

Young, Unexpected, and Ischemic: A Case Series of Varied Stroke Presentations

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Abstract

Stroke in young adults represents a distinct clinical entity with diverse and often unconventional etiologies. Unlike older populations where atherosclerosis predominates, young stroke frequently arises from cardioembolic, genetic, autoimmune, or vascular structural causes. Early identification of these conditions is critical to prevent recurrence and long-term disability. This case series illustrates the varied clinical presentations, diagnostic challenges, and underlying mechanisms of stroke in young individuals. Our findings highlight the necessity for a thorough etiologic workup—including advanced imaging and targeted laboratory evaluation—to uncover rare but actionable causes. Improved recognition of these atypical contributors enables personalized management strategies and better functional outcomes in this vulnerable population.

Keywords: Young stroke; Early-onset stroke; Unusual stroke etiology; Neurovascular disorders; Stroke in young.

1. Introduction

Stroke in young adults — typically defined as occurring under the age of 50 years — represents a distinct and increasingly recognised cerebrovascular disorder that differs substantially from the classic stroke presentations in older populations [1]. Historically, stroke was considered largely a disease of the elderly, but recent epidemiologic data indicate that approximately 10-15 % of all stroke cases occur in young adults, and in some regions even higher proportions have been reported. The mean age of onset in these populations is often in the fourth decade, though patients in their 20s and 30s are not uncommon.

Young-onset stroke presents unique clinical, etiologic and prognostic challenges. The etiological spectrum in younger patients is broader: in addition to conventional vascular risk factors (such as hypertension, diabetes mellitus and dyslipidaemia), young stroke is more often associated with non-atherosclerotic mechanisms such as arterial dissection, genetic vasculopathies, coagulopathies, and cardioembolic causes in the absence of overt vascular disease [2]. Moreover, the long-term social and economic impact of stroke at a younger age is profound — patients often live many decades post-event and may suffer significant disability during their most productive years.

Genetic and structural vascular contributors are gaining increasing attention in this cohort. Monogenic disorders, inherited arteriopathies and rare thrombophilic states may underlie otherwise cryptogenic strokes in the young, and advances in genetics and vascular imaging have begun to expand this landscape. Comprehensive etiologic evaluation — including advanced imaging, cardiac assessment, vascular imaging and prothrombotic work-up — is therefore essential to refine diagnosis, personalise management and potentially improve outcomes in young stroke.

In this case series, we describe a cohort of young adults presenting with stroke, delineate their clinical and etiologic heterogeneity, and highlight the value of a systematic and expansive diagnostic approach. Recognition of unusual or rare stroke mechanisms in younger adults may enhance diagnostic precision, influence therapeutic decisions and ultimately improve long-term prognosis.

2. Aim and Objectives

Aim:

To describe the clinical and etiological spectrum of stroke in young adults through a series of cases with diverse underlying mechanisms.

Objectives:

1. To document the clinical presentation, risk factors, and diagnostic findings in young stroke patients.
2. To analyze the etiological heterogeneity, including uncommon and non-atherosclerotic causes of stroke in the young population.
3. To explore the relevance of advanced imaging, laboratory evaluation, and cardiac work-up in identifying the underlying mechanisms.
4. To emphasize the importance of comprehensive evaluation for early diagnosis, personalized management, and prevention of recurrence in young stroke patients

3. Review of Literature

Stroke in young adults – often defined as stroke occurring before the age of 50 years – represents a distinct cerebrovascular entity with epidemiological, etiological and prognostic features that differ markedly from stroke in older age groups [3]. Globally, young-onset stroke accounts for approximately 10–15% of all stroke cases [4]. The incidence appears to be increasing in many regions, possibly driven by rising prevalence of vascular risk factors in younger populations [3].

From an epidemiologic standpoint, studies have reported incidence rates of first-ever stroke in young adults ranging from about 5 to 40 cases per 100,000 persons per year, though these figures vary widely depending on age-cutoff, region, ethnicity and study design [4]. Traditional vascular risk factors such as hypertension, dyslipidaemia, smoking and diabetes are increasingly observed in young stroke populations, though their relative impact differs from older cohorts [5].

