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Fulminant Thrombotic Thrombocytopenic Purpura presenting through the FAST Stroke Pathway

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Abstract

Presentation

A previously healthy woman in her mid-40s of African ancestry presented with sudden-onset left-sided weakness, aphasia, and collapse. On arrival, she was disoriented with a fluctuating GCS of 9–11. Neurological assessment revealed speech impairment and limb weakness.

Diagnosis

CT brain showed multifocal parietal and cerebellar infarctions. Laboratory tests revealed severe thrombocytopenia (20 × 10^3/mm^3), anaemia (Hb 9 g/dL), elevated creatinine (180 µmol/L), and high troponins (1139 ng/L). Peripheral smear confirmed red cell fragmentation, and ADAMTS13 activity was <10%, confirming fulminant Thrombotic Thrombocytopenic Purpura (TTP).

Treatment

Thrombolysis was contraindicated due to profound thrombocytopenia, and intensive supportive care was initiated, including vasopressors, transfusions, and planned plasma exchange. Despite aggressive management, she developed hemodynamic collapse and died within 24 hours.

Discussion

This case highlights fulminant TTP mimicking acute stroke, emphasizing the importance of early recognition in patients with severe thrombocytopenia. Prompt multidisciplinary intervention and TTP-specific therapy are essential to improve survival outcomes.

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Introduction

Thrombotic Thrombocytopenic Purpura (TTP) is a rare but life-threatening thrombotic microangiopathy caused by severe ADAMTS13 deficiency, leading to accumulation of von Willebrand Factor multimers and widespread platelet aggregation. This results in microvascular thrombosis, haemolysis, and multi-organ injury involving the brain, kidneys, and heart[1,2]. TTP typically presents with the classical pentad of thrombocytopenia, haemolytic anaemia, fever, renal impairment, and neurological dysfunction[3]. Neurological involvement can range from mild confusion to coma, and may mimic acute ischemic stroke[4]. We present a rare case of stroke-like presentation in active TTP with multi-organ dysfunction, demonstrating the critical need to recognize TTP in the setting of ischemic stroke.

Case Report

A 46-year-old woman of African ancestry presented with sudden-onset left-sided weakness and dysarthria. Prior to the event, she had complained of headache and speech difficulty. Her medical history included hypertension and migraines treated with sumatriptan. She was a non-smoker, did not consume alcohol, and had no family history of cerebrovascular or haematological disorders. ECG was normal sinus rhythm and troponins elevation were most likely due to microvascular ischaemia and global myocardial injury from TTP. Neurological assessment showed aphasia, dysarthria, and lower limb drift with fluctuating levels of consciousness with no reliable focal deficits. Cranial nerves I-XII and sensory examinations were unremarkable. Her NIH stroke scale score was 5.

Based on initial findings, an acute ischemic stroke was suspected and she was transferred for urgent neuroimaging. Her CT brain showed multi-focal posterior parietal infarctions and cerebellar parietal hypodensities suggestive of multi-focal ischemic changes (Figure 1). However, bloodwork revealed severe thrombocytopenia (20 × 10^3/mm^3), anaemia (Hb 9 g/dL), elevated creatinine (180 µmol/L), and markedly raised troponins (1139 ng/L). Peripheral smear showed fragmented red cells, consistent with microangiopathic haemolytic anaemia. These findings prompted reconsideration of stroke thrombolysis.

The decision was made to intubate the patient due to severe agitation causing respiratory distress. After intubation, the patient's blood pressure dropped despite aggressive vasopressor therapy including phenylephrine, adrenaline and noradrenaline and she suffered a cardiac arrest past midnight. At the time, a bedside ECHO showed global left ventricular dysfunction with an ejection fraction of 15% and a significant haemoglobin drop to 5g/dl for which she was administered packed red cells and fresh frozen plasma. Haematology teams were consulted early during illness, and the patient was taken to ICU for therapeutic plasma

exchange but unfortunately passed away before this was commenced. ADAMTS13 testing was sent and the result came back positive weeks later. Despite resuscitative efforts, the patient passed away. An autopsy was not performed as per the request of the family.

Discussion

TTP is a medical emergency and must be treated in a timely fashion wherein earlier diagnosis is associated with good prognosis. However, these patients can rapidly decline due to thromboembolic sequelae associated with haemolytic anaemia and severe thrombocytopenia[5]. A review of the literature within the past five years identified 8 cases where patients had confirmed stroke in the setting of active TTP (Table 1). All patients survived with prompt plasma exchange and corticosteroids; however, our patient's rapid deterioration underscores the catastrophic potential of fulminant disease with concurrent renal and cardiac dysfunction. Clinical manifestations of cardiac involvement in TTP can vary by case but there is limited knowledge on how to optimally evaluate patients from a cardiac standpoint[6]. As well, it is generally unclear whether the patient's cardiovascular demise was related to thrombocytopenia and anaemia leading to ischemia-induced damage to cardiomyocytes and its vasculature or due to a thromboembolic aetiology[7].

