Case Report

Permissive hypotension in traumatic brain injury with blunt aortic injury: How low can we go?

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Abstract

With an ever-increasing incidence of high impact collisions, polytrauma is becoming increasingly common. Patients with traumatic brain injury (TBI) may require urgent surgical intervention along with maintenance of an adequate mean arterial pressure (MAP) to maintain cerebral perfusion. On the other hand, patients who sustain blunt aortic injuries (BAI) have a high mortality rate, many of them succumbing to their injury at the site of trauma. Surgery has been the mainstay of the management strategy for the remaining survivors. However, in recent years, the paradigm has shifted from early operative management to conservative treatment with aggressive blood pressure and heart rate control, serial imaging, and close clinical monitoring. When TBI and BAI coexist in a patient, it becomes crucial to maintain the MAP within a narrow range to prevent secondary insult to the brain as well as to prevent aortic rupture. We present the management of a case of TBI with traumatic aortic pseudoaneurysm, which required stringent monitoring and maintenance of hemodynamics during decompressive craniectomy.

Key words: Aortic aneurysm, brain injuries, craniocerebral trauma, vascular system injuries

Introduction

In today's world with high speed collision, number of patients with complex trauma injuries has increased. These patients present with unique challenges as several injuries may require contrasting physiological management. Patients with traumatic brain injury (TBI) require maintenance of vital parameters within strict limits to prevent secondary brain insult. It is well-recognized that low mean arterial pressure (MAP) reduces cerebral perfusion resulting in poor outcome in patients with severe TBI.[1] Blunt aortic injury (BAI) with pseudoaneurysm <3 cm have been managed conservatively in our center. They can be managed by maintaining permissive hypotension (MAP of 40-50 mmHg or a systolic blood pressure [SBP] ≤80) and serial imaging. Here, we report a case of TBI with BAI (pseudoaneurysm >4cm), where an urgent decompressive craniectomy was warranted. BAI was managed conservatively both in intra-operative and postoperative period with permissive hypotension without causing neurological deficit in long-term. Our research revealed that no such case has yet been reported in the literature.

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Case Report

A 27-year-old male patient presented in our emergency department with a history of road traffic injury. On admission, his Glasgow coma score (GCS) was E1V1M3, heart rate (HR) was 85 beats/min and blood pressure (BP) was 135/82 mmHg. His computed tomography (CT) scan showed right temporoparietal subdural hematoma (SDH) with multiple underlying contusions [Figure 1]. He was immediately intubated with the help of injection propofol 60 mg intravenous (IV) and cricoid pressure, with manual in-line stabilization and cervical collar *in situ*. A surgical decompressive craniotomy was planned. Patient also had left sided chest trauma and an intercostal drain (ICD) was inserted, that drained around 200 ml of blood. CT scan and angiography of the chest revealed a pseudoaneurysm of the aortic arch, proximal to the origin of subclavian artery, measuring 4 cm in diameter [Figures 2 and 3]. The low GCS caused the evacuation of SDH to take precedence over BAI and an immediate evacuation was planned.

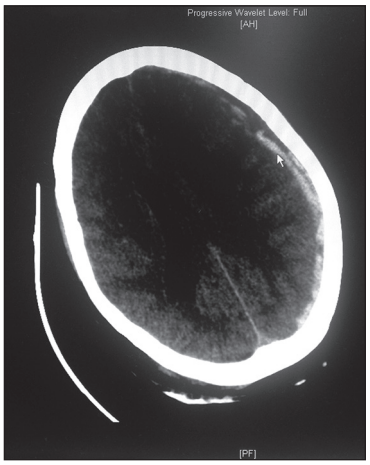

Figure 1: Non-contrast computed tomography head of the patient showing fronto-temporal subdural hematoma (white arrow)

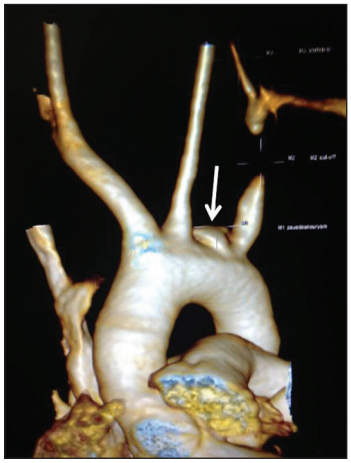

Figure 2: Three-dimensional reconstruction of the computed tomography scan showing heart and the great vessels (white arrow shows the aortic arch aneurysm)

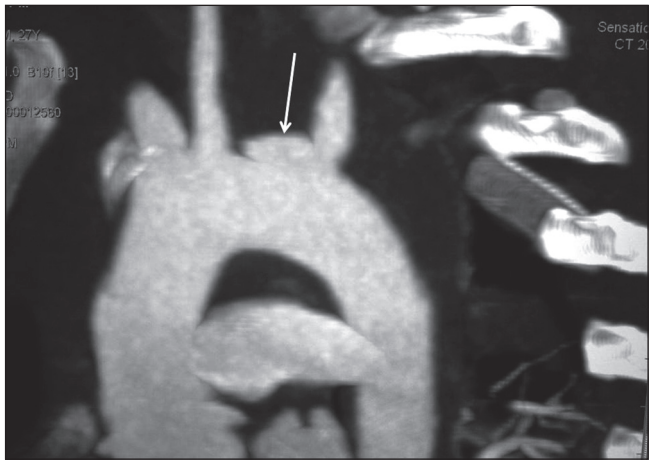

Figure 3: Computed tomography angiograph of the patient showing a small aneurysm proximal to the origin of the left subclavian artery (white arrow)

