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Endovascular stenting for Wallenberg syndrome secondary to symptomatic vertebral artery kinking: A case report

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Keywords
posterior circulation stroke, vertebral artery kinking, vascular anomaly, endovascular stenting, thunderclap headache

Abstract

Background: Anatomical anomalies of the vertebral arteries—such as looping or kinking—are frequently incidental findings on vascular imaging and often dismissed as clinically benign. Nonetheless, evidence is accumulating that such dolichoarteriopathies can alter hemodynamics or precipitate thrombogenic conditions, thereby contributing to posterior-circulation cerebrovascular events.

Case presentation: We report a 52-year-old male with no prior cardiovascular risk factors who developed a sudden, severe occipital ("thunderclap") headache during sexual intercourse, followed by dysarthria, dysphagia, truncal ataxia and hemisensory deficits. Imaging revealed an acute right dorsolateral medullary infarct consistent with Wallenberg syndrome and bilateral kinking of the vertebral arteries with contrast stagnation. Given the presumed causative vascular anomaly, the patient underwent bilateral rigid stenting via the endovascular route, tolerated the intervention uneventfully and achieved significant neurological improvement on discharge.

Conclusions: This case underscores that vertebral-artery kinking should not be dismissed as a benign anatomical variant. In selected patients presenting with posterior-circulation stroke without conventional atherothrombotic risk factors, recognition of this vascular anomaly may permit targeted endovascular intervention and reduce stroke recurrence. The case further highlights the need for heightened clinical vigilance, advanced imaging to assess flow dynamics, and individualised management strategies beyond conservative therapy.

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Introduction

Ischemic stroke remains a leading cause of mortality and disability worldwide, with posterior-circulation strokes accounting for an estimated 20–25 % of ischemic cerebrovascular events (15). Disease

affecting the vertebral and basilar arterial systems therefore represents a significant clinical entity. While atherosclerotic stenosis of the vertebral arteries (particularly at the ostium or proximal segments) is relatively well characterized, non-atherosclerotic vascular anomalies of the vertebrobasilar system receive comparatively less attention in the neurovascular literature.

Among those anomalies, dolichoarteriopathies of the vertebral artery (VA), including kinking, looping, and coiling, have been documented with variable prevalence and clinical associations (14). Tortuosity (undulating or S-shaped elongation) is the most frequent, followed by looping and then kinking (sharp angulation) (9). For example, recent computed tomography angiography (CTA) data demonstrated tortuosity in 36 % of examined V1 segments, with kinks accounting for 19 % and coils for 17%.

Although most VA kinks or loops are discovered incidentally and remain asymptomatic, mounting evidence supports their potential association with neurologic symptoms —including vertebrobasilar insufficiency, brainstem or cerebellar ischemia, and cervical radiculopathy due to nerve root compression (2,8). The proposed pathophysiological mechanisms include: (1) hemodynamic impairment – whereby the sharp angulation reduces perfusion pressure or generates turbulence; (2) thromboembolic risk – due to endothelial injury at the angulation, stagnation and micro-embolus formation; (3) direct mechanical stenosis of the vessel lumen; (4) dynamic compression – particularly with neck rotation or extension, as in the entity Bow Hunter's syndrome (rotational vertebral artery occlusion) (7).

In neurovascular practice, failure to recognize vertebral-artery kinking as a clinically relevant entity may lead to missed etiologies of posterior-circulation stroke, particularly in younger patients lacking conventional vascular risk factors. The management paradigm for symptomatic VA kinking is not well established; conservative antiplatelet or anticoagulant therapy has been the mainstay, whereas reports of surgical or endovascular correction remain limited to case series and individual reports (5).

Here we report a distinctive case of a 52-year-old male with acute dorsolateral medullary infarction in the context of bilateral vertebral-artery kinking and contrast stagnation, who underwent bilateral stent deployment with favourable neurological recovery. This case serves to raise awareness of this vascular anomaly as a potentially modifiable stroke substrate, and to discuss its imaging, pathophysiology and therapeutic implications.

