

A Rare Case of Ischemic Stroke Secondary to Polycythemia with Rapid Resolution After Phlebotomy

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Introduction: Ischemic stroke secondary to polycythemia is a rare but serious complication, particularly in young adults. Early recognition and intervention can lead to rapid neurological recovery.

Case Presentation: A 21-year-old male from Western Uganda presented with sudden-onset right-sided hemiparesis and slurred speech. He had no prior history of thromboembolic events but reported chronic headaches and fatigue. Physical examination revealed facial asymmetry, right-sided weakness (power 3/5), and hyperviscosity signs. Laboratory tests confirmed polycythemia (Hb: 22 g/dL, Hct: 68%). Brain CT showed an acute left middle cerebral artery (MCA) infarct.

Management: Emergency phlebotomy (500 mL) was performed, followed by hydration and low-dose aspirin. The patient showed significant improvement within 48 hours, with near-complete resolution of symptoms at one-week follow-up.

Conclusion: Polycythemia-induced stroke, though rare, should be considered in young patients with unexplained thromboembolic events. Phlebotomy remains a rapid and effective treatment, especially in resource-limited settings.

Keywords: ischemic stroke, polycythemia, phlebotomy, hyperviscosity syndrome, young adult stroke, Uganda

Introduction

Stroke in young adults represents a growing global health concern, accounting for 10–15% of all stroke cases, with distinct etiological profiles compared to older populations.¹ While traditional vascular risk factors dominate in elderly patients, younger individuals frequently present with uncommon causes, including hematologic disorders such as polycythemia.² Polycythemia, characterized by elevated hemoglobin and hematocrit, increases thrombotic risk through hyperviscosity, platelet dysfunction, and impaired microcirculatory flow.³ The condition may be primary (polycythemia vera, a JAK2-mutated myeloproliferative neoplasm) or secondary (due to hypoxia, erythropoietin excess, or other stimuli).⁴

Ischemic stroke secondary to polycythemia is rare, particularly in sub-Saharan Africa, where infectious and cardioembolic etiologies predominate.⁵ In high-income countries, polycythemia vera accounts for 1–5% of young strokes, but data from Africa are scarce due to diagnostic limitations.⁶ Secondary polycythemia—often driven by chronic hypoxia from high-altitude residence, smoking, or undiagnosed cardiopulmonary disease—may be underrecognized in regions like Western Uganda.⁷ Without treatment, polycythemia carries a 1.8% annual risk of arterial thrombosis, with stroke being the most devastating complication.⁸

Phlebotomy remains first-line therapy for acute hyperviscosity, rapidly reducing thrombotic risk by lowering hematocrit.⁹ However, delays in diagnosis are common in resource-limited settings due to limited access to hematologic testing and neuroimaging.¹⁰ This case highlights the diagnostic challenges, therapeutic efficacy of phlebotomy, and unique considerations of secondary polycythemia in young African adults.

Case Presentation

The patient was a 21-year-old male subsistence farmer from the highland region of Western Uganda (approximate altitude 1500 meters above sea level) who presented to a Bwera General hospital emergency department with sudden-onset right-sided weakness and slurred speech that began 6 hours prior to admission. He reported no preceding trauma, seizure activity, or loss of consciousness. The neurological deficits developed abruptly while he was working in his field, with no identifiable triggering factors.

Further history revealed a 6-month prodrome of progressively worsening symptoms including constant throbbing headaches (predominantly occipital, worse in mornings), persistent dizziness, frequent epistaxis, and generalized fatigue that limited his ability to work. He denied any history of visual disturbances, tinnitus, paresthesias, or weight loss. There was no personal or family history of thromboembolic events, hematologic disorders, or premature cardiovascular disease. The patient was a lifelong non-smoker with minimal alcohol consumption and no use of recreational drugs or performance-enhancing substances. His diet was typical of the region, consisting mainly of plantains, beans, and vegetables, with occasional meat consumption.

On physical examination, the patient appeared plethoric with notable facial redness and injected conjunctivae. Vital signs showed blood pressure of 140/90 mmHg (no prior baseline measurements available), heart rate of 88 beats per minute, respiratory rate of 18 breaths per minute, and oxygen saturation of 98% on room air. Cardiovascular examination revealed normal heart sounds without murmurs or gallops, and peripheral pulses were symmetric and full. Respiratory examination was unremarkable with clear lung fields bilaterally.

Neurological assessment demonstrated right-sided upper motor neuron facial weakness with sparing of the forehead. Motor examination revealed right hemiparesis with Medical Research Council (MRC) grade 3/5 strength in the upper extremity (shoulder abduction, elbow flexion/extension, hand grip) and grade 4/5 in the lower extremity (hip flexion, knee extension, ankle dorsiflexion). Muscle tone was slightly increased on the right side with hyperreflexia (3+) and a positive Babinski sign. Sensory examination was normal to light touch, pinprick, and vibration throughout. Cerebellar testing showed no dysmetria or intention tremor. The patient had moderate dysarthria but no evidence of aphasia or neglect.

