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Traumatic asphyxia: a rare clinical syndrome

創傷性窒息：一個罕見的臨床綜合徵

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Traumatic asphyxia is a rare clinical syndrome characterised by cyanosis, oedema and petechial haemorrhage of the face, neck and upper chest due to the sudden, transient and severe thoraco-abdominal compression trauma. The huge increase in intra-thoracic pressure caused by the compression constitutes all the symptoms. Although mortality and morbidity due to traumatic asphyxia can happen, most of the patients survive without any sequel. Herein, we report three cases of traumatic asphyxia assessed in our emergency department. (Hong Kong j.emerg.med. 2014;21:185-188)

創傷性窒息是由突然、短暫和嚴重的胸腹擠壓傷引起的罕見臨床綜合徵，特點是面、頸及上胸部的發紺、水腫和點狀出血。擠壓造成胸內壓力大幅增加導致所有的症狀。雖然創傷性窒息可能造成傷亡，多數病人生存，並且沒有任何後遺症。我們在此報告在我們急診室診治的三例創傷性窒息。

Keywords: Cyanosis, retrobulbar hemorrhage, purpura, thoracic injuries

關鍵詞：紫紺、眼球後出血、紫癜、胸創傷

Introduction

Traumatic asphyxia is a rare clinical syndrome. Although the exact mechanism is controversial, traumatic asphyxia patients presented to the emergency department after sudden, high energised thoracoabdominal compression trauma.[1-3] Cutaneous petechial haemorrhage of the face, neck and upper chest occurred due to the compression. Additionally, cervicofacial subcutaneous oedema, cyanosis and blue discolouration of face and neck can be seen depending on the contributing effect of 'fear response' including deep aspiration and closure of the glottis. The huge increase in central venous pressure caused by the compression of the chest transmitted to the head and neck capillaries. The pressure to the eye, and hence subconjunctival haemorrhages and bulbar injuries are almost always due to the relative lack of tissue support around this area.[1,4] Although most of the cases survive with good prognosis, some patients died or were left with a morbidity in spite of the clinical treatment modalities. Herein, we report three cases of traumatic asphyxia assessed in our emergency department.

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Case 1

A 30-year-old automobile mechanic presented to the emergency department after his chest was being crushed under an automobile while running repairs. He was fully conscious and cooperative on admission. Petechial and purpuric haemorrhages were seen at his upper chest and neck. His right shoulder was deformed and his face was found intensively oedematous and cyanotic (Figure 1). Despite the lack of a direct blow

to the head, a scalp defect sized approximately 3 cm was found at his occipital area during physical examination. On eye examination, eyes of the patient were protruded, the pupils were dilated and the response to light was absent bilaterally (Figure 2). On visual acuity examination, he could count fingers at 3 meters with his right eye; the left eye had total visual loss. Head, neck and chest tomography was done immediately. There were bilateral retrobulbar haematomas in the eyes. In the thorax, there was a 10 cm diameter mediastinal haematoma, and fractures of the sternum. Chest tomography also showed fracture of the left clavicle, left scapula, left and right first ribs and a non-displaced T10 vertebra fracture. Since the patient had severe proptosis and visual loss in the left eye, lateral canthotomy was performed in the emergency department for orbital decompression and the patient was admitted to the ophthalmology clinic for further treatment. His vision improved in the following days and the patient was transferred to orthopaedic surgery clinic for his fractures. He was discharged from the hospital with good recovery.



Figure 1. Petechia and blue-red discoloration on the upper chest region.

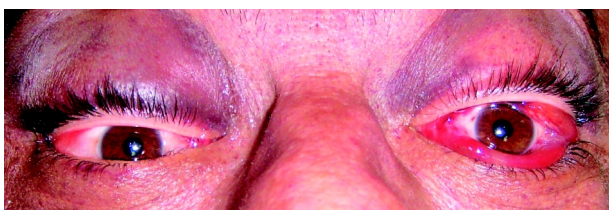


Figure 2. Protrusion of the left eye.

Case 2

A 56-year-old repairman presented to the emergency department after his chest was being crushed under a fishing boat while running repairs. He was conscious, however did not answer any questions. The oxygen saturation was 84% by pulse oximetry and his upper chest, neck and head were cyanotic. His head was found intensively oedematous and the eyes protruded. However the pupils were isochoric, their size and the response to the light were normal. Open crush injury was found at his right foot. Head, neck and chest tomography was performed for the patient. His head tomography was normal. However the chest tomography revealed multiple rib fractures, pulmonary contusions and pneumothorax. The radiographic imaging of the right foot revealed multiple tarsal and metatarsal bone fractures. The patient regained full consciousness in the emergency department and he was admitted to the intensive care unit for respiratory care. He was transferred to orthopaedic surgery clinic for his fractures after resolution of the pulmonary problems. He was discharged from the hospital with good recovery.

