Diplopia following microvascular decompression surgeries: illustrative cases

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BACKGROUND Microvascular decompression is an effective treatment strategy for trigeminal neuralgia. However, there may be inadvertent complications involving adjacent cranial nerves during or months after the operation. This case lesson highlights the potential manifestations, both optical and nonneurologic (monocular) and binocular diplopia, after microvascular decompression in two patients. Neurosurgeons should recognize monocular versus binocular causes of diplopia after neurosurgical microvascular decompression.

OBSERVATIONS The authors reported on two patients who presented with diplopia after microvascular decompression for trigeminal neuralgia. The first patient had binocular diplopia with a paradoxical head tilt potentially due to a contiguous trochlear nerve palsy. The second patient had monocular diplopia due to dry eye syndrome from trigeminal nerve dysfunction. However, within 2 years after their operations, both patients had resolution of their diplopia without additional surgical intervention.

LESSONS Both monocular and binocular diplopia can be presenting symptoms of cranial neuropathies after microvascular decompression for trigeminal neuralgia. Most cases of postoperative diplopia (both monocular and binocular) resolve spontaneously over time without additional neurosurgical treatment.

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KEYWORDS microvascular decompression; diplopia; monocular; binocular; trigeminal neuralgia

Microvascular decompression (MVD) is an effective treatment for vascular compressive etiologies for trigeminal neuralgia (TN) that fail maximal medical therapy.1 In MVD, the compressed nerve root is freed from the impinging vasculature, and a barrier, often made of Teflon, is placed to separate the aberrant vessel from the involved trigeminal nerve.2 Postsurgical side effects include tinnitus, hearing disturbances, vertigo, facial numbness and palsy, and diplopia.3,4 Additionally, up to 30% of patients report recurrent pain 4 years after MVD surgery for TN.1 Risk factors for eventual pain recurrence include age younger than 53 years at the time of surgery, symptoms lasting longer than 11.5 years, female sex, and pain on the left side.1

In general, approximately 16% of patients undergoing MVD for TN will experience symptoms related to cranial nerve (CN) or cerebellar dysfunction.4 The percentage of patients enduring postsurgical complications is decreased to approximately 5% after 3 years.4 Such nerve dysfunction is likely due to direct physical interaction with the affected CNs. Stretching of the CN, inadvertent trauma to the nerve, and acoustic trauma incited by the use of a drill have all been suggested as causative factors, particularly in explaining CN VIII–related symptoms after MVD.5

The occurrence of diplopia after MVD is uncommon, but neurosurgeons need to differentiate monocular from binocular etiologies for diplopia. Up to 3%–6% of patients with MVD reported immediate diplopia postoperatively, whereas only 1.3% cases of reported diplopia persisted for more than 3 years.4,6 Diplopia also seems to depend on the type of compressive pattern; MVD involving the
superior cerebellar artery (SCA) has shown lower rates of diplopia occurrence compared to MVD involving non-SCA arteries. The likelihood of other postoperative complications (e.g., facial numbness) increases with the number of MVD revisions. Ocular motor CN (e.g., III, IV, VI) palsies cause binocular diplopia. Of the reported post-MVD ocular motor palsies, most cases are described as transient or self-resolving causes of diplopia, whereas less than 0.2% are described as permanent. We report two cases of diplopia after MVD: one monocular and one binocular diplopia. We compare and contrast the two cases to emphasize the key and differentiating features between monocular and binocular diplopia.

Illustrative Cases

Case 1
A 54-year-old woman with a 2.5-year history of TN treated with MVD presented with binocular oblique diplopia associated with left hemifacial spasm, left facial droop, left facial weakness, left eye pain, and entire left side facial pain. Her medical history was significant for LASIK in both eyes, which caused dry eye and episodes of blurred vision that improved with blinking and topical artificial tears. She had been diagnosed 2.5 years earlier with idiopathic TN in a left V1 distribution. Radiographic imaging identified dolichoectasia of the anterior inferior cerebellar artery (AICA) compressing the ipsilateral left trigeminal nerve at its root exit zone. She received MVD of the left trigeminal nerve 1.5 years after diagnosis, and a Teflon pad was placed between the root exit zone of the trigeminal nerve and the AICA. No complications were noted during the surgery. However, upon awakening, she experienced sudden-onset, constant, binocular oblique diplopia consistent with a contiguous CN IV nerve palsy; this was associated with left facial droop, left eye pain, and facial pain in a V1-V3 distribution. She later developed left hemifacial spasms. Post-surgical magnetic resonance imaging did not show a Teflon granuloma between the trigeminal nerve and AICA.

