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Case Report

Blast injury causing extensive brain injury and elevated skull fracture

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ABSTRACT

Blast injuries are caused by rapid chemical transformation of a solid or liquid into a gas resulting in a high-pressure wave exceeding the speed of sound. We discuss a case 28 year gentleman who sustained severe traumatic brain injury and elevated skull fracture secondary to a blast of refrigeration gas cylinder.

1. Introduction

Blast injuries are caused by rapid chemical transformation of a solid or liquid into a gas, the gas expanding radially outward as a high-pressure wave exceeding the speed of sound.[1] Blasts injuries are responsible for about two-thirds of the war related traumatic brain injuries.[2,3] In present article, we report a case of young man who sustained fatal traumatic brain injury and elevated skull fracture following a blast in refrigeration gas cylinder discuss the underlying mechanism of such injuries.

2. Case report

A 28 year male patient sustained had injury following a blast of refrigeration gas cylinder. He came to the emergency about 3 h after the injury. He was unconscious since the time of injury. He had multiple episodes of vomiting and bleeding from the left ear. There was no history of seizures or oral bleed. At the time of examination in the emergency room his GCS was E1V2M1. Pupils were mid-dilated and sluggishly reacting. His respiration was shallow. Immediately endotracheal intubation was performed and the patient was put on

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elective ventilation. There was a large lacerated would over left temporo-parietal region with scalp avulsion and palpable fracture of the underlying bone. There was active bleeding from the wound. After stabilizing the patient a CT scan brain was performed. CT scan brain showed a large comminuted, compound fracture of left temporo-parietal bone with elevated fractured fragments (Figs. 1 and 2). There was massive scalp swelling and underlying contusion involving the left temporal and parietal lobe with intraventricular hemorrhage (Fig. 1). The wound debridement was performed and hemostasis was achieved. The patient was kept on elective ventilation, anti-edema measures, broad spectrum antibiotics and anti-convulsants were started. The patient did not respond to the treatment and had fatal outcome.

3. Discussion

Rapidly outwards expanding gas from the point of detonation displaces the surrounding medium (air or water) causing an immediate rise in pressure, creating a blast waves that subsequently dissipating over distance and time.[4–7] Highly compressed air on the leading edge of the blast wave creates a shock front with outward movement of ambient air (the "blast wind") following this shock front.[1] The mechanisms of injury from blast have been divided into primary blast injury (blast wave-induced changes in atmospheric pressure), secondary blast injury (from objects projected from the blasts into people) and tertiary blast injury (from people being propelled from the blast).[1,4,7] Blast waves, interacting with the body, will transfer energy at interfaces between tissues of differing acoustic impedance thus leading to cellular disruption, soft tissue destruction and bone micro-fractures.[8] Following blasts, the blast pressure wave exerts forces mainly at air–tissue interfaces within the body, and pulmonary, gastrointestinal, and auditory systems are being at the greatest risk.[2] It has been postulated that the blast wave–head interaction is a complex high rate flow phenomenon that is affected by the oncoming blast wave parameters and also by the geometric and anatomical variations of the human head.[9] Blast waves generated in the explosions impinge on the head–brain complex and induce mechanical pressure pulses in the brain resulting in traumatic brain injury.[9] Several theories have been proposed to explain the potential mechanisms of the blast induced traumatic brain injury and include vascular transmission,[10,11] direct transmission of the blast wave through cranium[12,13] and flexure of skull.[14,15] Blasts within 1 m of the brain can produce skull fractures and extensive epidural, subdural, subarachnoid, and intracerebral hemorrhages findings similar to the damage found after severe direct closed head injury.[2,16–19] Fractures following as a consequence of primary blast injury are usually considered a marker for a lethal injury and these can be due to direct coupling of the blast wave into the tissues.[20,21] It has been shown in animal models that the bones can be fractured by the blast wave alone, particularly when placed in close proximity (<50 cm) to the seat of the explosion.[22] Elevated skull fracture has been defined as the fracture in which fractured fragment is elevated above the level of the intact skull are rare injuries[23,24] and was first described in the Edwin Smith Surgical Papyrus, over 5000 years ago.[25] Elevated skull fractures are usually compound injuries and caused by a long sharp-edged heavy weapon or object; and an outward component is imparted by lateral pull of weapon while retrieving it, or by rotation of the head after impact, or while transferring the patient.[23–29] Tangential injuries which sliced off a portion of

