Chronic extradural hematoma presenting 33 years after penetrating cranial trauma

Case report

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A case is reported of a 53-year-old normotensive man who presented, 33 years after a penetrating cranial war injury, with dysphasia of 10 month's duration, which proved to be due to a chronic extradural hematoma. The pathogenesis and symptomatology are discussed, and it is proposed that the lesion had been present since the original injury.

KEY WORDS: penetrating trauma, hematoma, chronic extradural, ischemia, dysphasia

In their series of 125 cases of extradural hematoma, McKissock, et al., reported that only six patients presented more than 2 weeks after injury, and the longest delay was 31 days. However, chronic extradural hematoma is a recognized condition; Iwakuma and Brunngraber recently reviewed a series of 21 cases operated on from 13 to 41 days after injury, and Grant reported a case presenting 6 years after injury. In the present case the site of the lesion and the evidence of penetration of the skull indicate that the hematoma resulted from an injury received in 1943, and the contents are compatible with it having been present for 33 years.

Case Report

A 53-year-old right-handed bank manager presented in August, 1976, with a 10-month history of an initially intermittent, but later progressive language disorder. Ten months previously, while reading documents at work, he suddenly found that the written words before him had no meaning. This lasted for 30 minutes, and he remained well until 4 months later when, at a business meeting, he experienced a further transient episode, during which he was unable to comprehend spoken words. This also resolved completely and he again remained well until 3 months before presentation when he developed a steadily progressive deterioration in his ability to produce and understand spoken and written language. Throughout, he experienced no headache, loss of consciousness, or other neurological disturbance.

In 1943, while leading his Company in an attack at Salerno, he had been wounded when a fellow officer trod on an anti-personnel mine. The patient had been struck by multiple small fragments of metal and suffered a penetrating injury of the left eye (which was later enucleated), a perforation of the left tympanic membrane, multiple small puncture wounds of the left side of the head, neck, and upper limb, and compound fractures of the left third and fifth metacarpals. Apart from loss of the left eye and a mucoid discharge from the left ear, which persisted intermittently for 4 years, he made a full recovery and had been well until the present illness.

Examination. The patient had traumatic tattoos on the left side of his face and neck. His blood pressure was 140/80; there were no carotid bruits, and the cardiovascular,
respiratory, alimentary, and genito-urinary systems were normal. The neurological signs were a mixed expressive and receptive dysphasia with occasional jargon speech, especially when reading aloud, an upper quadrantic visual field defect in the right eye (the left eye was prosthetic), and a healed perforation of the left tympanic membrane with severe conductive deafness. The differential diagnosis was between carotid artery stenosis and a left temporal tumor.

A plain chest film showed several opacities of metallic density around the left hemidiaphragm. Plain skull films revealed an intact pituitary fossa. The pineal gland was not seen, and there were several metallic fragments on the left side of the head, most of which were extracranial but one was intracranial around the left petrous ridge. A radioisotope brain scan showed an area of abnormal activity in the left temporoparietal region. A left common carotid angiogram showed normal extracranial carotid arteries and an avascular left temporal mass. The preoperative diagnosis was left temporal glioma.

Operation. At a left temporal craniotomy, a cystic, subtemporal, extracerebral mass was found, which was extradural and 8 cm in maximum diameter. The wall of this cyst was continuous with the dura of the middle cranial fossa, the bare bone of the petrous ridge formed the base of the cyst, and it contained turbid, shimmering brown fluid. A 1.1-mm metallic fragment was found on the superior surface of the petrous bone. The cyst was emptied and the wall excised except for an area posteromedially where it encircled large veins draining the temporal lobe. The patient made an uneventful postoperative recovery. Within 2 months his neurological deficit had resolved completely and he has remained well.

Pathological Examination. The pathologist (Dr. R. O. Weller) reported as follows: The specimen consisted of a partly opened cyst with a tough, fibrous wall 2 to 3 mm thick; its inner surface was rough and yellow in color.
Chronic extradural hematoma

Histology of the wall showed dense fibrous tissue and a lining of granulation tissue. There were a few foci of lymphocytes within the wall and extensive areas of foreign body giant cell formation, often with two or three giant cells surrounding a small cholesterol cleft (Fig. 1 left). There was no evidence of an epithelium or of a parasitic infection. Cytology of the brown fluid within the cyst showed numerous cholesterol crystals, a few foamy macrophages, and a considerable amount of amorphous debris. The metallic fragment burst on cleaning to disclose a powder that proved to be principally sulphur and phosphorus.

Discussion

Pathology. The description of the lesion as a chronic extradural hematoma is based on its site, its composition, and its contents. The histological appearance of the wall of the cyst suggested that the granulation tissue and dense fibrous tissue had been formed in response to a chronic hematoma. The composition of a fibrous wall, probably the dura, with no additional capsule is in accordance with one series of 21 cases of chronic extradural hematoma, of which only two had definable capsules. The other 19 had a varying amount of granulation tissue which had ossified in some of the younger patients. The two cases in which a capsule was present were operated on more than 21 days and 41 days after injury. The first description of an encapsulated chronic extradural hematoma was of a case operated on 36 days after injury. In the present case no capsule had been formed although 33 years had elapsed between injury and operation.

The contents of the cyst were the product of the breakdown of erythrocytes, and were not keratin or mucus, which would have been present if the lesion had been an implantation dermoid derived from indriven epithelium of the outer or middle ear. The contents indicated that the lesion was old.

Pathogenesis. The patient sustained two distinct types of cranial trauma: penetrating and blast. Intracranial hematoma following penetrating head wounds are unusual. A series of 2000 war-time missile head injuries contained only 83 cases of intracranial hematoma, of which only three were extradural; all of these three occurred within 48 hours of injury and were due to laceration of middle meningeal arteries by bone fragments. The minute metallic fragment in the present case entered the cranium through the middle ear and, despite its small mass and retardation during penetration, caused local stripping of the dura and created the venous extradural hematoma.

Alternatively, the dura might have been stripped as a result of blast injury. However, because of the distance of the patient from the explosion and the interposition of the body of his less fortunate fellow officer, he sustained little blast injury. Furthermore, in a review of cerebral blast injuries, all patients who developed intracranial hematoma had suffered a blast severe enough to cause loss of consciousness (“blast concussion”).

A third possible etiology is that a small hematoma formed at the time of the injury and later enlarged by a process similar to that postulated in chronic fluid subdural hematoma, or as a result of subsequent repeated hemorrhages. However, these last explanations are incompatible with the symptomatology.

Symptomatology. Iwakuma and Brunngraber observed modes of presentation that included raised intracranial pressure, persistant hemiparesis, lack of expected improvement, and secondary deterioration in the level of consciousness. In the present case the symptoms probably resulted from focal ischemia of the temporal and inferior parietal lobes. There was no evidence of recent hemorrhage or of focal epilepsy, and recovery was complete. The absence of symptoms of raised intracranial pressure suggests that the lesion had been present for a considerable time, and this contention is consistent with the pathology.

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