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Late-onset Seizure and Left Hemiparesis after Unusual Craniocerebral Penetrating Injury by a Rusty Sickle: A Case Report

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Abstract

BACKGROUND: Penetrating head injuries are a significant public health problem in worldwide, with an estimated 35.000 civilian deaths annually. Patients that survive to reach the hospital require rapid triage and imaging evaluation. This case report describes a patient with late-onset seizures and left hemiparesis after unusual craniocerebral penetrating injury by a rusty sickle.

CASE PRESENTATION: A 26-year-old man reported that he had a sickle stabbed into the right-side parietal area. On admission, he had no neurological deficits. The rusty sickle was broken off just above the skull and did not protrude from the scalp. Computed tomography showed that the knife blade was in the parenchyma without underlying tract hematoma. Surgery was performed after 2 h of admission. Post-operatively, he recovered with no neurological deficit. After 16th days postoperatively, he was complaining of seizure and left hemiparesis. CT scan with contras showed edema and hypodense lesion at the right side of hemisphere. A course of intravenous phenytoin 100 mg/8 h and ceftriaxone 2 g/day was initiated. Physical therapy was done on the patient for 2 weeks.

CONCLUSION: Traumatic brain injury (TBI) is the result of energy being transferred from an object to the human skull and underlying brain. Post-traumatic epilepsy is a common complication and can occur as early or late manifestations related to penetrating TBI. Prophylactic treatment of post-traumatic seizures (PTS) is currently not routinely recommended beyond 1 week following head injury (role of antiseizure prophylaxis). Phenytoin is the most rigorously tested AED for PTS

Introduction

Penetrating the skull and brain injuries are relatively uncommon, representing about 0.4% of head injuries [1]. Approximately 70–90% of these victims die before arriving at the hospital, and 50% of those who survive reaching the hospital die during resuscitation attempts in the emergency department. Low velocity penetrating brain injuries are rare because the skull usually provides an effective protective barrier. Penetrating head injuries generally occur due to violence, including self-inflicted injuries, unexpected events, worker accidents, etc. A knife blade is the most common agent, but wooden objects, scissors, firearms, and nail guns have recently been featured. Transcranial stab wounds made with a knife or sickle mainly produce a classic slot skull fracture and underlying tract hematoma and often cause severe neurological deficits. We treated a patient with a self-inflicted penetrating skull injury at the right parietal portion [2].

Case Report

A 26-year-old man reported that he had recently had an argument with his friend over a bird with a sickle stabbed into the right-side parietal area. After 15 h, the patient was referred to Dr. Moewardi General Hospital due to lack of neurosurgeon. On admission, he had no neurological deficits. The rusty sickle was broken off just above the skull and did not protrude from the scalp. The wound was irrigated, and mannitol 25 g, phenytoin 100 mg, anti-tetanus serum 1500 IU, and ceftriaxone 2 g were administered intravenously. Computed tomography showed that the knife blade was in the parenchyma without underlying tract hematoma (Figure 1). Cerebral angiography could not be performed. However, venous flow around the sickle was prolonged because of tamponading. Surgery was performed after 2 h of admission under general anesthesia after obtaining informed consent and making careful preparations. His head was fixed in

the supine position, and a lazy "S" incision was made around the wound. After reflecting the skin and muscle, the knife was found firmly lodged in the parietal bone a few centimeters to the midline's right. 4 burr holes around the penetrating site: proximal, distal, medial, and lateral were made. A trapezoid bone flap pattern was also made. After preparations had been made for the massive hemorrhage management from the cerebral arteries, the anterior and posterior edges of the residual bone were carefully drilled, and the knife was removed with one bite of the surrounding bone to prevent any unnecessary movement of the knife. Finally, the knife insertion point was identified as the right hemisphere. We found that small amounts of subarachnoid hemorrhage (SAH) and subdural hemorrhage were evacuated, and massive bleeding from the Intracerebral could be stopped by cautery bipolar. The penetrating area was debrided by H_2O_2 and 2-liter NaCl 0.9%. The dural defect was not closed. The bone flap was not restored because of the risk of infection (Figure 3). Post-operatively, he recovered quickly with no neurological deficit. After 16th days postoperatively, the patient was complaining of seizure and left hemiparesis. On admission, he was afebrile and white blood cell count of 8.600/ml. Because we suspected brain abscess, the patient was subjected to a head computed tomography scan with contrast but showed edema and hypodense lesion at the right side of hemisphere (Figure 4). A course of intravenous phenytoin 100 mg/8 h and ceftriaxone 2 g/day was initiated. Physical therapy was done on the patient for 2 weeks. Follow-up results showed good progression within 2 days. After the discharged hospital, he could do his daily activity without limitation.

