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CHIASMAL ARACHNOID1S

Nabiev A.A.
Assistant of the department of Neurosurgery of
Samarkand State medical university.

Abstract

To many oculists chiasmal arachnoiditis is a nebulous entity, considered in differential diagnoses of chiasmal lesions but seldom encountered in practice. Duke-Elder (1949) describes the pathology. Proliferative changes in the arachnoid bind down the chiasma and optic nerves, compressing them, cutting into them, or infiltrating them along their connective tissue septa.

Introduction

The aetiology is obscure, but preceding infection (haematogenous, encephalitic, or spread from the nasal accessory sinuses) is mentioned, and he points out that the arachnoid, like the peritoneum, tends to continue an adhesive sclerosis long after the inciting agent has ceased to be active. Symptoms are headache with loss of vision appearing suddenly or (more often) gradually, and oculomotor pareses have occasionally been reported. The visual fields commonly show pleomorphic shifting changes, irregular concentric contraction, or incongruous bitemporal hemianopic defects. The diagnosis is based mainly on the lack of other signs, endocrine, nervous, or radiological. It seems worth recording this rather dramatic case because it presents many features of the disease.

Discussion

A male company secretary aged 38 years was referred to out-patients by his doctor on September 24, 1955. Apart from myopia (Right - 3-25D sph., - 5D cyl., axis 950; Left - 2D sph., - 0 5D cyl., axis 300) and congenital red-green blindness, he had had no previous trouble with his eyes.

For 10 days he had noticed dull occipital pain, particularly on lateral ocular movements, and for 3 days pain behind the left eyeball. Then on waking one morning he had found the vision in the left eye so defective that at first he thought his left eye was closed. He could see (in his own words) "a purple curtain with shimmering crosses of silver on it". This cleared within a few minutes, leaving a slight blur which persisted when he attended hospital 3 days later, although the corrected visual acuity was 6/5 in each eye. The fundi were normal and the visual fields showed no scotomata, but the left pupil was 1 mm. larger than the right, and showed a sluggish and poorly maintained reaction to light.

On October 20, 1955, he again reported pain on movement, this time of the right eye, followed soon after by blurred right vision. Visual acuity with glasses was 6/12 in the right eye and 6/5 in the left. The pupils remained unequal, but the right reaction was now poorly maintained, and there was weakness of the right medial rectus and inferior oblique muscles. The IV, VI, and VII nerves and corneal sensation were normal. The media were clear, and still no definite scotoma could be found. A fortnight later the vision in the right eye had improved to 6/9, but there was still some diplopia on looking to the left; a

further 4 weeks later the corrected vision was 6/5 in each eye, the pupils were equal, and the ocular movements normal.

On January 7, 1956, he again reported failure of vision, which had started in the left temporal field 8 days previously. He described a "bar" spreading across the field, above which he could see blurred images and below which he could see clearly, until on the fourth day his left central vision "blacked out". The vision in the right eye had also deteriorated over the last 24 hours. The corrected visual acuity was 6/36 in the right eye and hand movements in the left. Both pupils showed a retrobulbar type of reaction. The left field showed a dense central scotoma, and the right a defect in its upper part with a deep central core.

The visual fields improved during the next few days, and by January 13 the left central scotoma had fragmented, while the right had lost its dense core. 3 days later the left defect was receding from the macular region, and the right had disappeared. On January 23 the corrected visual acuity was 6/6 in each eye, and no scotomata were found to 1/1000 white or 3/1000 red for either eye.

This remission lasted only 5 days. He returned complaining of blurred vision in the left eye and on January 28 the visual field showed for the first time a bitemporal hemianopic tendency, mainly in the upper part.

2 days later the left scotoma had begun to recede, but the right eye improved more slowly.

On February 16 there was another recurrence. Again he described the sensation of a curtain passing across his sight, diagonally from the lower temporal to the upper nasal part of the right field. The corrected acuity was 6/60 in the right eye and 6/6 in the left. Whereas the left field was normal, the right showed a general lack of perception of 1: 1000 white.