Initial laboratory evaluation revealed striking abnormalities in his complete blood count: hemoglobin 20.6 g/dL (reference range 13–17), hematocrit 67.1% (40–50), RBC 55×10^{12} white blood cell count $12 \times 10^3/\mu\text{L}$ (4–11) with normal differential, and platelets $450 \times 10^3/\mu\text{L}$ (150–450). Peripheral blood smear showed marked erythrocytosis without leukoerythroblastosis or significant poikilocytosis. Basic metabolic panel was unremarkable with normal renal function and electrolytes. Liver function tests were within normal limits. Coagulation studies including prothrombin time and activated partial thromboplastin time were normal. C-reactive protein was mildly elevated at 8 mg/L (normal <5).

Given the clinical presentation of acute focal neurological deficits in the setting of severe polycythemia, an urgent non-contrast computed tomography (CT) scan of the brain ([Figure 1](#)) was obtained, which demonstrated a hypodense area in the left middle cerebral artery territory consistent with an acute ischemic stroke. No hemorrhage or mass effect was noted. Due to resource limitations, advanced neuroimaging such as MRI or CT angiography was not immediately available.

Additional diagnostic workup was initiated to determine the etiology of the polycythemia. JAK2 V617F mutation testing (performed by PCR) was negative, making polycythemia vera less likely. Serum erythropoietin level was elevated at 35 mIU/mL (normal 4–29), supporting a diagnosis of secondary polycythemia. Arterial blood gas analysis on room air showed pH 7.38, pO_2 72 mmHg, pCO_2 38 mmHg, and oxygen saturation 94%. Chest radiograph was normal without evidence of parenchymal lung disease or cardiomegaly. Abdominal ultrasound revealed no splenomegaly or renal abnormalities.

Treatment and Follow-Up

Emergency therapeutic phlebotomy (500 mL) was performed with concurrent IV hydration. Low-dose aspirin (75 mg daily) was started alongside supportive measures including bed rest, DVT prophylaxis, and supplemental oxygen. Within 24 hours, the patient's dysarthria improved significantly, and by 48 hours, right upper extremity strength increased to 4/5,

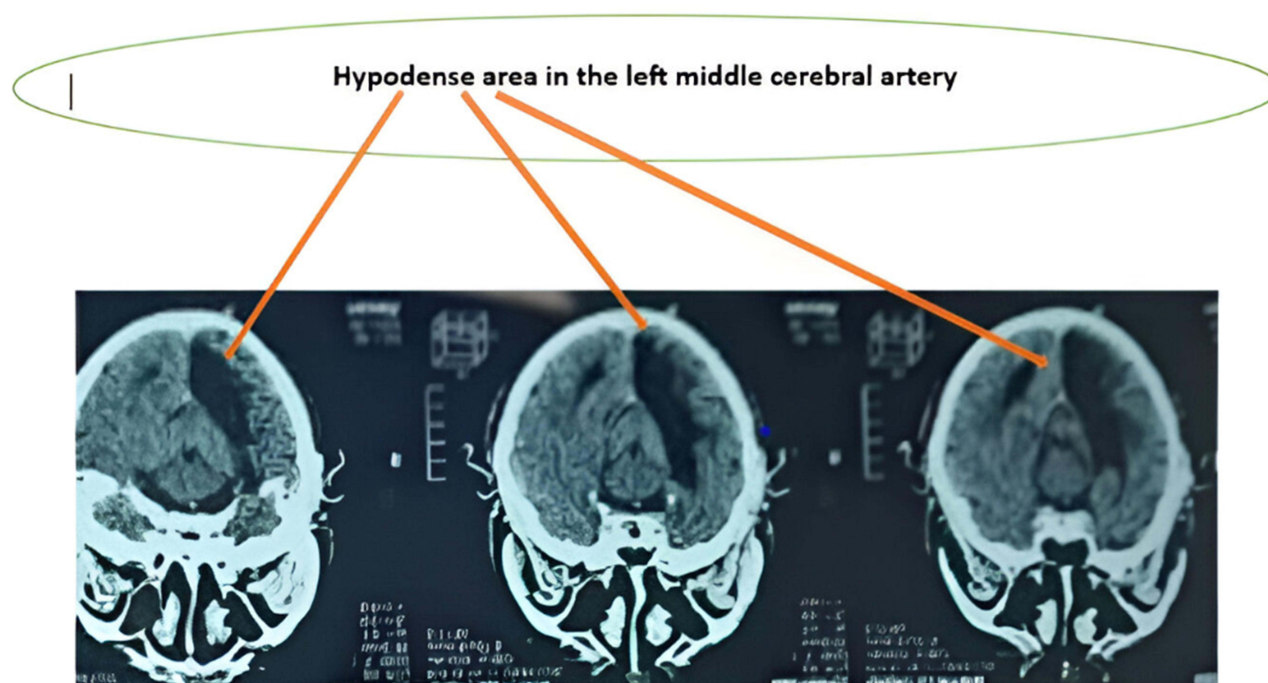


Figure 1 CT scan demonstrated a hypodense area in the left middle cerebral artery territory consistent with an acute ischemic stroke.

with hematocrit dropping to 58%. At one-week, neurological deficits had nearly resolved (MRC 5-/5) after a second phlebotomy (250 mL), with hematocrit at 48%. By one month, the patient was asymptomatic with a stable hematocrit of 45% (Table 1). He was referred for further evaluation of secondary polycythemia causes, with long-term management including maintenance phlebotomy (target Hct <45%) and continued antiplatelet therapy.