Etiologically, the spectrum of stroke in young adults is broad and heterogeneous. In contrast to older patients where large-artery atherosclerosis and small-vessel disease dominate, younger patients more often present with non-atherosclerotic mechanisms including cervical arterial dissection, cardioembolism (often related to congenital or structural heart disease), prothrombotic conditions, autoimmune vasculopathies, migraine with aura, substance abuse, and inherited vascular disorders (such as CADASIL) [6]. Notably, arterial dissection has been reported in up to 20–25% of young ischemic stroke cases in some series [8]. Moreover, despite advances in diagnostics, a large proportion of young stroke cases remain classified as cryptogenic – that is, without a definite identifiable cause – which underscores the diagnostic challenge in this group [7].

Genetic and structural vascular contributors are increasingly recognized in younger stroke cohorts. Monogenic causes, inherited arteriopathies and rare thrombophilias may underlie apparently idiopathic strokes in young adults [10]. In particular, the role of advanced imaging, extended cardiac monitoring, and comprehensive prothrombotic/genetic testing is emphasised in the evaluation of young stroke, to uncover these less-common but actionable mechanisms [8].

Clinically and prognostically, stroke in the young carries distinct implications. Although younger patients often have better immediate outcomes compared to older stroke survivors, the long-term burden – both personal and societal – is substantial. Young survivors may face decades of disability, recurrent events, and significant socioeconomic impact [5]. The presence of modifiable risk-factors in young stroke also highlights the opportunity for prevention and risk-factor modification at an earlier age [8].

Despite the growing body of literature, many gaps remain. There is heterogeneity in how “young” is defined, regional variations in incidence and etiology, variable diagnostic work-up completeness, and limited long-term follow-up data in young stroke cohorts [11]. More research is needed to establish standardized evaluation protocols, clarify genotype–phenotype links (in inherited causes), and optimize secondary prevention specifically tailored for young adults.

4. Material and Methods

CASE 1

An 18-year-old male, previously healthy with no known comorbidities, presented with sudden onset weakness of the right upper limb and right lower limb lasting 4 hours. There was no history of slurred speech, facial deviation, loss of consciousness, seizures, headache, or trauma.

On neurological examination, the right upper limb power was 4-/5, and the right lower limb power was 3/5, indicating mild to moderate motor deficit. Other cranial nerve examinations were normal. The NIHSS score was **6, suggestive of a moderate stroke severity.**

Given the acute focal neurological deficits, he underwent an MRI of the brain, which revealed multiple areas of diffusion restriction involving the left cingulate cortex and the left occipital lobe, consistent with acute ischemic infarcts.

As the patient presented within the therapeutic window and had no contraindications, he was thrombolysed with intravenous Tenecteplase.

Based on clinical presentation and imaging findings, he was diagnosed with acute ischemic stroke presenting as right hemiparesis, categorized as stroke in the young. Further evaluation for underlying etiologies—such as cardioembolism, arterial dissection, thrombophilia, vasculitis, or substance use—was planned as part of the young-stroke workup.

CASE 2

A 35-year-old male, with a significant past history of cerebrovascular accident (CVA) 6 months earlier and multiple substance abuse behaviors—including chronic cocaine use—presented to the emergency department with acute onset neurological deficits.

He developed sudden weakness of the left upper and lower limbs, accompanied by slurring of speech and deviation of the angle of the mouth to the right, indicating an acute right hemispheric event.

On arrival, he was alert, with muscle strength graded as 3/5 in the affected left-sided limbs with right UMN facial palsy. Based on clinical presentation and imaging findings, he was diagnosed with acute ischemic stroke presenting as left hemiparesis, categorized as stroke in the young.

