

Imaging-Based Diagnosis of Intracranial Vertebral Artery Dissection Leading to Non-Aneurysmal Subarachnoid Hemorrhage: A Vascular-Neuroimaging Case Report

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How to cite this paper: Ramírez-Mendoza, C., Rivero, M.N.-G., Palacios-Halaby, F., Mejía-Cuello, K., Plata-Mendoza, A., Barcenás-Contreras, S.I., Ramos-Lafaurie, M., Maciá-Caraballo, F., Torres, L.V., Puerta-Barboza, S., Matson-Sepulveda, W. and Macías-Alvarado, J. (2026) Imaging-Based Diagnosis of Intracranial Vertebral Artery Dissection Leading to Non-Aneurysmal Subarachnoid Hemorrhage: A Vascular-Neuroimaging Case Report. *Journal of Biosciences and Medicines*, **14**, 386-396.

<https://doi.org/10.4236/jbm.2026.141029>

Received: December 2, 2025

Accepted: January 23, 2026

Published: January 26, 2026

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Abstract

Background: Intracranial vertebral artery dissection (VAD) is an uncommon but clinically significant cause of non-aneurysmal subarachnoid hemorrhage (SAH). When hemorrhagic, its early mortality is driven largely by the elevated risk of rebleeding. Diagnosis relies heavily on advanced vascular imaging, as clinical manifestations often overlap with those of other causes of acute headache.

Case: We report the case of a 57-year-old man with hypertension and type 2 diabetes who presented with sudden, severe holocranial headache and posterior cervical pain immediately following neck hyperextension during public transportation. Initial non-contrast CT revealed a diffuse Fisher II SAH. Digital subtraction angiography demonstrated a dissection flap at the left vertebralbasilar junction with absence of opacification of the posterior inferior cerebellar artery (PICA), in the absence of aneurysms or arteriovenous malformations. MRI confirmed subarachnoid blood and an intramural hematoma signal consistent with arterial wall injury. **Conclusion:** This case highlights the critical role of multimodal vascular imaging—particularly CT, MRI, and catheter angiography—in diagnosing intracranial VAD presenting as non-aneurysmal SAH. Recognizing characteristic imaging features facilitates timely management, prevents diagnostic delay, and underscores the importance of considering VAD in patients with acute headache and neck pain, especially following

biomechanical stress to the cervical segment.

Keywords

Vertebral Artery Dissection, Subarachnoid Hemorrhage, Intracranial Arterial Dissection, Neuroimaging, Angiography, Digital Subtraction, Magnetic Resonance Imaging, Intramural Hematoma, Vertebrobasilar System, Endovascular Procedures, Posterior Inferior Cerebellar Artery

1. Introduction

Spontaneous intracranial artery dissection (IAD) is an uncommon but increasingly recognized cause of stroke, particularly in young and middle-aged adults, and encompasses a spectrum of ischemic and hemorrhagic phenotypes driven by intramural haematoma formation within small-calibre intracranial vessels [1]. Although cervical artery dissection is far more frequently reported in population-based cohorts, with an incidence of approximately 2.6 - 3.0 per 100,000 person/year, spontaneous vertebral artery dissection (VAD) alone accounts for roughly 1.0 - 1.1 per 100,000 and represents a leading cause of posterior circulation events in otherwise low-risk individuals [2] [3]. The intracranial segment of the vertebral artery is particularly prone to dissection-related complications because of its fixed course, abrupt angulations, and complex perforator anatomy, all of which modulate local haemodynamic stress and predispose to both pseudoaneurysm formation and rupture [2] [3].

Within the broader landscape of spontaneous subarachnoid haemorrhage (SAH), aneurysmal rupture still accounts for approximately 85% of cases, whereas the remaining 10% - 15% comprise a heterogeneous group of non-aneurysmal aetiologies that include perimesencephalic haemorrhage, arterial dissection, arteriovenous malformations, and dural fistulas, among others [4]. Non-aneurysmal SAH associated with intracranial VAD occupies a particularly high-risk niche: contemporary series suggest that vertebrobasilar dissections account for a small proportion of all SAH presentations, yet they carry disproportionate early mortality because of very high rebleeding rates in the acute phase [5]-[7]. Historical and modern cohorts consistently report recurrent haemorrhage in 30% - 60% of ruptured intracranial vertebral dissecting lesions within the first 24 hours, with some series describing rebleeding rates exceeding 70% when treatment is delayed [7].