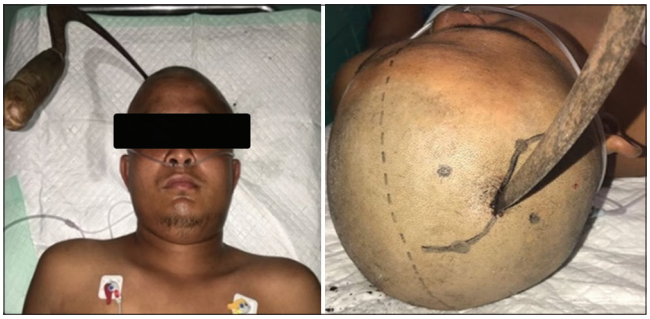

Figure 1: Skull radiographs on admission, showing a sickle penetrating deep into the right parietal region near the midline and incision design lazy "S"

Discussion

Traumatic brain injury (TBI) is the result of energy being transferred from an object to the human skull and underlying brain. The penetrating object has kinetic energy proportional to the projectile equal to the mass times the square of its velocity (Ek = ½ mv^2). Most non-bullet penetrating objects, such as nails or knives, impart minor damage to the skull and brain because they have less kinetic energy to transfer on impact. Wounds resulting from penetrating TBI (pTBI) can be classified into five categories: tangential, penetrating, perforating, ricochet, and careening. Tangential wounds are projectile injuries that do not penetrate through the skull. Penetrating injuries are those where the projectiles penetrate the skull and brain parenchyma and remain there. Perforating injuries, the most devastating of all penetrating traumatic brain injury (pTBI), usually the result of high-velocity projectiles or those fired at close range, penetrate the skull and then exit at a site distal from the entry point (Figure 5) [2], [3].

When surgery performed more than 12 h after the initial injury, there is an increased risk for infectious complications. No data exists to support the advantages of craniectomy over craniotomy, though recent data from the military suggesting improved mortality with early decompression. The placement of drains, either subgaleal, epidural, or both, are common and have shown in at least one analysis to result in a trend toward fewer postoperative complications [2].

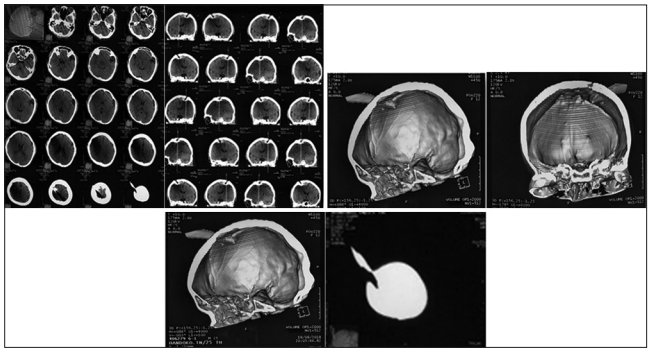

Figure 2: Computed tomography scans on admission, showing that a sickle is located in the right parietal region but no apparent tract hematoma

There is an extensive list of delayed complications that can arise from pTBI. Vascular complications are the most common and most devastating, occurs in 30% of transcranial penetration cases: aneurysm in 15%, carotid-cavernous fistula in 7%, other arteriovenous fistulae in 4%, occlusion in 4%, transection in 3%, and severe vasospasm in 3% [4]. SAH can result in delayed cerebral vasospasm and delayed cerebral ischemia. The highest vasospasm incidence occurs between days 5 and 11 but can occur anywhere between 3 and 21 days after injury. The risk of infectious complications increases following the 1st week post-injury, with recent post-surgical infectious complications occurring at a rate of 5–23% [2]. Brain abscess due to the retained foreign body usually develops 3–5 weeks after trauma [5]. Most of the contaminating organisms are skin flora, such as *Staphylococcus epidermidis*, but *Staphylococcus aureus* and Gram-negative bacilli are common pathogens. Effective debridement can reduce the risk of infection from such contaminants. Lin *et al*. reported antibiotic coverage with vancomycin,

gentamycin, and metronidazole for 2–3 days, while recent US military guidelines recommend cefazolin for 5–7 days and note high rates of multidrug-resistant organisms such as *Acinetobacter [6].* If dirt, debris, or clothing contaminates the wound, expansion to anaerobic coverage with metronidazole is recommended [7].