Acute stroke with underlying acute TTP presents a very unique challenge in terms of acute management. A multi-disciplinary approach from both the stroke team and the haematology team is crucial for these cases. Acute TTP-associated stroke necessitates immediate TTP-directed therapies such as therapeutic plasma exchange (TPE) with the addition of steroids and/or rituximab, though around 15-32% of patients are refractory[8]. These are now complemented by newer targeted agents such as caplacizumab and recombinant ADAMTS13. Patients who receive caplacizumab, an anti-von Willebrand factor humanised, bivalent immunoglobulin fragment, experienced reduced needs for plasma exchange, higher likelihood of platelet normalisation and shorter hospitalisations in a double-blind controlled trial[9].

This case reinforces the importance of early consideration of TTP in stroke-like presentations accompanied by cytopenias. Clinicians should maintain a high index of suspicion, as immediate recognition and initiation of TTP-directed therapy are life-saving.

Declarations of Conflicts of Interest:
None declared.

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Appendix:

Figure 1: *Computerised Tomography of the brain, demonstrating attenuated right M2 and M3 vessels of the right middle cerebral artery.*

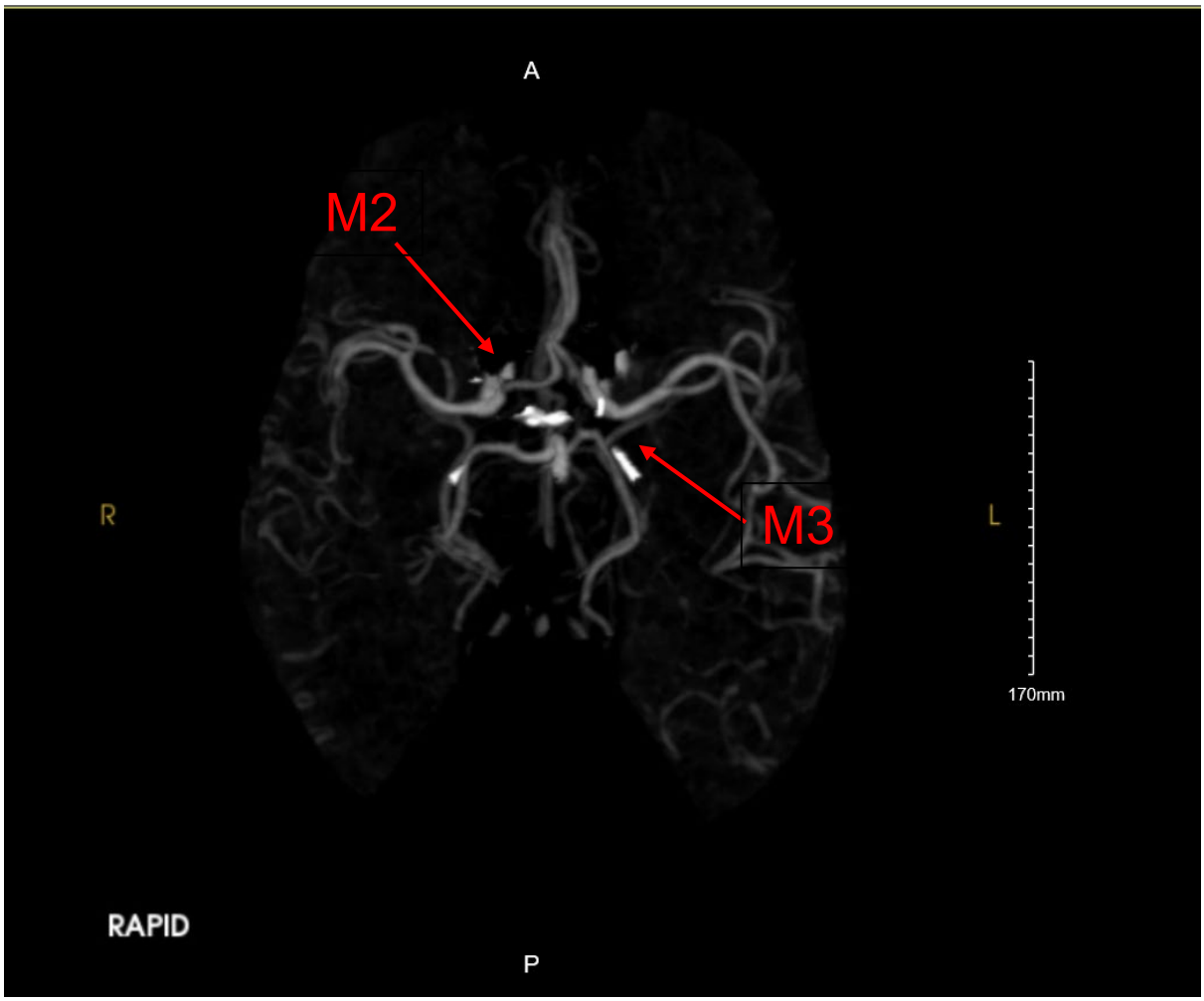

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Table 1: Review of case reports in the literature of stroke presentations with confirmed imaging in the setting of active TTP

First Author	Adimora I	Barreto F	Jameie M	Reichman N	Agyapong KO	Albo Z	Filatov	Ramesh
Year	2023	2024	2023	2025	2023	2022	2020	2023
Age	53	75	59	42	40	63	31	61
Gender	Female	Female (Madeira)	Male	Female	Female (AFR)	Female	Female	Male
P/C	3/7 dysequillibrium, nausea	Transient dysarthria, right arm paresis, labial commissure deviation to left, dysphagia, right headache	Left hemiparesis, dysarthria, and decreased consciousness	Sudden left hemiplegia	1/7 Slurred speech, facial asymmetry, left upper and lower limb weakness	Subacute dizziness, confusion, right-sided hemiparesis, sensory deficits	Incoherent speech, parasthesia	Right upper extremity weakness
NIHSS	N/A	N/A	10	N/A	N/A	6	N/A	N/A
