CEREBELLAR EXTRADURAL HEMATOMA

REPORT OF A CASE

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The literature contains 3 reports of extradural hematoma in the posterior fossa treated by operation.1,2,5 All 3 patients recovered. Fifteen additional cases, perhaps more, have been reported from autopsy material.3,4,6 Adequate clinical observation, however, is so scarce that an easily recognizable syndrome for this lesion can not yet be described. In some, it appears that the progress of neurological signs extends over a period of a week or more, allowing for relatively simple localization of the lesion. In others, the progress is rapid and no localizing features are observed until compression of the cerebellum and brain stem is far advanced. Unless relieved by prompt operation, the patient may then expire following a short interval of coma and hypotonia of all extremities.

The following case of cerebellar extradural hematoma is an example of the type that pursues a less emergent course.

CASE REPORT

A 27-year-old oil field laborer was admitted to the Veterans Hospital of Wichita on Aug. 31, 1947, for treatment of a head injury of 8 days' duration. His past history was not relevant.

On Aug. 22, 1947, he fell from a doorstep, striking the back of his head on the sidewalk. He was unconscious for 15 minutes, then recovered consciousness and was taken home. For the following 8 days he was confined to bed by headache, vertigo, nausea and vomiting. It could not be ascertained whether his state of consciousness fluctuated, improved or became progressively worse, since the patient was an unreliable witness in regard to this symptom. However, a mild degree of drowsiness was present on admission, and increased moderately while under observation.

General physical examination was not unusual. Pulse was 88, and blood pressure 135/90. There was nystagmus and papilledema. Headache was constant and he ate poorly because of nausea. On September 2, a lumbar puncture was performed. Xanthochromic spinal fluid was obtained under an initial pressure of 500 mm. of water. This was reduced to 220 mm. by removal of 7 cc. of fluid. Queckenstedt test: no evidence of subarachnoid block. Neurosurgical consultation was then requested.

Examination on September 2, 11 days after injury, elicited the following: The patient was drowsy but easily aroused. He complained of frontal headache. Speech was slurred and had a

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nasal quality. The neck was slightly stiff. There was contusion of the scalp 1 inch to the left of the inion. The patient preferred to lie on his side. Change of position consistently produced vertigo, nausea and a slow horizontal nystagmus of short duration. On attempting to fix his gaze straight ahead, the eyes would deviate slowly to the right. Pupils were equal and reacted well to light and near vision. External ocular movements were not restricted. There was mild papilledema bilaterally. No visual field defect could be demonstrated. There was complete paralysis of the left soft palate. Except for mild central paresis of the left side of the face, the remainder of the cranial nerve functions were normal.

The outstanding localizing abnormalities were cerebellar signs involving the left extremities. Adiadokokinesis, dysmetria, decomposition of movement, hypotonia and absence of check were found in the left upper extremity, and ataxia in the left heel-to-shin test. Tendon reflexes were more active on the left. Plantar reflexes were normal. There was no disturbance of pain perception, position sense, stereognosis or vibration perception. Roentgenograms of the skull failed to reveal a fracture owing to the omission of an occipital view. The clinical impression was hematoma in the left posterior fossa, with probable contusion of the right temporal lobe.

Operation, Sept. 2, 1947. A left suboccipital exposure was made, disclosing a fissure fracture extending diagonally across the left half of the occipital bone. A craniotomy over the center of the cerebellar hemisphere revealed an extradural clot of currant-jelly consistency confined to the left side of the posterior fossa. The clot was 2.5 cm. in thickness in a circumscribed area directly beneath the fracture line, but diminished rapidly in thickness toward the periphery, so that the total volume of the clot was estimated to be not more than 15 gm. Upon removal of the clot a laceration of the dura, 3 cm. in length, was exposed coinciding with the line of fracture, and at this point lacerated cerebellar cortex was visible. The cerebellar hemisphere was explored without encountering an intracerebellar hematoma. The dura re-expanded very slightly during the procedure. Bleeding points along the dural laceration were controlled without difficulty and the wound was closed without drainage.

The patient improved steadily following operation and was discharged on the 16th postoperative day. Examination on October 29, 38 days following removal of the hematoma, revealed no neurological abnormalities. The patient was free from symptoms and ready to resume work.

COMMENT

In this case, diagnosis of hematoma in the left posterior fossa was not difficult. The history of a moderately severe head injury followed by the appearance of papilledema, nystagmus, incoordination on the left, and paralysis of the left 10th cranial nerve directed attention inevitably to the left posterior fossa. No attempt was made preoperatively to predict the location of the hematoma, as to whether it was extradural, subdural or intracerebellar. The extensive functional loss appeared to favor the intracerebellar location. The patient had, however, been subjected to a Queckenstedt test and had withstood reduction of intracranial pressure by withdrawal of spinal fluid, with no ill effects. The fact that he did not succumb to these studies was thought to favor the extradural location.

The writer believes, with many others, that the Queckenstedt test should be carefully avoided in the diagnosis of intracranial lesions. It is of interest, however, to note that Kessel's case2 of cerebellar extradural hematoma did show a complete block by the Queckenstedt test.

The majority of the reported instances of cerebellar extradural hematoma have been encountered at autopsy. LeCount and Apfelbach3 reported 8 cases among 504 autopsies involving skull fracture. Vance4 encountered 4 cases in a series of 512 autopsies, also involving skull fracture. Both these authors describe the lesion as arising from lacerations of the transverse sinuses by fracture. In some of these, the