CASE 3

A 31-year-old female presented to the emergency department with acute onset giddiness that began abruptly a few hours prior to admission. The giddiness was described as a spinning sensation associated with multiple episodes of non-bilious vomiting. The symptoms started suddenly while at rest and were persistent, prompting medical attention. There was no history of dysarthria, drooping of eyelids, deviation of angle of the mouth, or altered facial sensations. She denied weakness, numbness, or incoordination of the upper or lower limbs. There were no gait disturbances, no preceding unsteadiness, and no classical cerebellar symptoms such as dysmetria, dysidiadochokinesia, or intention tremor. There was no history suggestive of seizures, headache, loss of consciousness, or head trauma.

Her past medical history was significant for a cerebrovascular accident in 2023, diagnosed clinically and radiologically as a left lateral medullary (Wallenberg) syndrome. She had recovered substantially from the previous event and had no residual neurological deficits. She was on regular follow-up and reported adherence to secondary stroke prevention medications.

On arrival, the patient was conscious, oriented, and hemodynamically stable. General physical examination was unremarkable. Neurological evaluation showed a right internuclear ophthalmoplegia (INO) characterized by impaired adduction of the right eye on horizontal gaze, with abducting nystagmus noted in the contralateral (left) eye. Pupillary reactions were preserved. There were no features suggestive of cranial nerve involvement apart from the ocular movement abnormality. Fundus examination was normal, and there were no signs of papilledema.

Her speech was normal, without dysarthria or dysphonia. Motor system examination revealed normal tone and full muscle power (Grade 5/5) in all four limbs. Deep tendon reflexes were present and symmetrical, and plantar responses were flexor bilaterally. Sensory system examination was intact to all modalities. There were no cerebellar signs, and gait could not be tested initially due to ongoing giddiness.

Given the acute vestibulo-ocular symptoms, presence of a definite brainstem sign (INO), and the patient's prior history of a posterior circulation stroke, a provisional diagnosis of recurrent ischemic posterior circulation stroke in a young adult was considered. The sudden onset, associated vomiting, and isolated ocular movement deficit raised suspicion for involvement of the medial longitudinal fasciculus (MLF) or adjacent pontine/mesencephalic structures.

In view of her young age and previous stroke, the clinical picture also prompted evaluation for stroke in the

young, including cardioembolic, vasculitic, prothrombotic, and arterial dissection etiologies. Immediate neuroimaging (MRI brain with diffusion-weighted imaging and MR angiography) and detailed stroke workup were warranted.

CASE 4

A 40-year-old male, previously healthy with no known comorbidities, presented with acute-onset giddiness accompanied by weakness of the left upper and lower limbs. There was no history of loss of consciousness, headache, seizures, visual disturbances, or speech difficulty.

On examination, the patient was conscious, alert, and fully oriented. Motor system evaluation showed reduced tone on the left side. And power 4/5 in left side with reduced reflexes and hemisensory loss

Given the combination of left-sided motor weakness and hemisensory loss, the overall clinical picture was indicative of a posterior circulation involvement, raising concern for a cerebrovascular event.

In view of the patient's age and recurrent presentation, a provisional diagnosis of recurrent cerebrovascular accident — stroke in the young was made, and further evaluation for underlying etiologies such as vertebrobasilar insufficiency, arterial dissection, cardioembolic sources, vasculitis, hypercoagulable states was planned.

Case 5

A 31-year-old right-handed female, a homemaker with no known comorbidities, and no significant family history or past history of stroke, presented with weakness of the right upper and lower limbs of 1-day duration. The weakness was sudden in onset and progressive, with no associated sensory symptoms. Her speech remained normal, and there was no history of facial deviation, headache, seizures, visual disturbances, or loss of consciousness.

Reviewing her past medical history, there was no history of arthritis, connective tissue disease, miscarriages, or the use of oral contraceptive pills, all of which may predispose to stroke in young.

On examination, the patient was conscious and oriented to time, place, and person. Cranial nerve examination was normal.