PHX /FHX TTP or related symptoms	19 y/o presented with prolonged uterine bleed in 1st pregnancy s/p splenectomy 25 y/o recurrence in 2nd pregnancy - 48 y/o post chole	None	None	None	None specific	Migraines, hypertension, hyperlipidemia, Hyothyroidism, peripheral vascular disease	None	Intermittent headaches
PHx/FHx Stroke	1st: Left middle cerebral artery (MCA) 2020 –	None	None	Recent cryptogenic R superior MCA	2x strokes 5 years ago	None	None	Transient weakness in left upper

	patent foramen ovale closure 2nd: Right corona radiata 2020 3rd: Left cerebellum 2020 On apixaban			and L posterior pericallosal ischemic stroke w/ hemorrhagic conversion in R parietal and L medial occipital lobe				extremity 4 weeks ago
Platelets	216	26	77	38	18	14	4	98
Hemoglobin	12	10.4	15	12.6	4.3	N/A	6.6	11.7
Renal Profile	Normal	Abnormal	Normal	Normal	Normal	N/A	Normal	Abnormal
LDH	281 U/L	662	1148	N/A	261	N/A	1154	537
TBili (REVISE)	N/A	1.1	1.9mg/dl	N/A	19.86 umol/L	N/A	N/A	N/A
ADAMTS-13 ACTIVITY	<3%	N/A	<0.3 IU/ml	<5%	N/A	<5%	<5%	<5%
Blood Smear	<1 schistiocyte per high power field (HPF)	Presence of schistiocytes and microspherocytes	Occasional schistiocyte	N/A	Schistocytes ++ with occasional normoblast	N/A	Schistiocytes present	0-2 schistiocytes per HPF
CT-Brain	Nil Acute	Nil Acute (angiography)	Acute stroke in the right middle cerebral artery (MCA) territory, including the right	acute vessel imaging demonstrated irregularity and mild narrowing of the right posterior	Periventricular bleed, ischemic changes	N/A	N/A	Remote infarcts

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			parietotemporal lobe infarct	M2/M3 MCA branches without large vessel occlusion				
MRI-Brain	New ischemic changes in left anterior cerebral artery involving frontoparietal paramedium cortices	N/A	Subacute infarctions with restriction and mild hemorrhagic transformation in the right parietotemporal, left frontal lobe, and left centrum semiovale	Ischemic infarction in superior MCA and posterior pericallosal artery.	N/A	Acute infarct of L posterior cerebral artery territory involving the left medial temporal lobe, left occipital lobe, left thalamus	N/A	N/A
TTP Treatment	None	Plasmapharesis Steroids	Plasmapheresis Steroids Rituximab Caplacizumab	Plasmapheresis Steroids Rituximab	Plasmapheresis Steroids HD with calcium gluconate	Plasmapheresis Steroids	Plasmapheresis Steroids Rituximab	Plasmapheresis Steroids Rituximab
Outcome	Alive	Alive	Alive	Alive	Alive	Alive	Alive	Alive