From a neuroimaging standpoint, intracranial VAD with SAH represents a diagnostic challenge where the incremental yield of each modality must be understood in a mechanistic framework. Digital subtraction angiography (DSA) remains the reference standard for delineating the dissected segment, characterising pseudoaneurysmal changes, and assessing perforator anatomy, but non-invasive techniques such as CT angiography, MR angiography, and high-resolution vessel-wall MRI have become essential both for initial suspicion and longitudinal follow-up.

In the acute setting, non-contrast CT provides rapid confirmation and grading of SAH, whereas multimodal vascular imaging is required to distinguish ruptured dissecting lesions from more common saccular aneurysms and from angiographically occult non-aneurysmal patterns, decisions that have direct implications for prognosis, intensity of haemodynamic management, and timing of intervention.

Therapeutically, ruptured intracranial VAD sits at the intersection of haemorrhagic and thromboembolic risk, and there is no universally accepted standard of care. Observational data and expert consensus favour early “deconstructive” strategies—such as parent-artery occlusion or trapping—for unstable dissecting aneurysms at high risk of rebleeding, while “reconstructive” approaches using stent-assisted techniques or flow diversion aim to preserve vertebrobasilar flow in anatomically or haemodynamically unfavourable configurations [5]-[7]. However, the comparative effectiveness and long-term safety of these strategies remain incompletely defined, particularly outside Asian and highly selected tertiary cohorts. In this context, carefully phenotyped single cases with detailed multimodal imaging are valuable for refining diagnostic algorithms, illustrating mechanistic pathways linking biomechanical triggers, vascular injury and haemorrhage, and informing the design of prospective registries and interventional studies focused on non-aneurysmal SAH due to intracranial VAD.

We therefore present a case of non-aneurysmal SAH secondary to intracranial vertebral artery dissection in which the diagnostic and therapeutic strategy was guided predominantly by multimodal vascular imaging, and we discuss the implications of an imaging-based approach for risk stratification and management of this rare but clinically consequential entity.

2. Case Presentation

A 57-year-old right-handed man, employed as an intercity bus dispatcher, with a medical history notable only for well-controlled hypertension and dyslipidemia, presented to the emergency department after experiencing an abrupt, severe occipitonal headache while stepping off a moving bus. The event occurred immediately after an episode in which he turned his head sharply to the left to respond to a colleague, followed by a sudden forced counter-rotation as he lost balance on the ramp. He described the pain as instantaneous, explosive, and unlike any prior headache, accompanied by abrupt neck tightness, intense photophobia, and a sensation of “warm fluid” spreading through the posterior scalp. Within minutes, he developed nausea and repeated vomiting, prompting emergency transport. He denied syncope, seizure activity, or focal neurological symptoms. He reported no recent chiropractic manipulation, heavy lifting, cough paroxysms, or cervical hyperextension events. There was no family history of aneurysmal subarachnoid haemorrhage, connective tissue disease, or early cardiovascular deaths.

Upon arrival, the patient was alert, oriented, and cooperative, with a blood pressure of 158/92 mmHg, a heart rate of 92 bpm, and no respiratory compromise. Neurological examination showed prominent meningismus with resistance to

passive neck flexion, yet cranial nerves II - XII were intact. Motor strength was full and symmetric, deep tendon reflexes were normoreactive, and plantar responses were flexor bilaterally. Sensory modalities were preserved. Cerebellar testing—including finger-to-nose and heel-to-shin—was normal, and gait could not be assessed due to symptom intensity. Fundoscopy revealed sharp optic disc margins with no evidence of papilledema or retinal hemorrhages. Cardiopulmonary and abdominal examinations were unremarkable. The patient had no stigmata of connective tissue disorders or vasculopathies on physical inspection.

