



RESEARCH ARTICLE

An Unusual Culprit: Vertebrobasilar Dolichoectasia Presenting as Trigeminal Neuralgia

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ABSTRACT

Background: Trigeminal neuralgia (TN) is a debilitating neuropathic pain disorder most commonly caused by neurovascular compression at the trigeminal nerve root entry zone. While classical cases are typically due to small arterial loops, secondary causes such as vertebrobasilar dolichoectasia (VBD) are rare but clinically significant and often under-recognized.

Case Presentation: We report a 62-year-old male presenting with a 6-month history of recurrent, severe, right-sided, electric shock-like facial pain involving the maxillary and mandibular divisions of the trigeminal nerve. The pain was triggered by routine activities such as chewing and speaking, consistent with classical trigeminal neuralgia. Neurological examination revealed mild hyperesthesia over the affected dermatomes without motor deficit.

Magnetic resonance imaging with angiography demonstrated an abnormal vertebrobasilar configuration with significant rightward lateral displacement and high bifurcation of the basilar artery. Although the arterial diameter did not meet conventional criteria for ectasia, the vessel was seen abutting and indenting the cisternal segment of the right trigeminal nerve, confirming neurovascular compression. These findings were consistent with vertebrobasilar dolichoectasia based on Smoker's criteria.

The patient was treated with carbamazepine, resulting in significant symptomatic improvement, and remains under follow-up for potential surgical intervention if symptoms become refractory.

Discussion: This case highlights an important diagnostic and pathophysiological consideration: vascular geometry and displacement may be more critical than arterial diameter in producing symptomatic neurovascular conflict. Unlike classical TN, VBD-associated TN involves a large, tortuous vessel capable of exerting sustained mechanical distortion on the trigeminal nerve. Recognition of such structural causes is essential, as they carry distinct prognostic and therapeutic implications.

Conclusion: Vertebrobasilar dolichoectasia is an uncommon but important cause of secondary trigeminal neuralgia. Detailed neurovascular imaging is crucial for diagnosis, as identification of vascular morphology—not merely vessel caliber—can influence clinical decision-making and long-term management.

Keywords: Trigeminal neuralgia; Vertebrobasilar dolichoectasia; Neurovascular compression; MRI; Basilar artery; Smoker's criteria
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INTRODUCTION

Trigeminal neuralgia (TN) is a chronic neuropathic pain disorder characterized by recurrent, unilateral, brief episodes of electric shock-like facial pain in the distribution of one or more divisions of the trigeminal nerve. It is most commonly caused by neurovascular compression at the root entry zone (REZ), a region particularly vulnerable due to the transition between central and peripheral myelin typically by the superior cerebellar artery[1].

While most cases are classified as classical or idiopathic, secondary TN due to structural causes is increasingly recognized. These include tumors, demyelinating diseases, and vascular anomalies such as vertebrobasilar dolichoectasia (VBD)[2].

VBD is defined by elongation, dilatation, and tortuosity of the vertebrobasilar arterial system, which may compress adjacent cranial nerves or brainstem structures.³

Although uncommon, it represents an important and potentially underdiagnosed cause of TN, particularly in elderly patients.

However, literature on VBD-related trigeminal neuralgia remains limited, especially from resource-constrained settings, and carries distinct diagnostic and therapeutic implications.

This report highlights a case of TN secondary to VBD, emphasizing the role of detailed neurological examination and targeted neurovascular imaging.

Case Presentation

A 50 yearsold female presented with a 3-month history of recurrent, severe, right-sided facial pain involving the maxillary (V2) and mandibular (V3) divisions of the trigeminal nerve. The pain was described as sudden, electric shock-like, lasting for a few seconds, and triggered by

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activities such as chewing, speaking, brushing teeth, and light touch over the face. Over time, the frequency and intensity of episodes increased, significantly impairing daily activities. There was no history of similar illness in the past, trauma, visual disturbances, hearing loss, or features suggestive of demyelinating disease.

On examination, the patient was conscious and oriented, with blood pressure of 120/70 mmHg. Cranial nerve examination revealed preserved motor function of the trigeminal nerve. Sensory examination demonstrated mild hyperesthesia over the right V2 and V3 dermatomes. Corneal reflex was intact bilaterally. No facial asymmetry or involvement of other cranial nerves was noted. Motor and sensory examination of the limbs was normal, and cerebellar signs were absent.