At this stage he was admitted to hospital for a 3 weeks' course of rest, anticoagulants (Dindevan), and ACTH. Regular prothrombin times ensured a satisfactory anticoagulant effect, and no toxic reactions occurred. The vision showed a steady improvement during this treatment until the last day.

Then on March 7, while he was still on ACIH, a relapse occurred. He described a shadow passing across the left field from the temporal side. Corrected visual acuity was 6/12 in the right eye and 6/36 in the left. The visual fields showed a dense left centrocaecal scotoma, and a right hemianopic defect. He was discharged from hospital on cortisone 25 mg. twice daily, iodides were started as well, and it was observed that bodily exertion or hot baths produced an appreciable but temporary falling off of vision.

On March 17 there was a rapid bilateral deterioration of of vision, which may have been connected with the fact that he had run out of cortisone 48 hours previously.

When he began to take cortisone again improvement set in, and the field defects dwindled, but for the first time optic atrophy was noted. The visual fields on April 5 are shown in Figs 7 and 8 (opposite).

On April 9 it was suggested that there might be a definite connexion between his disease and dental infection. A right lower premolar had been aching, and before one of his visual failures in March there had been an exacerbation of pain. Furthermore, after the extraction of this tooth on March 26, there had been a steady improvement in vision.

A bacteriological examination made from a left upper incisor extracted on April 28 grew Strep. viridans and N. catarrhalis, sensitive to penicillin, streptomycin, aureomycin, and chioramphenicol. These may well have been contaminants from the mouth, but nevertheless a course of vaccine was tried. Parke-Davis anti-catarrh vaccine was given in Julne, starting with 0-1 ml. and increasing by seven doses to 1 ml. In July the visual acuity improved, and by September, although bitemporal defects were present, both maculae were spared.

The patient has been on prednisone 5 mg. daily up to time of writing, and on the last few occasions when the visual fields were recorded they showed no change.

The corrected visual acuity is now 6/18 in the right eye and 6/9 in the left and both discs show optic atrophy. The patient feels confident enough of his sight, however, to drive a car in the country.

X-Ray of Skull.-Some asymmetry of the lesser wings of the sphenoid and secondary asymmetry of the superior orbital fissures, within normal limits.

Dr. Simon Behrman saw the patient on February 7, 1956, and considered this to be a case of arachnoiditis rather than of a demyelinating disease:

"The episode on September 24, 1955, as also the initial variability of perimetric defects, their rapid fluctuation, and the absence of neurological, radiological, and serological-abnormalities served as a basis for the clinical diagnosis.

The extremely rapid, not to say apoplectiform, fluctuations have been in my experience a significant feature in several cases of chiasmal arachnoiditis confirmed at operation. These phenomena indicate circulatory disturbances which are presumably caused by embarrassment of the blood vessels in their passage through the subarachnoid space in the regions affected by the inflammatory process.

The blood vessels supplying the spinal cord are in similar anatomical relationship with the leptomeninges to those supplying the chiasma and optic nerves. Spinal cord lesions are also apt to arise in an apoplectic fashion as a consequence of chronic spinal leptomeningitis (arachnoiditis).

When present these apoplectic events help to distinguish chiasmal arachnoiditis from chiasmal or optic nerve lesions of demyelinating pathology.

The symptoms are described and the central fields recorded of a case of chiasmal arachnoiditis, showing seven episodes of visual deterioration during the course of some 8 months. The field fluctuations were at first " extremely rapid, not to say apoplectiform" and recovery was complete. Later the field defects developed a bitemporal hemianopic tendency, and with the onset of optic atrophy a permanent defect of this type remained.

This patient was under the care of Mr. H. McNeil Symons, and I wish to thank him for his permission and encouragement to write the case summary.

We are indebted to Dr. Simon Behrman for his help with the diagnosis and treatment, and for allowing his comments to be quoted. Last, but not least, I am grateful to the patient, for cheerfully submitting to the tedious process of recording his fields on so many occasions.

Conclusion

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