

Anesthetic Management of Penetrating Nail Injury Brain: A Case Report

Abstract

Penetrating injury brain is common after accidental falls, sharp projectile injury, suicidal attempts etc. We present an unusual case of accidental penetrating nail injury brain in a 19 year old male patient presenting for emergency craniotomy. This case report highlights the difficulties faced in the anesthetic management of patients presenting with penetrating injury brain.

Keywords: Cerebral perfusion pressure; Emergency craniotomy; Nail injury; Penetrating brain injury

Abbreviations: ASA: American Society Anesthesiologist; CBF- Cerebral Blood Flow; MAC: Minimum Alveolar Concentration; RSI: Rapid Sequence Induction; MAR: Mean Arterial Pressure; ICU: Intensive Care Unit; CPP: Cerebral Perfusion Pressure; CMRO₂- Cerebral Metabolic Oxygen Requirement

Introduction

Penetrating injury brain commonly occurs as a result of accidental fall into shrapnel, suicidal attempts, low velocity projectile injury during construction work, high velocity projectile during bomb blasts etc [1-3]. Usually low velocity injuries have a good prognosis. Surgical extraction of such projectiles can be successfully done, when they are partly impacted to the skull bone but their blind removal carries the risk of secondary brain injury [4]. This case report illustrates the anesthetic challenges encountered in the surgical management of a patient presenting with a penetrating nail injury brain at a tertiary care hospital.

Case Report

A 19 year old male patient presented in the emergency department of Gauhati Medical College and Hospital with a 2 day history of traumatic nail injury brain. Patient was referred to this institute for urgent neurosurgical intervention. The patient had a post traumatic loss of consciousness, now complaining of severe headache with frequent nausea vomiting and weakness of the right upper limb. Household repairs were going on in his when he accidentally slipped and fell head on a nail. He was immediately rushed to the nearby health facility, from where he was referred to Gauhati Medical College. Patient was examined by the emergency neurosurgery and neuroanaesthesia team.

He had a Glasgow Coma Score of 13/15, no other external injuries were noted. A nail head entry wound was visible on his scalp with bloody discharge around it (Figure 1). His right upper limb power was 3/5 as per Medical Research Council grading with poor hand grip in comparison to the opposite side with no other focal neurological deficit. Routine blood investigations, coagulation profile were normal. Skull X-ray showed a vertically oriented radio-opaque nail penetrating through the frontal bone of skull and into the brain parenchyma (Figure 2). Computerized

Case Report

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tomography scan revealed a 7cm long nail penetrated through the frontal bone and into the left frontal lobe of brain (Figure 3). It was evident that nail required immediate surgical removal. An informed consent was obtained from the patient for the operative procedure & general anaesthesia. Patient had received Tetanus Immunoglobulin 250 IU and Inj Tetanus Toxoid I.M in the emergency department.



Figure 1 & 2: Showing entry wound over the scalp and in x ray.

After being shifted to the neurosurgical operating room Standard American Society of Anaesthesiologists (ASA) monitors were attached and baseline vitals were recorded. 18G IV cannulation was done in each forearm and warm normal saline was started slowly. Patient received broad spectrum antibiotics as per hospital protocol. Inj Levepiracetam 1000 mg IV and Inj Fentanyl citrate 60 micro grams IV were also administered. Patient was preoxygenated for 3 minutes with 100 % oxygen.

Rapid sequence induction (RSI) was done with 80 mg Propofol and muscle relaxation was achieved with 40 mg of Inj Rocuronium Bromide. Airway was secured with a 8 millimeter internal diameter reinforced endotracheal tube which was fixed at 22cms after bilateral air entry was checked to be equal and adequate. Anaesthesia was maintained with oxygen:air in 1:2 ratio (flow 2 L/minute) with 0.4 to 0.8% Isoflurane. Patient was ventilated with a tidal volume of 6-8ml/kg, with a respiratory rate of 12 to 14 breaths perminute. Divided doses of Inj Rocuronium Bromide were used intraoperatively. After antiseptic dressing & draping, local infiltration of the surgical site with 20ml of 1% lignocaine was done. A 9 cm curved incision was made across the nail head to raise a scalp flap (Figure 4).

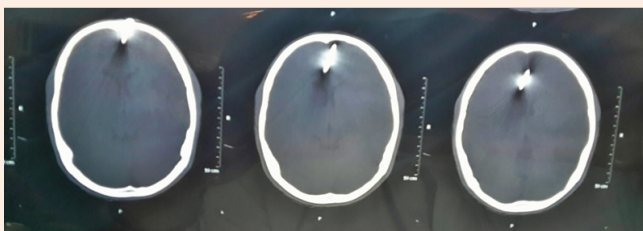


Figure 3: CT SCAN (bony window) showing intra brain parenchymal extension of the nail.