Case Presentation

A 52-year-old man with no significant medical history presented with a 30-hour evolution of a sudden, severe occipital headache (10/10 on the visual analog scale) that began abruptly during sexual intercourse. The headache was followed by gait instability, persistent hiccups, dysarthria, nausea, photophobia, and dysphagia. On neurological examination, he displayed asymmetric elevation of the right palatal arch, an abnormal bedside water swallow test, left-sided hemisensory loss predominantly affecting temperature perception, right appendicular dysmetria and dysdiadochokinesia, and right truncal ataxia. The remainder of the cranial nerve and motor examination was unremarkable, consistent with a right lateral medullary (Wallenberg) syndrome.

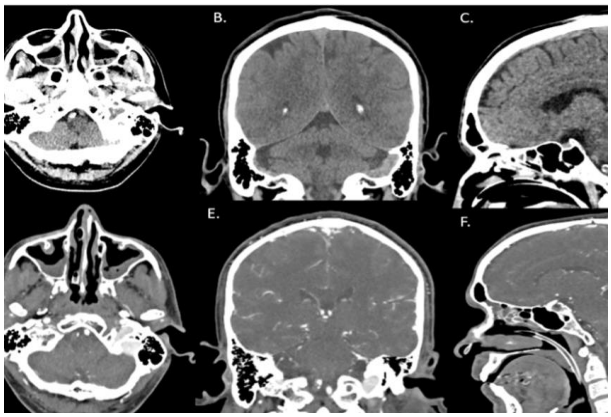

Figure 1. Non-Contrast Head CT: A). Axial view showing a hypodensity in the right lateral bulbar region, with evidence of atheromatosis in the right vertebral artery. B). Coronal view demonstrating the hypodensity in the right lateral bulbar region. C). Hypodensity observed at the bulbar level, with additional evidence of basilar artery atheromatosis. D, E, F). Obstruction of the right vertebral artery at the intracranial (V4) segment is observed.

An initial non-contrast head CT demonstrated a subtle hypodensity in the right dorsolateral medulla, and CT angiography (Figure 1) identified non-opacification of the intracranial (V4) segment of the right vertebral artery. Given the concern for posterior-circulation infarction, MRI of the brain and digital subtraction angiography (DSA) were obtained

(Figures 2–3). MRI confirmed an acute lateral medullary infarct, while DSA revealed bilateral vertebral artery kinking with contrast stagnation, a configuration considered highly thrombogenic and presumed to be the source of the ischemic event.

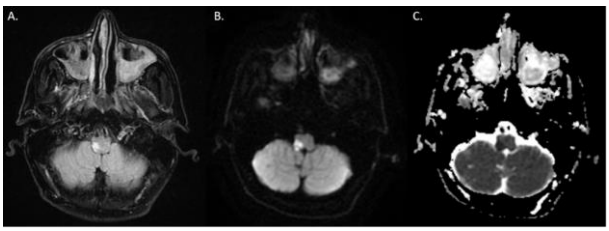

Figure 2. Brain MRI: A, B, C). T2 Flair sequence, diffusion-weighted imaging (DWI), and ADC map demonstrating diffusion restriction consistent with an acute non-hemorrhagic infarct in the dorsolateral medulla.

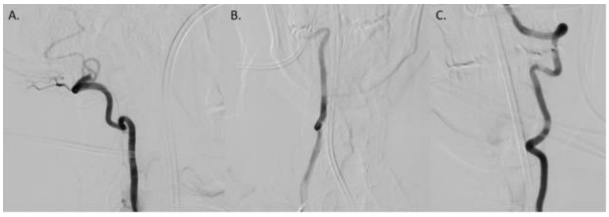

Figure 3. Diagnostic Cerebral Angiography: A). Lateral view of the right vertebral artery showing kinking in the V2 segment at the C3-C4 level, with evidence of distal occlusion at the V4 segment. The origin of the PICA (Posteroinferior Cerebellar Artery) is observed from the V3 segment of the right vertebral artery. B). Anteroposterior view of the right vertebral artery demonstrating vertebral kinking with contrast stagnation in the V2 segment at the C4-C5 level. C). Anteroposterior view of the left vertebral artery also showing evidence of kinking.

The imaging results confirmed an acute ischemic stroke in the right dorsolateral medulla. Findings also included bilateral vertebral artery kinking with evidence of contrast stagnation, a recognized thrombogenic risk. This kinking was identified as the likely etiology of the ischemic event. We therefore decided to proceed with endovascular correction of the vascular anomaly (Figure 4) by deploying rigid stents bilaterally to mitigate the thrombogenic risk.