Motor system examination revealed Hypotonia of the right upper limb and right lower limb, Motor power of 4/5 in both the right upper and lower limbs, No involuntary movements or abnormal posturing. Sensory system examination was normal, with intact modalities. Cerebellar signs were unremarkable, with no dysmetria or coordination deficits. The examination of other systems, including cardiovascular and respiratory systems, was within normal limits. Based on the acute focal motor deficit, in a young adult with no vascular risk factors, the working diagnosis is acute ischemic stroke presenting as right hemiparesis — stroke in the young, warranting further evaluation.

Case 6

A 30-year-old male with h/o patent foramen ovale presented with acute onset left-sided weakness involving both the upper and lower limbs. The weakness was sudden in onset, noted on waking/while performing routine activities, and was progressive over minutes to hours. There was no associated loss of consciousness, seizures, headache, or vomiting at onset.

The patient also reported difficulty in using the left hand for fine tasks and clumsiness while walking, consistent with a motor deficit. Additionally, he described impaired ability to recognize objects by touch, difficulty localizing touch, and trouble appreciating joint position on the affected side, suggestive of cortical sensory loss. No visual aura or transient monocular blindness was reported.

There was no history of speech disturbances (aphasia, dysarthria); and no sensory symptoms on the right side. No bladder or bowel involvement.

On examination, the patient was conscious, alert, and oriented. Cranial nerves were apparently normal. Motor examination revealed left-sided hemiparesis, more pronounced distally, with reduced muscle tone and power %. Sensory examination showed deficits predominantly in higher cortical modalities on the left side, including agrophesthesia, astereognosis, impaired two-point discrimination, and loss of proprioception, while primary modalities were relatively preserved, indicating parietal cortical involvement.

5. Results (Including Observations)

Case 1

Routine baseline laboratory evaluation showed normal CBC, renal and liver function tests, ESR, and CRP. ECG

and 2D echo normal. MRI brain showed multiple areas of diffusion restriction seen in left centrum semiovale, corona radiata, left parietooccipital and cerebellar hemisphere. Chest X-ray was normal. Etiology Screening was positive lupus anticoagulant and positive anti-cardiolipin IgG and IgM antibodies, while β 2-glycoprotein IgG and IgM were negative. Rheumatologist opinion was taken and was started anticoagulant

Case 2

Routine stroke-in-young evaluation was carried out. Initial CT brain was normal. MRI brain showed infarct in corona radiata. Basic laboratory investigations including CBC, ESR/CRP, fasting glucose, HbA1c, fasting lipid profile, renal and liver function tests, and thyroid profile were within normal limits. 2D echocardiography was normal without evidence of a cardioembolic source. A complete prothrombotic panel was done during his previous stroke including antiphospholipid antibody testing (lupus anticoagulant, anti-cardiolipin, anti- β 2GP1), Protein C, Protein S, Antithrombin III, Factor V Leiden mutation, and homocysteine levels was negative. Autoimmune and inflammatory screening including ANA, ANCA, complement levels, panel was negative, and CRP normal, making systemic vasculitis unlikely. Infectious screening including HIV, syphilis serology, and hepatitis B and C markers was also negative. In the absence of other etiologies, the stroke was attributed to cocaine-induced ischemic stroke

Case 3

Routine stroke-in-young evaluation was performed. MRI brain showed acute infarct in right hemispheres and left globus pallidus. Basic laboratory panel including CBC, ESR/CRP, fasting glucose, HbA1c, fasting lipid profile, renal and liver function tests, and thyroid profile were normal. 2D echocardiography showed no cardiac source of embolism. A prothrombotic and hypercoagulable work-up including antiphospholipid antibody panel (lupus anticoagulant, anti-cardiolipin, anti- β 2GP1), Protein C, S, Antithrombin III, Factor V Leiden mutation, Prothrombin G20210A mutation, and homocysteine level was done, in which Protein S was found to be deficient while the rest were normal. Autoimmune panel including ANA, ANCA, complement levels and CRP remained normal throughout evaluation. Infectious screening including HIV, syphilis (VDRL), and hepatitis B and C serology was negative. Rheumatology opinion was obtained in view of Protein S deficiency, and the patient was discharged in stable condition