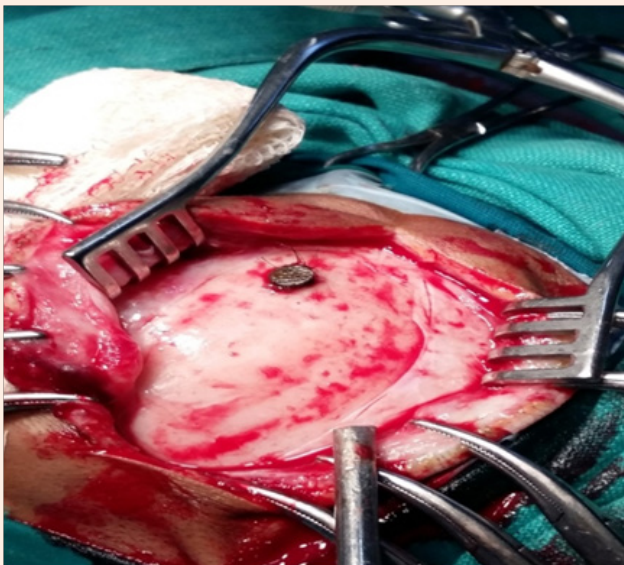


Figure 4: Nail lodged head visible through the skull bone.

A small 4cm diameter craniotomy trephine was created adjoining the nail to widen the interphase between it and the skull. Bone adjoining the nail was gradually nibbled off, the nail became loose, the circular piece of bone was removed and under direct vision using a plain forceps, the nail head was stabilised and gradually pulled out of the brain parenchyma (Figure 5 & 6). The surgical field was dry and the only bleeding was noted from the scalp wound. The nail measured 7 centimeters (Figure 7). The surgical site was irrigated copiously & gently with warm normal saline from a 10 ml syringe. The opening in the skull was covered by the trephined piece of bone & the wound was closed in layers.

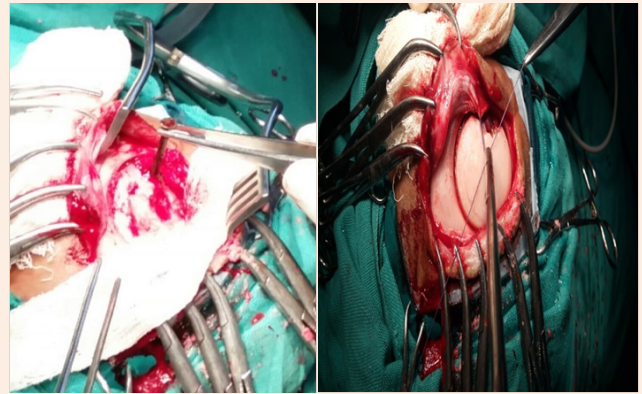


Figure 5 & 6: Intraparenchymal nail extraction and craniotomy closure.



Figure 7: The lodged nail.

Patients received Inj Paracetamol 1000mg slows IV intraoperatively. Intraoperatively patients was normocapnic and no episodes of hypoxaemia ($SaO_2 < 92\%$) developed. Throughout the procedure we maintained a mean arterial pressure; of greater than 70 mm Hg. At the end of the procedure patient was reversed adequately and later shifted to neuro surgical intensive care unit. The operative procedure was completed within 60 minute. Patient received 1200 ml of isotonic saline intraoperatively; with an estimated blood loss of 200ml. Intraoperative urine output was 80 ml. The patient recovered uneventfully over the next 10 days and he was subsequently discharged on the fourteenth postoperative day. He regularly followed up at the neurosurgical outpatient department.

Discussion

Penetrating nail injury brain is a emergency situation which requires prompt medical management & neurosurgical intervention. Following penetrating injury to the brain, pre-hospital care involves patient stabilization & securing the penetrated object to prevent further brain injury. If the patient comes in a life threatening situation, adequate resuscitation with thorough assessment of any form of neurological deficit is important before any surgical intervention to remove the penetrating object is attempted [5]. In addition to nails, penetrating injury brain has also been reported with other objects like screw driver, scissors, knives, crowbar, spears etc [4,5]. Usually penetrating objects do

not intracranial bleed at the time of impalement but due to high velocity of impact have a tamponade effect. But they may bleed once that tamponade effect ceases to act i.e. during their extraction [6]. The visible nail head in our patient guided the surgeons in the successful extraction. Some authors report using this tamponade effect of projectile injury by delaying extraction, hence allowing a stable clot formation around the object [7].

