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# Isolated Abducens Nerve Palsy Associated with Neurovascular Compression by a Vascular Loop of the Anterior Inferior Cerebellar Artery: A Case Report

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## ABSTRACT

Neurovascular compression is an uncommon cause of isolated abducens nerve palsy. We report a 67-year-old woman who presented with acute-onset diplopia caused by an isolated left abducens nerve palsy. Computed tomography of the brain, performed with and without contrast on the day of presentation, was unremarkable. Glycaemic investigations were normal, although the patient had dyslipidaemia. Because her symptoms persisted, MRI and MR angiography of the brain were performed 2 weeks after symptom onset. High-resolution three-dimensional T2-DRIVE imaging demonstrated a loop of the left anterior inferior cerebellar artery contacting the cisternal segment of the left abducens nerve and producing focal bowing at the root exit zone. She was managed conservatively with an eye patch and empirical corticosteroid therapy, which had been commenced before MRI for presumed inflammatory neuropathy. Approximately 3 months after symptom onset, she reported near-complete improvement, with only minimal residual diplopia and no further requirement for the eye patch. No follow-up imaging was obtained. This case highlights the value of dedicated high-resolution MRI in identifying nerve deformation associated with a neurovascular conflict. However, the causal relationship should be interpreted cautiously because spontaneous recovery and a competing microvascular mechanism could not be excluded.

Keywords: abducens nerve palsy, anterior inferior cerebellar artery, diplopia, neurovascular compression

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## INTRODUCTION

Isolated abducens nerve palsy is a well-recognised cause of diplopia and may result from lesions occurring anywhere along the nerve's long intracranial course. Important causes include microvascular ischaemia, brainstem infarction or demyelinating disease, intracranial aneurysm, raised intracranial pressure, and lesions involving the cavernous sinus or orbit. In older adults, giant cell arteritis must also be considered when the clinical context is suggestive (Jain, 2022). Although these aetiologies account for most cases, neurovascular compression of the abducens nerve is an uncommon and likely under-recognised cause.

Among reported cases of neurovascular compression, the offending vessels most commonly include the basilar, vertebral or anterior inferior cerebellar arteries (AICA) (Aggarwal *et al.*, 2023). AICA loops are also frequent incidental findings in the cerebellopontine angle; a 3D-CISS study identified them in 47.6% of asymptomatic individuals, suggesting that vascular contact alone is insufficient to establish symptomatic compression (G *et al.*, 2024). Diagnostic confidence therefore depends on concordant symptoms, exclusion of alternative causes and objective nerve deformation on dedicated imaging.

Recognition of this rare mechanism is clinically important, as failure to identify the underlying cause may lead to unnecessary investigations, diagnostic uncertainty, or delayed management. We present a case of isolated left abducens nerve palsy in which high-resolution MRI demonstrated focal bowing of the cisternal segment of the left abducens nerve by an AICA loop at the root exit zone. The clinical course, differential diagnoses, imaging findings and limitations in establishing causality are discussed.

