Edwin Smith Papyrus Case 8: a reappraisal

Historical vignette

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There are now been 3 translations of the Edwin Smith Papyrus: Breasted’s (1930), Allen’s (2005), and Sanchez and Meltzer’s (2012). Case 8 is similarly presented in all 3 translations, although with increasing detail in the later works. The patient in Case 8 had a comminuted skull fracture under intact skin. There were palpable pulsations at the fracture site. There was deviation of the eye on the side of the injury and an ipsilateral spastic hemiparesis in an ambulant patient with bleeding from the nose and ear.

Explanations of the paralysis have included a contrecoup lesion and compression of the contralateral cerebral peduncle against the tentorial incisura. Brainstem compression due to herniation is accompanied by loss of consciousness. Extensive contrecoup lesions consistent with the extent of the described paresis would also be associated with probable coma. The paralysis was spastic, but spasticity takes weeks to develop after trauma. Yet this patient’s trauma was fresh, as there was still bleeding from the nose and the ear.

It is suggested the paresis antedated the trauma, which was not its cause. The reasons for this suggestion are presented in this paper.

Key Words • Edwin Smith Papyrus Case 8 • ipsilateral hemiparesis • history

Clinical Description

The components of the clinical picture are taken from the most detailed translation, by Sanchez and Meltzer.3

1. A smashed in/burst fracture of his skull under the skin of his head
2. The eye is askew under it
3. Rigidity in the back of the neck
4. Bleeds from his nostrils/nasal cavities and from his ears
5. He walks shuffling with his foot on his side which is under that blow which is in his braincase
6. One whose thumbnail/fingernail bends down into the middle of his hand
7. Shuffles with his foot ... toes contracting/curled up toward the instep. [Breasted2 calls this region the sole. I have been in telephone communication with the authors of the two later papers,2,3 who agree that the term instep is an error and the correct term is sole.—J.C.G.]
8. Cannot loosen the forked bone in his shoulder joint

Interpretation of the Clinical Picture

Fracture Type

The image on the left in Fig. 1 shows diagrammatically the sort of fracture which could be involved. The image on the right shows that this sort of fracture could be associated with an intraorbital component.

Eye Deviation

A fronto-orbital fracture with depression of the orbital roof could produce a mechanical rather than a neurological deviation of the eye. There are two common mechanisms whereby cerebral trauma can produce eye deviation based on neurological damage. Damage of the intermediate frontal gyrus can cause both eyes to deviate...
Fig. 1. Left: This image shows semi-diagrammatically the kind of fracture described in this case. The fracture is comminuted and not impacted, and this lesion could well be associated with the extracranial transmission of intracranial pulsations. Right: This image is the same as the one on the left except for the addition of an intraorbital fragment that could well be associated with a fracture of this kind in this location. Such a fragment could produce a mechanical rather than a neurological deviation of the eye on the side of the fracture.

toward the damaged side. Only one eye is described as deviated in this case. Moreover, such patients are most commonly unconscious when an injury is fresh, and the injury described is fresh as explained below. The second cause of posttraumatic eye deviation is compression of the oculomotor nerve at the tentorial edge. This compression is the result of tentorial herniation associated invariably with loss of consciousness. Yet the patient in Case 8 is ambulant. A mechanical deviation is more consistent with a conscious ambulant patient.

Neck Rigidity

This is most likely due to posttraumatic subarachnoid hemorrhage.

Bleeds From the Nose and Ear

This suggests two things. The fracture is most probably basal. Moreover, as the bleeding is ongoing, the fracture is fresh.

Ipsilateral Hemiparesis

Two explanations have been given for this. The first, in Breasted's translation, suggests a contrecoup lesion. The most recent, in Sanchez and Meltzer's book, suggests compression of the opposite cerebral peduncle against the tentorial incisura. However, there are findings that make these interpretations less likely.

Firstly, why is it unlikely that the patient had a contrecoup lesion? In this case, while no mention is made of the face, the paresis affects the shoulder, arm, and leg. Contrecoup lesions involve the cerebral cortex on the opposite side to an injury striking the inside of the cranium and sustaining injury. Inevitably such lesions are located peripherally in the opposite hemisphere. Cerebral function is most spread out in the cortex. Thus, to affect shoulder, arm, leg, and foot, such an injury would be very extensive. While not certain, it is to be expected that such a patient would suffer from a deterioration of consciousness. In addition, fresh cortical injury would produce a flaccid, not a spastic, paresis. The patient would be unable to walk. In the case described it is documented that the lesion is fresh, because the patient is bleeding from the nose and ear. So, for the reasons of the extent of trauma, the spastic paresis, the fresh trauma, and the capacity to walk, it is suggested a contrecoup lesion is not a likely cause of the described ipsilateral hemiparesis.

Secondly, why is it unlikely that compression of the contralateral cerebral peduncle could cause the ipsilateral hemiparesis? This is simpler to answer. For such compression to occur there would be tentorial herniation. This is invariably associated with unconsciousness, and a patient in this situation would be unable to walk.

Spastic Hemiparesis

The case presentation includes a detailed description of a spastic hemiparesis, with inability to abduct the shoulder, contracture of the fingers into the palm of the hand, and contracture of the toes into the sole of the foot. As mentioned above, the injury described must be fresh, since there is bleeding from the ear and nose. It is common experience that hypertonia and hyperreflexia take time to develop after cranial trauma. It has been recorded that several weeks must pass after cerebral damage before spasticity develops. Since the injury discussed in Case 8 is fresh, for the reasons given above, it seems unlikely that a spastic paresis can be the result of the recent injury.

Conclusions

In view of the evidence presented above, it is suggested that the ipsilateral hemiparesis recorded in Case 8 of the Edwin Smith Papyrus is not the result of the described injury but is more probably preexisting. Previous cerebral injury, infection, or cerebral palsy could be possible explanations.

Disclosure

The author reports no conflict of interest.

References


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