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Traumatic asphyxia during stadium stampede

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Abstract

Objectives: To present a series of cases of survivors and non-survivors of traumatic asphyxia from a single mass casualty incident in Zimbabwe and a review of the literature.
Design: Descriptive case review.
Setting: Parirenyatwa Hospital is a tertiary referral 1 000 bed teaching hospital in Zimbabwe.
Results: Survivors (n = 4) displayed the classic signs of traumatic asphyxia of conjunctival haemmorhages, petechial blue-purple discoloration of head and neck and neurological findings of confusion or unconsciousness and convulsions. Non-survivors (n = 12) showed more varied signs but all showed petechiae and with a history of being crushed. On-site resuscitation and triage was absent, reducing the chance of identifying potential survivors at the scene.
Conclusion: The outcome in traumatic asphyxia is improved by rapid restoration of ventilation and circulation. The epidemiology of traumatic asphyxia in Zimbabwe is unknown but the conditions predisposing to it are present. Closer integration between hospital and pre-hospital services will permit better management of major trauma patients and mass casualty events.

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Introduction

On the 9 July 2000 there was a stampede at the National Sports Stadium in Harare after police threw CS tear-gas into the crowd on the terraces to quell disturbances during a soccer match. This resulted in 13 deaths, four admissions to intensive care and 35 others injured. Since then, several similar incidents have occurred across Africa with more or less numbers of casualties. We describe below the clinical cases and the *post mortem* findings.

The term '*traumatic asphyxia*' is commonly used to describe the clinical signs of craniocervical cyanosis and ecchymosis, conjunctival haemorrhage petechial haemorrhage in skin and mucous membranes of the chest and head. However, the definition refers to the mechanism of injury in which external mechanical forces (trauma) prevent ventilatory movements, thereby causing asphyxia (hypoxia). Death may ensue if the asphyxia persists or from associated injuries. A wider spectrum of clinical signs may, therefore, be expected. A wide variety of terms

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are in current use, synonymous with traumatic asphyxia: compression, mechanical, entrapment asphyxia etc.

Ollivier first described the condition he called '*masque ecchymotique*' in the *post mortem* examination of stampede victims of riots in Paris in 1837.[1] Perthes, however, is credited with the first complete description of the condition in 1900.[2] Most early descriptions, like the one presented, were of people crushed while stampeding during riots, fires etc. A wide variety of crush situations have since been reported producing the condition of 'traumatic asphyxia'.

Clinical Record

Case 1. A 15 year old boy arrived unconscious in the Casualty department. He had been crushed during the stampede at the stadium. The specific details were not available. A Glasgow Coma Scale (GCS) of 5/15 was recorded on admission. He was haemodynamically stable and self-ventilating. He was intubated and ventilated promptly. Clinical findings were those of conjunctival haemorrhages, facial oedema. He was extubated and discharged to the general ward the next day. On discharge he could recollect the event.

Case 2. A 32 year old male accountant also crushed in the stampede. When seen in Casualty, he had a GCS of 6/15. He was intubated and ventilated. He also had conjuctival haemorrhages, facio-cervical oedema, proptosis and orbital ecchymosis. Plain chest and skull X-ray were normal and CT scan the next day showed cerebral oedema. He was extubated and discharged from ICU on day four. During his ward stay he continued to complain of headaches and sore throat. He had an episode of breathlessness, which appears to have resolved without major intervention. An ENT surgeon who diagnosed subglottic stenosis followed him up and he had tracheal dilatation six and eight weeks later. He had (amnesia) no recollection of the event and has continued to have problems with his short term memory which has affected his ability to function in his job.

