CASE REPORTS AND TECHNICAL NOTES

TUBERCULOUS OPTICOCHIASMATIC ARACHNOIDITIS

REPORT OF A CASE*

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(Received for publication November 27, 1950)

The surgical freeing of arachnoidal adhesions about the optic chiasm was first described by Balado1 in 1929 and this condition is often referred to in the South American literature as Balado's disease. In 1931 Heuer and Vail6 reported a series of cases in which trauma or infection was the probable cause. These authors emphasized early operative intervention. Since that time the syndrome of opticochiasmatic arachnoiditis has become well known.2,4,8 In many succeeding reports the etiology has remained obscure.2,3,8 In 1937 Hausman8 mentioned 5 cases all thought to be due to syphilis of the central nervous system. Bruetsch2 in his review of the subject listed trauma, infections of the facial cavities and nonpurulent cerebromeningeal infections as among the leading causes.

Our case represents the first case of opticochiasmatic arachnoiditis that was proven due to tuberculosis and surgically treated. One can merely conjecture that this may be due to the high mortality rate in tuberculous meningitis prior to the advent of streptomycin therapy. Ivey, Phillips and Meirowsky7 recently reported the successful extirpation of a cerebellar fibrocaseous tuberculoma. These authors stated that intramuscular and intrathecal streptomycin may encourage surgical extirpation of tuberculous lesions of the brain. It seems also likely that as antibiotic therapy advances, chronic arachnoiditis may be more commonly met with in the future as the immediate mortality rate falls and the later sequelae appear.

CASE REPORT

#172126. J.G., a 25-year-old Mexican male laborer was admitted on Oct. 12, 1948. Seven months previously a "boil" on the left side of his neck had been lanced. This continued to drain serosanguinous fluid for several months and finally healed spontaneously. Two weeks prior to admission he began to have generalized headaches, which became progressively more intense and were accompanied by stiffness of the neck and low-grade fever. For the last several days he had been severely nauseated and unable to retain food or fluids. There was no history of convulsions nor of exposure to tuberculosis.

Examination. The patient appeared acutely ill. Temperature 103°, pulse 110, respi- rations 22. B.P. 180/90. Abnormal physical findings were limited to strongly positive Kernig's and Brudzinski's signs, horizontal and vertical nystagmus, and pathological plantar responses on the left. RBC 4,100,000; WBC 8,500, with a normal differential, and a sedimentation rate of 23 mm./hr. Urinalysis and serology negative.

* Reviewed by the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions of the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.
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**Hospital Course.** Lumbar puncture on admission revealed turbid and xanthochromic CSF with a pressure of 190 mm. of water and normal dynamics. The CSF contained RBC 28; WBC 913, with 75 per cent lymphocytes; sugar 25 mg. per cent; total protein 368 mg. per cent; globulin 4+; chlorides 640 mg. per cent; Wassermann negative, and colloidal gold curve 2355444443. A thick pellicle formed rapidly which failed to reveal acid-fast bacilli both on smear and culture. The chest x-ray was negative for evidence of pulmonary tuberculosis.

The patient was treated with streptomycin, promin, parenteral fluids and bed rest. During the first 10 hospital days he steadily improved and the temperature fell to normal. On the 16th day, however, he began to complain again of severe headaches, became nauseated and vomited continuously. He went progressively downhill, became mentally clouded, actively hallucinated, incontinent, and had rapid, successive, generalized convulsions. Repeated examinations revealed no papilledema or other neurological changes.

![Visual fields 2 months before operation, showing symmetrical bitemporal hemianopsia.](image1)

![Visual fields 1 month before operation, showing progressive constriction, and loss of central vision.](image2)

Despite intensive therapy in the succeeding weeks there were repeated bouts of stupor, high temperature, convulsions, and nuchal rigidity. Left facial nerve palsy of infranuclear type developed. The CSF protein continued to be elevated; on one occasion it was 6,400 mg. per cent. Repeated cultures and smears remained negative for acid-fast bacilli.

Over a period of several months the patient gradually improved, became lucid and coherent, and his temperature again returned to normal. However, he continued to have occasional, generalized convulsions without localizing signs. The facial nerve palsy disappeared spontaneously. He was given dilantin and phenobarbital which controlled his convulsive attacks. In June 1949, 9 months after admission, he first complained of diminished vision without associated headaches, nausea or vomiting, or temperature elevation. At this time there was evidence of early primary optic atrophy. No papilledema or retinal hemorrhages were noted. Visual field studies revealed a bitemporal hemianopsia (Fig. 1). Serial visual field studies showed a rapid progressive loss of both peripheral and central vision (Fig. 2). In addition, the optic pallor increased. Roentgenograms of the skull revealed a normal sella turcica.

**Operation.** On Sept. 13, 1949 a subfrontal chiasmal exploration was performed. The optic nerve and chiasm were found to be surrounded by dense, matted adhesions of leptomeninges. Both optic nerves were markedly flattened and the chiasm was densely bound down at the base of the brain. The adhesions were carefully divided across the chiasm and both the chiasm and optic nerves were liberated.

**Postoperative course** was smooth and uneventful. His temperature remained normal and he had no further headaches, vomiting or convulsions. Repeated examinations revealed progressive and dramatic improvement in the peripheral visual fields and visual acuity (Fig. 3). Objectively, there was no change in the optic atrophy. He was discharged to the Chest Medical Service on Oct. 24, 1949.

Shortly thereafter a review of his previous chest x-rays revealed a destructive process involving the body of the 11th thoracic vertebra and a paraspinal abscess, which apparently had been present since December 1948, and had been overlooked. Coincident with this,