Case Report

Thrombo-occlusive left lateral medullary syndrome (Wallenberg syndrome) with iron deficiency anemia: A case report with clinical insights

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ABSTRACT

Left lateral medullary syndrome (Wallenberg syndrome) is a rare ischemic stroke caused by infarction in the lateral medulla, typically presenting with vertigo, ataxia, and sensory deficits. This case reports a rare presentation of Wallenberg syndrome in a 48-year-old female with coexisting severe iron deficiency anemia (IDA). The patient presented with acute dizziness, vomiting, left-sided ptosis, and contralateral limb weakness. Magnetic resonance confirmed infarcts in the left lateral medulla and cerebellum, as well as the anterior capsulo-ganglionic region, explaining the atypical presentation of limb weakness. Angiography revealed complete thrombo-occlusion of the left vertebral artery. Laboratory evaluation identified significant IDA as a potential exacerbating risk factor. The patient was managed with antiplatelets, statins, and intravenous iron replacement, resulting in significant neurological recovery. This case underscores the importance of evaluating IDA as a potential contributing factor in ischemic stroke, particularly in women without traditional vascular risk factors.

Key words: Iron deficiency anemia, Ischemic stroke, Posterior inferior cerebellar artery, Vertebral artery occlusion, Wallenberg syndrome

Wallenberg syndrome, first diagnosed by Vieusseux in 1808 and fully described by Wallenberg in 1895, is a neurological condition caused by ischemia in the lateral medulla oblongata [1]. It is most commonly caused by obstruction of the posterior inferior cerebellar artery or the vertebral arteries [2]. The obstruction causes an infarction of critical brain structures such as the spinothalamic tract, the trigeminal nerve nucleus, the inferior cerebellar peduncle, and the vestibular nuclei, resulting in the syndrome's distinguishing symptoms [1,2]. Wallenberg syndrome accounts for 2–3% of all ischemic strokes and typically affects people aged 40–70. While major risk factors include hypertension and atherosclerosis, iron deficiency anemia (IDA) has been increasingly recognized as a potential risk factor for ischemic events, particularly in younger women [3]. Mechanisms such as reactive thrombocytosis and hyperviscosity associated with IDA may contribute to a pro-thrombotic state [4].

We report this case to highlight a rare "Wallenberg-Plus" presentation accompanied by significant limb weakness and to emphasize the clinical relevance of identifying and treating coexisting anemia in stroke patients [5].

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CASE REPORT

A 48-year-old female with no prior medical history presented to the hospital approximately 5 h after the onset of symptoms. She reported sudden-onset dizziness followed by 4–5 episodes of non-bilious vomiting. Her relatives reported left eye drooping (ptosis) and rightward deviation of the mouth that started the same morning.

On neurological examination, she was conscious and oriented, with stable vital signs (pulse rate 82 bpm, blood pressure 120/80 mmHg, respiratory rate 20/min, SpO_2 96%, afebrile). A formal National Institutes of Health Stroke Scale score was not calculated at the time of acute admission; however, clinical severity was assessed through physical examination. Clinical signs included left eye ptosis and miosis (constricted pupil) suggestive of partial Horner's syndrome, left upper limb weakness with a positive pronator drift, tongue deviation

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to the left, and left upper limb dysmetria indicating cerebellar involvement. Deep tendon reflexes were normal bilaterally.

Initial laboratory investigations revealed significant IDA (Hb 7.10 g/dL, low mean corpuscular volume and mean corpuscular hemoglobin, serum iron 24.52 µg/dL, ferritin 10.51 ng/mL), elevated white blood cell count (15,700 cells/mm³), and mildly prolonged prothrombin time (19.2 s) with an International Normalized Ratio of 1.54. Renal and liver functions were within normal limits.

Magnetic resonance imaging (MRI) of the brain showed hyperintense signals on T2/FLAIR sequences in the left lateral medulla and left cerebellum (Fig. 1). In addition, acute infarcts were noted in the left anterior capsulo-ganglionic region and parieto-temporal lobes. Magnetic resonance angiography (MRA) demonstrated complete thrombo-occlusion in the cervical and intracranial segments of the left vertebral artery (Fig. 2), confirming the diagnosis.