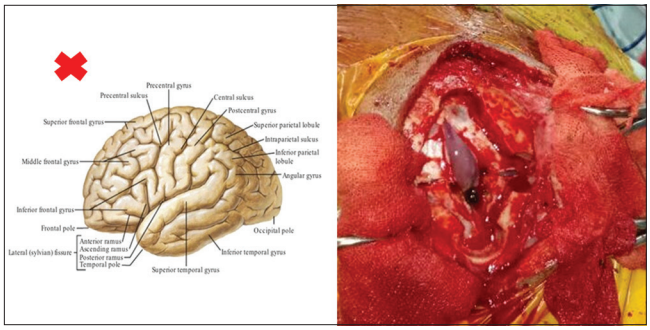

Figure 3: Illustration picture, red cross showing penetration area at the right-side gyrus precentralis [3]

Moreover, cerebrospinal fluid leaks are a complication arising from the Dura's laceration where basilar skull fractures or orbitonasoethmoid injuries are most prevalent. Post-traumatic epilepsy is a common complication and can occur as early or late manifestations related to pTBI. Early seizures, occurring within the 1st week after injury, are often generalized and occur in 2–8.9% of pTBI patients. Late-onset seizures, occurring up to 15 years following the injury, can present as focal seizures or focal seizures with secondary generalization in as many as 50% of patients. Early seizures following TBI can be prevented with anti-convulsant therapy typically instituted during the 1st week following pTBI even though there is no evidence that early anti-convulsant therapy reduces the incidence of late posttraumatic seizures, nor does it affect functional outcome [2].

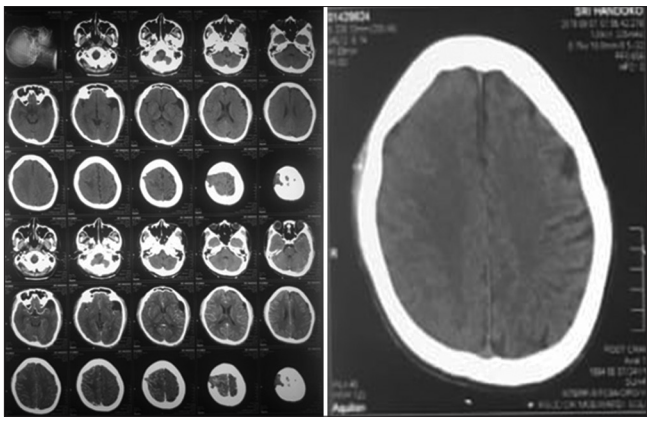

Figure 4: Computed tomography scans with contrast, after seizure and left hemiparesis, day 16th after the surgery

Early damage following TBI is due to interruption in blood flow and oxygenation, which activates an ischemic cascade. Calcium ion (Ca^{2+}) influx leads to glutamate release, and decreased glial uptake by specific glutamate transporters further elevates glutamate levels. Excess glutamate binds to NMDA receptors and promotes Ca^{2+} and sodium (Na^{+}) influx, which further activates enzymes responsible for cell damage. Upregulation of Ca^{2+} also increases Nitric Oxide (NO) synthesis, causing further oxidative damage. Intracellular accumulation of Ca^{2+} promotes mitochondrial sequestration of Ca^{2+} stores, the elevation of which can impair oxidative phosphorylation and cause the production of reactive oxygen species (ROS). ROS production can lead to further brain cell damage and underlies seizure formation [8].

