



Rare Event Captured with Intracranial Pressure Monitor: Malignant Spike in Intracranial Pressure During Delayed Chest Closure in Heart-Transplant Patient with Ischemic Stroke

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Key words

- Chest wound closure
- Heart transplant
- ICP monitor
- Intracranial pressure
- Malignant cerebral edema
- Sternal closure ischemic stroke

Abbreviations and Acronyms

CT: Computed tomography
DSC: Delayed sternal closure
EVD: External ventricular drain
ICP: Intracranial pressure
MCA: Middle cerebral artery
OCM: Open chest management
POD: Postoperative day

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INTRODUCTION

Patients with prolonged open chest wounds, either related to trauma or cardiothoracic procedures, who undergo closure are subjected to various physiologic alterations. At the time of wound closure, increased intrathoracic pressure reduces venous return volume. This affects intracranial pressure (ICP) due to increased total blood volume within the skull. A patient with intact cerebral autoregulation is able to respond to such alterations by adjusting cerebral blood flow in order to maintain overall ICP. In cases where cerebrovascular autoregulation is impaired, such as with stroke or trauma, these fluctuations in ICP cannot be compensated for appropriately. Patients with open chest wounds and concomitant brain damage are particularly vulnerable to experience pathologic

■ **BACKGROUND:** Patients with open chest wounds, either related to trauma or cardiothoracic procedures, are subjected to significant physiologic alterations when undergoing chest wound closure. Sudden increased intrathoracic pressure at closure decreases venous return from the brain to the heart; consequently the total blood volume in the ridged skull increases, resulting in increased intracranial pressure (ICP). Patients with intact cerebrovascular autoregulation can compensate for these sudden changes by adjusting cerebral blood flow to maintain physiologic ICP. In cases where the autoregulatory mechanisms are compromised, such as stroke or trauma, the ICP can become pathologically elevated. If the patient does not have an ICP monitor, the spike in ICP can go unrecognized, compromising cerebral perfusion pressure and resulting in increased morbidity and mortality.

■ **CASE DESCRIPTION:** Herein we describe a case of pathologic elevations in ICP captured with an ICP monitor at the time of chest wound closure in a patient with delayed chest wound closure after orthotopic heart transplant and comorbid embolic stroke.

■ **CONCLUSIONS:** This case report demonstrates the potential utility of ICP monitoring in patients with impaired cerebrovascular autoregulation with open and manipulated chest wounds from transplant or trauma.

changes in ICP during manipulation of the chest wound or wound closure. Stroke and trauma patients often have difficulty following examinations because of deficit or sedation. In these patients, ICP alterations may be difficult to detect except for once an hour when the nurse transduces the pressure at the external ventricular drain (EVD). ICP monitoring allows for real-time tracking of ICP and can help identify patients who may be going through unrecognized elevations in ICP, especially on chest wound closure, that can further injure the brain and affect recovery.

Open chest management with delayed sternal closure (DSC) is a commonly used technique in open heart surgery when the patient is hemodynamically unstable, has intractable arrhythmias, or develops severe coagulopathy during the procedure.^{1,2} The timing of the DSC is largely determined by risk-benefit analysis as the risk of infection, air and fat embolic events, and need for hemodialysis due to substantial requirement

for vasoconstrictive medications increase with time to DSC.³ Patients undergoing open chest management (OCM) for a prolonged period of time are subject to various physiologic alterations on wound closure. One such phenomenon is the reduction in venous return to the heart from the jugular veins as intrathoracic pressure increases. This can lead to elevations in ICP if the cerebrovascular autoregulatory mechanisms are damaged or absent.

Normally, cerebral blood flow, and thus ICP, are preserved despite moderate variability in cerebral perfusion pressures.⁴ However, in cases where the cerebral autoregulation is damaged, such as stroke or traumatic brain injury, these mechanisms are compromised. Therefore patients with an open chest wound undergoing OCM may be especially susceptible to pathologic fluctuations in ICP during wound closure if they have any comorbidity that has altered their normal cerebrovascular autoregulatory mechanisms.

Perioperative ischemic stroke is a well-known complication in up to 13% of orthotopic cardiac transplantation.⁵⁻⁸ We present a case illustrating massive changes in ICP during DSC in a patient who experienced an ischemic stroke during heart transplant surgery.

CASE DESCRIPTION

A 68-year-old male with congestive heart failure underwent an orthotopic cardiac transplant with DSC secondary to excessive intraoperative bleeding. The cardiac transplant surgeons originally planned to

do DSC on postoperative day (POD) 1 in the operating room as this patient was now immunosuppressed and at a particularly high risk of infection with OCM. Immediately postoperatively, the patient was found to have left upper extremity paralysis but was awake and following commands on the right upper and bilateral lower extremities. Computed tomography (CT) of the brain demonstrated a large right middle cerebral artery (MCA) fat embolus with extensive cerebral edema and midline shift (**Figure 1**). Because the patient had significant comorbidities, he was initially treated conservatively with

standard medical management for cerebral edema. By POD 2, the patient progressively became more lethargic and repeat imaging showed worsening edema and impending herniation. Decompressive right hemicraniectomy was performed emergently on POD 2 after orthotopic heart transplant. There was adequate decompression with resolution of midline shift (**Figure 2**). Immediately after decompression, the patient was able to localize in his right upper extremity with flexion in bilateral lower extremities and a plegic left upper extremity. An EVD with an ICP monitor was placed with opening pressure of 13 mm Hg. The catheter used was an Integra Camino Flex ventricular catheter (Integra Lifesciences, Plainsboro, New Jersey, USA), which allows continuous, simultaneous cerebrospinal fluid drainage and ICP monitoring by the sensor located at the tip of the catheter. The ICP trend line was then stored in the monitor for up to 24 hours. Physiologic ICP values range from 8–15 mm Hg. From placement of the Camino EVD with fiberoptic ICP monitor until sternal closure was completed, the patient had normal ICP ranging from 9–15 mm Hg.