She was managed with intravenous Augmentin (1.2 g 8-hourly) to address leukocytosis, mannitol (20 g 8-hourly) for anti-edema measures, pantoprazole, and ondansetron. Specific treatment for the stroke included oral aspirin (75 mg daily), clopidogrel (75 mg daily), and atorvastatin (40 mg at bedtime) [6]. Ferric carboxy-maltose was administered for anemia correction. The patient's iron deficiency anemia was managed with intravenous ferric carboxymaltose. Concomitant administration of pantoprazole may impair gastrointestinal iron absorption, while dual antiplatelet therapy with aspirin and clopidogrel increases the risk of occult gastrointestinal blood loss, potentially worsening anemia. In the setting of acute brainstem ischemia (Wallenberg syndrome), optimization of hemoglobin levels is crucial to ensure adequate cerebral oxygen delivery; therefore, close hematological and hemodynamic monitoring was maintained throughout therapy [6].

A physiotherapy program was initiated to support motor and functional recovery. Her neurological symptoms gradually improved during hospitalization, and she remained hemodynamically stable. Upon discharge, she was prescribed oral ferrous sulfate, Vitamin C, aspirin, clopidogrel, atorvastatin, and pantoprazole. Follow-up was scheduled to monitor neurological recovery and iron levels.

DISCUSSION

This case report describes a 48-year-old female diagnosed with left lateral medullary syndrome due to thrombo-occlusive disease of the left vertebral artery, alongside significant IDA. Wallenberg's syndrome is typically characterized by vertigo, ataxia, and sensory deficits without limb weakness [1,5]. However, our patient presented with hemiparesis. This atypical "Wallenberg-Plus" presentation is explained by the MRI findings (Fig. 1), which revealed concurrent infarcts in the anterior capsulo-ganglionic region, a supratentorial area supplied by distal embolic showers from the occluded vertebral artery [7]. This highlights the importance of thorough imaging when clinical signs do not align perfectly with a single vascular

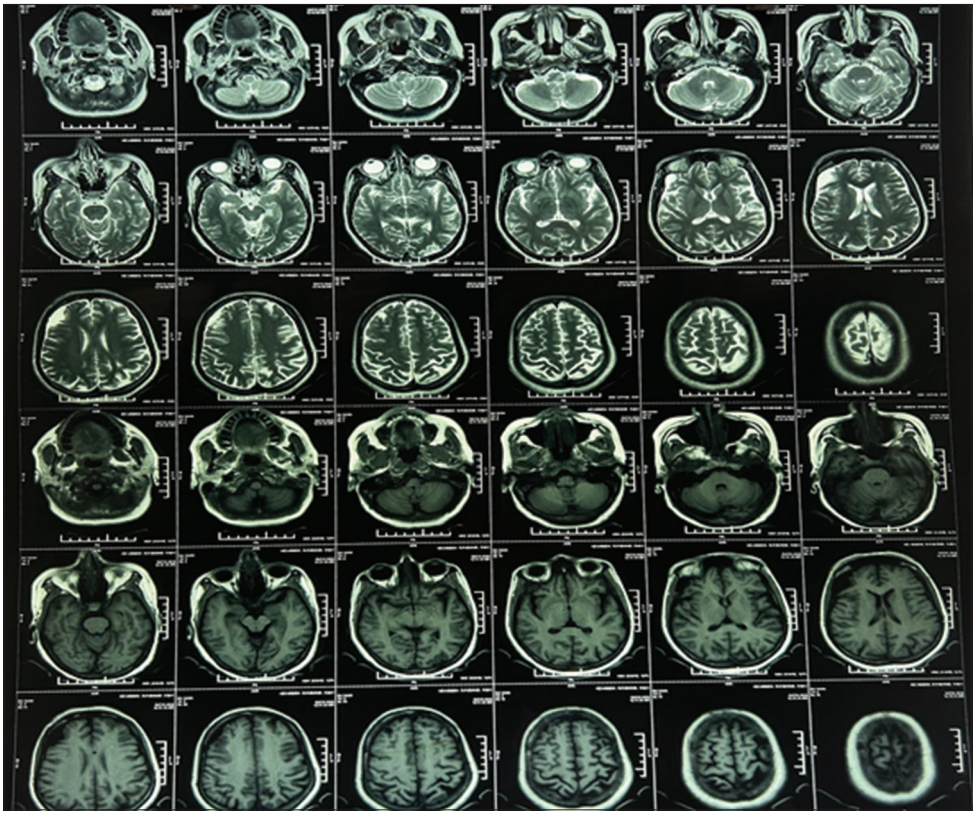

Figure 1: Magnetic resonance imaging of the brain showing hyperintense areas in the left lateral medulla and cerebellum, consistent with acute infarction. Note the additional involvement of the capsulo-ganglionic region