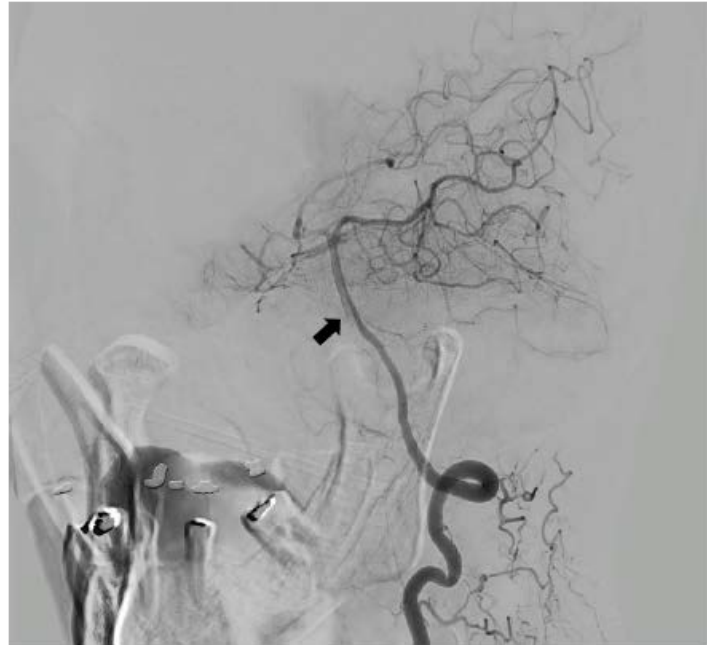


Figure 1. Non-contrast head CT demonstrating diffuse subarachnoid hemorrhage predominantly within the basal cisterns and perimesencephalic spaces, consistent with a Fisher grade II pattern.

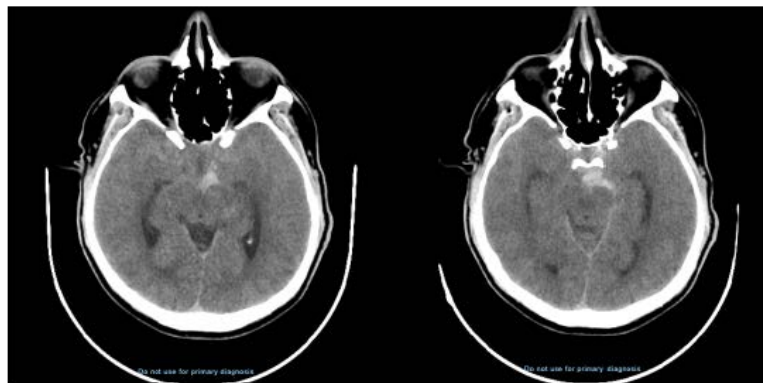


Figure 2. DSA shows luminal irregularity and focal narrowing at the left vertebralbasilar junction (arrow), compatible with intracranial vertebral artery dissection.

An emergent non-contrast head CT revealed diffuse acute subarachnoid

haemorrhage predominantly involving the prepontine cistern, perimesencephalic cisterns, and ventromedullary cistern, with asymmetric extension into the left cerebellomedullary cistern (**Figure 1**). No intraparenchymal haematoma, subdural collection, cortical sulcal haemorrhage, or hydrocephalus was observed. The distribution pattern was compatible with a modified Fisher grade II. Notably, the haemorrhage was centered on the anterior brainstem and foramen magnum region, raising suspicion for a vascular lesion within the posterior circulation.

CTA of the head and neck demonstrated subtle luminal irregularity and calibre variation along the left V4 segment of the vertebral artery, without definitive evidence of a saccular aneurysm or high-flow vascular malformation. The posterior communicating arteries were diminutive, and no other aneurysms were visualised. Given the haemorrhage pattern and the indeterminate CTA findings, urgent DSA was pursued to further evaluate the vertebrobasilar circulation.