## CASE REPORT

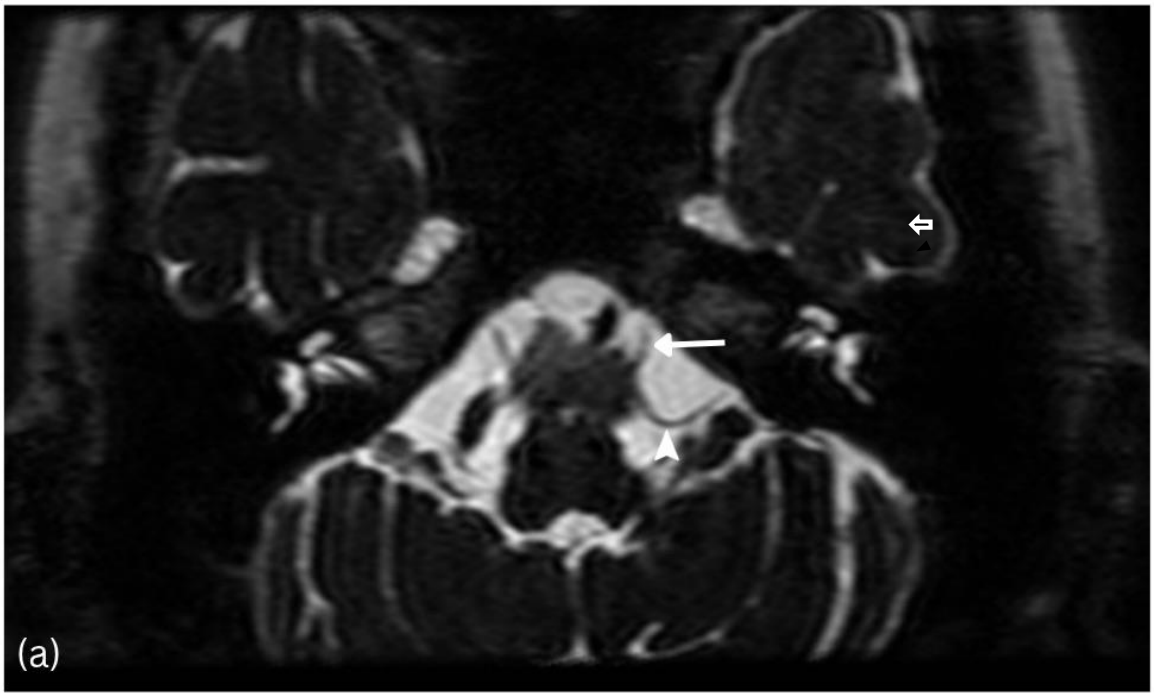
A 67-year-old woman presented with acute-onset diplopia. She had no known history of diabetes mellitus or hypertension, and her blood pressure remained non-hypertensive during admission. She denied any history of trauma, headache, facial numbness, ptosis, symptoms suggestive of raised intracranial pressure, or other systemic illness.

Assessment by the emergency, ophthalmology and neurology teams demonstrated an isolated left abducens nerve palsy, with no evidence of associated orbital or paranasal sinus pathology.

Laboratory testing showed HbA1c 5.6%, fasting blood glucose 4.7 mmol/L and random blood glucose 6.9 mmol/L. C-reactive protein was 1.23 mg/L, erythrocyte sedimentation rate 9 mm/hour, white-cell count 10.89 x $10^9$/L and platelet count 255 x $10^9$/L. The initial lipid profile showed total cholesterol 6.35 mmol/L, HDL cholesterol 1.51 mmol/L, LDL cholesterol 4.28 mmol/L and VLDL cholesterol 0.56 mmol/L. Repeat testing confirmed dyslipidaemia, with total cholesterol 6.50 mmol/L, triglycerides 1.78 mmol/L, HDL cholesterol 1.26 mmol/L and LDL cholesterol 4.43 mmol/L. Plain and contrast-enhanced CT of the brain and orbits on the day of presentation showed no acute haemorrhage, hydrocephalus, infarction, intracranial enhancing lesion, paranasal sinus disease or orbital abnormality.

In view of persistent diplopia and the absence of an identifiable cause on CT, further evaluation with magnetic resonance imaging (MRI) and MR angiography (MRA) of the brain were performed.

MRI and MRA were performed 20 days after symptom onset, using a 3 T scanner (Figures 1 and 2). High-resolution imaging comprised a three-dimensional T2-DRIVE sequence with a 1.0-mm slice thickness, a repetition time 2000 ms and an echo time of 246.2 ms. Phase-contrast MRA of the circle of Willis with SENSE acquisition was performed using a 1.2-mm slice thickness, repetition time 20 ms and echo time 3.5 ms. The images demonstrated a vascular loop of the left AICA abutting the left abducens nerve at its root exit zone near the pontomedullary junction. Compared with the normal contralateral nerve, the left abducens nerve showed focal bowing at the point of contact. There was no restricted diffusion, abnormal enhancement or focal structural lesion involving the left abducens nerve along its course, and MRA showed no intracranial or cavernous internal carotid artery aneurysm.