Laboratory investigations were within normal limits, including hemoglobin of 12.8 g/dL, total leukocyte count of 7,600/mm³, platelet count of $2.4 \times 10^5/\text{mm}^3$, serum creatinine of 0.9 mg/dL, blood urea of 28 mg/dL, and normal liver function tests. Fasting blood glucose was 108 mg/dL, and serum electrolytes were within normal range.

Magnetic resonance imaging (MRI) of the brain, along with magnetic resonance angiography (MRA), revealed a markedly dilated, elongated, and tortuous basilar artery deviating laterally toward the right side. The ectatic vessel was seen abutting and compressing the root entry zone of the right trigeminal nerve at the level of the pons. There was no evidence of demyelinating plaques, space-occupying lesions, or infarcts. The findings were consistent with vertebrobasilar dolichoectasia causing neurovascular compression.

Magnetic resonance imaging (MRI) of the brain with magnetic resonance angiography (MRA) revealed an abnormal configuration of the vertebrobasilar arterial system. The basilar artery demonstrated significant lateral displacement toward the right side, reaching the lateral margin of the clivus, along with a high bifurcation above the level of the suprasellar cistern, consistent with vertebrobasilar dolichoectasia as per Smoker's radiological criteria.

The measured basilar artery diameter was approximately 3.5 mm, which did not meet the threshold for ectasia; however, the presence of marked lateral deviation (score 2) and elevated bifurcation (score 2) supported the diagnosis of dolichoectatic morphology.

On high-resolution imaging, the dolichoectatic basilar artery was seen abutting and indenting the cisternal segment of the right trigeminal nerve, consistent with neurovascular compression. No evidence of trigeminal nerve atrophy, demyelinating lesions, infarction, or space-occupying lesions was identified.

These findings correlated with the patient's clinical presentation and confirmed the diagnosis of secondary trigeminal neuralgia due to vertebrobasilar dolichoectasia.

Treatment Course

The patient was initiated on carbamazepine 100 mg twice daily, gradually titrated to 200 mg three times daily, resulting in marked reduction in pain frequency and severity. The patient is currently under regular follow-up, with consideration of microvascular decompression in case of medically refractory symptoms.

(A) Axial TOF MR angiography demonstrating high bifurcation of the basilar artery above the suprasellar cistern (arrow), corresponding to Smoker's score 2.

(B) Image showing rightward lateral displacement of the basilar artery reaching the lateral margin of the clivus (arrow), consistent with Smoker's score 2 for laterality.

(C) Axial image depicting dolichoectatic basilar artery indenting the cisternal segment of the right trigeminal nerve near Meckel's cave (arrow), indicating neurovascular compression without nerve atrophy

DISCUSSION

The present case highlights the importance of detailed vascular and anatomical assessment in patients with trigeminal neuralgia (TN), particularly when an atypical neurovascular configuration is suspected. Although focal neurovascular compression at the root entry zone (REZ) of the trigeminal nerve remains the most recognized mechanism underlying classical TN, variations in vascular

anatomy may significantly influence nerve irritation and symptom development. The REZ represents a vulnerable transitional region between central and peripheral myelin, where chronic pulsatile vascular contact can result in focal demyelination and aberrant ephaptic transmission, producing characteristic paroxysmal facial pain.¹ In the present case, the presence of vertebrobasilardolichoectatic morphology with significant displacement of adjacent structures emphasizes the potential role of vascular geometry in symptomatic trigeminal nerve compression.

While classical TN is most commonly associated with compression by the superior cerebellar artery, secondary causes including tumors, demyelinating disorders, and vascular abnormalities should be considered, particularly in patients with atypical clinical or radiological features[2]. Vertebrobasilardolichoectasia (VBD) represents an uncommon but clinically important vascular abnormality that may produce cranial nerve symptoms through mechanical distortion of adjacent neural structures. Unlike typical arterial loops causing focal compression, dolichoectatic vessels may cause more extensive displacement because of their increased size, tortuosity, and altered vascular course.

