Trigeminal Neuralgia as the Presenting Symptom of a Tuberculoma of the Cerebellopontine Angle

Case Report

G. BORNE, M.D.
Neurosurgical Service, University Hospital Center, Oran, Algeria

Although manifestations of sensory deficit of the fifth cranial nerve are part of the classical cerebellopontine angle syndrome, classical trigeminal neuralgia of the first division as due to local compression of the fifth cranial nerve is rather rare. Acoustic neurinoma is the most common cause, followed by meningioma of the posterior surface of the petrous bone, epidermoid cyst, neurinoma of the trigeminal nerve, arachnoiditis of the lateral recess, choroidal papilloma of the fourth ventricle, basal skull carcinoma, and basilar aneurysm. Tuberculoma of the cerebellopontine angle as a cause of trigeminal neuralgia is very rare. Although Elkins and Rack,3 Descuns, et al.,2 and Obrador1 have seen tuberculomas of the cerebellopontine angle with trigeminal deficit, they do not mention trigeminal neuralgia.

Case Report

A 25-year-old man was admitted on March 23, 1965, with a complaint of severe "electric shock-like" pain over the right eye of 3 years' duration. There had been intermittent episodes of headache and vomiting for 3 months. He had blurred vision for 4 months, and actual blindness for the last 2 months. He had never consulted a physician during this time.

Examination. There was hypesthesia in the right supraorbital and maxillary region, diminished right corneal reflex, and "trigger zone" at the right supraorbital notch. Between paroxysms of pain the patient described a burning sensation associated with numbness of the same region. Other positive findings were right hypoacusia, right peripheral facial paresis, lateral nystagmus with the slow component to the right, and deviation of the extended arms to the right. There was bilateral papilledema with hemorrhages. The clinical impression was tumor of the posterior fossa, located at the petrous apex near the right fifth cranial nerve. Skull x-rays including Stenvers' views were made and showed no lesion on the petrous apex or in the internal auditory meatus. Ventriculography with Lipiodol was performed through a right frontal burr hole and showed deviation of the fourth ventricle to the left and difficult passage of the Lipiodol (Fig. 1). X-rays of the lungs showed no lesion.

Hemogram, blood sugar, proteins, and urea were within normal limits. The sedimentation rate was 7, 9, 92; urine analysis was normal. Ventricular fluid examination showed cells 4/mm³ (lymphocytes), proteins 35 mg/100 ml, glucose 55 mg/100 ml, and chloride 720 mg/100 ml.

Operation. On April 10, 1965, a right unilateral suboccipital craniotomy was performed. Excision of the lateral third of the cerebellum revealed a yellowish mass lying between the petrous apex and the internal auditory meatus. It was attached to the dura, pushing and compressing the right fifth, seventh, and eighth cranial nerves. There was no sign of arachnoiditis. This mass seemed to protrude outward from the right cerebellar lobe as well as anteromedially compressing the brain stem. The tumor measured 30×25 mm, and had a yellow mottled surface. It was easily dissected and removed from the cranial nerves, the cerebellum, and the dura of the petrous apex without any hemorrhage. The dural attachments were coagulated. The cut surface was soft, whitish, with caseous material, without any blood. It was recognized grossly as a tuberculoma, and streptomycin was put into the operative cavity. The patient was given streptomycin, P.A.S., and Rimifon for 2 months, as well as intrathecal streptomycin daily for the first 15 days.

Histopathological Examination. Conglom- erated tubercles with several typical giant cells were shown along the margin of the caseation (Fig. 2).

Postoperative Course. Immediately after the operation, the symptoms of trigeminal neural-
Tuberculoma of the Cerebellopontine Angle

FIG. 1. Lipiodol ventriculograms showing deviation and constriction of the fourth ventricle.

gia disappeared. The patient was discharged from the hospital on May 5, 1965. Two months later the only findings were blindness and slight hypoacusia. All other symptoms had disappeared. The optic discs showed advanced secondary optic atrophy.

Discussion

We believe that this tuberculoma started in the right cerebellar lobe and gradually increased in size. As it telescoped forward and laterally toward the cerebellopontine angle, it caused local compression on the fifth cranial nerve, followed by the neighboring seventh and eighth cranial nerves. This compression explains the clinical findings of hypoacusia, facial paresis, numbness, loss of corneal reflex, and trigeminal neuralgia. These neurological pains were not exactly like those in “tic douloureux,” in which motor and sensory functions are usually normal.

In the second stage of its growth, lateral displacement toward the internal auditory meatus brought about the compression of the seventh and eighth cranial nerves, and their deficits.

In the third stage, when the growth was stopped at the lateral wall, it turned back inward, medially and downward, progressively pushing and constricting the fourth ventricle. This resulted in increased intracranial pressure, which may explain the long

FIG. 2. Photomicrograph of tubercles with giant cell.