Initial neuroophthalmic examination showed visual acuity of 20/25, and color plates were 14/14 in both eyes. Pupils were reactive with no anisocoria or relative afferent pupillary defect. Slit-lamp examination and intraocular pressures were normal in both eyes. External examination showed she had an intermittent hemifacial spasm on the left. Motility findings showed a 7° compensatory head tilt to the left. There was a left hypertropia (LHT) of 10 prism diopters (PD) in primary gaze. The LHT increased in right gaze (16 PD) compared with 4 PD LHT in left gaze. The LHT increased in left head tilt (14 PD) versus 5 PD LHT in right head tilt. These findings were consistent with a left CN IV palsy. Double Maddox rod test showed exyclotorsion in the left eye. Dilated fundus examination also showed exyclotorsion in the left eye.

Case 2
A 66-year-old white man presented with a chief report of monocular diplopia and pain in his left eye after undergoing an MVD procedure with Teflon cushion for TN. He had been originally diagnosed with TN in the 1980s, at which point he described multiple paroxysmal attacks of pain in a left V3 distribution. Despite being treated with medical therapy, he reported continued symptoms. Imaging studies were performed, and no tumors or masses were noted. Approximately 10 years later, he received initial MVD on the left side that provided symptom relief for approximately 27 years, and then his facial pain returned. Approximately 30 years after the initial MVD, an MVD revision for TN was performed with the placement of a Teflon cushion between the trigeminal nerve and the dolichoectatic artery, which once again resolved his symptoms. Several weeks after the surgery, he noticed persistent numbness on the left side of the face as well as dry eye, irritation, redness, monocular diplopia, eye pain, and headache. None of these symptoms were present prior to the MVD revision. His symptoms of “double and triple vision” persisted for a full year before resolving spontaneously. Symptoms of blurry vision, eye irritation, and headache were still present at the time of the patient’s last visit.

His medical history included hypertension, rheumatoid arthritis, hypothyroidism, diabetes mellitus type 2, congestive heart failure, and gastroesophageal reflux disease.

A computed tomography scan of the head without contrast obtained in July 2020 showed expected postoperative changes from his prior craniotomies and no evidence of a Teflon granuloma.

On clinical examination, his visual acuity was 20/20 in the right eye and 20/40 in the left eye. Silt-lamp examination revealed a few punctate epithelial erosions in the interpalpebral fissure zone in the left eye. Intraocular pressure was 20 mm Hg in each eye. Fundus examination was unremarkable. Numbness and decreased corneal sensation were present on the left side of the face. Both eyes exhibited full motility, straightness in the primary position, and diagnostic positions of gaze. There was no evidence of ocular motor palsy or ocular misalignment. The monocular diplopia resolved with the pinhole test.

Discussion

Observations
Diplopia is defined as seeing two images when viewing a single object. Table 1 summarizes the similarities and differences between monocular and binocular diplopia. Diplopia can be divided into monocular and binocular diplopia. Monocular diplopia occurs when two images are viewed with only one eye but can be either unilateral or bilateral. Monocular diplopia is typically optical (e.g., dry eye, refractive error, cataracts) and not neurological. It is important to note that trigeminal nerve dysfunction may lead to neurotrophic corneal changes and dry eye that can produce monocular diplopia.

The second image in monocular diplopia is typically characterized by a clearer image associated with a second “ghost image” or “shadow image.”

Rarely, monocular diplopia or even triplopia can be bilateral and cerebral in origin. In cerebral polyopia, the images are all equally clear and do not resolve with the pinhole test. The differential diagnosis includes postoperative occipital lobe strokes or seizures. The pinhole test can be used to distinguish optical from cerebral causes of monocular diplopia.

Binocular diplopia occurs when seeing two images using both eyes, and the diplopia resolves when either eye is covered. There are typically two distinct images viewed in equal clarity, and they may be arranged in vertical, horizontal, or oblique orientations. Binocular diplopia is considered dangerous because it is often due to serious underlying neurological conditions.