Case 4

Routine laboratory tests including CBC, renal and liver function tests, ESR, CRP, fasting glucose, HbA1c, and fasting lipid profile were normal. MRI brain showed infarct in right gangliothalamic region. Chest X-ray and 2D echocardiography, holter monitoring were normal. Thrombophilia screening including Protein C, Protein S, and Factor V Leiden mutation was negative. Autoimmune testing including ANA and ANCA, antiphospholipid antibody panel, homocysteine, complement level, Prothrombin G20210A mutation, infectious screening was also negative. With all investigations unremarkable, he was diagnosed cryptogenic stroke and was treated with antiplatelet and statins.

Case 5

Routine laboratory tests including CBC, renal and liver function tests, ESR, CRP, fasting glucose, HbA1c, and fasting lipid profile were normal. Chest X-ray revealed evidence of straightening of right heart border but 2D echocardiography were normal. FLP was abnormal with dyslipidemia. MRI revealed infarct in the left corona radiata. On the 4th day of course in hospital patient developed chest pain and palpitations for which ECG was done which was normal, hence proceeded with holter monitoring which revealed evidence of Paroxysmal supra ventricular tachycardia as the etiology of ischemic stroke

Case 6

Routine laboratory investigations, including complete blood count, renal and liver function tests, ESR, CRP, fasting glucose, HbA1c, and fasting lipid profile, were all within normal limits. A chest X-ray was also normal. The patient was a known case of patent foramen ovale (PFO) and had previously been advised to continue antiplatelet therapy, which he had discontinued for the past two years. In view of his recent ischemic stroke, he was again counselled regarding definitive management with PFO closure; however, the patient declined surgical intervention

6. Discussion

Stroke in young adults, defined as occurring in individuals under 45 years of age, is an increasingly recognized clinical entity with distinct etiologies and risk factors compared to older populations[12]. Unlike conventional stroke, which is primarily associated with atherosclerosis and traditional vascular risk factors, stroke in the young often involves uncommon mechanisms such as cardiac anomalies, thrombophilic disorders, vasculitis, substance abuse, and genetic predispositions[13]. Early recognition and etiological evaluation are critical for secondary prevention and long-term functional outcomes[14,15].

While strokes in the young are less common than in older adults, they carry a significant burden due to potential long-term disability[16]. Etiologies are heterogeneous and often multifactorial[17]. In this series, we observed diverse causes including: Cardioembolism: patent foramen ovale (PFO) and paroxysmal supraventricular tachycardia (PSVT), Prothrombotic states: Protein S deficiency and antiphospholipid antibody syndrome, Substance-induced ischemia: chronic cocaine use, Cryptogenic stroke: cases where extensive evaluation revealed no identifiable cause. Patients presented with a spectrum of neurological deficits depending on the vascular territory involved: Anterior circulation strokes: Sudden-onset hemiparesis (Cases 1, 2, 5, 6), often affecting upper and lower limbs asymmetrically. Some patients had associated facial palsy and slurred speech (Case 2). Posterior circulation strokes: Acute vertigo, vomiting, and ocular motor deficits (Cases 3 and 4), including internuclear ophthalmoplegia (INO) and cerebellar signs. Neurological examination typically revealed focal motor deficits graded between 3/5 and 4/5, with corresponding cranial nerve involvement in select posterior circulation strokes. Cognitive and sensory deficits were observed in cortical strokes, with higher cortical sensory loss evident in one case (Case 6). Stroke severity scores (e.g., NIHSS) ranged from mild to moderate. Diagnostic Evaluation includes all patients underwent thorough clinical and laboratory evaluation to identify potential underlying causes: Neuroimaging: MRI brain with diffusion-weighted imaging revealed acute infarcts in diverse regions including the corona radiata, cingulate cortex, occipital lobe, gangliothalamic region, cerebellar hemisphere, pons, and globus pallidus. MR angiography was performed in posterior circulation strokes to assess vertebrobasilar and intracranial arteries. Cardiac assessment: 2D echocardiography, Holter monitoring, and evaluation for PFO or arrhythmias identified cardioembolic sources in select cases. Laboratory evaluation: Routine hematology, biochemistry, lipid profile, and fasting glucose were largely normal. Thrombophilia workup revealed Protein S deficiency in one patient and positive antiphospholipid antibodies in another. Autoimmune and inflammatory panels (ANA, ANCA, complement levels, ESR/CRP) were negative in most cases. Infectious screening (HIV, hepatitis B/C, syphilis) was unremarkable. Substance use: Chronic cocaine exposure was implicated in one patient after exclusion of other causes. Management includes Acute management followed standard stroke protocols: Thrombolysis: Patients presenting within the therapeutic window without contraindications received intravenous thrombolysis (e.g., Tenecteplase, Case 1). Secondary prevention: Based on etiology, patients were started on antiplatelets, anticoagulants, or statins. Cardiac arrhythmias were managed with rhythm control as appropriate. PFO closure was advised in the relevant patient, although declined in one case. Lifestyle modification and substance cessation counseling were provided where indicated. Rehabilitation included physiotherapy, occupational therapy, and, in cases with posterior circulation involvement, vestibular and ocular motor training. Multidisciplinary follow-up ensured optimization of neurological recovery and prevention of recurrent events.

