ACUTE BRAIN SWELLING DURING EVACUATION OF INTRACEREBRAL HEMATOMA CAUSED BY PRIMARY PROGRESSIVE CONTRLATERAL SUBDURAL HEMATOMA: A case report

İntraserebral hematom boşaltılması sırasında karşı taraf yerleşimli büyüyen subdural hematoma bağlı akut beyin şişmesi

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Summary: *In this paper, a patient who experienced severe brain swelling during the evacuation of traumatic intracerebral hematoma is presented. Postoperative computed tomographic (CT) scans revealed primary progressive subdural hematoma on the side opposite the intracerebral hematoma. Subdural bleeding occured in the area of the fractured skull.The reduction of intracranial pressure after the evacuation of intracerebral hematoma was postulated to be the most important factor contributing to the formation of the delayed intracranial hematoma. We want to emphasize that acute brain swelling during surgery for traumatic intracerebral hematoma may be caused by primary progressive contrlateral subdural hematoma.*

Key Words: *Brain swelling, Primary progressive intracranial hematoma, Subdural hematoma, Traumatic intracerebral hematoma*

Özet: *Bu makalede, travmatik intraserebral hematomun cerrahi olarak boşaltılması sırasında karşılaşılan akut bir beyin şişmesi takdim edildi. Ameliyat sonrası çekilen bilgisayarlı beyin tomografisinde, intraserebral hematomun karşı tarafında, kranial kemik kırığının olduğu bölgede genişleyen bir akut subdural hematom tesbit edildi. İntraserebral hematom boşaltılmasını takiben kafa içi basıncında meydana gelen azalmanın geciken intrakranial hematomların oluşumuna katkıda bulunan en önemli faktör olduğu ileri sürülmektedir. Bu makalede, travmatik intraserebral hematom boşaltılması sırasında karşılaşılan akut beyin şismesine, karşı taraf yerleşimli genişleyen bir akut subdural hematomun neden olabileceği vurgulanmak istenmiştir.*

Anahtar Kelimeler: *Beyin şişmesi, birincil genişleyen intrakranial hematom, Subdural hematom, Travmatik intraserebral hematom*

Acute brain swelling during the evacuation of a traumatic intracerebral hematoma is one of the most tragical complication in neurosurgery. Cerebral edema, CO_2 retantion, poor positioning of the patient, hyperemia and delayed intracranial hematoma may be possible pathological mechanisms leading to such a complication (1-3). To our knowledge, the occurrence of delayed subdural hematoma during the evacuation of a contrlateral traumatic intracerebral hematoma has not previously been reported in the literature.

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Received 29 May, 1995

CASE REPORT

A 55-year old man was admitted to our hospital within an hour of a motor-vehicle accident. The patient was comatose (localizing pain with no eye opening and no verbal response, i.e. Glasgow coma score 7/15). He had anisocoria on the left. There was a subgaleal hematoma in the right frontotemporal region. X-Ray examination of the skull revealed right frontotemporal linear fracture.An intracerebral hematoma in the left temporal lobe leading to 13 mm mid-line shift was determined on the CT scans (Figure 1). The patient was immediately taken to the operating room while receiving 1 g/kg mannitol 20% intravenuosly. At the operation, left temporoparietal craniotomy was performed. After opening the dura, intracerebral hematoma was exposed beneath cortical contusion of the left temporal lobe. At the end of the evacuation

of the hematoma, acute swelling of the brain was noted. In spite of the efforts to decrease the brain swelling (i.e additional mannitol infusion, hyperventilation), the exposed cortex continued to bulge. Therefore, an internal decompression of anterior temporal lobe was necessery. At the end of the procedure, prior to extubation, it was noted that the right pupil was larger than the left. The patient was immediately taken to the CT room and a second CT scan showed a large frontoparietotemporooccipital subdural hematoma on the opposite side of the evacuated intracerebral hematoma and a left posterior temporooccipital intracerebral hematoma (Figure 2). The patient was returned to the operating room, where a right temporoparietal craniotomy was performed. Dural tear and active arterial bleeding on the parietal cortex was noted. The arterial bleeding was controlled and the subdural hematoma was totaly removed. The patient left the intensive care unit on 12th day with a favorable motor response (localizing pain). He started obeying commands on Day 20 and was discharged home moderately disabled after 4 months of intensive rehabilitation (Glasgow outcome score 4).

