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Brain Tamponade after a Cardiac Arrest from Cervical Fracture and Acute Intracranial Monitoring

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Tamponade can be defined as a process where pressures are equal across opposing forces. In a physiological sense, it is the equalization of pressure in two separate chambers of the body where these are either connected or exerting pressure against each other due to a common cavity. The best example in clinical practice is cardiac tamponade.[1] Brain tamponade is a rarely used term in clinical practice due to the limitation of detailed physiological data to confirm it. Here we show intracranial pressure in real-time monitoring with equalization of pressure inside the skull and arterial pressure waveform.

A 35-year-old patient was brought to the emergency room followed by admission to neurointensive care for a high cervical injury, cardiac arrest, and anoxic brain injury. He fell from a ladder and was found to be apneic by the first responder. On emergency services arrival, cardiopulmonary resuscitation was in progress. It took over 20 minutes for the return of spontaneous circulation. His admission cervical spine magnetic resonance imaging (MRI) is shown in ►**Fig. 1**. High cervical cord injury and edema were noted. Neurologically, he was not awake, with minimal cough on deep suction and sluggish pupillary light reflex. MRI of the brain showed extensive bilateral cerebral edema and anoxic changes (►**Fig. 1**). Due to his young age, a bedside intracranial pressure fiberoptic monitoring catheter was inserted. The pressure was not high and typical diastolic collapse was seen consistent with brain tamponade (►**Fig. 2**). A follow-up computed tomography (CT) confirmed diffuse edema, loss in gray–white demarcation, and minimal to no ventricles (►**Fig. 2**). The critical care team proceeded to a brain death examination and pronounced him expired shortly afterward.

The description of the brain tamponade case above is not a usual phenomenon that can be seen in critical care practice. Here the real-time monitoring shown is unique to the brain. Another close example can be compartment syndrome of the lower extremities.[2] Due to the Monro–Kellie doctrine,[3] the brain cannot expand, which leads to tamponading itself. In the lower extremities, fasciotomies can break that balance.[4] In a global insult to the brain, it is not possible to break the pressure differential, and also anoxic insult is so advanced that brain death is not avoidable. Other examples of tamponade include acute abdominal compartment syndrome,[5] cardiac tamponade, retroperitoneal hemorrhage,[6] subcapsular hemorrhage around vital organs (liver, spleen, renal),[7,8] tension pneumothorax with or without shift (in intubated patient), neck hematoma after carotid surgery leading to airway compromise, and ring finger injury due to the metallic ring.

Nornes et al initially showed a noninvasive measurement of intracranial circulatory arrest with Doppler,[9] which led to the modern transcranial Doppler examination for brain death confirmation. The same group a few years later explained the circulatory changes with brain tamponade.[10,11] These explanations were based upon diminished to no circulation due to diffuse brain edema, limiting any flow into the skull. Lundar et al measured epidural pressure in cardiac surgery patients who underwent circulatory arrest; one of the patients developed brain tamponade and died.[12] Extensive subarachnoid hemorrhage leading to the diffuse collection of blood can present as brain tamponade, and emergent decompression should be done. This is usually with cerebrospinal diversion or bilateral decompressive craniectomy.[13] The mechanics of true brain tamponade should not be confused with an external tamponade of the brain, which is seen after decompressive hemicraniectomy for acute pressure increase such as malignant cerebral edema. The process is a control surgical trauma to protect the brain from a focal pathological process.[14,15] The Windkessel effect can explain the process of no flow into the brain due to the equalization of pressures. The vessels

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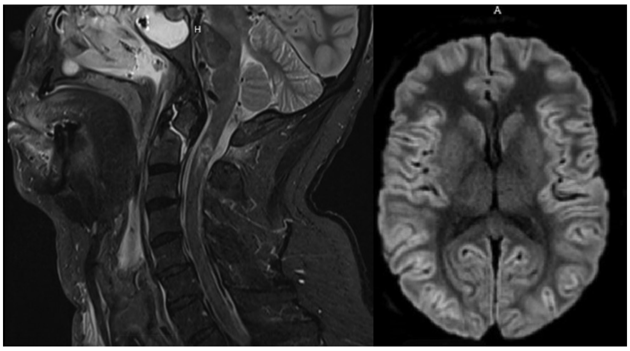

Fig. 1 Magnetic resonance imaging of the cervical spine with acute injury of C1–C2. Brain diffusion weighted imaging with extensive anoxic injury.

inside the brain cannot expand with brain tamponade, while extracranial larger vessels can expand, thus leading to low-flow state.[16] There is no capacitance left of intracranial vessels due to brain tamponade, which leads to absent diastole or diastolic collapse as seen in ►**Fig. 2**.

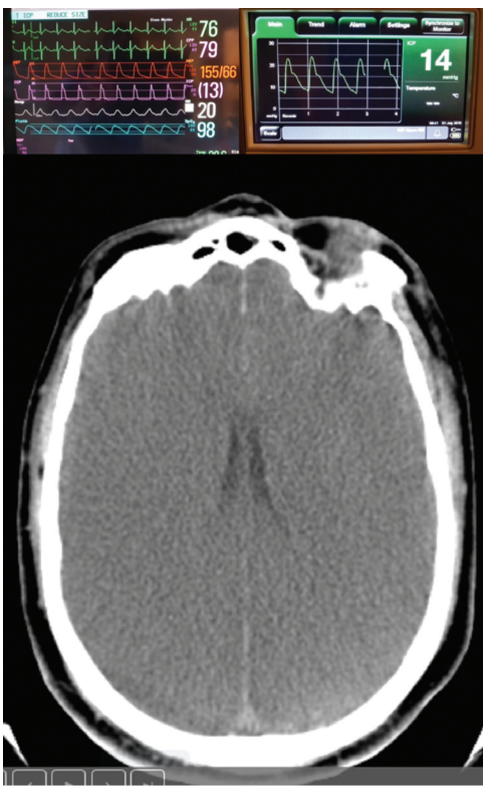

Fig. 2 Computed tomography scan of the brain with extensive edema, minimal ventricles. Intracranial pressure monitoring showing diastolic collapse (*purple waveform*) consistent with no blood flow due to the tamponade effect from edema.

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Conflict of Interest
None declared.

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