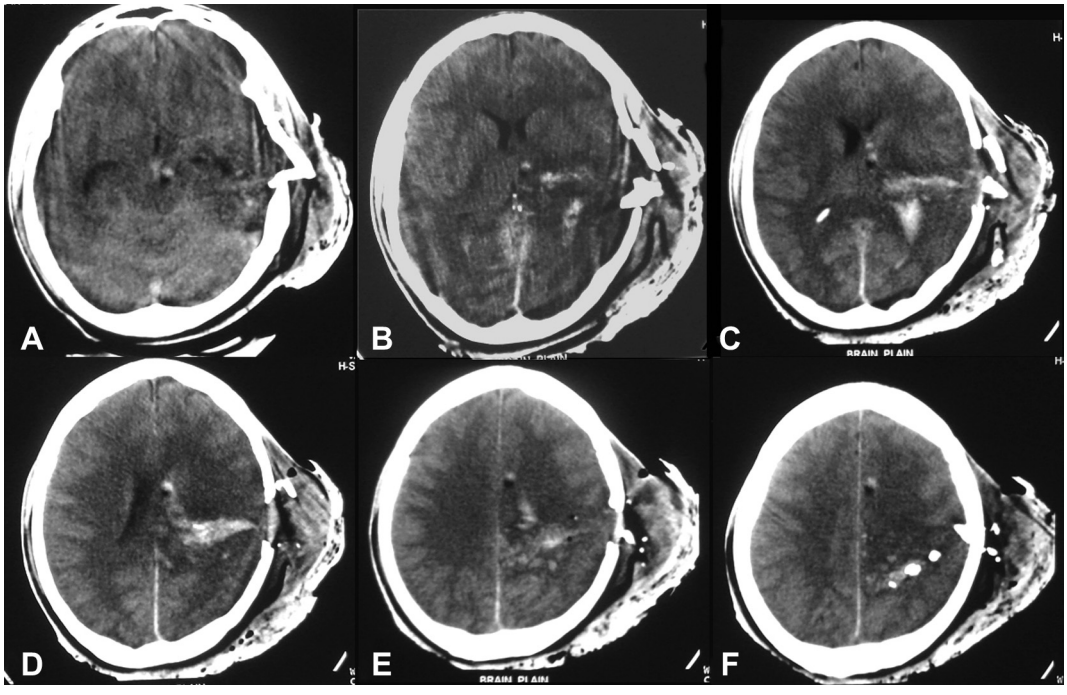

Fig. 1 – CT scan brain plain showing extensive scalp swelling elevated bone fragments, extensive underlying contusion and intraventricular hemorrhage.

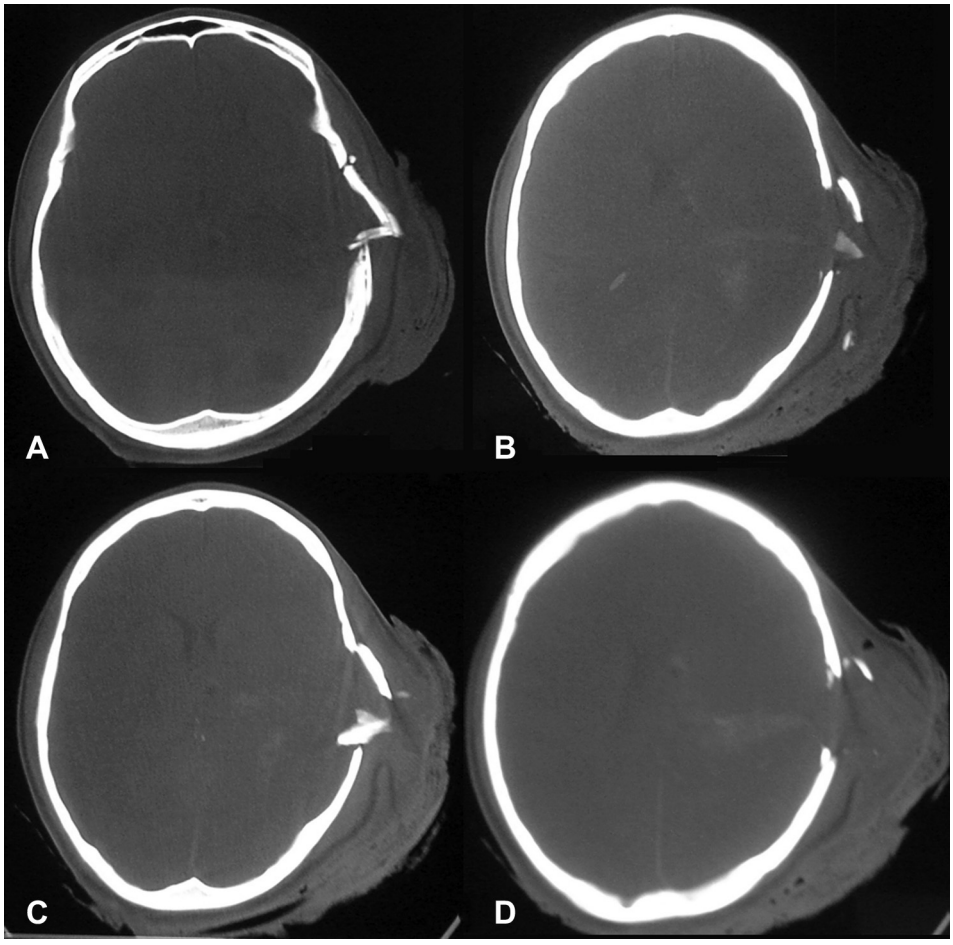

Fig. 2 – CT scan brain bone window showing extensive scalp swelling and elevated bone fragments.

the scalp, skull and the underlying dura and brain were mainly responsible for compound elevated skull fractures.[25,29]

4. Conclusion

In present case multiple mechanisms were responsible for a fatal traumatic brain injury (compound elevated skull fracture, multiple underlying contusions and intraventricular hemorrhage) and elevated skull fracture.

Conflicts of interest

All authors have none to declare.

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