Visual loss from optochiasmatic arachnoiditis after tuberculous meningitis

Case report

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While recovering from tuberculous meningitis, a 5-year-old boy developed a profound visual deficit because of optochiasmatic arachnoiditis. Following micro-neurosurgical decompression of the optic nerves and chiasm, prompt visual recovery occurred.

Key Words  meningeal tuberculosis  arachnoiditis  optic nerve  optic chiasm

Although ocular abnormalities occur frequently in tuberculous meningitis, visual loss due to optochiasmatic arachnoiditis is unusual in the convalescent phase. There are scattered case reports in the literature, and the syndrome is reviewed in the text of Walsh and Hoyt.

We recently treated a young patient in whom severe visual loss occurred during the convalescent phase of tuberculous meningitis. A pneumoencephalogram revealed a mass in the region of the optic nerves and chiasm. At neurosurgical exploration, dense scar tissue was found enveloping these structures; after microneurosurgical excision of the scar tissue, prompt visual recovery occurred.

Case Report

This 5-year-old boy was readmitted on June 12, 1975, to the New England Medical Center because of progressive visual loss.

First Admission. He had been admitted 2 months previously because of a grand mal seizure following a 2-week history of chills, fever, and progressive lethargy. His father was under treatment for active pulmonary tuberculosis.

Examination at that time revealed a febrile, obtunded child with neck stiffness and occasional decerebrate and decorticate posturing. Lumbar puncture revealed an opening pressure of 140 mm H₂O, with a protein of 144 mg%, glucose of 38 mg%, and a white blood cell count of 100/cu mm (35 polymorphonuclear cells and 65 lymphocytes). A smear was positive for acid-fast bacilli. The child was begun on daily doses of streptomycin, 30 mg/kg, INH (isonicotinic acid hydrazide), 15 mg/kg, and ethambutol, 25 mg/kg. An ophthalmologic consultant noted a ptosis and mild limitation of adduction of
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the left eye. The pupils were 5 mm and equal, with very sluggish direct and consensual reactions to light. On indirect ophthalmoscopy, whitish-yellow choroidal tubercles, measuring approximately 300 μ in diameter, were noted in the posttemporal fundus. A few days later, as the child’s neurological status began to improve, an accurate acuity of 15/15 in both eyes was obtained. The ptosis and adduction of the left eye improved, and the choroidal tubercles appeared faded in color. X-ray films of the skull and chest were normal. One month after his first admission, all neurological signs and symptoms had cleared, and the patient was discharged on ethambutol 400 mg by mouth daily, rifampin 300 mg by mouth daily, and twice weekly injections of streptomycin 340 mg.

Although the patient continued to feel well, he began to experience increasing difficulty with his vision. He was unable to catch a ball thrown to him, and ignored the television set while seated in front of it. After missing several clinic visits, he returned for examination on June 5, 1975. Acuity was now reduced to 2/400 in the right eye, and light perception only in the left. Visual field examination revealed marked bitemporal cuts and large central scotomata. The discs were now pale, but the choroidal tubercles were diminished in size. Because of suspected ethambutol toxicity, the medication was discontinued and INH was instituted, 100 mg four times daily by mouth.

Second Admission. When visual deterioration continued, the child was readmitted. He was unable to see color plates or an Amsler grid. The Marcus-Gunn pupil in the left eye was again noted. Acuity was 3/30 on the right with Allen cards and light perception only on the left. The extraocular muscle movements again were full, and there was no ptosis. The choroidal tubercles and pallor of the optic discs were unchanged. A pneumoencephalogram revealed obliteration of the chiasmatic cistern, and splaying and elevation of the optic and infundibular recesses of the anterior third ventricle (Fig. 1). Moderate hydrocephalus was noted, and the findings suggested a mass, possibly tuberculoma, in the region of the optic chiasm.

Operation. On June 23, 1975, the patient underwent a right frontal craniotomy. The right optic nerve was identified and in its most anterior portion appeared normal. Posteri-
FIG. 2. Photomicrograph of resected tissue, showing numerous thin-walled blood vessels interspersed with chronic inflammatory cells and fibroblasts. The appearance is typical of granulation tissue. H & E, × 400.

tained 2 months postoperatively revealed no evidence of hydrocephalus or mass.

Discussion

Chronic optochiasmatic arachnoiditis is the most likely cause of a chiasmal compression syndrome following tuberculous meningitis. In 15 patients with treated tuberculous meningitis with findings suggestive of chiasmal lesions, Feld and Sicard reported that 13 had chronic arachnoiditis and only two had tuberculomas. Symptoms of chiasmal compression from chronic arachnoiditis may occur late in the convalescent phase of adequately treated tuberculous meningitis. Our patient's deficit was first noted 4 weeks after initial treatment, at which time he was otherwise asymptomatic; Coyle reported a case in which the visual disturbances began almost a year following active infection. He points out that antimicrobial and steroid therapy has been unsuccessful in preventing further progression of the visual deficit.

An aggressive surgical approach to these lesions, with a thorough dissection of the scar tissue from the chiasm and optic nerves, appears to be the only effective treatment. The major cause of the visual impairment seems to be ischemia secondary to continued gradual compression by the tough scar tissue. Block has suggested that a local pressure effect on the optic nerves and chiasm from hydrocephalus may also be a factor, and he reported a case in which vision was "significantly improved" by a ventriculoatrial shunt. Although our patient had moderate hydrocephalus before surgery, the findings on both pneumoencephalography and surgical exploration were indicative of a mass in the region of the chiasm, and it is doubtful that shunting alone would have altered this phenomenon. The fact that postoperative CT scanning revealed only minimal ventricular dilatation does suggest that removal of the adhesions relieved an obstruction to flow of cerebrospinal fluid in the chiasmatic cistern, and thereby reduced pressure on the visual apparatus. Our patient’s lumbar puncture pressures, however, were always within normal range.

Acknowledgment

We wish to thank the Department of Neuroradiology at the New England Medical Center for providing the pneumoencephalogram reproduction shown in Fig. 1.

References


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