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Radiologic-Clinical Correlation
Junctional Visual Field Loss

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Nerve fibers originating in the retina follow a specific topographical arrangement in the optic nerve and chiasm. Compressive lesions at the junction of the intracranial optic nerve and optic chiasm can produce characteristic visual field defects. Optic nerve involvement at the junction of the optic chiasm results in unilateral visual field loss. If fibers from the inferonasal retina of the contralateral eye (Wilbrand's knee) are involved, there will also be a superotemporal visual field defect in the contralateral eye. These defects can be produced by pituitary tumors, suprasellar meningiomas, supraclinoid aneurysms, craniopharyngiomas, gliomas, and other entities (1–4). We describe two patients with intracranial lesions and junctional visual field defects.

Clinical Histories

Case 1

A 46-year-old morbidly obese woman presented with painless, progressive loss of vision in the right eye over 1 year. Her symptoms began as mild, gradually progressive visual loss in the right eye 1 year before, which worsened over the next several months to complete blindness. She felt that her left

eye was normal. She had no other neurologic or ophthalmologic symptoms. Her medical history was unremarkable, and she was taking no medications. Neuroophthalmologic exam revealed a visual acuity of no light perception in the right eye and 20/20 in the left eye. She correctly identified 14 of 14 Ishihara color plates in the left eye. The pupils were isocoric, and the left pupil reacted normally to light. The right pupil was nonreactive. Motility examination showed full ductions and versions, but there was a 20-prism diopter sensory exotropia present. Findings of slit lamp biomicroscopy, intraocular pressure measurements, and external examination were normal. Ophthalmoscopic examination revealed diffuse pallor of the optic disk on the right with a normal optic disk on the left. The remainder of the retinal examination in both eyes was normal. Static visual field testing of the left eye revealed a superior temporal arcuate defect extending from the blind spot to the vertical midline (Fig 1A).

Magnetic resonance (MR) imaging could not be performed because of the morbid obesity of the patient. Computed tomography (CT) revealed a 2.5 × 2.1 × 2.0-cm well-circumscribed parasellar mass on the right, consistent with a giant aneurysm of the internal carotid artery (Fig 1B). The lesion showed homogenous enhancement after the administration of contrast material. Before cerebral arteriography could be performed, the patient became acutely obtunded at home and died. No postmortem study was obtained, but it was presumed that there had been a fatal rupture of the giant carotid artery aneurysm.

Fig 1. Case 1. *A,* Static visual field testing (Humphrey 30-2 program) of the left eye reveals a superotemporal arcuate defect extending from the blind spot to the vertical midline. There was no light perception in the right eye.

B, Postcontrast CT reveals a 2.5 × 2.1 × 2.0-cm mass (*arrow*) arising from the right internal carotid artery.
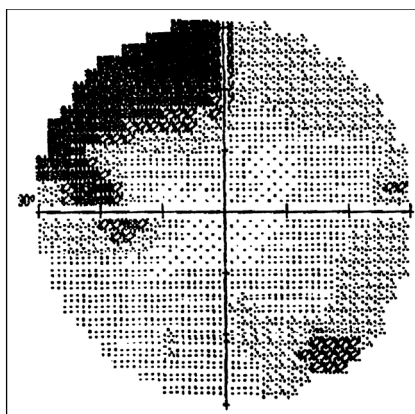
A   B

Case 2

A 61-year-old woman presented with painless, progressive visual loss in the left eye over the previous year. The patient denied any other neurologic or ophthalmologic symptoms. Medical history was significant for hypertension, hypothyroidism, and degenerative joint disease. Her medications included propranolol and levothyroxine. Neuroophthalmologic examination revealed visual acuity of 20/20 in the right eye and 20/200 in the left eye. The pupils were isocoric, and the right pupil reacted normally to light. The left pupil reacted sluggishly to light, and there was a left afferent pupillary defect. She correctly identified 14 of 14 Ishihara color plates with the right eye, but could see only gross colors with the left eye. Findings of slit lamp biomicroscopy, intraocular pressure measurements, and external and motility examinations were all normal. Ophthalmoscopic examination revealed diffuse optic atrophy on the left. The remainder of the retinal examination was within normal limits. Static visual field testing of the left eye revealed a temporal visual field defect involving central fixation (Fig 2A). Static visual field testing of the right eye was normal.

MR of the head revealed a 3.0 × 2.0 × 2.0-cm suprasellar mass extending from the left anterior clinoid region and the tuberculum sellae with compression of the left optic nerve and chiasm anteriorly. The lesion was isointense to brain on T1-weighted images (Fig 2B) and showed homogenous contrast enhancement (Fig 2C and D). On the T2-weighted images, the lesion was homogenously slightly hyperintense to gray matter. The MR findings were consistent with meningioma.