Our patient presented 2 days after the injury & we planned urgent removal as he was having focal neurodeficit and signs of raised intracranial pressure. Our other immediate concerns were infection & seizures. Majority of the penetrating nail injury brain cause minor neurological deficit [3,8]. The injuries are limited locally along the nail track [9]. Our patient complained of mild weakness in the right upper limb post trauma. Hypoxia and hypotension correction prevent secondary brain injury & are the chief priorities during the perioperative management of patients with penetrating brain injury. A single episode of associated hypotension increases morbidity and doubles mortality [10].

Surgical blood loss and anesthetic drugs further predispose these patients to hypotension. Both invasive and non invasive monitoring are important and should include electrocardiogram, pulse oxymetry, capnography, temperature, urine output, invasive arterial pressure monitoring, arterial blood gas analysis and perioperative blood glucose monitoring are important. Central venous access may be useful for administration of vasoactive drugs and central venous pressure monitoring during resuscitation. Intracranial pressure (ICP) monitoring has gold standard recommendation in patients presenting with traumatic brain injury. Anesthetic goals in these patients include maintainance of adequate cerebral perfusion pressure & intracranial pressures, proper plane of anaesthesia & perioperative analgesia, observing normocapnia & hypoxia prevention during mechanical ventilation, euglycaemic state with hypothermia and hyperthermia prevention.

Analgesia is most essential, as any noxious stimuli may cause sympathetic surge increasing cerebral blood flow (CBF), cerebral metabolic oxygen requirement (CMRO₂), and ICP. All volatile agents upto 1 minimum alveolar concentration (MAC), usually reduce CMRO₂, cause cerebral vasodilation, increase cerebral blood flow (CBF) further increasing ICP. Inhalational anesthetics reduce cerebral responsiveness to carbon dioxide. Sevoflurane below 1 MAC is best suited for neurosurgical interventions. Use of inhalational nitrous oxide is obsolete in neurosurgical cases. Intravenous induction agents on the other hand reduce CMRO₂, CBF, and ICP.

Propofol induction in these patients causes significant hypotension which may further reduce cerebral perfusion pressure (CPP). Neuromuscular blocking drugs allow controlled ventilation & prevent coughing or straining reflexes in the operated patient. Rocuronium has minimal cardiovascular effects with no histamine releasing property & is the muscle relaxant of choice in neuroanaesthesia. Patient positioning with extremes of flexion or rotation of the head impedes venous drainage from brain, further raising the ICP. Controlled ventilation aims at maintaining normocapnia, while preventing hypoxia. Hyperventilation to maintain a PaCO₂ value of 30-35 mm Hg is

useful in patients with intracranial hypertension (ICP≥20mm Hg) with jugular venous oxygen saturation estimation as a measure of adequacy of cerebral oxygenation. A reduction in PaCO₂ reduces cerebral blood volume and ICP due to cerebral vasoconstriction. But hyperventilation may cause cerebral hypoperfusion, further causing ischaemia [11]. Adequate perioperative hydration along with the use of vasopressors to maintain a CPP of 60mm Hg maintains CBF and tissue oxygenation. Maintainance of perioperative euglycaemic state is essential. 20% Mannitol may be used perioperatively for control of acute increase in ICP, but may cause intravascular volume depletion, hypotension, renal complications, and hyperkalaemia and rebound intracranial hypertension [12].

Induced hypothermia (32-35.8) C is known to have neuroprotective action demonstrated in animal studies. Moderate hypothermia effectively reduces ICP and reduces CMRO₂ and is useful in the management of raised ICP refractory to medical therapy [11] I.V. Barbiturates although lower ICP but cause significant cardiovascular instability & are indicated in cases of refractory intracranial hypertension. Postoperative intensive care unit (ICU) based care is necessary for these patients. Our patient received prophylactic anticonvulsants and antibiotics prior to induction. We used Propofol for induction in our patient as we went for RSI. Our patient was kept normocapnic, normothermic, nonhypoxic intraoperatively maintaining a MAP of >70mm Hg, with adequate planes of anaesthesia and analgesia. His perioperative course was uneventful & recieved intensive care unit (ICU) based care postoperatively.

Conclusion

Careful planning & preparation with maintainance of anaesthetic goals is necessary in the anaesthetic management of patients with penetrating brain injury. Perioperative management continues to focus on prevention of secondary brain injuries and maintenance of CPP. Although advanced medical treatment has significant reduced mortality, still no single treatment modality alone has been shown to improve outcome in these group of patients.

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