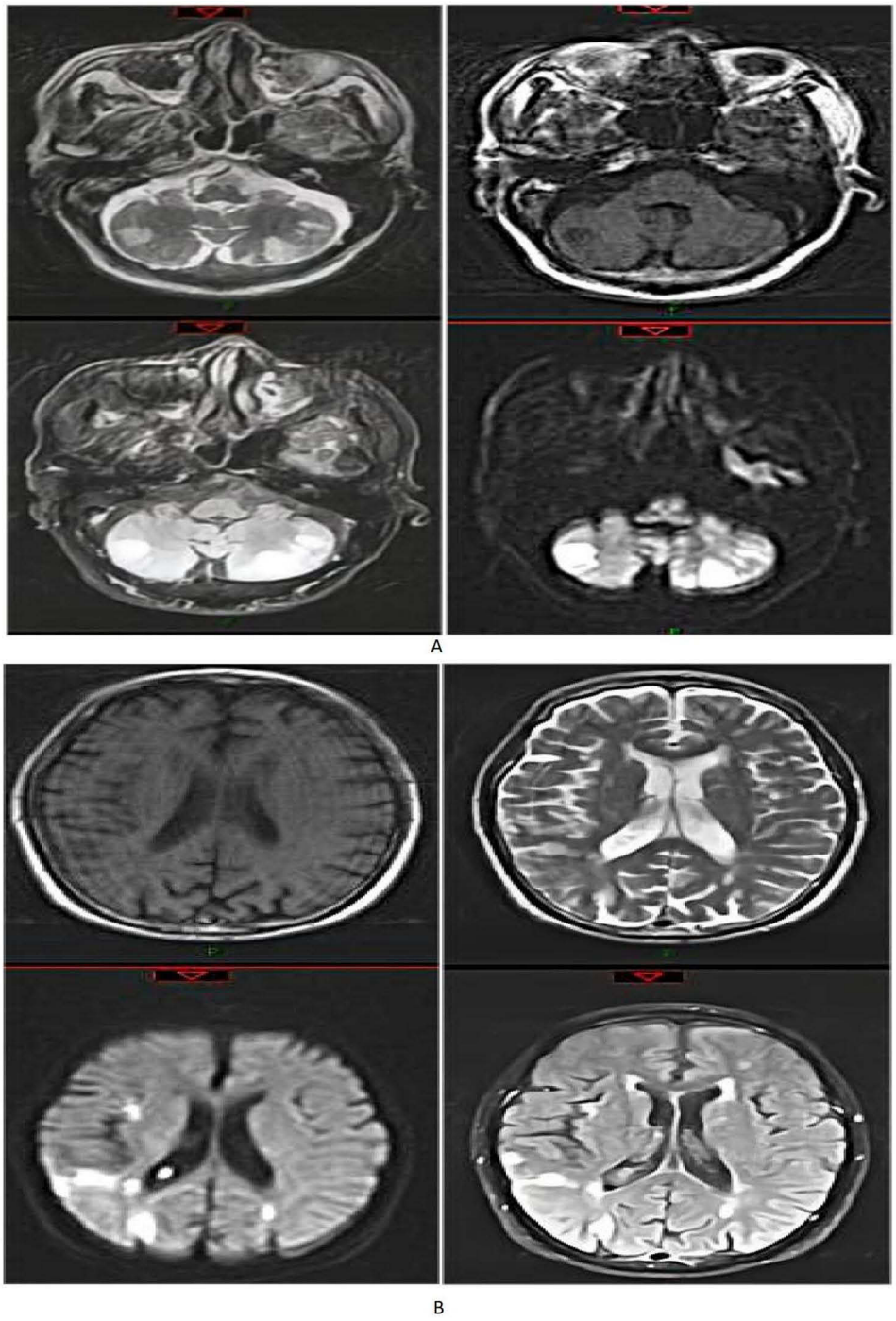

Figure 2 Cerebral magnetic. Cerebellar hemispheres (**A**) and basal ganglia (**B**).The upper left shows T2-weighted imaging, the upper right shows T1-weighted imaging, the lower left shows T2-weighted fat-suppressed imaging, and the lower right shows diffusion-weighted imaging.

Comprehensive Literature Review on TTP Etiology and Differential Diagnosis

TTP is primarily driven by a severe deficiency in ADAMTS13 activity (<10%), which can be congenital due to mutations in the ADAMTS13 gene or acquired through autoimmune inhibitors.[1,2] Congenital TTP, also known as Upshaw-Schulman syndrome, often presents in childhood and requires prophylactic plasma infusions, whereas acquired TTP is more common in adults and is frequently associated with autoantibodies against ADAMTS13.[8] Secondary TTP can arise from various triggers, including infections, malignancies, pregnancy, and drugs.[3,9,10] For instance, drug-induced TTP has