DSA revealed a focal eccentric narrowing at the left vertebrobasilar junction with an intimal flap and delayed contrast washout, representing a dissecting lesion arising within the intradural V4 segment (**Figure 2**). The contour of the affected segment appeared irregular and corrugated, consistent with intramural haematoma. No saccular or fusiform aneurysm was identified. The left posterior inferior cerebellar artery (PICA) failed to opacify, suggesting flow impairment secondary to the dissection. Collateral supply to the PICA territory via the contralateral vertebral artery was limited, though no frank territorial infarction was observed angiographically.

MRI with high-resolution vessel-wall sequences, obtained within 24 hours, demonstrated crescentic T1 hyperintensity and mural thickening in the corresponding V4 segment, confirming the presence of intramural blood products consistent with acute intradural vertebral artery dissection. T2-weighted imaging revealed subtle periarterial oedema. Diffusion-weighted imaging showed no acute infarction within the posterior circulation, and the brainstem appeared structurally intact. No signs of reversible cerebral vasoconstriction or inflammatory vasculopathy were present.

The patient was admitted to the neurocritical care unit for strict blood pressure management, early detection of vasospasm, and prevention of rebleeding. Continuous neurological monitoring, scheduled transcranial Doppler assessments, and close cardiopulmonary surveillance were instituted. Given the intradural location of the lesion, its angiographic instability, and the historically poor natural history of ruptured vertebrobasilar dissections, the multidisciplinary neurovascular board advised early endovascular intervention. During the first 12 hours of monitoring, the patient remained clinically stable with preserved neurological function and no evidence of rebleeding, new symptoms, or delayed ischemia.

Following definitive endovascular repair, his hospital course was uneventful, without vasospasm, delayed cerebral ischemia, or further hemorrhagic events. He was discharged home neurologically intact, and at 3-month follow-up, he remained symptom-free with stable vascular imaging and no recurrent bleeding.

3. Discussion

Intracranial vertebral artery dissection (VAD) remains an uncommon but clinically formidable cause of subarachnoid haemorrhage (SAH), distinguished by its uniquely aggressive natural history and diagnostic complexity. Unlike cervical artery dissection, which typically manifests with local pain and ischemia, intradural VAD more frequently presents with haemorrhage due to the absence of an external elastic lamina and the intrinsic structural vulnerability of the intradural vertebral segment. These anatomical characteristics render intradural dissections particularly prone to rupture, pseudoaneurysm formation, and recurrent bleeding—features that influence both the diagnostic strategy and the therapeutic approach [8]-[10].

3.1. Pathophysiological Considerations

The mechanism underlying SAH in intracranial VAD involves extension of an intramural haematoma toward the adventitia, leading to rupture into the subarachnoid space. The vertebral artery's V4 segment, which courses intradurally after perforating the dura mater at the foramen magnum, is especially susceptible to high shear stress and abrupt haemodynamic transitions. These biomechanical vulnerabilities are further amplified at the vertebrobasilar junction, where abrupt curvature and complex flow dynamics create oscillatory shear zones that may predispose the intima to disruption [9] [10]. This anatomic stress point is directly relevant to the present case, in which the dissecting focus at the vertebrobasilar junction exemplifies the haemodynamic vulnerability described. Epidemiological data support this mechanistic framework: although spontaneous VAD is relatively rare, ruptured intracranial dissections disproportionately involve the V4 segment and are associated with high early fatality rates [8]-[11].

Mild biomechanical triggers—including sudden rotation, axial loading, or minor hyperextension of the neck—have been implicated in up to one-third of spontaneous VAD cases [12]. The index patient experienced a sudden forced rotation-counterrotation event, consistent with observational studies suggesting that low-energy cervical motion may suffice to initiate arterial wall injury in individuals with underlying microstructural susceptibility [12] [13]. The absence of trauma does not diminish diagnostic suspicion, as most ruptured VADs occur without any significant precipitating event [13].

