Carotid-ophthalmic artery aneurysm masquerading as optic neuritis

Sir: Acute monocular visual loss secondary to optic nerve compression is unusual, but has been associated with aneurysms, tumours, fibrous dysplasia, and sphenoid sinus mucoceles and pyoceles.1-5 Generally, compressive optic nerve and chiasmal injuries are associated with fluctuating or progressive monocular visual loss, concomitant central scotomas, and contralateral visual field defects.5-9 Significant, spontaneous visual recovery has not been recognised. This case illustrates that compressive optic nerve lesions may clinically mimic optic neuritis.

A 48-year-old woman presented to her ophthalmologist with a complaint of blurred vision of the left eye. Additional symptoms included left orbito-temporal pain that increased with eye movement and brief “flashing light” phenomena. The initial examination revealed an afferent pupillary defect and mildly constricted visual fields by perfimetry in the left eye. In the ensuing 24 hours the patient’s visual acuity decreased to counting fingers concomitant with the development of hyperemic disc oedema and a large central scotoma by temporal fields. A pattern reversal VEP revealed a prolonged latency of 124 ms (control 110 ms). The visual acuity spontaneously and rapidly improved over one month, was associated with the disappearance of the afferent pupillary defect, and was 6/9 by 6 weeks. The right eye remained normal. Two months after the development of eye symptoms the patient began to complain of left face, arm, and leg numbness. No additional subjective or objective clinical findings were noted, and the contralateral eye showed no visual field defects. A CT scan revealed a left supraclinoid enhancing lesion suggestive of an aneurysm. Angiography subsequently demonstrated left carotid-ophthalmic and incidental right intracavernous carotid artery aneurysms (fig).

Operative exposure revealed a left carotid-ophthalmic artery aneurysm that arose immediately distal to the origin of the ophthalmic artery, and was compressing and indenting the optic nerve only. The aneurysm was clipped successfully. Postoperative visual field testing revealed a small, superior, relative scotoma. Visual acuity has remained essentially unchanged.

Recovery of visual function after optic nerve decompression is a frequently observed phenomenon. However, recovery during chronic compression is generally incomplete, and is usually fluctuating or protracted.5 10-13 Spontaneous visual recovery without optic nerve decompression probably represents remyelination and restoration of central conduction. Partial remyelination of optic nerves has been observed after 5 weeks of chronic compression.14 Recovery of visual acuity probably parallels remyelination, and coincides with the restoration of central conduction as observed in the demyelinated lesions of cat spinal cords.15 By the pattern of acute monocular visual loss, remyelination, and rapid, spontaneous recovery of visual acuity, optic nerve compressive lesions masquerade as optic neuritis.

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References

Orbital apex syndrome caused by rheumatoid nodules

Sir: Common clinical features of orbital apex syndrome are paresis, painless ophthalmoplegia, sensory disturbance, distribution of the first division of the trigeminal nerve and visual disturbance with or without exophthalmos in one eye. These symptoms are caused by involvement of the optic nerve and vessels nerves which pass through the superior orbital fissure and the optic foramen.1-12 Various kinds of pathological conditions1-5 (trauma, tumours, syphilis, tuberculosis, non-specific local inflammatory processes, infections or mucoceles spreading from neighbouring structures) manifest as the orbital apex syndrome, or similar syndromes such as the superior orbital fissure syndrome and the Tolosa-Hunt syndrome. In this report, we describe a case of rheumatoid arthritis which showed the orbital apex syndrome during exacerbation of general symptoms of rheumatoid arthritis. This rare neurological complication of rheumatoid arthritis, which was confirmed pathologically, has not previously been reported.

A 58-year-old female suffered from repeated arthralgia and swellings of joints in the feet, hands and extremities for 10 years. She showed typical rheumatoid deformities of joints in the feet and hands on admission to our hospital in September 1975 at the age...