CASE REPORTS AND TECHNICAL NOTES

TUBERCULOUS OPTICOCHIASMATIC ARACHNOIDITIS

REPORT OF A CASE*

ERIC T. YUHL, M.D.,† AND CARL W. RAND, M.D.;‡

Neurosurgical Service, Wadsworth General Hospital, Veterans Administration Center,
Los Angeles, California

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The surgical freeing of arachnoidal adhesions about the optic chiasm was first described by Balado in 1929 and this condition is often referred to in the South American literature as Balado's disease. In 1931 Heuer and Vail 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. In many succeeding reports the etiology has remained obscure. In 1937 Hausman mentioned cases all thought to be due to syphilis of the central nervous system. Brustsch 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 Meirowsky recently reported the successful extirpation of a cerebellar fibrocaceous 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, respirations 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.

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† Resident in Neurosurgery.

‡ Senior Neurosurgical Consultant.
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.

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,
there developed severe pain in both his legs and hips and a left foot drop. On Nov. 18, 1949 a paraspinal abscess was drained. He did not improve. Serial x-ray examination revealed extension of the abscess, and on Dec. 20, 1949 another incision and drainage was carried out. The wound failed to heal and continued to drain purulent material. In January 1950 x-ray of the chest showed a soft area of pnemonic infiltration in the left base. Sputum cultures and smears showed acid-fast bacilli. Despite intensive therapy with PAS, streptomycin, and thiosemicarbazone the patient ran a slow, progressive, downhill course. Re-examination 1 month before death revealed no gross contraction of the visual fields or alteration of the visual acuity.

Nuchal rigidity and tetanic convulsions developed, and following a brief period of coma the patient expired on March 11, 1950.

Autopsy. Extensive, disseminated, arachnoiditis was present over the entire brain stem and base of the brain. Caseo-cavernous tuberculosis was present in both lungs. Miliary nodules were found in the spleen and kidneys.

COMMENT

Although this patient died of widely disseminated tuberculosis, two important points need emphasis. The gratifying and dramatic improvement in his visual fields remained until death. At autopsy the optic nerves and chiasm remained remarkably free of arachnoidal adhesions, although such were found over the rest of the base of the brain. In retrospect, it appears that the focus of infection in the 11th thoracic vertebra was of far more importance as the cause of death than the operative procedure. One might almost conjecture that had the tuberculosis of the spine been controlled, the patient may well have survived.

SUMMARY

1. A brief review of opticochiasmatic arachnoiditis has been reported.
2. A case of tuberculous meningitis with secondary involvement of the optic chiasm has been reported. Surgical liberation of the adhesions was successfully performed.

REFERENCES