This case series highlights the diverse etiologies of stroke in young adults. Notably: Cardioembolic and structural heart disease (PFO, PSVT) remain important contributors even in the absence of traditional vascular risk factors. Prothrombotic disorders (Protein S deficiency, antiphospholipid antibody syndrome) should be systematically evaluated in young patients with cryptogenic strokes. Substance-induced ischemia, particularly with cocaine use, represents a preventable cause of stroke. Posterior circulation strokes may present subtly with isolated vertigo or ocular motor deficits, underscoring the need for high clinical suspicion and early imaging. The heterogeneous presentation and multifactorial etiology of stroke in young adults necessitate comprehensive evaluation, including neuroimaging, cardiac assessment, thrombophilia and autoimmune workup, and substance use history. Early diagnosis and targeted management can significantly reduce morbidity and prevent recurrence.

Conclusion

Stroke in young adults is a multifaceted clinical entity with distinct etiologies and varied presentations. Identification of underlying causes, prompt acute management, and individualized secondary prevention are crucial to optimize outcomes. Our case series emphasizes the importance of systematic evaluation for

prothrombotic, cardioembolic, substance-induced, and cryptogenic strokes in this population, with multidisciplinary care forming the cornerstone of long-term management.

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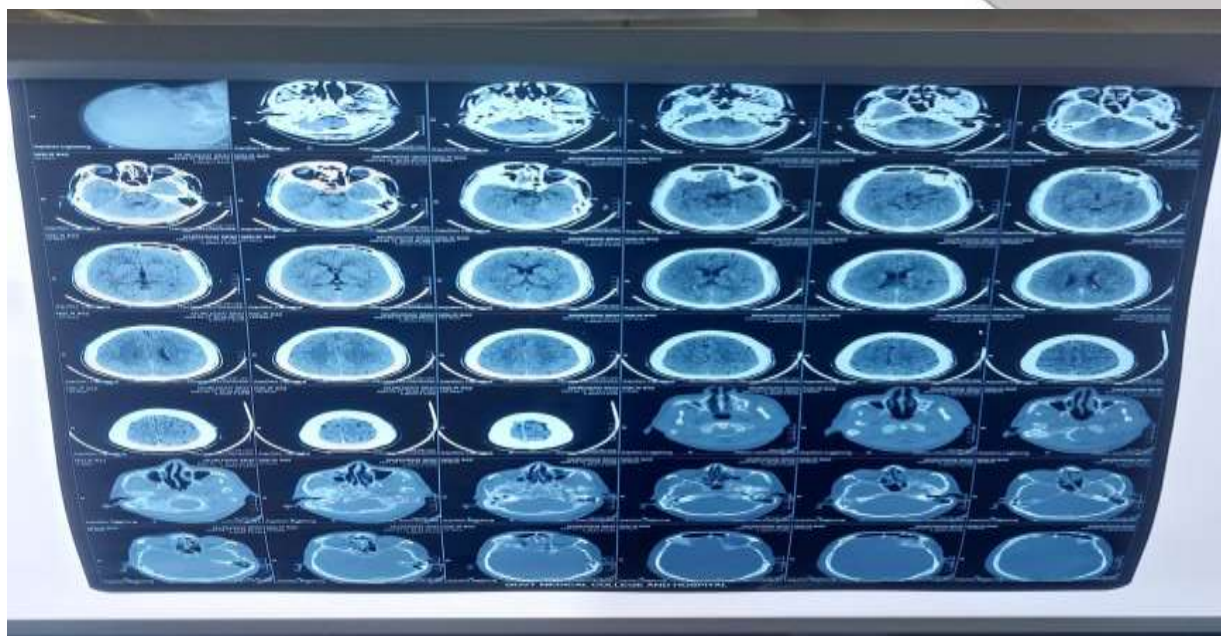
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8. Figures and Illustration

