

INTRACRANIAL MENINGIOMA FOLLOWING TRAUMA

Report of a Case

J. CORNALL HOWARTH, M.B., Ch.B., and ALEXANDER T. BUNTS, M.D

Department of Neurosurgery

WITH any subject as controversial as that of traumatic and post-traumatic tumors, critical analysis of case histories and physical findings is essential. Strict criteria must be established, against which the data on each patient may be tested, and these are detailed by Ewing¹ as the following:

- (1) the adequacy and authenticity of the trauma must be established;
- (2) the previous integrity of the injured part must be confirmed;
- (3) the tumor must originate from the exact site of the injury;
- (4) there must be a reasonable lapse of time between the injury and the onset of the tumor symptoms, and
- (5) a positive diagnosis of the nature of the tumor must be made.

If such a test were applied to all recorded cases of tumor following trauma, much confusion could be avoided.

Case Report

A 59-year-old man was referred to the Cleveland Clinic on August 19, 1949, with the complaint of repeated convulsive seizures. The relevant history concerned an automobile accident 21 years previously, in which he had sustained cerebral concussion and a depressed fracture in the left frontoparietal region. The calvarial depression had never been elevated.

Following recovery from the immediate effects of the accident, he had been completely free from symptoms until approximately 1 year prior to the present examination. At that time while in bed and apparently asleep, his wife had noticed that he was making clutching movements with both hands. She spoke to him, got no response, and was unable to arouse him. The attack developed into a generalized tonic seizure followed by flaccidity and unconsciousness for approximately 15 minutes. When he awoke, he was apparently perfectly normal, with no headache. There had been no generalized clonic phase, no lateralizing sign, no incontinence of bladder or bowel, and no aura.

Similar attacks had occurred about ten times during the year following the original episode. The later spells had occurred during waking hours, accompanied by a short aura of lightheadedness and an indescribable odor, and commencing with a tonic turning of the face to the right side and jerking movements of the right arm. The duration of each episode was about 15 minutes, and there was no subsequent paralysis or headache.

The family history was clinically unimportant and the patient's previous medical history showed no relevant factors except the remote possibility of beryllium toxicity, as he had been exposed to this substance while working in a foundry for several years.

General examination disclosed a well-built man with a normal mental status. He had a circular depression in the left frontoparietal region about $\frac{3}{8}$ inch deep and 2 inches in diameter.

Neurologic examination on August 19, 1949, revealed no abnormal signs and no evidence of disease of the central nervous system. There was no papilledema. Roentgen films of the skull showed evidence of an old depressed fracture in the left frontoparietal area (fig. 1a).

An air encephalogram on September 1, 1949 (fig. 1b), showed dilatation of both lateral ventricles and the third ventricle with a slight displacement of the whole ventricular system to the right. The cerebrospinal fluid was not under increased pressure, and contained 41 mg. of protein and 1 cell per cu. mm.