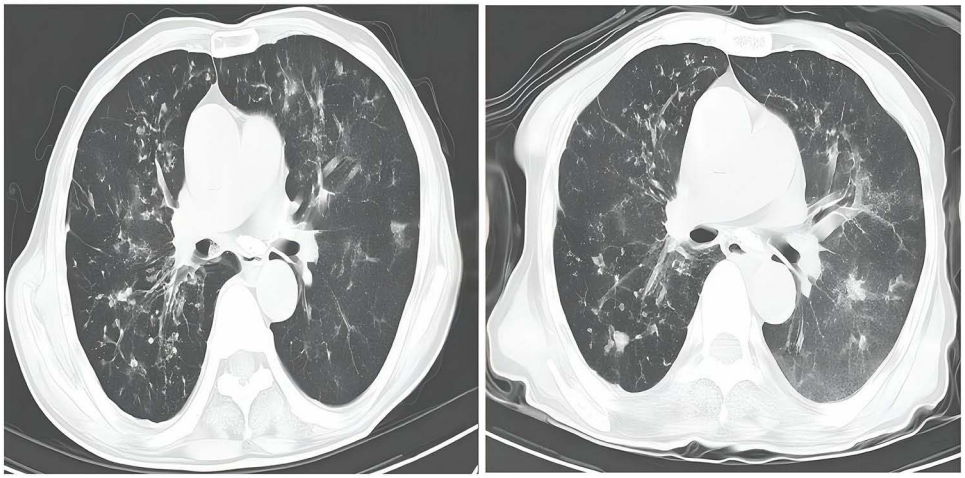

Figure 3 Chest CT. One month prior to admission (left) and at admission (right).

been documented with agents like Infliximab, Dasatinib and ipilimumab, where immune-mediated mechanisms lead to ADAMTS13 inhibition.[7,9] In the case of ipilimumab, a CTLA-4 inhibitor, TTP manifests through antibody production that disrupts ADAMTS13 function, similar to hypotheses for rifampicin in our case.[9,]

Beyond drug triggers, it is essential to differentiate TTP from other thrombotic microangiopathies (TMAs) and conditions causing thrombocytopenia. Hemolytic uremic syndrome (HUS) typically involves predominant renal failure and is often associated with Shiga toxin-producing Escherichia coli or complement dysregulation, distinguishing it from TTP's broader organ involvement.[11] Disseminated intravascular coagulation (DIC) presents with coagulopathy, such as prolonged prothrombin time and elevated D-dimer, which were absent in our patient.[12] Immune thrombocytopenia (ITP) is characterized by isolated thrombocytopenia without microangiopathic hemolysis or schistocytes.[13] Sepsis or infection-related thrombocytopenia usually shows signs of systemic inflammation, like leukocytosis or positive blood cultures, which were not evident here.[14] Our patient's presentation—lacking renal failure, coagulopathy, or infection markers—strongly supports TTP as the diagnosis, consistent with literature emphasizing ADAMTS13 testing for confirmation.[1]

In-Depth Comparison with Similar Cases

Our patient's course shares similarities with reported drug-induced TTP cases, such as those involving ipilimumab, where patients developed acute thrombocytopenia within months of drug initiation, and TTP was confirmed by detected ADAMTS13 deficiency.[9] However, key differences exist: most literature focuses on anticancer agents. A review by

Table 2 Naranjo Scale

Item	Yes	No	Do not Know	Score
1. Are there previous conclusive reports on this reaction?	1	0	0	1
2. Did the adverse event appear after the suspected drug was administered?	2	−1	0	2
3. Did the adverse reaction improve when the drug was discontinued or a specific antagonist was administered?	1	0	0	0
4. Did the adverse reaction reappear when the drug was readministered?	2	−1	0	0
5. Are there alternative causes (other than the drug) that could on their own have caused the reaction?	−1	2	0	2
6. Did the reaction reappear when a placebo was given?	−1	1	0	0
7. Was the drug detected in the blood (or other fluids) in concentrations known to be toxic?	1	0	0	0
8. Was the reaction more severe when the dose was increased, or less severe when the dose was decreased?	1	0	0	0
9. Did the patient have a similar reaction to the same or similar drugs in any previous exposure?	1	0	0	0
10. Was the adverse event confirmed by any objective evidence?	1	0	0	1
Total score				6

Notes: It is comprised of 10 questions with each response assigned a score. Total score corresponds to likelihood of drug-related ADR. Score ≥9 = definite; 5–8 = probable; 1–4 = possible; ≤0 = doubtful.

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Kremer Hovinga et al highlights that drug-induced TTP cases often have a delayed diagnosis due to non-specific symptoms, which may explain the poor outcome in our case.[10] Compared to typical TTP, which has a relapsing nature and better response to rituximab, our case underscores the challenges in managing rare triggers like anti-TB therapy. This comparison illustrates the novelty of our report in highlighting underrecognized drug associations.

This case finds strong support in a published case report that directly documents rifampin as the causative agent of TTP.[4] That report describes a nearly identical clinical presentation, characterized by the classic pentad of symptoms and similar hematological parameters, following rifampin administration. The resolution of symptoms upon withdrawal of the drug and initiation of appropriate supportive care, as seen in the present case, is a recurring and critical theme in that and other anecdotal reports, strengthening the argument for a causal relationship.