Case 1



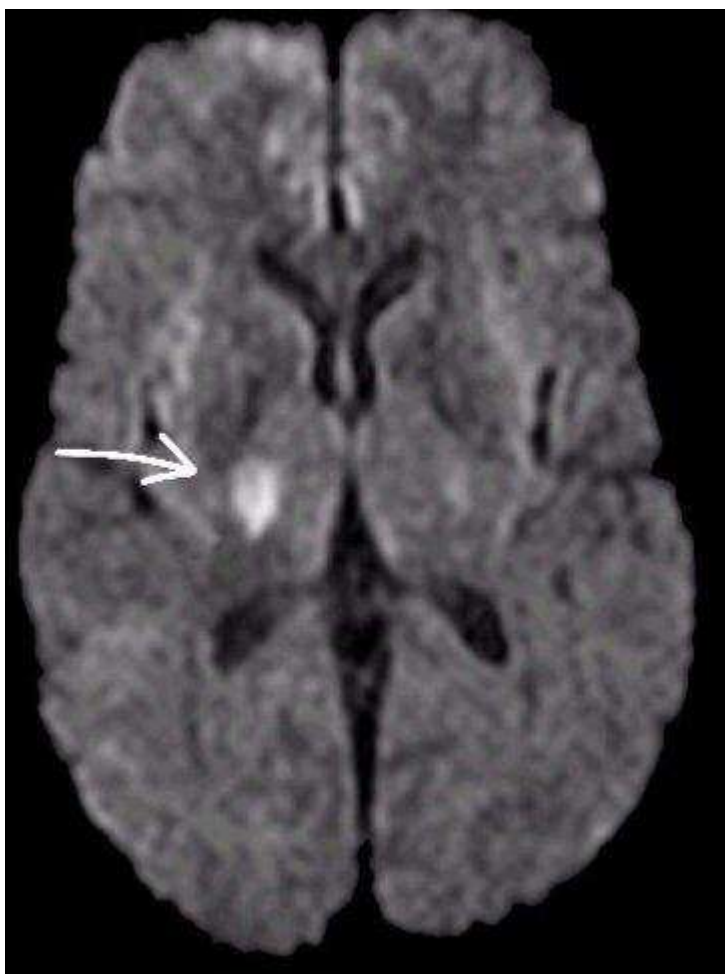
Case 2



Case 3



Case 4



Case 5

A 31-year-old right-handed female, a homemaker with no known comorbidities, and no significant family history or past history of stroke, presented with weakness of the right upper and lower limbs of 1-day duration. The weakness was sudden in onset and progressive, with no associated sensory symptoms. Her speech remained normal, and there was no history of facial deviation, headache, seizures, visual disturbances, or loss of consciousness.

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Case 6