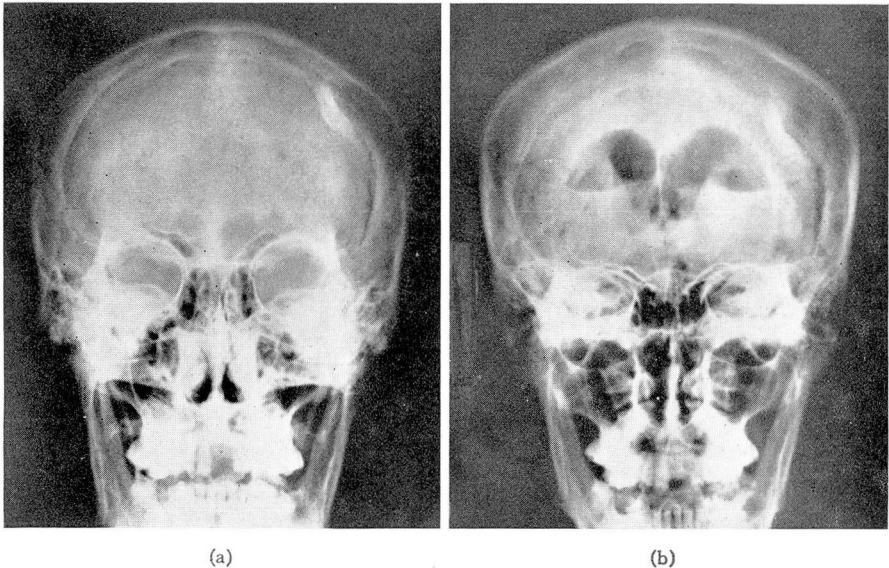


FIG. 1. (a) Plain skull film showing evidence of old depressed fracture in left frontoparietal region. (b) Air encephalogram showing dilatation of lateral ventricles and third ventricle with slight displacement of midline structures to the right.

Electro-encephalogram showed a borderline record with no evidence of a focal, cortical lesion.

It was our diagnostic impression that the depressed fracture was the cause of the patient's Jacksonian convulsive seizures.

On September 2, 1949, an operation was performed for the purpose of elevating the depressed calvarial fragment. The description of the operation reads as follows:

"A large scalp flap was turned down around the old depressed fracture in the left frontoparietal area. The depressed portion of the skull was about 2 inches in diameter and was situated directly over the motor arm area on the left side. The depression was about $\frac{3}{8}$ inch deep and the bone fragments had healed firmly during the 20 odd years since the injury. A drill hole was made through the center of the depressed area and the depressed bone was gradually removed by means of a rongeur until there was a bony opening about 3 by 2.5 inches in size. The underlying dura was quite adherent to the

inner depressed table of the skull but no fragments of bone had broken through the dura. The bone was thick and avascular and extremely hard at the site of the depressed fracture. The underlying dura was somewhat vascular and tense. The operator decided to open the dura in order to examine the cortex and to determine the presence or absence of adhesions between the dura and the cortex. The thickened dura was opened with a curved incision and a flat retractor was placed beneath it in an upward direction toward the sagittal sinus. At a point just beneath the site of the old depressed fracture, the retractor met resistance. It was the operator's impression that the retractor had encountered some adhesions between the cortex and the dura. The dura was then opened more widely in an upward direction, and to the surprise of the surgeon, a tumor was encountered at the exact location of the previous depressed fracture; an encapsulated meningioma slightly larger than a robin's egg attached to the dura was lying in a pocket of the cortex in the motor arm area. The dura was cut through all the way around the base of the attachment of the tumor and the tumor was gradually separated from the cortex by cotton dissection. The neoplasm was easily removed in one piece with its overlying dural attachment. There was no excessive bleeding. The tumor had the gross appearance of a meningioma (fig. 2). The dura was partially closed with interrupted nylon sutures.

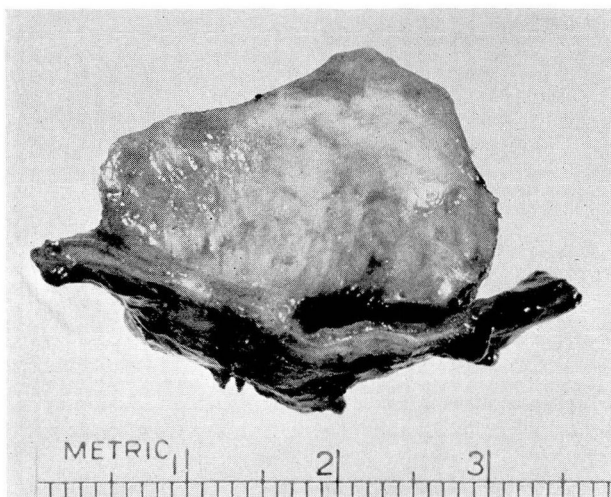


FIG. 2. Meningioma after removal, complete with its dural attachment.