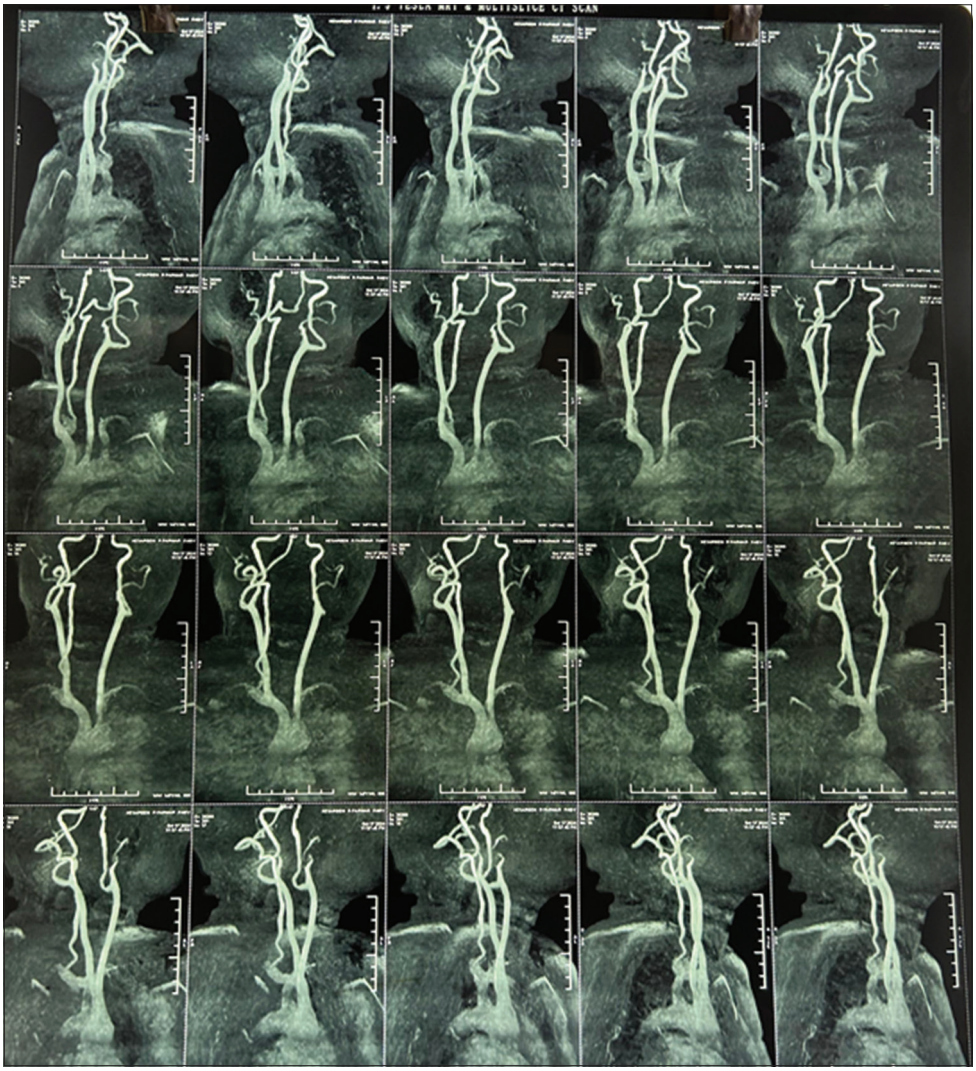

Figure 2: Magnetic resonance angiography demonstrating complete thrombo-occlusion (absence of flow signal) in the left vertebral artery compared to the patent right vertebral artery

territory [8]. The presence of atypical limb weakness was successfully correlated with multi-territory infarction on MRI [9].

IDA is a recognized risk factor for stroke, especially in younger female patients [3]. In individuals with compromised circulation, anemia can worsen cerebral ischemia by contributing to hypoxia and decreased oxygen-carrying capacity. Mechanisms linking IDA to stroke include reactive thrombocytosis and hyper-viscosity [4,10]. The study by Chang *et al*. highlighted that patients with IDA have a significantly higher risk of ischemic stroke compared to those without [3].

Due to the acute presentation and resource constraints, a specific autoimmune vasculitis profile was not performed. However, the acute onset, the presence of severe anemia, and the clear thrombo-embolic occlusion on MRA strongly supported the diagnosis of ischemic stroke exacerbated by hematologic factors.

CONCLUSION

This case of left lateral medullary syndrome underscores the complex interplay between ischemic stroke and IDA. Effective management required a tailored therapeutic approach addressing both the cerebrovascular occlusion (antiplatelets) and the hematologic deficit (iron supplementation). This approach emphasizes the need for clinicians to investigate IDA as a potential contributing factor in ischemic stroke to improve patient outcomes.

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