The radiological diagnosis of VBD is primarily based on assessment of vertebrobasilar artery morphology using established imaging criteria, including Smoker's criteria, which evaluate basilar artery diameter, lateral displacement, and bifurcation height.³ In the present case, although the basilar artery diameter (~3.5 mm) did not meet the conventional threshold for ectasia, marked lateral displacement (score 2) and elevated bifurcation (score 2) demonstrated significant dolichoectatic morphology. This finding suggests that clinically relevant neurovascular conflict may occur even in the absence of pronounced arterial enlargement.

An important observation from this case is that vascular configuration and spatial relationship with the trigeminal nerve may be more clinically significant than vessel diameter alone[3-4]. In classical TN, a relatively small arterial loop may produce focal pulsatile compression at the REZ, whereas VBD-associated TN involves a large, elongated, and tortuous vascular structure capable of producing sustained mechanical distortion.⁴ Continuous pressure from such vessels may contribute not only to demyelination but also to displacement and deformation of the nerve, potentially explaining differences in symptom severity, treatment response, and recurrence patterns.

Comparison with previously reported literature

indicates that most cases of VBD-associated TN involve marked vertebrobasilar dilatation, frequently exceeding 4.5 mm, with associated brainstem compression or involvement of multiple cranial nerves. However, the present case demonstrates that symptomatic trigeminal nerve involvement can occur despite the absence of significant basilar artery enlargement. The presence of substantial lateral displacement and abnormal vascular trajectory may therefore represent important contributors to neurovascular conflict. Similar observations have suggested that morphological characteristics, including tortuosity and displacement, may have greater clinical relevance than diameter measurements alone. This differs from classical TN, where focal compression by a smaller vascular loop generally occurs without major anatomical distortion of surrounding structures[5-7].

From a therapeutic perspective, VBD-associated TN may have a more complex clinical course compared with classical TN. Carbamazepine remains the first-line pharmacological therapy for TN and provided symptomatic relief in the present case. However, persistence of the underlying vascular abnormality may predispose patients to recurrence or progression of symptoms, necessitating long-term clinical monitoring[8].

For patients with medically refractory symptoms, microvascular decompression (MVD) remains the definitive surgical treatment option. However, VBD-related TN presents unique technical challenges because of the size, rigidity, and reduced mobility of the dolichoectatic artery.⁶ Adequate mobilization and repositioning of the vessel may be difficult, and the risk of incomplete decompression or vascular complications may be higher compared with conventional neurovascular compression syndromes. Therefore, careful preoperative imaging assessment is essential for surgical planning.

A limitation of the present case report is the absence of intraoperative confirmation of the neurovascular relationship, which could have provided direct anatomical evidence of the compression pattern. Nevertheless, high-resolution magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) demonstrated characteristic vascular displacement and morphological abnormalities supporting the diagnosis of VBD-associated trigeminal neuralgia.

This case emphasizes that evaluation of trigeminal neuralgia should extend beyond simple identification of vascular contact and should include comprehensive assessment of vascular morphology, displacement, and

anatomical relationships. Recognition of these imaging characteristics may improve diagnostic accuracy, assist in treatment planning, and provide better understanding of the mechanisms underlying atypical forms of trigeminal neuralgia. Further studies are required to determine the prognostic significance of vascular displacement parameters compared with arterial diameter in patients with VBD-associated TN.

Learning Point

- Not all cases of trigeminal neuralgia are idiopathic; secondary causes should always be actively sought.
- A detailed trigeminal nerve examination remains a crucial bedside tool for identifying patients who require further etiological evaluation.
- MRI with vascular imaging is essential for detecting neurovascular conflict and differentiating classical from secondary TN.
- Vertebrobasilar dolichoectasia, although rare, should be considered in elderly patients with TN.

CONCLUSION

Vertebrobasilar dolichoectasia is an uncommon but clinically significant cause of secondary trigeminal neuralgia. This case underscores that vascular tortuosity and displacement, even in the absence of marked arterial dilatation, can result in significant neurovascular compression.

Early identification using high-resolution MRI and MR angiography is essential, as management extends beyond symptomatic therapy and may require surgical intervention in refractory cases. Recognition of this entity is crucial for accurate diagnosis, prognostication, and individualized treatment planning.

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