A tantalum plate about 3 by 4 inches in size was fashioned and screwed down to the skull with four tantalum screws, covering the bony defect. The scalp flap was then replaced over the tantalum plate and united in layers with interrupted black nylon sutures. Microscopic examination of the tumor revealed a meningioma of meningotheelial type."

Discussion

For many years the relationship between trauma, irritation and neoplastic growth has been debated, and in the case of the intracranial tumors, no less than with neoplasms of other organs, authorities have differed widely in their views.

Stewart² disapproved of attempts to correlate the incidence of a single injury with subsequent cancer. Ewing¹ believed that coincidence is of greater importance than is generally recognized.

It must be remembered that a tumor may be the indirect cause, rather than the effect, of trauma. This is especially true with intracranial new growths, in which disorders of equilibrium, visual defects and convulsions may lead to head injuries.

Thus, even though injury may occur over the site of a neoplasm and may be followed by the onset of signs and symptoms referable to the growth itself, the traumatic incident should be regarded as an aggravating rather than a causative factor. The aggravation in these cases is usually believed to be the result of edema or hemorrhage in the tumor area. In fact, a small proportion of previously undiscovered intracranial neoplasms is disclosed in this manner.

Many authorities now appear to believe to some extent in the possibility of tumor formation following chronic irritation.

Keen and Ellis³ in 1918 described a case in which a depressed fracture of the skull involving both tables was followed about 20 years later by manifestations of irritation and compression of the brain. These symptoms were shown to be due to neoplasm. The tumor lay directly beneath the area of the depressed fracture and was diagnosed at that time as a fibroma. Complete removal was effected, and the patient survived the operation by 30 years. Keen's opinion was that the constant motion of the brain and dura, due to arterial and respiratory pulsations, caused friction against a spicule of bone fractured from the inner table, and gave rise to the tumor.

Cushing and Eisenhardt⁴ supported the view that the importance of trauma could not be overlooked. They believed the relationship between a blow on the head and the location of an ensuing meningioma to be so precise as to make the conclusion inescapable that an etiologic factor was involved. In support of this belief they quoted several case histories, and noted that almost one-third of their patients with meningiomas at that time had an associated history of trauma. Among these cases was the interesting history of General Leonard Wood in whom the onset of Jacksonian attacks occurred 4 years after a blow on the vertex, and from whom a neoplasm was removed. Cushing and Eisenhardt also call attention to another interesting case in which an apparently normal dura was exposed at operation for removal of thickened bone at the site of an old fracture. Three years later a meningioma was found at exactly the same site. In this case the onset of symptoms occurred 10 years after the original injury. Other cases of parasagittal tumors quoted by Cushing and Eisenhardt show a somewhat convincing relationship between injury and symptomatic onset.

As a result of observations on these and other cases, Cushing and Eisenhardt suggested the following factors which might play a part in the pathogenesis of such neoplasms. "(1) A local contusion of the bone; (2) a bruised meninx most likely to occur along a suture line; (3) a local outpouring of meningocytes to take up the extravasation, and (4) a pathologic continuance of their active cell division."

These factors were believed to have explained the relative frequency of meningioma at the bregma and the pterion where four suture lines meet and where there are four adherent sections of dura. However, the fact that meningiomas never result from the operation of craniotomy, in which all four conditions are probably fulfilled, would appear to militate against this theory of pathogenesis.

Confusion may be caused in some of these patients by the recognized tendency of certain types of meningioma to form bone. It may be difficult to state whether a bony fragment found to be embedded in the tumor is the cause, or one of the pathologic effects of such neoplastic growth. Undoubtedly many fractures involve the inner table of the skull, with consequent irritation of the meninges by pointed spicules or the sharp bony edges of the fracture. However, the incidence of meningioma following such injuries is low.

Summary

In the case reported a depressed fracture of the cranial vault was followed some 21 years later by manifestations of cortical irritation at the site of the old fracture. The findings at craniotomy and the other available facts suggest that the tumor originated as a direct result of the depressed fracture. It must be emphasized however that the cause of such neoplasms still remains obscure.

References

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