Case 3. A friend brought a 30 year old female patient with a known history of asthma into the Casualty department in private transport. She had marked facial oedema and conjunctival haemorrhages, proptosis and exophthalmos. GCS was 6/15 and she, had bruises on the abdomen but no evidence of other injuries. She was sedated, intubated and ventilated on 40% oxygen. Chest X-ray and, plain skull X-ray were normal. A CT scan of the head done the next day showed diffuse cerebral oedema. Ophthalmological assessment of the eyes was normal except for the conjunctival oedema and haemorrhage. On day three the patient developed bronchospasm which was treated with intravenous hydrocortisone and aminophylline and nebulised salbutamol. The patient was extubated on day eight and discharged to the High Dependency Unit. She did not remember of the event. Recovery was uneventful.

Case 4. A 24 year old female patient was admitted to Casualty with a depressed consciousness level, agitation, bruises on the chest and conjunctival heamorrhages. She was sedated, intubated and ventilated. She was extubated the next day and discharged to the HDU and then a ward on day four. Recovery was uneventful and she had no recollection of the incident.

Table I: The deaths.

Name	Age	Sex	Injuries	Cause of Death
Case I	20 years	F	Marked chest and abdominal bruising, surgical emphysema, fractured 1st and 2nd ribs, haemopheumothorax, bilateral lung contusions.	Multiple Injuries
Case II	6 years	M	Petechiea on the lungs.	Asphyxia
Case III	27 years	M	Bruising on the chest, forehead and left hip. Lacerated right pulmonary hilum, subpleural petechiae. Pneumothorax.	Asphyxia Pneumothorax
Case IV	17 years	F	Bilateral pleural petechial haemorrhages. Extensive cervical and thoracic paravertebral haematoma.	Asphyxia
Case V	27 years	M	Peripheral cyanosis.	Asphyxia
Case VI	16 years	M	Haemorrhagic pulmonary oedema, subpleural haemorrages.	Asphyxia
Case VII	10 years	M	Subpleural petechial oedema.	Asphyxia
Case VIII	41 years	M	Haemorrhagic pulmonary oedema.	Asphyxia
Case IX	25 years	F	Haemorrhagic pulmonary oedema.	Asphyxia
Case X	25 years	F	Bruises on both lower legs and trunk. Petechial haemorrhages on pleural surfaces.	Asphyxia
Case XI	19 years	M	Abrasions on left leg and right chest. Fractured 3 ribs on right side. Haemorrhagic pleural oedema.	Asphyxia
Case XII	30 years	M	Multiple bruises and abrasions on extremities and truck. Petechial haemorrhages on pleura surfaces of both lungs.	Asphyxia
Case XIII		M	*Post mortem* report could not be traced.	Asphyxia

In eight patients the only *post mortem* findings were of petechial haemorrhages with or without pulmonary oedema.

There was some superficial bruising in some to suggest trampling or crushing against some surface. In three patients there were other injuries severe enough to be lethal, as in cases I, II, and IV. One patient had fractured ribs, a non lethal injury. In the last patient the *post mortem* report could not be traced although the entry in the register was of asphyxia only.

Pathophysiology.

The accepted mechanism of injury is that a sudden severe compressive force is applied to the upper abdomen and or chest. Forces as great as several thousand kilograms have been described. The high intrathoracic pressure prevents venous return resulting in high venous pressures that cause petechial haemorrhages.[3] In the presence of an unobstructed airway the chest wall is relatively compliant and the pressures do not rise excessively despite high compression forces. The classic signs of 'ecchymotic mask' may not then appear, as is the case in the fatalities presented.[4,5] The static blood desaturates resulting in the cyanotic appearance.

However, the airway may be obstructed by the glottis closing (as in coughing) during the 'fright' response in which the victim '*takes a deep breath, holds it and braces himself for the impending disaster*'.[6] In this situation the chest wall is less compliant to the compressive forces and the very high intra thoracic pressures are achieved, leading to retrograde venous flow from the atrium and superior *vena cava* resulting in ecchymosis of the head, face, neck and upper thorax.