In light of the stroke, the subsequent hemicraniectomy, and ongoing coagulopathy, the DSC had to be delayed beyond POD 1 as planned. Once the coagulopathy was adequately resolved, the cardiac transplant surgeons elected to undergo DSC; however, the patient was deemed too critically ill for transfer to the operating room. Thus the cardiac transplant surgeons performed DSC at the bedside in the intensive care unit with an anesthesiologist present to assist. Fentanyl and propofol were used for sedation. During and after the procedure the patient remained intubated with neither a significant change in peak airway pressure nor a change in his already minimal oxygen requirement. There were no episodes of hypotension, and cerebral perfusion pressure remained above 60 mm Hg until the sternum was closed. During the procedure the patient's head was kept at least 30 degrees elevated over his body.

Immediately after the last step of the procedure, the actual sternal closure, ICP spiked into the 70s and despite aggressive medical management, pressure was sustained at that level for approximately

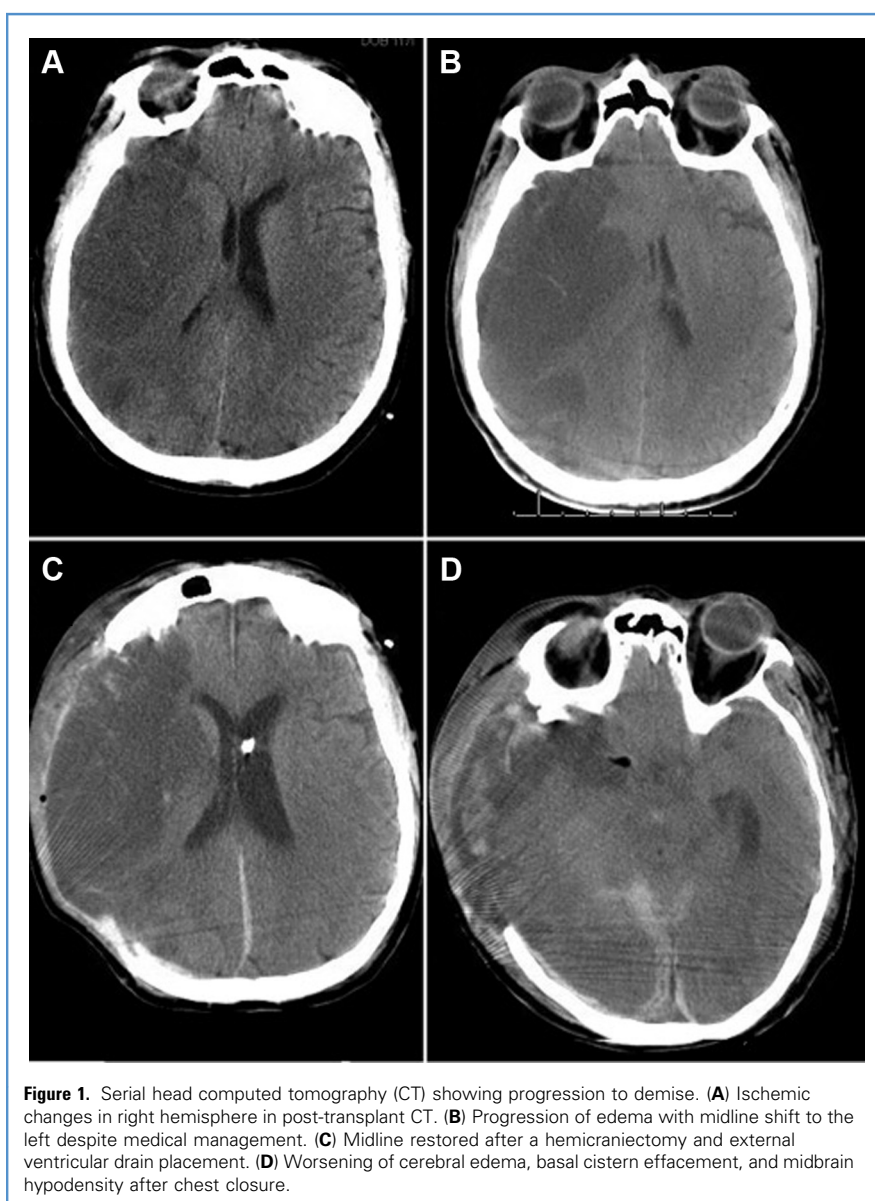


Figure 1. Serial head computed tomography (CT) showing progression to demise. (A) Ischemic changes in right hemisphere in post-transplant CT. (B) Progression of edema with midline shift to the left despite medical management. (C) Midline restored after a hemicraniectomy and external ventricular drain placement. (D) Worsening of cerebral edema, basal cistern effacement, and midbrain hypodensity after chest closure.

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