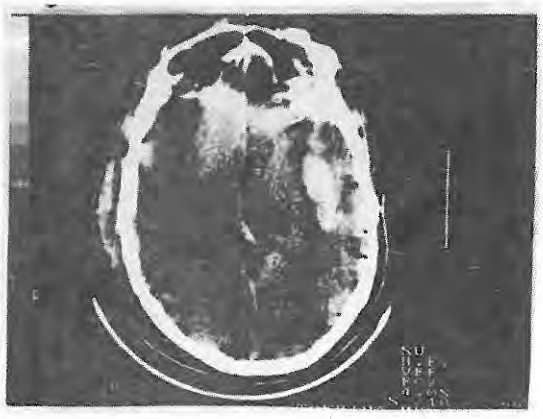

Figure 1. Preoperative CT scan showing a left temporal intracerebral hematoma and an obliteration of the third ventricle and basal cisterns

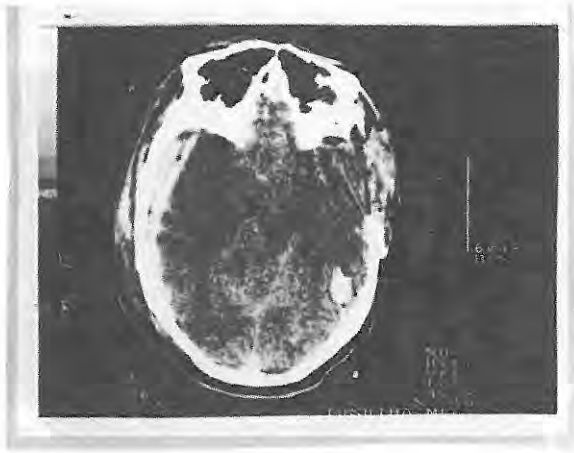

Figure 2. Postoperative CT scan showing primary progressive right subdural hematoma. Note the third ventricle and basal cisterns cannot be distinguished

DISCUSSION

Operative management of traumatic intracerebral hematomas may be complicated by acute brain swelling, especially in cases of severe brain injury. Effective decompressive measures should be tried either by pharmacological agents or by surgical decompression (1, 2, 4).The first includes barbiturate coma and hyperosmolar agents such as mannitol. Induced systemic hypotension to reduce hyperemic processes was also advocated (1, 2). In order to avoid hypercapnia, it is essential to ensure a proper airway and ventilation (2).

We could not find article concerning with the primary progressive contrlateral subdural hematoma in a patient with traumatic intracerebral hematoma. In 1987, Meguro et al. (3) reported two cases who developed acute brain swelling during evacuation of subdural hematoma caused by delayed contrlateral extradural hematoma and they noted that: a) both patients had skull fractures at the site of impact b) the initial lesion treated was the contrecoup injury c) delayed extradural hematoma presented on the side of injury and skull fracture. These peculiarities are true for our case with the exception of location of the hematoma.

Several etiological mechanisms have been proposed to explain the production of delayed intracranial hematoma after severe head injury. While Evans and Scheinker (5) suggested an impairment of vasomotor mechanism, Kaufman et al. (6) noted evidence suggesting that coagulopathy may play some role in the evolution of delayed intracranial hematoma. However, loss of the tamponade effect due to surgical decompression or use of hyperosmolar agents such as mannitol may be the likely mechanism of delayed intracranial hematoma (7, 8). Similarly, Meguro et al. (3) proposed that evacuation of the first hematoma reduced the tamponade effect on the contrlateral bleeding, allowing new intracranial hematoma to form.

Fukamachi et al. (9) in their report on delayed traumatic intracerebral hematoma with extradural hemorrhages emphasized that the incidence of post surgical intracerebral hematoma with extradural hemorrhage is high and acute brain swelling during surgery for extradural hematoma is largely caused by the delayed intracerebral hematoma.

Early diagnosis of traumatic delayed intracranial hematoma requires a high index of suspicion and awarenes of the condition. Close observation for clinical deterioration alone is not sufficient. In a recently reported series (4), 11 patients with delayed extradural hematoma were diagnosed by clinical observation alone, resulting in four death and two disabilities. However, in the remaining 11 patients, close observation was supplemented by either continuous ICP (intracranial pressure) monitoring or serial CT (computerized tomography); no death and two moderate disabilities were documented.

We, therefore, suggested that high-risk patients (with persistently impaired levels of consciousness and skull fracture, especialy with contributory factors such as hyperventilation, osmotic dehydradation, barbiturate administration, otorrhea, surgical decompression and hypovolemia) should certainly be considered for ICP monitoring and/or serial CT. But, because of the institution of ICP monitoring requires expertise, takes time, and has complication, repeated CT scanning may be more practical.

The present case supports the notion that brain injury leading to formation of hematoma should be considered as a dynamic event rather than a static process. Therefore, serial CT scans have become an important tool in monitoring of such patients. Surgeon must be aware of the possibility of a contrlateral subdural hematoma, when the exposed brain becomes swollen during evacuation of intracerebral hematoma. In his textbook, Schnieder (10) drew attention to Mckenzie's statement; "When in doubt, trephine over fracture line first". Although this statement, made in 1938, still applies, we suggest that performing exploratory burr holes on the fractured side is the best strategy to be chosen in case of dramatic deterioration with obvious contrlateral intracranial hematomas. Awaiting a postoperative CT scan is wasting valuable time and should be considered after a negative exploration.

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