CASE REPORTS AND TECHNICAL NOTE

ABSCESSES OF THE CEREBELLOPONTINE ANGLE*

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Among inflammatory lesions that may cause a syndrome of the cerebellopontine angle, most authors\(^1\) refer only to arachnoiditis. However, tuberculoma and gummatita also may be considered. Tuberculoma developing in the cerebellopontine angle and simulating acoustic neuroma is rare; the only reference found in the literature was a report by Elkins and Rack.\(^2\) In the cases reported by Rosenthal\(^3\) and also in a personal case still unpublished, the lesions were primarily tuberculoma of the pons that extended towards the region of the angle.

Finally, chronic abscesses may also be located in the pontomedullo cerebellar space. As the following cases will show, their clinical picture may not always be easy to distinguish from that produced by tumors in the same location.

CASE REPORTS

Case 1. A married woman, aged 29 years, was admitted on March 4, 1955 (referred by Dr. A. Subirana, Barcelona).

In May 1954, after normal delivery of her third child, she had onset of dysarthria and left facial paralysis. A little later, asthenia, somnolence, and marked dizziness occurred, accompanied by frequent vomiting. In October, she complained of sharp pains in the left ear. The left 9th, 10th and 11th cranial nerves were involved. Roentgenograms revealed no alteration in the internal auditory meatus, nor in the foramen lacerum posterius. However, an oto logic, suspecting a tumor of the glomus jugulare, prescribed radiotherapy in December, 1954.

In January, 1955, left-sided headaches, nausea, vomiting, diplopia and staggering gait kept the patient in bed.

Early in March, otoscopic examination revealed redening of the tympanic membrane and thickening of its posteriusuperior portion; a paracentesis was performed but there was no exudate.

Biopsy of the thickened tympanic membrane disclosed no abnormality.

Examination. On admission to the Neurosurgical Department, positive neurologic findings were bilateral papilledema of slight degree, bilateral cerebellar syndrome, predominating on the left, left facial paralysis, deafness of left ear with Weber lateralized to the right side, and hypoexcitability of the left vestibularis on the caloric test. There was paralysis of the palatal and pharyngeal muscles on the left side. She could not elevate the left shoulder.

Roentgenograms showed no erosion of the porus acusticus and a normal foramen lacerum posterius.

Operation. On March 7, 1955, with the patient in the sitting position, a left suboccipital craniectomy was performed with ablation of the lateral half of the cerebellar hemisphere. This exposed a mass the size of a small walnut; tapping through its smooth surface showed that the lesion was a multilocular abscess located in the inferior half of the cerebellopontine angle. The cavity of the abscess was injected with antibiotics; its medial wall was dissected from the medulla, and the lesion was removed radically, leaving only a small portion of its wall firmly attached to the 9th, 10th and 11th nerves in the neighborhood of the foramen lacerum posterius. The field was constantly irrigated with solutions of penicillin and streptomycin. The 5th, 6th and 7th nerves were not directly adherent to the wall of the abscess. The dural incisions were closed tightly and the operative wound was closed in the usual manner.

Bacteriological Report. There were no organisms in the pus.

Course. For 20 days an intensive treatment with parenteral penicillin and streptomycin was given. On the first 5 days, penicillin was also injected into the ventricles. Primary healing of the wound was obtained.

The patient now leads a very active life. The mobility of her face is normal and the degree of auditory impairment has diminished notably.

Case 2. M.S., a 51-year-old male, was admitted on Sept. 10, 1956 (referred by Dr. A. Subirana, Barcelona and Dr. Gorgues, Huesca).

He had always been in good health until 1 year previously, when he began to have left-sided headaches and tinnitus. In the course of the following month he noticed progressive impairment of hearing in the left ear until he became completely deaf. Two weeks before admission, left facial paralysis and staggering gait with deviation to the left appeared. Ultimately vomiting and disturbances in swallowing liquids occurred.