The external jugular vein has valves at its entrance to the subclavian and at about 4cm above the clavicle. The valves do not prevent regurgitation.[7] The internal jugular vein has valves just above its inferior bulb, 2 to 3 cm above its junction with the subclavian vein. This is believed to be competent at pressures of up to between 45 to 100mmHg, in about one third of people.[8] The internal jugular vein valves have a protective effect on the brain, while the external jugular vein takes the full force. In addition the cranium is thought to produce counter pressure that limits intra cranial haemmorhage.[9]

The high intra abdominal pressures collapse the inferior *vena cava* and prevent retrograde flow into the lower trunk or legs. The clinical signs of '*masque ecchymotique*' can be produced by the other conditions such as epilepsy, severe vomiting, coughing, sneezing, severe asthma, obstetric labour etc. Children can sustain severe pressure with relatively little signs of injury due to their compliant tissues.[10] Traumatic asphyxia must be distinguished from positional asphyxia. This is the adoption of a position, voluntary or forced, that mechanically interferes with ventilation such as the 'head-down', prone position with hand and feet tied at the back or falling into confined spaces.[11] The proviso is that it must be impossible to change position to extrinsic factors (e.g. coercion) or intrinsic factors such as alcohol, drugs etc.

All the patients, including those admitted to Intensive Care, had their first medical attention in the hospital. In the patients that were certified dead no cardio-respiratory resuscitation had been attempted. The injuries of most of the victims may have been treatable if emergency services had been readily available. The worst injured were two whose injuries were fractured 1st and 2nd ribs, bilateral pulmonary contusions and haemopneumothorax (Case I) and ruptured pulmonary hilum and pneumothorax (Case III).

Although traumatic asphyxia is often a *post mortem* diagnosis, the circumstances in which it occurs are often strongly suggestive. The mortality of traumatic asphyxia is related to the duration of compression, associated injuries and the rapidity with which ventilation, oxygenation and restoration of perfusion take place. Therapy is generally supportive and prognosis is good. Patients with specific injuries may be more serious, requiring directed therapy.

Table II: Summary of organ injury in traumatic asphyxia.

Neurological	**Brain:** oedema micro haemorrhages loss of consciousness, disorientation, coma without spinal inury, amnesia, mental dullness, convulsions. **Eyes:** conjuctival petechiae or echymosis, exophthalmos, proptosis, orbital oedema visual loss. **Periphral nerves:** bachial plexus inhury, paraplegia. **Autonomic:** hyperpyrexia.
Cardiovascular	**Cardiac:** myocardial contusion, electrocardiographic changes, pericardial effusion. **Vessels:** venous stasis, capillary slugging.
Respiratory	**Oral and nasal:** pharyngeal haematoma, lingual and palatine oedema epistaxis. **Trachea:** laryngeal oedema, haemorrhages and hymoptysis. **Lungs:** contusions, oedema and haemorrhages, surgical emphysema, pneumothorax.
Gastro-intestinal	**Oesophagus:** haematoma and heamatemesis. **Stomach and intestines:** mucosal bleeding.
Renal	**Kidneys:** haemoturia and albuminuria.
Musculoskeletal	**Muscle:** haemorrhagic infiltrates in axillary, pectoral upper back muscles. **Bone:** periostial haemorrhages, fractures especially ribs, sternum and clavicle.
Ear	Tinnitus, hearing loss, petechiae and haematoma on tympanic membrane, internal and external meatus. Drum perforation CSF leak even in the absence of drum perforation,
Dermatological	**Colour:** blue-red to blue-black discolouration from ecchymotic blood with sparing of pressure areas. **Integrity:** abrasions, lacerations, oedema.