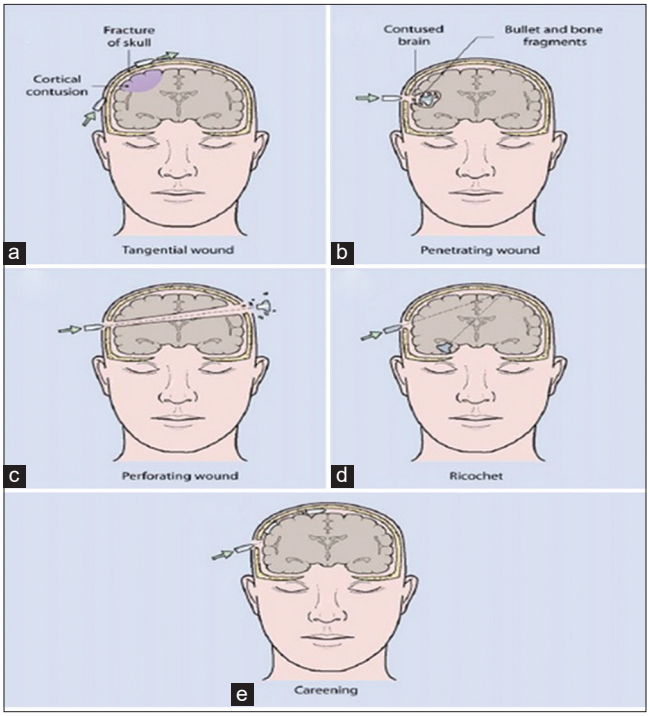

Figure 5: (a-e) Patterns of penetrating head wounds

Late seizures are thought to be caused by cortical damage due to iron deposition and elevated excitatory amino acid levels. The post-injury self-recovery process causes reorganization of brain circuitry, and the newly created connections cause hyperexcitability between the new synapses, particularly in the dentate gyrus, which further increases

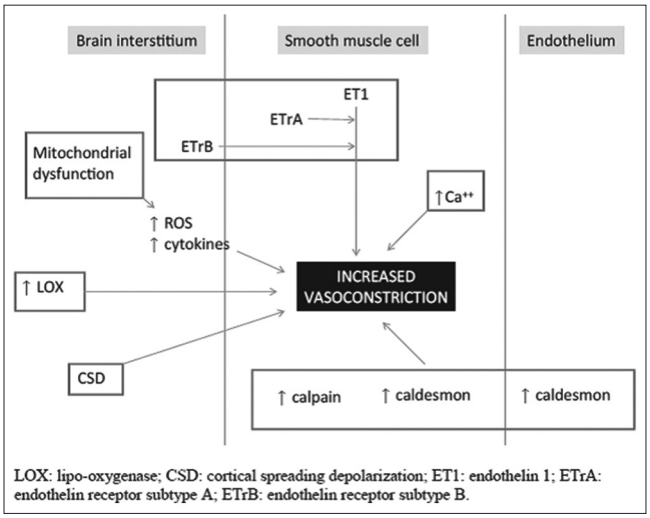

Figure 6: Mechanism of post traumatic cerebral vasopasm

hippocampal excitability. Blood-brain barrier disruption and inflammation following TBI leads to further damage and seems to influence PTE [8].

Anti-epileptic drugs (AEDs) for preventing post-traumatic seizures (PTS) have been evaluated to varying degrees by randomized controlled trial. Evidence supports that Phenytoin and Carbamazepine are useful for prophylactic treatment of Early Post Traumatic Seizures not for Late Post Traumatic Seizures. Carbamazepine requires monitoring of serum drug levels and has a large interaction profile with other medications. Levetiracetam is a newer generation of AED with several advantages over the older generation, including decreased drug interaction and side-effect profile. It requires no loading dose or typical serum monitoring. Beyond the scope of Early PTS prophylaxis, for patients who have experienced two or more PTS events, it is generally agreed that these patients should be treated with an AED. A trial of withdrawing from AED use may be considered after 2 years with no seizure activity. In these cases of drug-resistant epilepsy, surgical treatment may be considered as an option to attain seizure control and potentially reduce the amounts of AEDs required [9].

