Bilateral pupillary dilatation with normal intracranial pressure

Sir,

The standard management of non-operable severe head injury is ventilation with intracranial pressure (ICP) monitoring (brain trauma foundation).[1] Since the patient is often sedated for ventilation, neurological monitoring is restricted to examination of the pupils. We report the case of a patient who almost forced a fundamental change in our management protocol.

A 50-year-old diabetic female patient was admitted 2 h following a road traffic accident with a Glasgow coma scales core of 8/15, minimally asymmetric, but briskly reacting pupils and no lateralizing deficit. A computerized tomography (CT) scan of the brain showed bilateral frontal and a small right temporal contusion with preserved basal cisterns [Figure 1]. She was managed conservatively with ventilation and ICP monitoring. The ICP remained below 15 mmHg for the 1st 3 days and then showed a minimal increase up to a maximum of 18 mmHg. It was therefore decided to repeat a CT scan before weaning from the ventilator. The CT scan was performed 76 h after the trauma showed resolving contusions with increased edema around them [Figure 2]. Hence, mannitol was started and later vasopressors were added to maintain an adequate cerebral perfusion pressure.

At 16 h after the second scan, her pupils became large and non-reacting, though ICP remained below 20 mmHg. Assuming the IC*P* values were wrong, patient was taken up for a bifrontal decompressive craniectomy. After craniectomy, dura and brain were lax, excluding raised ICP. After craniectomy, blood pressure dropped further and had severe metabolic acidosis with a pH of 6.91 and a chloride of 123 mmol/L. Despite vigorous treatment of acidosis, blood pressure never recovered and she died 4 h later.

This report highlights the importance of hemodynamic changes and escalating needs of vasoactive drugs while

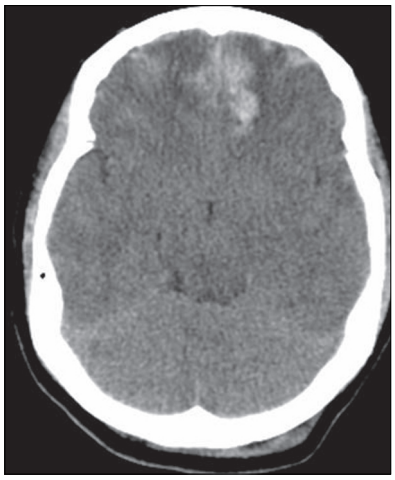

Figure 1: Axial computerized tomography scan 3 h following trauma showing bilateral frontal contusions adjacent to the falx with some surrounding edema. cerebrospinal fluid cisterns are seen

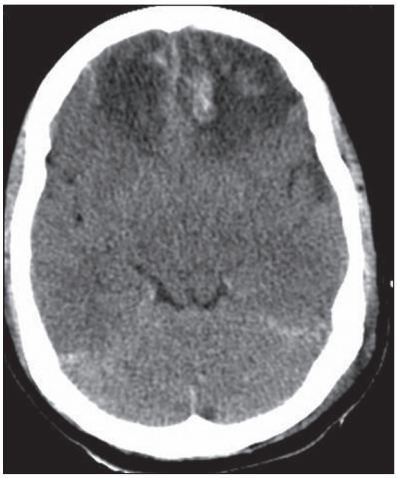

Figure 2: Computerized tomography scan 3 days later showing a decrease in the volume of hemorrhage and an increase in the edema. CSF cisterns are better visualized than in the earlier scan

managing a severe head injury with ICP monitoring. The hypotension was assumed to be due to the effects of acidosis on the heart and vasculature.[2] Though hyperchloremic acidosis is not fatal, Kellum *et al*. have reported that saline resuscitation significantly shortened survival. The survival time inversely correlated with the decrease in pH and the rise in serum chloride.[3]

Ritter *et al*. demonstrated reduced blood flow in the midbrain in patients with pupillary dilatation, independent of the ICP and brainstem lesions on CT scan and concluded that brainstem ischemia was an important causal factor in pupillary dilatation.[4] It is possible that the severe acidosis could have caused local vascular changes in the region of brainstem in our patient resulting in pupillary dilatation since there were no clinical or surgical features of raised ICP or herniation.

In conclusion, we wish to reiterate that pupillary changes in head injury need not always be caused by herniation. This case also shows that excessive importance given to one parameter (in our case, ICP) can result in decreased attention to other equally important clinical and laboratory findings and less than optimal care.

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