Concurrent delayed temporal and posterior fossa epidural hematomas

Case report

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A case of delayed epidural hematoma presenting in two different intracranial compartments is described. The presumptive mechanism of the lesion is discussed and the value of early reevaluation by computerized tomography is stressed.

KEY WORDS • computerized tomography • epidural hematoma • head injury

TRAUMATIC extradural hemorrhage is usually considered as an acute complication of head injury. Clinical manifestations of compression of intracranial contents often appear quickly, requiring prompt therapeutic intervention. The original description of this lesion in 1885 by Jacobson identified the middle meningeal artery as the source of bleeding, a fact that was later corroborated by Ford and McLaurin. This classical concept of arterial origin of such hemorrhages has changed, however, after several cases were reported of delayed extradural hemorrhage of venous origin.

A case is presented of a patient who developed delayed extradural hematomas in two different intracranial compartments. The pathophysiology and clinical significance of this condition are discussed.

Case Report

This 16-year-old white girl was transported to our shock and trauma facility by helicopter after being struck by a moving vehicle 1 hour before arrival. She was rendered unconscious at the scene, and during transportation she recovered consciousness and vomited once.

Examination. On arrival at 4:40 p.m., her blood pressure was 110/60 mm Hg and pulse 90/min. Her initial Glasgow Coma Scale score was 15 but, since she was mildly agitated, the shock and trauma team administered 75 gm of mannitol intravenously. She then underwent peritoneal lavage which was considered positive for blood. At that time neurosurgical evaluation was requested and revealed her to be conscious but disoriented to time, place, and recent events; she had a good verbal response. There was active left otorrhagia due to a lacerated external auditory meatus; neurological examination was otherwise normal.

First Operation. The patient underwent an exploratory laparotomy under general anesthesia and a laceration of the transverse mesocolon was found and repaired. No hypotension or complications were reported during the procedure. After surgery, she was taken to the surgical intensive care unit where she remained intubated. By midnight she was awake, obeying commands, and without any evidence of neurological dysfunction. At 5:45 a.m., there was sudden deterioration of her level of consciousness associated with a left dilated unresponsive pupil and a right hemiparesis. Repeat CT head scan (Fig. 2) showed a left posterior fossa epidural hematoma associated with a left dilated unresponsive pupil and a right hemiparesis. Repeat CT head scan (Fig. 2) showed a left posterior fossa epidural hematoma associated with a left temporal epidural hematoma. There was a left-to-right midline shift, and a left thalamic and right frontal hemorrhage, both of them small.

Second Operation. With the patient in the prone position, a left temporal craniectomy was carried out with evacuation of a dark epidural blood clot.
that, a left posterior fossa craniectomy was performed with evacuation of another dark epidural blood clot. In both instances no dural or vascular lesions were encountered and no areas of active bleeding could be found. The patient was then extubated in the operating room, and she was found to be responsive and adequately moving all her limbs. An immediate postoperative CT head scan was obtained and did not reveal any new intracranial lesions. She had an uneventful recovery and was discharged on the 15th postoperative day.

Discussion

Reports of delayed intracerebral and extradural hemorrhage of traumatic origin have appeared previously in the literature, but concomitant unilateral hematomas in two different compartments presenting in a delayed fashion have not been mentioned.

The venous source of delayed extradural hemorrhage has been described by Adeloye and Onabanjo, who implicated the dural sinuses as the main contributors to the hematoma. It has also been said that the administration of hyperosmolar agents, such as mannitol, could contribute to the formation of delayed intracerebral hematoma by loss of the tamponade effect of intracranial pressure on small oozing venules. This cause has been mentioned in regard to epidural clots. The only case in which alleviation of the tamponade effect resulted in the appearance of a contralateral epidural hematoma was that reported by Koulouris and Rizzoli; evacuation of a right frontal intracerebral hematoma in that case precipitated the development of a left temporal epidural hematoma. Goodkin and Zahniser reported a case of delayed extradural hemorrhage very similar to this one, in which the diagnosis was made by means of serial angiography. They mentioned that the elevation of blood pressure and the restoration of blood volume in a previously hypovolemic and hypotensive patient could have accounted for the development of the lesion. In the present case, a combination of the above-mentioned mechanisms may have been responsible for the development and accumulation of the epidural hematomas, since the clots were apparently of venous origin and the patient had received hyperosmolar agents as well as rapid transfusions.

Serial CT head scanning has been strongly recommended in the follow-up review of patients suffering head injury, and remains the best method of diagnosing delayed intracranial lesions. This is especially true in cases where intracranial pressure monitoring is not available or is not used due to the satisfactory condition of the patient as evaluated clinically.
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References


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