Discussion

This case illustrates several critical aspects of polycythemia-associated stroke in young adults, particularly in resource-limited settings. The patient's rapid neurological improvement following phlebotomy underscores the direct role of hyperviscosity in stroke pathogenesis. Studies demonstrate that hematocrit levels >55% increase stroke risk exponentially due to impaired cerebral perfusion and platelet activation.^{3,8} Our patient's hematocrit of 68% placed him at extreme thrombotic risk, consistent with prior reports linking severe erythrocytosis to large-vessel ischemic stroke.¹¹

The absence of JAK2 mutation and elevated erythropoietin confirmed secondary polycythemia, likely from chronic hypoxia due to high-altitude adaptation (Western Uganda averages 1500m elevation).¹² Similar cases have been reported

Table 1 Timeline of Hematocrit Levels, Phlebotomy Interventions, and Neurological Recovery in the Patient with Ischemic Stroke Secondary to Polycythemia

Timepoint	Hematocrit (%)	Intervention	Neurological Status
Admission	67.1	None	Right hemiparesis, dysarthria
24 hrs	58	500 mL phlebotomy	Speech improved, limb strength ↑
1 week	48	250 mL phlebotomy	Nearly full recovery
1 month	45	Maintenance monitoring	Asymptomatic

Notes: ↑, increased limb strength.

in Andean and Himalayan populations but are rarely documented in Africa.¹³ This gap may reflect underdiagnosis rather than true epidemiological differences, as hematologic workups for young stroke patients are often incomplete in low-resource settings.^{5,10}

Notably, the patient's rapid clinical improvement mirrors findings from a 2023 systematic review, where 78% of polycythemia-related stroke patients showed significant recovery within 72 hours of phlebotomy.¹⁴ This supports the theory that hyperviscosity-induced strokes often involve reversible hemodynamic insufficiency rather than irreversible thrombosis.¹⁵ However, our patient's MCA infarct on CT suggests some thrombotic component, emphasizing the need for combined antiplatelet therapy alongside viscosity reduction.¹⁶

Diagnostic challenges in this case reflect broader limitations in sub-Saharan Africa. While high-income countries routinely employ MRI, CT angiography, and genetic testing for young strokes,¹⁷ our diagnosis relied on clinical suspicion, basic labs, and non-contrast CT. This aligns with data from the Stroke Investigative Research and Educational Network (SIREN), which found that 62% of young African stroke patients lack access to advanced etiologic workups.¹⁸

Therapeutic implications are significant. Phlebotomy's efficacy in this resource-limited setting aligns with 2022 World Health Organization (WHO) guidelines for managing hyperviscosity syndromes where cytoreductive drugs are unavailable.¹⁹ However, long-term hematocrit control remains challenging without regular monitoring. Studies in similar settings advocate for community-based hematocrit screening in high-altitude regions to prevent complications.²⁰

Although secondary polycythemia was the leading cause in this patient, alternative stroke etiologies in young adults must be considered. Paroxysmal atrial fibrillation is a recognized source of embolic stroke, and prolonged cardiac monitoring or echocardiography could help exclude this possibility. Vascular imaging (CTA/MRA) may identify stenosis or arterial dissection, particularly in high-altitude populations where endothelial dysfunction is prevalent. Inflammatory vasculitides, including Takayasu arteritis and primary CNS vasculitis, should also be excluded through autoimmune panels (ANA, ANCA) and CSF analysis where feasible. Finally, inherited thrombophilias such as Factor V Leiden mutation and antiphospholipid syndrome remain important considerations in recurrent or unexplained strokes. Unfortunately, most of these advanced tests were unavailable in our setting, underscoring diagnostic limitations in resource-constrained hospitals.

Conclusion

This case highlights polycythemia as a rare but treatable cause of ischemic stroke in young adults, particularly in high-altitude populations. The dramatic response to phlebotomy underscores the importance of early recognition and intervention in hyperviscosity syndromes. In resource-limited settings, simple diagnostic approaches and therapeutic phlebotomy can yield excellent outcomes. Increased awareness of hematologic causes of stroke among young adults in Africa is needed, along with improved access to basic diagnostic testing. Secondary prevention should focus on maintaining normovolemia and adequate hematocrit control through periodic phlebotomy when needed.

Ethical Approval

Ethical clearance for this case report was obtained from the Ethics Committee at Bwera General Hospital (Approval Number: BGH-EC-2025-20-3).

Consent for Publication

Written informed consent was obtained from the patient for the publication of this case report and any accompanying images.

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Disclosure

The authors declare no conflicts of interest related to this study.

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