Furthermore, a broader perspective is provided by a recent systematic review that examined thrombocytopenia of all severities induced by various anti-tubercular drugs.[5] This review identifies rifampin as one of the most frequently implicated drugs in immune-mediated hematological reactions. While it notes that profound thrombocytopenia is more common than the full-blown TTP syndrome, the review confirms that TTP remains a rare but devastating potential outcome of ATT. The mechanisms discussed often involve the formation of drug-dependent antibodies, leading to platelet destruction and endothelial injury, which aligns with the pathophysiology suspected in this case.

Correlation of Laboratory Findings with Pathogenesis and Literature

The laboratory timeline in our case (Table 1) showed progressive thrombocytopenia (platelets dropping to 9×10^9/L), elevated LDH (754.4 U/L), and ADAMTS13 activity <5%, which are hallmark features of TTP pathogenesis.[1] ADAMTS13 deficiency leads to accumulation of ultra-large von Willebrand factor multimers, promoting platelet aggregation and microthrombi formation, as seen in the cerebral infarctions on imaging.[1,10] Literature indicates that such laboratory changes—particularly a rapid rise in LDH and persistent thrombocytopenia—correlate with severe organ damage and poor prognosis.[10,15] For instance, LDH elevation reflects ongoing hemolysis and ischemia, which aligns with our patient's neurological decline. This correlation underscores the importance of serial monitoring.

Analysis of Treatment Response and Preventative Strategies Based on Literature

Standard TTP therapy involves plasma exchange to replenish ADAMTS13 and remove inhibitors, combined with corticosteroids for immunosuppression.[16] Our patient received this regimen, which improves survival in 80–90% of cases when initiated early.[10,16] However, the refractory nature here may be attributed to delays in diagnosis or drug-specific factors, such as anti-TB therapy potential to induce irreversible immune damage. Literature supports the use of adjunctive therapies like rituximab for refractory TTP, which targets B-cells to reduce antibody production.[17] Rituximab can reduce relapse rates in autoimmune TTP, but its accessibility in resource-limited settings remains a barrier. Preventative measures could include routine platelet and LDH monitoring in patients on high-risk drugs, enabling earlier intervention.

Innovative Insights and Limitations

This case provides innovative insights by linking anti-TB therapy to TTP through a detailed literature-integrated analysis. Unlike previous reports, it systematically excludes alternative causes using established criteria and emphasizes the need for vigilance in TB-endemic regions. The comparison with drug-induced TTP cases and treatment analysis adds depth to the sparse literature on this topic, offering practical recommendations for early detection and resource-aware management.

However, several limitations must be acknowledged. The study is based on a single case report, which limits generalizability and precludes causal inferences. Reliance on a single ADAMTS13 activity measurement may have led to an overestimation or underestimation of the deficiency's severity, potentially affecting the interpretation of drug association. The retrospective design and incomplete patient history could introduce bias, influencing treatment decisions and follow-up assessments. For instance, the absence of anti-ADAMTS13 antibody testing due to resource constraints restricts mechanistic insights. These limitations suggest that the observed association may be influenced by unmeasured confounders, and future studies should adopt prospective designs with comprehensive data collection to mitigate such issues.

Conclusions

This case illustrates TTP as a rare but fatal complication of anti-TB therapy. For routine practice, we recommend implementing vigilant monitoring for patients on high-risk medications, including regular platelet counts during the initial treatment phases to facilitate early detection of hematological abnormalities. Clinicians should have a low threshold for ADAMTS13 testing in cases of unexplained thrombocytopenia. Policy-wise, establishing national registries for drug-induced TTP could improve data aggregation and risk assessment. Future research should focus on prospective multicenter studies to accumulate similar cases, investigate underlying immune mechanisms (eg, anti-ADAMTS13 antibodies), and evaluate the efficacy of adjunctive therapies such as rituximab in refractory scenarios 10. These steps will help refine management strategies and enhance patient safety.

In summary, this case reinforces the importance of clinical suspicion for TTP in complex pharmacological contexts, contributing to awareness without overstating causality. It calls for collaborative efforts to advance understanding and optimize care for rare adverse drug events.

Data Sharing Statement

All data generated or analyzed during this study are included in this published article.

Ethics Approval and Consent to Participate

This single case report was exempt from ethics review as it did not involve any interventions beyond standard clinical care. Therefore, institutional approval for the publication of case details was not required. Written informed consent was obtained from the deceased patient's next-of-kin for the publication of this case report, including all clinical details and images. The patient's identity remains protected through: 1) Removal of all personal identifiers (name, ID number, birthdate, location); 2) Avoidance of unique physical descriptors in text; 3) Approval of anonymized figures by the consenting relative. This consent process adhered to CARE guidelines and journal policy. A copy of the signed consent form is retained by the authors and available for editorial review.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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