3.2. Diagnostic Challenges and the Role of Multimodal Imaging

The diagnosis of intradural VAD in the setting of SAH is frequently delayed due to its imaging subtleties. CTA may demonstrate only minimal luminal irregularity or calibre variation, findings that lack sensitivity for detecting non-aneurysmal dissecting lesions. This contributes to a substantial proportion of SAH labelled as “angiogram-negative” on initial evaluation [14]. Digital subtraction angiography (DSA), however, remains the gold standard, offering superior spatial and temporal resolution for characterising intimal flaps, pseudoaneurysm formation, and

delayed contrast washout patterns that signify disrupted laminar flow [15]. The patient's angiographic features—focal eccentric narrowing, intimal flap, and impaired PICA opacification—are emblematic of the classical angiographic spectrum of vertebral dissections.

High-resolution vessel-wall MRI has emerged as a powerful adjunct, enabling direct visualisation of mural haematoma, wall thickening, and intramural blood products. Its incremental yield is especially valuable when CTA is equivocal and DSA is inconclusive or when serial monitoring is required [16]. In the present case, vessel-wall MRI confirmed mural T1 hyperintensity consistent with intramural haematoma, contributing decisively to the diagnostic certainty. The absence of diffusion-restricted lesions highlights the dissociation commonly observed between haemorrhagic and ischaemic phenotypes of VAD, reinforcing that SAH may occur in the absence of overt infarction [16].

Table 1 summarizes the imaging-based diagnostic features observed in this case and their clinical interpretation, highlighting the complementary roles of CT, CTA, DSA, and high-resolution vessel-wall MRI in intracranial VAD.

Table 1. Imaging-based diagnostic features and clinical correlates of intracranial vertebral artery dissection presenting with subarachnoid haemorrhage.

Domain	Key Findings in This Case	Clinical/Pathophysiological Interpretation
Clinical Presentation	Sudden, explosive occipitotuchal headache; neck stiffness; photophobia; emesis following forced cervical rotation.	Classic pattern for posterior circulation SAH; minor biomechanical trigger is consistent with spontaneous intradural dissection.
Initial CT (Non-contrast)	Diffuse SAH in the prepontine, perimesencephalic, and ventral medullary cisterns (Modified Fisher II).	Haemorrhage centered in brainstem cisterns strongly suggests ruptured vertebrobasilar pathology; it is less typical for saccular aneurysms.
CTA Findings	Subtle luminal irregularity in the left V4 without a discrete aneurysm.	CTA may be insufficiently sensitive; equivocal findings necessitate DSA.
Digital Subtraction Angiography	Eccentric narrowing, intimal flap, delayed contrast washout at the vertebrobasilar junction; absent PICA opacification.	Diagnostic triad of intradural VAD: focal stenosis, mural defect, haemodynamic impairment; high risk of rebleeding.
High-Resolution Vessel-Wall MRI	Crescentic T1 mural hyperintensity; intramural hematoma; circumferential wall thickening.	Confirms active mural injury and intramural blood products; MRI increases diagnostic certainty when CTA/DSA are ambiguous.
Ischemic Findings	No infarction on DWI.	Hemorrhagic phenotype of VAD may occur without ischemia; this is a distinct pathophysiology from cervical VAD.
Haemodynamic Stability	Persistent neurological integrity; no early vasospasm; no hydrocephalus.	Typical of perimesencephalic-predominant patterns; however, the rebleeding risk remains substantial.
Therapeutic Decision Drivers	Intradural location, angiographic instability, impaired PICA flow.	Favors early endovascular repair due to the high risk of early rebleeding and the poor natural history of ruptured V4 dissections.

3.3. Natural History and Prognostic Considerations

Ruptured intracranial VAD carries a markedly worse prognosis than its

unruptured or cervical counterparts. Multiple cohorts have documented early rebleeding rates between 30% and 60% within the first 24 - 48 hours, with some series reporting even higher rates in the absence of urgent treatment. Rebleeding is the principal determinant of early mortality and is strongly associated with angiographic instability, pseudoaneurysm morphology, and intradural location. The patient's lesion—located at the vertebrobasilar junction with angiographic evidence of wall instability—falls within this high-risk category, justifying early endovascular consideration.