Discussion

There is little experimental work on traumatic asphyxia. Most of the progress in our understanding and management is through analysis and reporting of clinical cases. The cases we report occurred in the context of a mass gathering. The epidemiology of traumatic asphyxia in Zimbabwe is

unknown but there are many situations predisposing to traumatic asphyxia and mass casualties: bus or train crashes; church, sport or political mass gatherings; domestic and occupational environments where crushing may occur. Analysis and management policies of these situations may reduce or even prevent injuries in future.[12]

The medical management of mass gatherings has become topical because of the heightened fear and expectation of terrorist attacks and the consequent potential for mass casualties.[13,14] Emergency services in the public sector in Zimbabwe are generally very basic.[15] The typical ambulance crew is trained only to ferry patients (scoop and run) rather than resuscitate and carry. In situations where traumatic asphyxia is present or suspected, immediate resuscitation is essential.[16] Integration between the pre-hospital and hospital emergency service would facilitate participation of hospital teams in the pre-hospital care and triage on-site and transportation of patients. The response of one central hospital in Harare to the same incident, as a receiving hospital, is reported elsewhere.[17]

Conclusion

Presented are 17 patients who presented following a stampede at a football stadium. All had the diagnosis of traumatic asphyxia: 13 died and four survived. Of the 13 only two had associated injuries severe enough to cause death. It is debatable whether the remaining 11 would have had a different outcome, but if the diagnosis of 'traumatic asphyxia' is accepted, the literature suggests that early and aggressive therapy may have helped some patients.

References

1. Ollivier d'Angers. Relation medicale des evenements survenus au Champs-de-Mars le 14 Juin 1837. *Annales d'hygeine* 183;8:485.
2. Fred HL, Chandler FW. Traumatic asphyxia. *Am J Med* 1960;29:508-17.
3. Ely SF, Hirsch CS. Asphyxial deaths and petechiae: a review. *J Forensic Sci* 2000; 45(6):1274-77.
4. Williams JS, Minken SL, Adams JT Traumatic asphyxia reappraised. *Ann Surg* 1968;167:384-92.
5. Sklar D, Baark B, McFeeley P, Osler T, Marder E, Demarest G. Traumatic asphyxia in New Mexico: a five year experience. *Am J Emerg Med* 1988;6(3):219-23.
6. Ming-Chung Lee, Sing-Seng Wong, Jaw-Ji Chu, *et al*. Traumatic asphyxia. *Ann Thoracic Surgery* 1991;51:86-8.
7. Giorgio Gabella E, Chapter 10. Cardiovascular. In: Grey's Anatomy. Williams PL, editor. 38th ed. Churchill Livingstone, 1995:1578-9.
8. Sum-Ping ST (John). Internal jugular valves: competent or incompetent? *Anaesthesia and Analgesia* 1994:78:1039-40.
9. Sandiford JA, Sickler D. Traumatic asphyxia with severe neurological sequelae. *J Trauma* 1974;14(9):805-9.
10. Nishiyama T, Hanaoka K. Traumatic asphyxia in a child. *Canad J of Anaesthesia* 2000;47(11):1099-02.
11. Belviso M, De Donno A, Vitale L, Introna F. Positional asphyxia: reflection on two cases. *Am J Forensic Med Pathology* 2003;24(3):292-7.
12. De Angelo D, Schurr M, Birnbaum M, Harms B. Traumatic asphyxia following stadium crowd surge: stadium factors affecting outcome. *World Med J* 1998;97(9):42-5.
13. De Lorenzo RA. mass gathering medicine: a review. *Prehospital and Disaster Med* 1997;12(1):68-72.
14. Milsten AM, Maguire BJ, Bissell RA, Seaman KG. Mass gathering medical care: a review of the literature. *Prehospital and Disaster Med* 2002:17(3):151-62.
15. Dickinson G. Emergency medicine in Zimbabwe. *Canad J Emerg Med* 2000;2(3):
16. BBC News. Fighting for life at Ellis Park. 15 April 2001. (photo) *'Medics administered treatment on the pitch'*. Last accessed 8th July 2004. http://news.bbc.co.uk/1/hi/programmes/from_our_own_correspondent/1278246.stm
17. Madzimbamuto FD. A hospital response to a soccer stadium stampede in Zimbabwe. *Emerg Med J* 2003;Nov 20(6):556-9.