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NEUROCRITICAL CARE SOCIETY

LESSONS LEARNED

Considerations for Intracranial Monitoring and Surgery in Severe Traumatic Brain Injury with Temporal Lobe Contusion

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A 27-year-old healthy man is struck by a car. After stabilization, he has a Glasgow Coma Scale (GCS) score of 9 (M5/V2/E2), normal pupils, and no focal neurologic deficits. Head computed tomography (CT) demonstrates scattered subarachnoid blood, subdural and bifrontal contusions, and a small left temporal intraparenchymal hemorrhage (Fig. 1a) His examination result worsens to a GCS score of 7 (M5/V1/E1), with new bradycardia and a larger left pupil with delayed reactivity on quantitative pupillometry.

What is the Relevance of the Clinical Examination Change in this Patient?

In this patient, decreased arousal can be localized to the reticular activating system, which radiates from the top of the midbrain through the thalamus. Small amounts of midline shift can disrupt this pathway and may herald cerebral herniation [1]. The pupillary changes may also reflect third nerve compression due to expansion of the temporal contusion and uncal herniation.

New-onset bradycardia can be the first indication of Cushing's triad (bradycardia, hypertension, and irregular respiration), a clinical manifestation of the brain's compensation for increased intracranial pressure (ICP) [2]. Subtle changes in pupil size or reactivity may indicate early third cranial nerve compression from tissue shifts resulting from temporal hematoma expansion. An urgent noncontrast head CT scan is indicated.

Head CT shows interval expansion of bifrontal and left temporal lobe contusions (Fig. 1b). The examination result remains the same.

How Should this Patient Be Clinically Optimized, and Should Invasive Intracranial Monitoring Be Used in this Case?

Escalation of therapy for herniation syndromes follows a tiered method [2] and should not be delayed for invasive intracranial monitoring (multimodal monitoring [MMM]). Reasonable initial options are placing the patient upright, avoiding neck manipulation, addressing agitation and fever, a trial of hyperosmolar therapy, and ultimately deciding if urgent surgery is needed. These maneuvers, if there is clinical improvement (e.g. pupils or examination), may suggest the patient is at the end of the intracranial volume–pressure curve. Alternatively, the patient is also possibly herniating independent of an ICP crisis due to the temporal lobe contusion causing focal uncal herniation.

MMM should not be used in this case as either a "bridge" to or justification of escalated therapy, including surgery. Notably, MMM is time and resource intensive and is dependent on the location of probe placement. A

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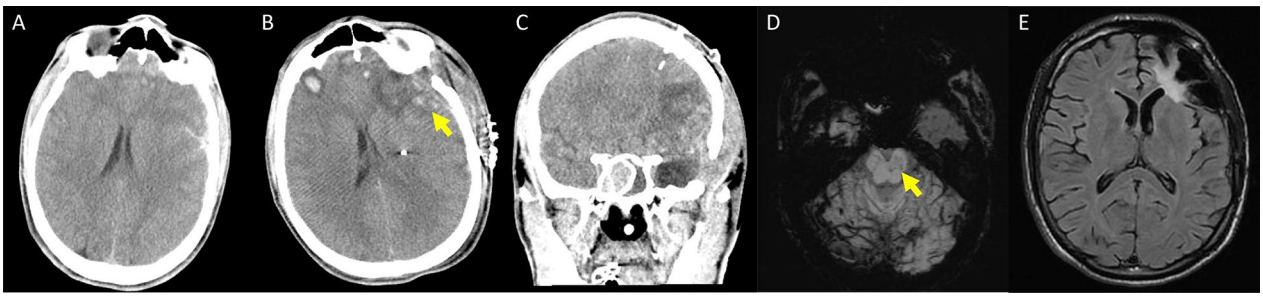

Fig. 1 Serial intracranial imaging. **a** Initial axial head computed tomography (CT) scan showing small left subarachnoid, subdural, and bifrontal contusions with small left temporal intraparenchymal hemorrhages. **b** Repeat coronal head CT scan prompted by worsening examination with increased contusion size, including temporal lobe contusion (arrow). **c** Coronal head CT scan post craniotomy demonstrating temporal hematoma resection. **d** Magnetic resonance imaging (MRI) of the brain on Hospital day (HOD) 8, suseptibility weighted imaging (SWI) sequence, and axial image showing small SWI changes in midbrain consistent with grade III diffuse axonal injury (arrow). **e**, MRI of the brain at 4-year follow-up and fluid attenuated inversion recovery (FLAIR) sequence showing left frontal gliosis

regional focus of uncus herniation may not be captured by the probe if it is distant to the injury site. Similarly, MMM values may be divergent from a concerning clinical picture, for example, in a study of malignant stroke, ICP monitoring showed an ICP < 22 mm Hg in a large number of patients despite evidence of midline shift and pupillary changes [3].

What Factors in this Case Support Immediate Surgical Decompression?

This patient should undergo immediate surgical decompression. The clinical picture of early pupillary changes, Cushing's triad, and expanding hematomas (especially in the temporal lobe) suggests medialization of the uncus in a tight temporal fossa and early herniation. Though the aforementioned tiered method should be followed to stabilize the patient [2], these therapies will not outpace the rate of hematoma expansion and edema, therefore making decompression a lifesaving step.

The large clinical trials on traumatic brain injury (TBI) surgical decompression do not apply to this patient with a temporal lobe hematoma and rapid clinical herniation. Decompressive Craniectomy in Diffuse Traumatic Brain Injury (DECRA) [4] and Randomised Evaluation of Surgery with Craniectomy for Uncontrollable Elevation of Intracranial Pressure (RESCUE-ICP) [5] compared decompressive surgery versus standard medical therapy [5]. DECRA did not reflect common clinical practice, as it was a study of early aggressive surgical decompression and only allowed bifrontal decompressive craniotomies, and the ICP threshold for surgery was lower than current guidelines. RESCUE-ICP represented a study of failed medical therapy, had a high crossover rate, and used hypothermia in the medical arm. As the field of TBI care moves toward precision medicine, large-scale studies may not capture the individual patient, and caution should be taken to not overgeneralize conclusions.

The patient undergoes urgent craniotomy with subtemporal and subfrontal decompression with anterior temporal lobectomy (Fig. 1c). Magnetic resonance imaging of the brain on day 9 shows grade 3 diffuse axonal injury (Fig. 1d). He was discharged to home with outpatient rehabilitation with a GCS score of 14 (E4/V4/M6) and a modified Rankin score of 4. At the 4-year follow-up in the clinic, repeat magnetic resonance imaging is done (Fig. 1e). He has no focal deficits, a modified Rankin score of 1, and intermittent mild behavioral outbursts. He lives independently and works as a mail carrier.

Lessons Learned

1. Hematoma expansion should be ruled out with acute decompensation in severe TBI. Temporal lobe lesions are prone to early uncal herniation independent of midline shift. Subtle symptoms include altered awareness, hemodynamic changes (e.g., hypertension, bradycardia), and pupillary changes.
2. MMM may not reflect regional herniation, and surgical decision-making can be made on clinical examination.
3. Avoid premature prognostication, as patients with TBI can have good functional outcomes at 1 year and beyond [6].

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Ethical Approval/Informed Consent
As a retrospective case, this article is approved by the institutional review board (UCI IRB #2283).

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