The patient was taken to the operating room after obtaining high-risk consent. He was hemodynamically stable. Anesthesia was induced with propofol 80 mg and fentanyl 100 μg and muscle relaxation achieved with vecuronium 4 mg IV. A central venous catheter (central venous pressure [CVP]) was placed in the right internal jugular vein and right radial artery was cannulated for invasive BP and serial arterial blood gas monitoring. Anesthesia was maintained with isoflurane in a mixture of air and oxygen (2:1) along with intermittent boluses of vecuronium and fentanyl. IV fluid therapy was guided by CVP, urine output and blood loss, and hemodynamics were meticulously maintained. Our aim was to maintain SBP around 100 and MAP of 60 mmHg to maintain cerebral blood flow and also to prevent the aortic aneurysm from rupture. For permissive hypotension propofol infusion was started at 50-60 mg/h.

Blood loss was approximately 1 L, which was adequately replaced. Surgery lasted for 4 h, at the end of which the patient was shifted to the Intensive Care Unit (ICU) for elective ventilation. Controlled ventilation was established with a tidal volume of 400 ml, respiratory rate of 15 breaths/min, positive end-expiratory pressure of 6 cm H_2O and a FiO_2 of 0.5. He was sedated using continuous infusion of midazolam (2 mg/h) and fentanyl (50 μg/h). The aortic pseudo aneurysm was managed conservatively with frequent imaging for aneurysmal size. Esmolol infusion (50-100 μg/kg/min) required for a period of 22 h on the first postoperative day to control unexplained tachycardia, following which the MAP and HR remained stable. Intracranial pressure (ICP) monitoring was carried out and a microdialysis catheter was also inserted to measure the lactate/pyruvate (L/P) ratio on day 2 of ICU. Both ICP and the L/P ratio remained within acceptable limits throughout the ICU stay (<20 cmH_2O and <25, respectively). Mean cerebral perfusion pressure was maintained around 70 mmHg. The ICD was removed on day 5 and ICP monitoring and microdialysis catheter were removed on day 7 of ICU. The patient was tracheostomized and discharged with a GCS of E4VTM6 and a resolving aneurysm on postoperative day 10.

Discussion

With extensive modernization and an exponential growth in motor vehicles, polytrauma has become increasingly common. Polytrauma patients form a special cohort since the management of one injury may interfere with the optimal treatment of the other, creating a dilemma for the treating physician. Such a situation arises, as in our patient, where TBI and BAI coexist, which poses a challenge to the anesthesiologist during surgical management of either of the injuries.

Traumatic brain injury is a major cause of disability and mortality worldwide. Secondary injury in TBI is described as the consequence of physiological insults such as ischemia,

reperfusion and hypoxia to areas of "at risk" brain in the period after the initial injury.[2] It has been widely studied and reported that hypotension and hypoxia have deleterious effects on the outcomes of TBI patients. The IMPACT study suggested that hypotension and hypoxia were significantly associated with unfavorable 6-month outcome.[3] Moreover, the duration of intra-operative hypotension was also inversely associated with functional outcome.

BAI have a higher mortality rate with the majority of the patients succumbing at the site of trauma. With improvement in pre hospital care the number of patients with BAI reaching hospital has increased. The patients may be managed by early surgery or nonoperative therapy depending on the severity of injury. The principle of nonoperative management revolves around differentiating BAI into minimal aortic injury (MAI) and severe aortic injury (SAI). Patients with MAI (usually lesions <3 cm) have a low risk of progression and aortic rupture and are better managed conservatively with aggressive negative inotropic and antihypertensive therapy, routine serial imaging and close clinical observation. SAI mandates surgical or endovascular repair in most of the cases if the risk-benefit ratio is favorable. Surgeons at our institution follow similar guidelines for management of aortic aneurysms. In our patient the management of TBI took precedence over BAI, therefore we decided to manage the BAI conservatively and operate the SDH. The pseudoaneurysm, although large (>4 cm), was managed conservatively in our patient.

According to the recent retrospective analysis of BAI by Caffarelli *et al.*,[4] aortic injuries, especially pseudoaneurysms are different from nontraumatic atherosclerotic aneurysms, because in the majority of the trauma patients, the underlying pathology does not exist. This Stanford Philosophy implies that nonoperative management may be more successful in traumatic aortic aneurysms as compared to pathological ones. However, application of permissive hypotension in patients with neurological involvement must always be carried out in the presence of monitoring of cerebral function, such as cerebral oximetry. At our institution, intra-operative cerebral oximetry is not used, but the use of ICP monitors and cerebral microdialysis catheters in the postoperative period are frequent.

Our patient, however, presented with the unique situation of TBI with BAI (pseudoaneurysm measuring 4 cm). Although according to the aneurysmal size the patient required surgery, the surgical management of TBI was more urgent. The management of the intra-operative period required meticulous monitoring of BP so that we could maintain cerebral perfusion without rupturing pseudoaneurysm.

Conclusion

Patients with BAI with large size pseudoaneurysm may be managed intra-operatively with permissive hypotension in the presence of appropriate monitoring of cerebral function such as cerebral oximetry. However, more cases are required to be studied for validation of this hypothesis and formulation of guidelines.

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