The patient's postoperative course was uneventful, with significant clinical improvement. He was discharged from the hospital with instructions for outpatient follow-up with imaging and started on dual antiplatelet therapy. (Figure 5).

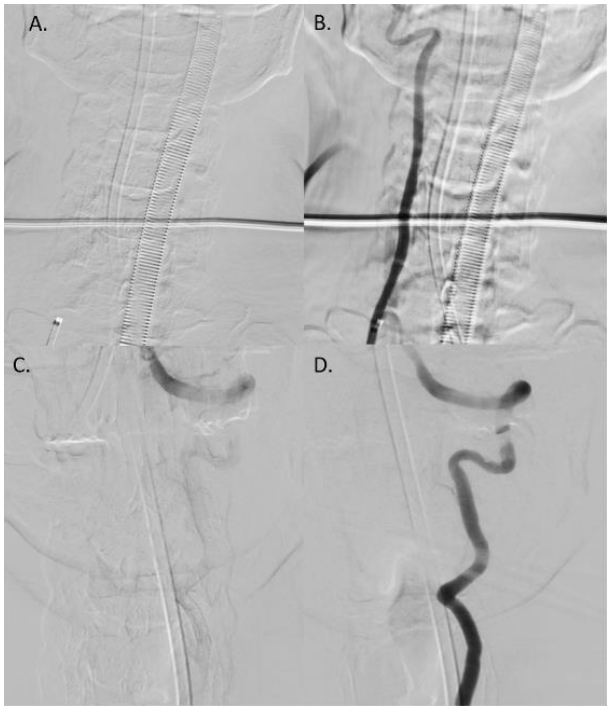

Figure 4. Stent Angioplasty + Diagnostic Cerebral Angiography: A, B). Deployment of a rigid coronary stent achieving anatomical correction of the right vertebral artery kinking. C, D). Deployment of a rigid coronary stent with no evidence of persistent contrast stagnation.

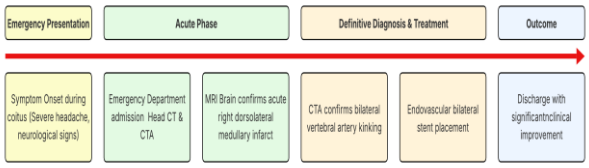

Figure 5. Timeline of diagnostic and therapeutic management from symptom onset to intervention.

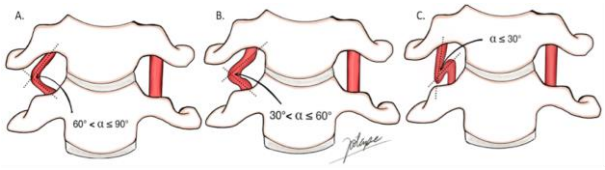

Figure 6. Classification of Vertebral Artery Kinking Severity by Angulation: A). Mild angulation (61-90°). B). Moderate angulation (31-60°). C). Severe angulation (0-30°).

Discussion

Posterior fossa cerebrovascular disease due to posterior circulation involvement has an estimated

prevalence of 20-25% (16). Vertebral artery stenosis is a leading cause, with an incidence of up to 40%, where atherosclerotic disease most commonly affects the ostium and proximal segment.

Vertebral artery kinking has been reported with a prevalence ranging from 0.6% to 7.5% in angiographic studies and may arise from congenital elongation, postural or degenerative cervical changes, or chronic mechanical stress (7, 16). While many cases remain asymptomatic, symptomatic kinking is typically linked to vertebrobasilar insufficiency, cervical radiculopathy, or, more rarely, ischemic stroke. The V2 segment is most often affected, although kinking may occur anywhere along the vessel (7). Diagnosis is typically made in patients over 40, either during workup for cervical spine surgery or upon presenting to the emergency department with cervical radicular symptoms or neck pain (11).