Hydrocephalus is less common in SAH from dissecting VAD compared with aneurysmal SAH, likely due to the predilection for prepontine and perimesencephalic cisterns rather than diffuse cortical involvement. Consistent with this, the patient exhibited no ventricular enlargement on initial imaging.

3.4. Therapeutic Decision-Making

The goals of treatment in ruptured intracranial VAD include preventing rebleeding, maintaining adequate posterior circulation perfusion, and mitigating secondary complications such as cerebral vasospasm. Historically, deconstructive techniques—most often parent artery occlusion (PAO) with or without trapping—were considered first-line therapy due to their straightforward mechanical elimination of the rupture source [17]. However, PAO is only feasible when the contralateral vertebral artery is robust enough to preserve basilar flow, and when collateralisation of the PICA territory is sufficient. Inadequate collateral flow may result in lateral medullary or cerebellar infarction.

Reconstructive approaches, including stent-assisted coiling, overlapping stents, or flow diversion, aim to preserve vessel patency while promoting endothelial remodelling and pseudoaneurysm thrombosis. Flow diverters have shown promising results in small modern series, particularly for lesions unsuitable for deconstructive therapy, although the need for dual antiplatelet therapy raises significant haemorrhagic concerns in the acute post-SAH period [17] [18]. The therapeutic decision for the index patient was shaped by the angiographic instability of the lesion and impaired PICA opacification, highlighting the importance of integrating radiographic, anatomical, and haemodynamic considerations into management planning. In this patient, early reconstructive endovascular treatment with stent-assisted stabilization of the V4 segment was performed to preserve vertebrobasilar flow while mechanically excluding the rupture focus. This strategy was selected due to insufficient collateral support for safe parent artery occlusion and the need to minimise the risk of rebleeding while maintaining posterior circulation integrity.

3.5. Epidemiological and Methodological Implications

Intracranial VAD with SAH exemplifies a condition in which the rarity of the disease poses significant challenges to evidence generation. Most clinical recommendations derive from small case series, heterogeneous institutional experiences,

and expert consensus. Standardised prospective registries remain scarce, limiting the capacity to derive accurate estimates of incidence, rebleeding risk, and the comparative effectiveness of endovascular strategies. This underscores the epidemiological value of detailed case reports that employ standardised imaging terminology, rigorous diagnostic criteria, and transparent clinical reasoning. From a population-health perspective, improved reporting contributes to cumulative meta-analytic power and supports the development of risk-stratification tools that may guide intervention timing.

The present case adds to the growing literature demonstrating that a structured, imaging-driven diagnostic algorithm can meaningfully reduce diagnostic delay and improve the safety of therapeutic decision-making. In particular, the complementary roles of vessel-wall MRI and DSA provide a reproducible framework for evaluating challenging cases in which non-invasive imaging is equivocal. [18]

4. Conclusions

Intracranial vertebral artery dissection remains a rare yet clinically devastating cause of subarachnoid haemorrhage, demanding a high index of suspicion and a structured, imaging-driven diagnostic strategy. This case illustrates how subtle biomechanical triggers, non-specific early symptoms, and initially equivocal non-invasive imaging can mask an underlying intradural dissecting lesion with substantial early rebleeding risk. The integration of multimodal vascular imaging—particularly high-resolution vessel-wall MRI and digital subtraction angiography—proved essential in establishing diagnostic certainty, delineating haemodynamic instability, and guiding timely therapeutic decision-making.

From an epidemiological and clinical standpoint, this case reinforces the need for systematic diagnostic pathways and vigilant early management in patients presenting with posterior fossa-predominant SAH, even in the absence of overt aneurysmal morphology. As evidence remains limited by the rarity of this entity, rigorously phenotyped individual cases continue to play an important role in refining diagnostic criteria, informing therapeutic strategies, and supporting collaborative efforts to improve outcomes in ruptured intracranial vertebral dissections.

Ethical Considerations

This case was conducted in accordance with the principles of the Declaration of Helsinki and institutional standards for clinical research ethics. All identifying patient information was removed to ensure full confidentiality. Written informed consent for publication of the clinical details and imaging studies was obtained from the patient.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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