Discussion

The intracranial optic nerves extend posteriorly from the optic foramen and join at the optic chiasm. Within the chiasm, fibers from the nasal retina of each eye cross into the contralateral optic tract, and fibers from the temporal retina pass uncrossed into the ipsilateral optic tract (5). Within the intracranial optic nerve, the crossed (nasal retinal) and uncrossed (temporal retinal) fibers are anatomically separated at the junction of the optic nerve and chiasm. In addition, inferior nasal crossing fibers can loop anteriorly for a short distance into the contralateral optic nerve. These fibers are often referred to as the *anterior knee* or *Wilbrand's knee* (5) (Fig 3).

Lesions at the junction of the optic nerve and chiasm can produce specific types of visual field defects that allow topographical localization (6–8). Selective compression of the crossed or uncrossed visual fibers at the junction can result in a unilateral temporal (4) or nasal (2) hemianopic field defect, respectively. In addition, involvement of the inferonasal fibers of the anterior knee results in a superotemporal visual field defect contralateral to the lesion (5).

In 1927, H. M. Traquair (6) used the term *junction scotoma* to refer to a unilateral temporal hemicentral field defect caused by compression of the nasal fibers crossing in the intracranial optic nerve at the junction of the optic nerve and chiasm (Fig 3). Miller (7)
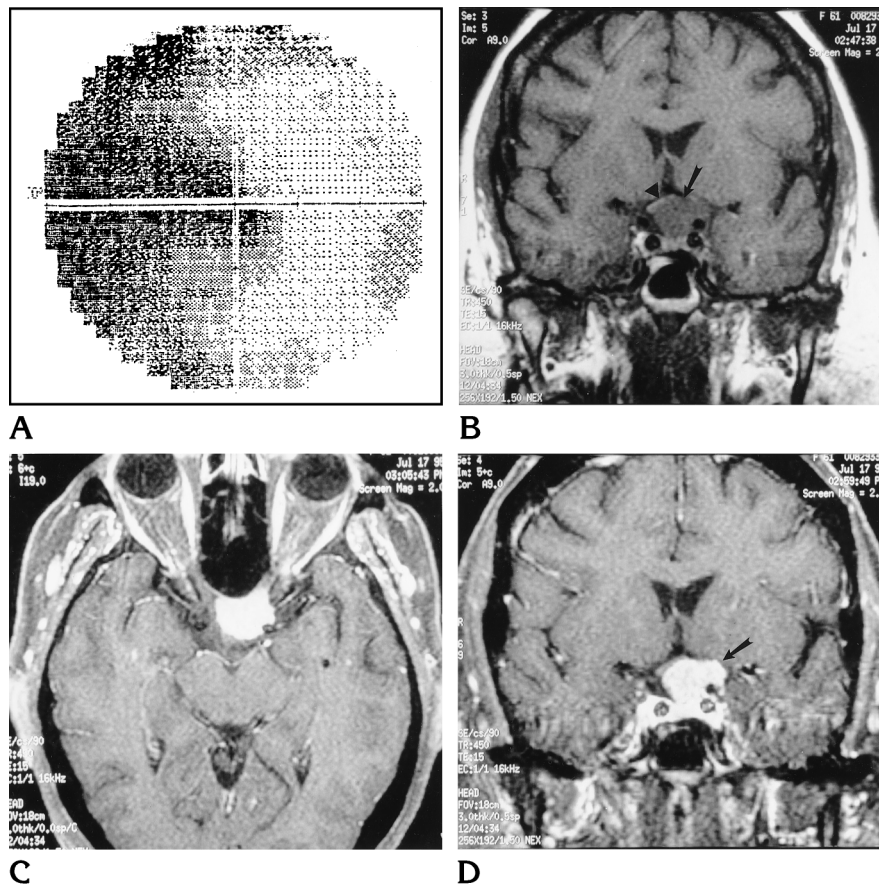
A

B

C

D

Fig 2. Case 2. *A*, Static visual field testing (Humphrey 30-2 program) of the left eye reveals a temporal hemifield defect with involvement of central fixation. Findings of visual field testing in the right eye were normal.

B, Coronal T1-weighted MR images (450/15/1 [repetition time/echo time/excitations]) reveals an isointense lesion (*arrow*) arising at the level of the anterior clinoid and compressing the left optic nerve and anterior optic chiasm (*arrowhead*).