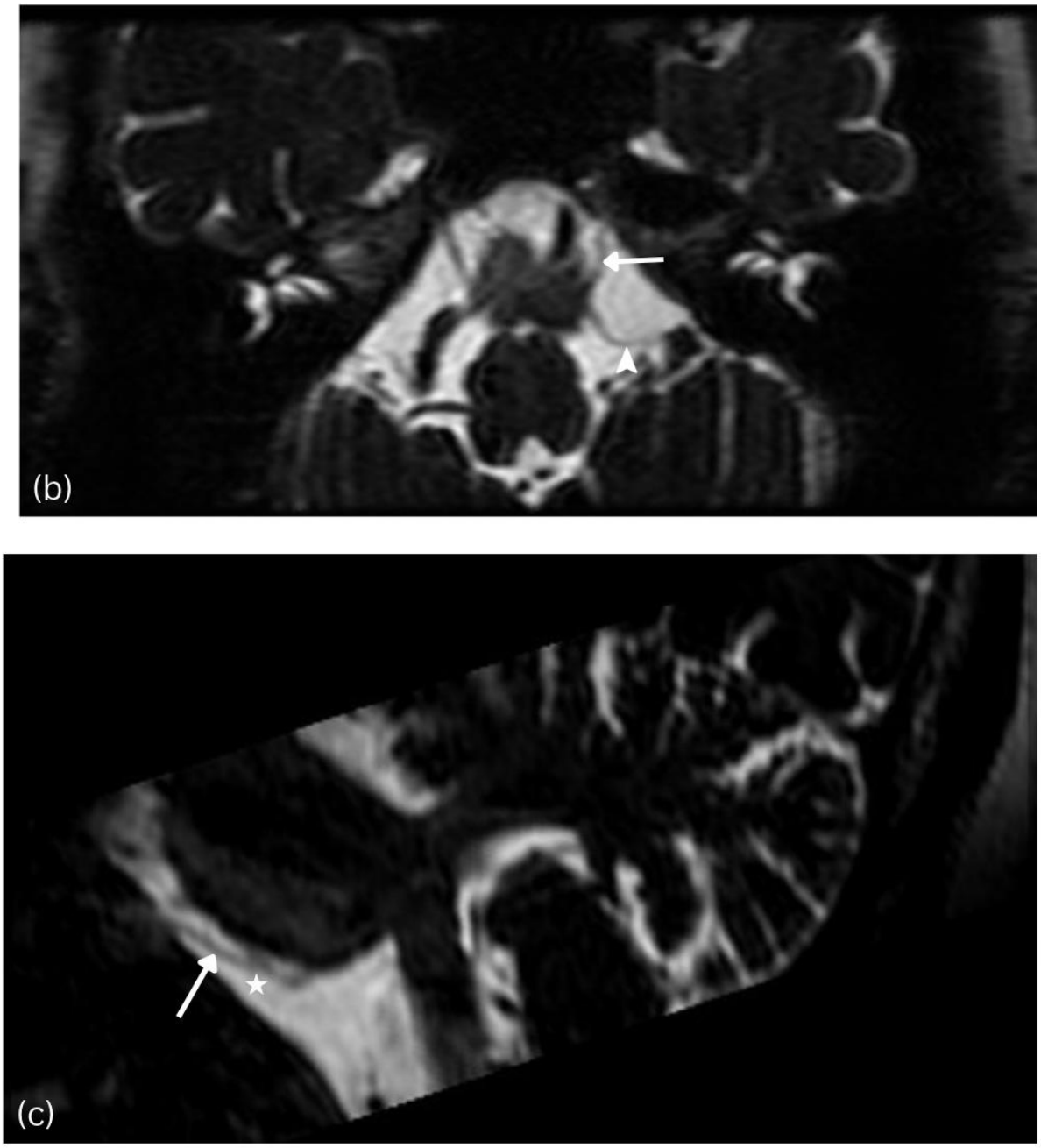



Figure 1. High-resolution 3D T2-DRIVE MRI (slice thickness 1.0 mm; TR 2000 ms; TE 246.2 ms). Axial view (Fig. 1a) and axial-oblique view (Fig. 1b) at the pontomedullary junction, and oblique-sagittal reformats (Fig. 1c) demonstrate the left AICA loop (arrowhead) contacting the cisternal segment of the left abducens nerve (arrow) at the root exit zone. Focal bowing of the nerve is evident compared with the contralateral side. The star denotes the point of neurovascular contact.

