Expansion of Contralateral Extradural Hematoma following Mannitol Therapy

Amit Agrawal[1] G. Malleswara Rao[1]

[1] Department of Neurosurgery, Narayana Medical College Hospital, Chinthareddypalem, Nellore, Andhra Pradesh, India

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Address for correspondence Dr. Amit Agrawal, MCh, Department of Neurosurgery, Narayana Medical College Hospital, Chinthareddypalem, Nellore 524003, Andhra Pradesh, India (e-mail: dramitagrawal@gmail.com).

Dear Sir,

Traumatic brain injury can be complicated by edema and mass effect resulting in elevation of the intracranial pressure (ICP), reduction in cerebral perfusion pressure, and global ischemia.[1] The use of osmotic agents (mannitol and hypertonic saline) is recommended to lower the ICP and to improve cerebral blood flow to hypoperfused brain regions in patients with traumatic brain injury.[2] Mannitol has traditionally been used as nonsurgical treatment options to control intracranial hypertension, tissue shifts, and ultimately herniation in these patients.[1,3–5] A 16-year-old girl sustained traumatic brain injury after being hit by a two-wheeler. She presented to the emergency department ~6 hours after the incident. She had a history of loss of consciousness for 20 minutes, multiple episodes of vomiting, and left ear bleed. There was no history of seizures. On neurological examination, she was opening eyes to call, disoriented, and localizing to pain (E3V4M5). Pupils were bilaterally equal and reacting to light. There were no focal neurological deficits. General and systemic examination was unremarkable. A computed tomography (CT) scan of the brain showed thin right fronto-temporo-parietal acute subdural hematoma with mass effect and minimal midline shift; in addition, there was a thin temporal extradural hematoma (EDH) of the left side also (►**Fig. 1A**). The patient was started on antiedema measures and prophylactic antiepileptics. She regained consciousness the next day after admission but was complaining of persistent headache. In spite of the improvement of her neurological status, in view of initial early scan, persistent headache, and presence of multiple hematomas on early scan, a decision to perform a follow-up scan was made. Follow-up scan revealed that there was almost complete resolution of right-sided acute subdural hematoma; however, there was an increase in the size of left-sided EDH with minimal mass effect to the opposite side (►**Fig. 1B, C**). Her blood investigations including coagulation profile were normal. In view of the small size of the extradural blood clot and improving neurological status, the patient was managed conservatively and mannitol was discontinued immediately. The patient recovered completely without any neurological deficits and was doing well at follow-up.

It has been shown that EDH can attain maximum size within minutes of formation following traumatic brain injury.[6–9] In majority of the cases, the enlargement of the EDH is heralded by arterial thrombosis at the injury site and also by the tamponade effect by the clot and adjacent brain, thus preventing continued EDH enlargement.[10] In a subgroup of patients, EDH can further enlarge (may be continued hemorrhage or rehemorrhage from an arterial or venous origin) as venous hemorrhage may not generate enough pressure to overcome the tamponade effects of the clot itself and the adjacent brain.[11] In most of the patients who were managed conservatively, the EDH enlargement occurred by 8 hours after injury (maximum up to 36 hours).[12,13] Several mechanisms have been suggested to explain this enlargement of the EDH and include hyperventilation, mannitol, Pentothal, cerebrospinal fluid leak (otorrhea or rhinorrhea), surgical decompression (removing the tamponade effect and there may be contralateral EDH), low blood pressure, or shock.[14] It has been shown that those patients who had underlying skull fracture are more prone to develop contralateral EDH as well as at increased risk for the enlargement of the contralateral EDH.[14,15] It is suggested that the presence of a contralateral acute subdural hematoma may tamponade an EDH at the coup site[16]; however, as we observed, there is a tendency for EDH to enlarge even in the presence of a contralateral acute subdural hematoma.[12] Furthermore, the use of mannitol resulted in reduction in ICP, thus reducing the tamponade effect. It is important to remember that delayed or enlarging EDH can only be diagnosed by scanning the patient twice, and high index of suspicion especially in a patient who is not

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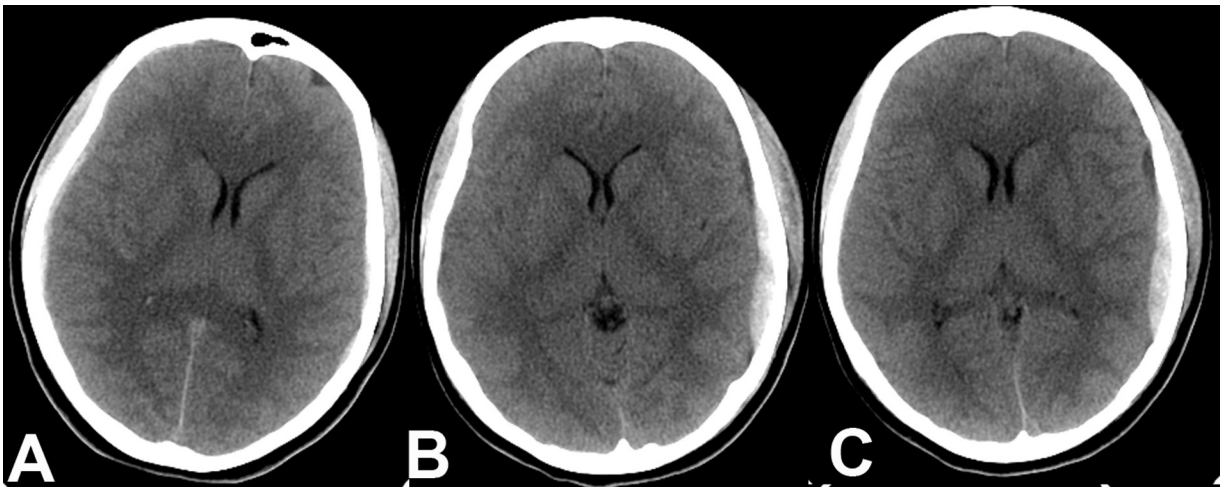

Fig. 1 (**A**) Early CT scan showing thin right temporoparietal acute subdural hematoma with mild mass effect and midline shift. (**B, C**) Follow-up repeat CT scan after administering mannitol showing almost complete resolution of the right acute subdural hematoma and increase in the size of the left temporoparietal extradural hematoma. Also, note the mass effect due to right-sided subdural hematoma has decreased.

improving neurologically or deteriorating is the key for an early diagnosis of delayed or enlarging EDH.[14,17–19]

Conflict of Interest
None declared.

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