Furthermore, post-traumatic vasospasm (PTV) is most detected on the large basal intracranial arteries: the internal carotid, the middle cerebral artery (MCA), and the basilar artery and less frequently on posterior cerebral arteries. The delayed type is related to the presence of SAH and mimics aneurysmal vasospasm. PTV is associated with traumatic SAH; its onset can be delayed by several days after the hemorrhage [10]. Delayed vasospasm in TBI is thought to relate to hemoglobin degradation and subsequent inflammatory cascade [11]. Age under 30 seems a critical risk factor for PTV. The identification of ischemia due to PTV is very difficult. Some authors used delayed clinical deterioration with ischemic symptoms; other studies employed delayed low densities on computed tomography (CT) scans. PTV seen on angiography was deemed responsible for delayed clinical deterioration and focal ischemia in many case reports of adults and infants. CT low densities had only a sensitivity of 41% in identifying patients with severe vasospasm, and 19% of patients with low densities on CT scans had no or only mild spasm on angiography [10].

PTV suspicion on transcranial doppler (TCD) should be confirmed by direct measurement of vessel diameter and cerebral blood flow (CBF). For this purpose, cerebral angiography has long been used, and a significant correlation was found between the lowest CBF and the highest MCA velocity in patients during the period of vasospasm. Nowadays, because of its very good accessibility and high performance, CT has become the first-line technique to confirm vasospasm with CT angiography showing the narrowing of vessels and assess the ischemic [11].

Mechanisms of PTV after TBI – pathophysiology (Figure 6)

Calcium dysregulation

After TBI, excitotoxicity leads to an increase in Ca2+ and Na+ influx. An increase in cytosolic calcium leads to the stimulation of extracellularly regulated kinase pathways and modulates the cytoskeletal actin organization leading to hypersensitivity to contractile stimuli [10].

Endothelin 1

In the last 15 years, endothelin 1 (ET1) has become the most studied mechanism. Its two receptor subtypes, ETrA and ETrB, have distinct brain locations under physiological conditions: pericytes of microvessels in the thalamus, hypothalamus, and distinct neural cell types. The shifts in both cellular receptor locations are one of the major factors inducing vasospasm after moderate to severe brain injury. The TBI-induced upregulation of ET1 (synthesis and utilization) seems to be primarily responsible for changes in CBF, in so far as the interaction between ETrA and ET1 makes vasospasm occur. Indeed, after TBI, the increased transcription of protein-encoding ET1 in pericytes located in microvessels lasts about 4 h, coinciding with the early increased vasoreactivity period [10].

Contractile proteins

Calponin and caldesmon are actin-binding proteins involved in the regulation of smooth muscle tone during blood vessel contraction. *In vitro* observations after TBI have found a migration of calponin from the cytosol to a location subjacent to the smooth muscle cell membrane, leading to a significant increase of calponin expression results in a sustained vasocontractility and hypoperfusion. Caldesmon is increased in both smooth muscle cells and endothelial cells *in vitro*. These proteins remain elevated in smooth muscle cells for more than 48 h, whereas their concentration decreases in endothelial cells [10].

Products of cerebral metabolism

The normal brain is known to be a significant source of a variety of arachidonic acid metabolites, synthesized by both cyclooxygenase and lipoxygenase (LOX) pathways. LOX generated by human glial cells

eicosanoids is highly vasoactive. They are also potent mediators of increased vascular permeability. In so far as mechanical perturbation of cell membranes leads to the release of arachidonic acid from phospholipidic membranes, vasospasm can occur this way [10].

Dysfunctions in mitochondrial mechanisms and oxidative stress

They can be considered as factors inducing vasospasm. Indeed, posttraumatic mitochondrial dysfunction is deemed to lead to a lower CBF after acute TBI.

Cerebral infarction was found in the territories supplied by the vasospastic arteries, but severe PTV did not consistently induce focal ischemic areas on the late CT scans. A correlation was found between the main side of subarachnoid blood, the localization of severe PTV, and focal ischemia [10].

Conclusion

Pre-operative and post-operative angiography is mandatory to document the vascular damage when the foreign body is located just beneath the major arterial or venous system. Tetanus immunization, anti-convulsant, and vigorous antibiotic treatment should be added to surgery. Surgery is performed under 12 hours to remove contaminated foreign bodies, repair vascular or Dural damage, and drainage intracranial mass lesions. PTV is suspected on daily TCD; the diagnosis is best confirmed by angiography, CT angiography or magnetic resonance angiography, and perfusion and ischemic consequences by perfusion CT or magnetic resonance imaging. These techniques may guide treatment decisions: balloon or chemical angioplasty with nimodipine or other appropriate agents. Extensive prospective studies are needed to assess the incidence more precisely, time course, risk factors, and best treatments of PTV.

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