In symptomatic patients, the geometric change in the vessel affects blood flow, leading to cerebral ischemia through several mechanisms:

1. Hemodynamic Mechanisms: Turbulence at the kink site causes energy loss, reducing distal perfusion pressure.
2. Thromboembolic Mechanism: Altered flow damages the local vessel endothelium, potentially leading to micro-emboli formation and embolic events.
3. Vascular Injury: Extreme kinking can directly lead to stenosis of the affected vessel.
4. Mechanical Cause: Neck rotation can cause temporary arterial obstruction, triggering symptoms of insufficiency (11, 16).

The symptomatology and clinical course depend on the degree and severity of the vessel angulation, classified into three grades: mild (61-90°), moderate (31-60°), and severe (0-30°) (Figure 6) (16). Currently, this condition is most frequently associated with radicular pain due to vascular compression of cervical nerve roots at the foramen. Consequently, worldwide management often involves microvascular decompression to release the affected foramen and alleviate symptoms (7). However, although rare, this entity should be suspected in young patients, like the one presented, as an etiology for acute ischemic events in the posterior fossa.

From a diagnostic perspective, multimodal imaging plays an important role. Non-contrast CT may identify ischemic hypodensity, but MRI with diffusion-weighted imaging remains the most sensitive tool to confirm medullary involvement (6). CTA and MRA are useful for identifying vascular anomalies such as kinking or looping, while digital subtraction angiography continues to be the gold standard for evaluating vessel angulation, flow stagnation, and associated thrombogenic risk, particularly in suspected mechanogenic ischemia. (12). These imaging modalities not only establish the diagnosis but also guide the decision between conservative and interventional management.

Management of vertebral artery kinking depends on clinical presentation. In patients without ischemic symptoms, conservative treatment with antiplatelet therapy, risk factor modification, and regular imaging surveillance is generally sufficient (4). However, in cases where kinking is associated with ischemic stroke, as in the patient described, invasive treatment should be considered. Surgical options, including microvascular decompression, are mainly reserved for patients with radiculopathy caused by nerve root compression (3). For ischemic presentations attributed to abnormal flow dynamics, endovascular approaches, angioplasty or stenting, have been increasingly reported as viable options, offering the theoretical benefit of correcting abnormal geometry, eliminating flow turbulence, and reducing thromboembolic risk. Evidence remains limited to case reports and small series, but available data suggest favorable long-term patency and reduced recurrence in carefully selected patients (1).

The case presented supports this approach. Bilateral rigid stent placement corrected the anatomical anomaly, stabilized blood flow, and prevented further embolic events, resulting in significant neurological recovery. These findings are consistent with recent series that demonstrate improved long-term patency and lower recurrence rates following endovascular treatment of vertebral artery disease compared to medical therapy alone (10, 13). Although randomized trials are lacking, the accumulating evidence highlights endovascular management as a promising option, particularly in anatomically complex or bilateral cases where medical therapy alone may be insufficient. Overall, vertebral artery kinking should not be dismissed as a

benign or incidental radiological finding. Its potential to cause severe posterior circulation strokes through hemodynamic and thromboembolic mechanisms underscores the need for clinical suspicion, timely diagnosis, and individualized management.

The present case illustrates this rationale. Bilateral stent deployment successfully corrected the anatomical configuration, restored flow, and was followed by substantial clinical recovery. Such cases reinforce the importance of not dismissing vertebral artery kinking as merely incidental—particularly when posterior-circulation stroke occurs in patients without traditional vascular risk factors.

Conclusions

Although vertebral artery kinking is often reported as an incidental imaging finding, this case demonstrates that it can have significant clinical implications. Sharp angulation may generate hemodynamic disturbance, promote thrombosis, and precipitate posterior-circulation ischemia. Clinicians should maintain a high index of suspicion in patients who present with thunderclap headache or brainstem symptoms in the absence of conventional risk factors. Multimodal vascular imaging is essential, and early identification of flow stagnation should prompt consideration of interventional management. Timely diagnosis and individualized treatment may prevent recurrent events and reduce the risk of disabling neurological sequelae.

Abbreviations

ADC – Apparent Diffusion Coefficient
CTA – Computed Tomography Angiography
CT – Computed Tomography
DWI – Diffusion-Weighted Imaging
HALE – Healthy Life Expectancy
MRA – Magnetic Resonance Angiography
MRI – Magnetic Resonance Imaging
PICA – Posterior Inferior Cerebellar Artery
TIA – Transient Ischemic Attack

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