C, Axial and *D*, coronal T1-weighted postcontrast MR images (450/15/1) show homogeneous enhancement of the mass (*arrow*).

emphasized that the junctional scotoma described by Traquair refers to a strictly unilateral temporal scotoma, which is assumed to arise from the junction of the optic nerve and chiasm. Unfortunately, some confusion has arisen regarding the use of the term *junctional scotoma*. Unlike Traquair (6), some authors have used the term to refer to an ipsilateral optic neuropathy with a contralateral superotemporal visual field defect. This superotemporal defect is caused by compression of the inferonasal fibers from the contralateral eye travelling in Wilbrand's knee (5).

To clarify this distinction, Miller (7), citing J. Lawton Smith, recommended that the unilateral temporal visual field defect described by Traquair should be referred to as the *junctional scotoma of Traquair* in order to differentiate it from the contralateral superotemporal defect more commonly referred to as the junctional scotoma.

Recently, the existence of Wilbrand's knee has come into question. Wilbrand was restricted to examining human subjects who

had undergone enucleation. In the enucleated eye, the nerve fibers atrophied and became distinct from the nerve fibers of the normal eye on myelin staining. J. C. Horton ("Wilbrand's Knee of the Optic Chiasm Is an Artifact of Long-Term Monocular Enucleation," presented at the annual meeting of the North American Neuro-Ophthalmology Society, Snowbird, Utah, February 1995) used axon labeling techniques in nonenucleated monkeys but was unable to demonstrate crossing fibers looping into the contralateral optic nerve (Wilbrand's knee). In one monkey that had undergone enucleation 4 years previously, however, nerve fiber topography similar to that described by Wilbrand was found. Horton hypothesized that Wilbrand's knee could be an artifact of enucleation caused by atrophy of the optic nerve, not a normal anatomic finding. Nevertheless, whether the Wilbrand's knee exists anatomically, the localizing value of junctional visual field loss to the junction of the optic nerve and chiasm remains undiminished since chiasmal com-
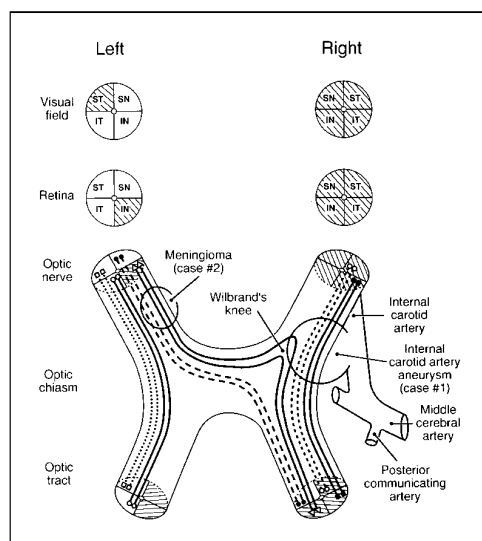
Fig 3. Schematic diagram illustrates the nasal fibers crossing from the ipsilateral retina to the contralateral optic tract via the optic chiasm (*solid* and *dotted lines*). The decussating inferonasal fibers, representing superotemporal visual field, pass for a short distance into the contralateral optic nerve as Wilbrand's knee (*solid line*, not drawn to scale). The *hatched area* represents the visual field deficit and the corresponding retinal areas as described in case 1. *ST* indicates superotemporal; *SN*, superonasal; *IT*, inferotemporal; and *IN*, inferonasal.

pression alone can result in the contralateral superotemporal visual field defect (junctional scotoma).

Trobe and Glaser (8) felt that junctional visual field loss would be caused by a mass lesion in 98 of 100 cases. The differential diagnosis of a junctional syndrome includes pituitary tumors, suprasellar meningiomas, supraclinoid aneurysms, craniopharyngiomas, and gliomas (1–4). Chiasmal neuritis and trauma are rare causes of the junctional syndrome.

Patients with the junctional scotoma of Traquair or the junctional scotoma should be considered to have a compressive lesion at the junction of the optic nerve and chiasm until proved otherwise. Neuroimaging studies should be directed to this location. Patients with the junctional scotoma can be unaware

of a small superotemporal visual field defect, and patients with strictly unilateral visual symptoms can be misdiagnosed as having an optic neuritis or other unilateral neuropathy. Therefore, in any patient with presumed unilateral visual loss, careful visual field testing should be performed in the contralateral asymptomatic eye.

Both of our patients presented with painless progressive loss of vision and clinical evidence of optic neuropathy. These findings are very suggestive of a compressive lesion of the anterior visual pathway. Junctional visual field defects in these patients were of localizing significance. Case 1 presented with an ipsilateral optic neuropathy and a contralateral superotemporal defect consistent with the junctional scotoma. Case 2 presented with temporal visual field loss consistent with the junctional scotoma of Traquair. Loss of visual acuity in this case was probably the result of further compression of the macular fibers in the intracranial optic nerve. Both cases illustrate the localizing value of visual field defects at the level of the junction.

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