Examination. Positive neurologic findings were slight, bilateral peripapillary edema and anisocoria, the right pupil being larger than the left; hypoesthesia in the distribution of the 5th nerve on the left side with complete abolition of the corneal reflex; paralysis of the 6th nerve; nerve deafness with vestibular hyporeflexia on the caloric test; hypoesthesia in the area of the 9th
nerve and paralysis of the left side of the soft palate, with abolition of the left palatal reflex. Movement of rotation of the head to the right was weak. There was a left cerebellar syndrome with dysmetria, dysdiadochokinesia, hypotonia, spontaneous nystagmus to the left and past-pointing to the left of the extended arms. There was a tendency to fall to the left in the Romberg test.

Roentgenograms showed no expansion of the porus acusticus.

Operation. On Oct. 18, 1956, a left cerebellar exposure was made with abrasion of the external half of the cerebellar hemisphere. A firm encapsulated mass was exposed and tamped, and 15 cc. of a thick yellowish pus were aspirated. Penicillin was injected into the cavity of the abscess. The wall of the abscess was dissected carefully and excised completely with the exception of a narrow diverticulum branching from the medial wall of the abscess and firmly attached to the pons. Examination of the angle now showed a second abscess, 15 mm. in diameter, which came into contact with the 9th, 10th and 11th nerves. It was made up of a collection of pus cells and its consistency was rather hard. This lesion was implanted in the dura mater, behind the porus acusticus, but was not adherent to the 7th and 8th nerves. It was removed by cutting its small pedicle. There was no direct connection between the two pyogenic lesions. The first was impressed in the anterior surface of the cerebellum and the second was located laterally. The operative field was irrigated with penicillin and streptomycin solutions. The dural incisions were occluded tightly, and the operative wound was closed in the usual manner.

Bacteriological Report. Smears and cultures of the pus revealed no germs.

Course. There were no immediate complications. Parenteral penicillin and streptomycin were given for 40 days. There was primary healing of the wound. The patient was discharged on Nov. 19, 1956.

On reexamination on Oct. 26, 1957, he was free from symptoms of increased intracranial pressure but still showed loss of hearing on the left, slight left facial hypoexcitability on the Barany test, and left facial weakness. Swallowing was normal and sensibility of the face was also normal.

This case shows that a clinical picture very similar to that of the acoustic neuromas may be caused by a double pyogenic lesion of the cerebellopontine angle. The smaller abscess was responsible for the disturbances of the 9th, 10th and 11th nerves while the larger one accounted for the cerebellar syndrome. The 7th and 8th nerves were probably compressed by the diverticulum at its point of emergence from the brain stem whereas, in contrast to what happens in cases of neuroma, the nerves were completely free at their entrance into the porus acusticus.

COMMENT

Two cases of chronic intracranial abscesses are presented involving the region of the cerebellopontine angle. In Case 1 the duration of symptoms was 6 months, in Case 2 it was 1 year.

In both cases, the course was completely afebril, lacking any symptoms of a previous infectious disease.

Both lesions clinically were considered as tumors, the first as a tumor of the glomus jugulare and the second as an acoustic neurinoma or a pontine glioma extending towards the region of the cerebellopontine angle.

In the neurosurgical literature reference to the type of abscess presented herein was rarely found. In the classical work of Cushing on tumors of the nervus acusticus, the possibility of a chronic abscess was mentioned only in discussing the diagnosis, but no case of abscess of the angle was presented.

The only cases of abscesses of the cerebellopontine angle to which the writer can refer are those of Matson and Werner.

The existence of these lesions must be kept in mind since their chronic evolution and the lack in the anamnesis of an apparent pyogenic process as etiological factor, make their distinction from neoplasms very difficult. It is improbable that examination of the cerebrospinal fluid may afford the clue to the diagnosis.

Regarding the pathogenesis of the abscess, nothing definite may be said, as otologic and roentgenographic examinations did not reveal any gross primary pyogenic focus in the ear. However, there were slight indications pointing to an otogenous origin. In Case 1 there was reddening and thickening of the tympanic membrane, although no pus or any granulomatous tissue could be found in the tympanum. In Case 2, the implantation of one of the abscesses in the dura mater covering the petrous pyramid, seems to suggest the path followed by the infection. Hence we may presume that the primary infectious focus in the ear was so slight that no objective otologic signs could be detected.

From the standpoint of treatment, it is interesting to note that both patients recovered satisfactorily from the operation and that primary healing of the wounds was obtained. It was not necessary to sever any of the nerves that cross through the region of the angle.

REFERENCES