Binocular diplopia occurs due to misalignment of the eyes. Etiologies of binocular diplopia after MVD includes a lesion anywhere along the efferent ocular motor pathway. This pathway includes the supranuclear, nuclear, and infranuclear segments. Furthermore, the infranuclear pathway is subdivided into the peripheral nerve, the neuromuscular junction, and extraocular muscular components. Damage or disorder of any part may lead to binocular diplopia.
TABLE 1. Comparison, monocular diplopia and binocular diplopia after MVD

<table>
<thead>
<tr>
<th>Onset after op</th>
<th>Monocular Diplopia After MVD</th>
<th>Binocular Diplopia After MVD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duration</td>
<td>Immediate to delayed (days to wks)</td>
<td>Variable, typically noted within a few days of op</td>
</tr>
<tr>
<td>Affected CN(s)</td>
<td>V (trigeminal neuropathy)</td>
<td>III (least likely), IV (common), VI (less common)</td>
</tr>
<tr>
<td>Associated Sx</td>
<td>Ipsilateral dry eye, pain, irritation, facial numbness</td>
<td>Binocular horizontal or oblique diplopia; may have other cranial neuropathies</td>
</tr>
<tr>
<td>Visual acuity</td>
<td>May be affected from corneal changes; improves/resolves with pinhole test</td>
<td>Unaffected</td>
</tr>
<tr>
<td>Exam</td>
<td>May show ipsilateral decreased corneal sensation, full extraocular motility, &amp; alignment</td>
<td>May have ductional deficit &amp; ocular misalignment on cover/uncover testing</td>
</tr>
<tr>
<td>Tx</td>
<td>Self-limited, conservative topical Tx</td>
<td>Patching, prism, strabismus op (if unresolved despite conservative management)</td>
</tr>
</tbody>
</table>

Sx = symptom; Tx = treatment.

Supranuclear causes of binocular diplopia include decompensation of preexisting phoria (a tendency for the eye to drift that is kept in check with fusion). This break- down of fusion can occur after any surgery, including MVD. CN nuclear lesions may involve the oculomotor, trochlear, or abducens nuclei from ischemia or hemorrhage in the brainstem after MVD. Infra- nuclear causes include cranial neuropathies (e.g., CN III, IV, and VI palsies) and may occur after MVD. Barker et al. described 15 patients (out of 1,336) who developed binocular diplopia from extracranial muscle palsies after MVD. There were 11 transient cases of CN IV palsy and two transient cases of CN VI palsy; the last two cases of diplopia were deemed due to permanent CN IV palsy after the diplopia did not resolve in the following 1 year.

In our first case, the diplopia was due to a left CN IV (trochlear) palsy. Surgery involving the root of the trigeminal nerve (e.g., MVD for TN) occurs in close proximity to the trochlear nerve and can produce an ipsilateral hypertropia and an “oblique” diplopia. Typically, patients who have CN IV palsies have a head tilt to the contralateral side to compensate for loss of intorsion of the superior oblique muscle. However, approximately 3.4% of patients (including our patient) with trochlear nerve palsies have a “paradoxi- cal” head tilt to the ipsilateral side of the palsy. If a head tilt contralateral to the palsy is unable to minimize the hypertropia adequately, binocular image fusion and single vision cannot be attained; in these cases, a “paradoxi- cal” head tilt ipsilateral to the palsy may be adopted to sufficiently increase the hypertropia and completely split the two images. This approach allows one image to more easily be ignored.

Treatment of binocular diplopia includes observation, patching of one eye to relieve symptoms, prism therapy, or, if unresolved with conservative therapy, strabismus surgery. Mizobuchi et al. reported a “delayed therapeutic effect” after MVD, in which half of the 10% of patients experiencing headaches 1 week after surgery had spontaneous relief after 3 years. Notably, nonpain complications 1 week post-operation, such as diplopia, were also noted to resolve without treatment after 3 years. The exact physiology and timeline of these delayed cases remain unclear. CN IV palsy usually resolves in the reported cases between 1 and 3 years after MVD.

Lessons

These cases illustrate that both monocular and binocular diplopia may occur after MVD for trigeminal neuralgia. Neurosurgeons should be able to differentiate between monocular and binocular diplopia. Most cases involve CN IV palsy and typically resolve over time.

References


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**Disclosures**

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

**Author Contributions**

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