Intravenous dexamethasone at a dose of 4 mg three times daily was commenced on day 2 of admission for presumed inflammatory neuropathy and continued for three days, during which only slight symptomatic improvement was observed. She was discharged with an eye patch and oral dexamethasone 4 mg once daily for one week, followed by 2 mg once daily for one week. At clinic review 3 months after symptom onset, she reported near-complete improvement with minimal residual diplopia. She was able to perform daily activities without the eye patch. No objective orthoptic measurement was recorded at that visit. She subsequently defaulted from follow-up, and neither complete clinical resolution nor radiological change could be confirmed.

## DISCUSSION

This case demonstrates a focal neurovascular relationship between an AICA loop and the cisternal

segment of the left abducens nerve. The imaging diagnosis was based not merely on contact but on focal bowing of the symptomatic nerve at the root exit zone compared with the unaffected side. This distinction is important because AICA loops are common in asymptomatic individuals (G *et al.*, 2024). Nevertheless, radiological evidence of nerve deformation remains supportive rather than absolute proof of causality, particularly when a competing vascular risk factor is present.

The abducens nerve emerges from the brainstem at the pontomedullary junction, traverses the prepontine cistern and Dorello's canal, and then enters the cavernous sinus before reaching the lateral rectus muscle (Nguyen *et al.*, 2022). Its cisternal segment can lie close to the AICA. In a cadaveric study, AICA branches pierced the abducens nerve in 5 of 44 hemispheres, illustrating the potential for clinically relevant neurovascular relationships (Esmer *et al.*, 2010). High-resolution three-dimensional heavily T2-weighted imaging and MRA improve diagnostic confidence by clearly depicting the relationship between vascular structures and the abducens nerve. Incorporating these sequences into routine imaging protocols in selected cases may help detect rare but clinically relevant compressive lesions that could otherwise be overlooked (Hara *et al.*, 2022; Aggarwal *et al.*, 2023).

The Chavda and Gorrie classifications were not applied in this case. These systems were developed to describe AICA loop extension into the internal auditory canal and their relationship with the facial-vestibulocochlear nerve complex, rather than contact with the abducens nerve (Kim *et al.*, 2019). We did not identify a validated classification specific to AICA-related compression of cranial nerve VI. Accordingly, the present case is characterised descriptively by the affected nerve segment, site of vascular contact and presence of focal bowing.

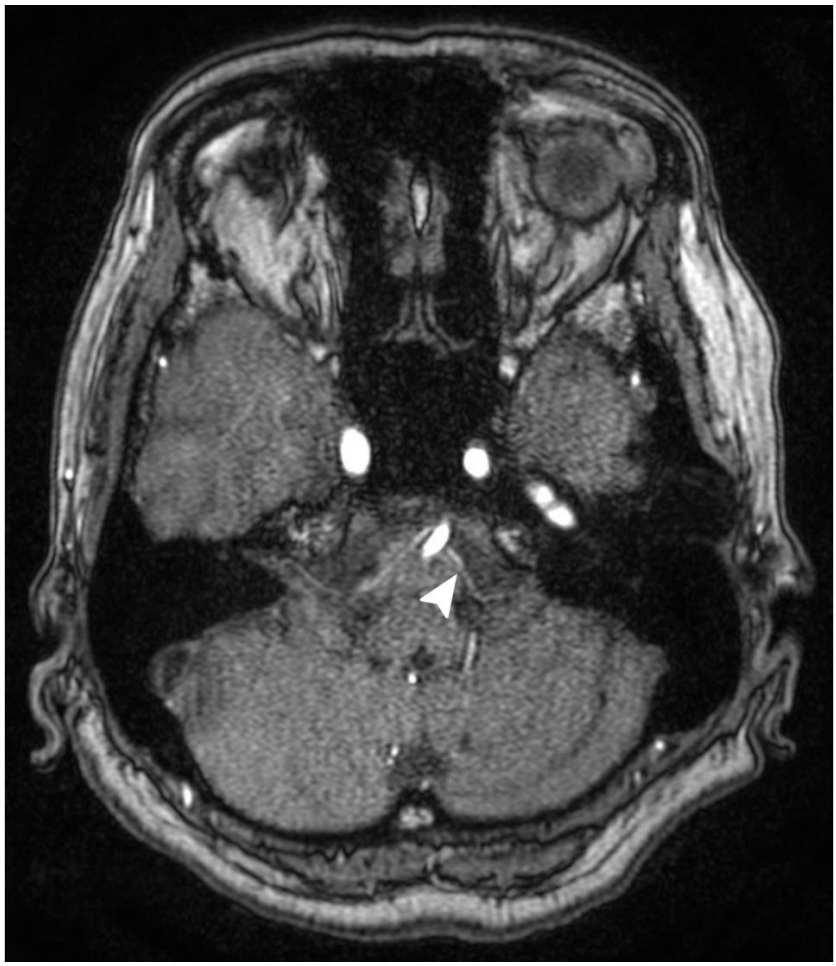


Figure 2. Non-contrast phase-contrast MRA of the circle of Willis with SENSE acquisition (slice thickness 1.2 mm; TR 20 ms; TE 3.5 ms) demonstrates the left AICA loop (arrowhead) coursing towards the pontomedullary junction and the expected root exit zone of the left abducens nerve. No intracranial or cavernous internal carotid artery aneurysm is identified.

Alternative causes were assessed systematically. Normal HbA1c and glucose measurements, together with the absence of known hypertension and non-hypertensive inpatient readings reduced the likelihood of diabetic or hypertensive microvascular neuropathy. However, repeated elevation of total and LDL cholesterol demonstrated dyslipidaemia, so a microvascular mechanism could not be fully excluded. CT and MRI showed no brainstem infarction, mass, demyelinating-appearing focal lesion, hydrocephalus, petrous apex, cavernous sinus or orbital lesion. Phase-contrast MRA showed no intracranial or cavernous internal carotid artery aneurysm. Giant cell arteritis was considered less likely because no headache or other systemic symptoms were reported and the ESR, CRP and platelet count were not elevated. However, specific contemporaneous documentation of GCA-associated symptoms was unavailable, and normal inflammatory markers alone do not definitively exclude the disease (Hellmich *et al.*, 2020).

Dexamethasone was started before MRI as empirical treatment for presumed inflammatory neuropathy, with only slight initial improvement observed. The later near-resolution therefore cannot be confidently attributed either to corticosteroid therapy or to relief of neurovascular compression. Eye patching provided temporary symptomatic control. If disabling diplopia had persisted, options would have included neuro-ophthalmic reassessment, prism therapy or occlusion, followed by consideration of botulinum toxin or strabismus surgery once the deviation stabilised. In a carefully selected patient with persistent symptoms and convincing nerve deformation, multidisciplinary neurosurgical assessment for microvascular decompression may be considered, although the evidence is limited to small case reports and series (Miyamoto *et al.*, 2020; Jain, 2022; Aggarwal *et al.*, 2023).

The overall prognosis of unilateral isolated sixth nerve palsy is often favourable. In a cohort of 213 non-traumatic cases, 78.4% recovered spontaneously, including 36.6% by 8 weeks and 73.7% by 24 weeks (King *et al.*, 1995). These figures are not specific to neurovascular compression. In a small review of reported neurovascular compression cases, all three patients treated with microvascular decompression achieved full recovery, whereas only one of five observed patients showed slight improvement; the very small, selected sample precludes comparative conclusions (Miyamoto *et al.*, 2020).

This report has several limitations. The patient had dyslipidaemia, so microvascular ischaemia remained a competing explanation. Although inflammatory markers were not elevated, specific GCA symptoms were not itemised contemporaneously. No objective orthoptic measurement was recorded at the final visit, and the patient subsequently defaulted from follow-up. No follow-up MRI was obtained; consequently, complete clinical resolution and radiological relief of compression were not demonstrated. These limitations support the interpretation of the AICA loop as a probable symptomatic neurovascular conflict rather than as definitive evidence of causation.

## CONCLUSION

An AICA loop associated with focal bowing of the cisternal segment of the abducens nerve at the root exit zone is a rare but important potential cause of isolated sixth nerve palsy. High-resolution three-dimensional T2-weighted MRI and MRA can demonstrate the nerve-vessel relationship when routine CT is unrevealing. Objective nerve deformation and comparison with the contralateral side provide stronger evidence than vascular contact alone. Nevertheless, neurovascular compression should be interpreted cautiously after assessment of more common causes, particularly when vascular risk factors such as dyslipidaemia coexist.

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