Case 3

An 18-year-old man presented to the emergency department after being crushed under a tractor accidentally in the farm. He was conscious but tachypnoeic with a respiratory rate of 30/per minute and oxygen saturation of 89% by pulse oximetry. Bilateral exophthalmia and limited eye movements were found in the examination. Petechial and purpuric haemorrhages and cyanosis were seen at his neck and face. Head, neck and chest tomography was performed for the patient. The head tomography was normal. However, orbital screening revealed right retrobulbar haematoma and chest tomography revealed sternocostal detachment and minimal pneumopericardium (Figure 3). Since his visual acuity was found to be normal, no intervention for retrobulbar haematoma was performed. His oxygenation and general appearance returned normal in the emergency department and he was admitted to the cardiac surgery clinic for further treatment. He recovered with

conservative treatment and did not required invasive surgical intervention. He was discharged from the hospital with good recovery.

Discussion

Traumatic asphyxia is a rare condition which can be seen after a heavy load compressing the thoracoabdominal region, such as being pinned or crushed by a vehicle or heavy machinery, wedging of the body within a narrow space or exposing to a gradual and prolonged crushing force in large and panic crowds.[1-3] Besides, there are a few different examples of the syndrome described following suicide attempts, diving, epileptic seizures, difficult obstetric delivery or just a t-shirt wrapped around thorax.[5] The classical presentation of the patients is blue-red cyanotic discolouration and oedema of the face, neck and upper chest, petechial purpura and subconjunctival haemorrhage. Although the exact mechanism is controversial, the main problem is thought to be the effect of the huge increase in intra-thoracic venous pressure. This pressure is transmitted to the mediastinum and causes reversal of venous flow from the heart through the valveless superior vena cava and other mediastinal veins to produce the cyanotic discolouration.[1] Pressure transmission to the venules and capillaries produces the characteristic cutaneous petechial haemorrhages, subconjunctival haemorrhage and retrobulbar haemorrhage with capillary stasis or rupture. The 'fear response' in which the victims realising that a problem was about to occur and reflexively brace themselves in preparation, by taking a deep breath and holding it and by tightening the accessory chest muscles is also contributory to the increase in the pressure of veins.[5,6] The fear response is necessary for producing clinical features of traumatic asphyxia. Occlusion of superior vena cava or compression of the chest alone fails to produce the clinical symptoms of the syndrome; however obstruction of the airway with simultaneous chest compression reproduces typical clinical appearances in experimental studies.[7]

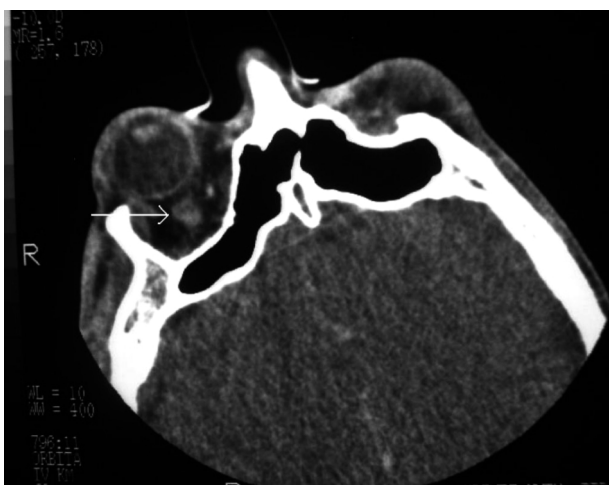


Figure 3. Right retrobulbar haematoma on computed tomography scan.

In case of traumatic asphyxia the lower torso is usually spared. It has been postulated that protection is afforded by the venous valves and also by compression and obliteration of inferior vena cava with fear response. The most commonly associated injuries are pulmonary injuries – including pulmonary contusion, pneumohaemothorax, lung lacerations and rib fractures with or without flail chest. Cardiac injuries are rare. Abdominal injuries including liver and splenic lacerations, gastrointestinal haemorrhage, diaphragmatic rupture, transient microscopic haematuria and proteinuria due to increased venous pressure in the kidneys may occur.[5]

The transmission of the increased pressure to the cerebral venous pressure causes a decrease in brain perfusion and consequent cerebral hypoxic and ischaemic cortical dysfunction and other neurologic sequels without any direct head trauma. Despite the lack of a direct blow to the head, we have found a scalp defect in our first patient and we postulated that the defect was caused by the pressure transmitted to skin surface.

Severe proptosis can occur due to traumatic displacement of orbital fat or retrobulbar haemorrhages caused by ruptured capillaries, sometimes resulting in visual loss. Lateral canthotomy for orbital decompression is an effective treatment for regaining the visual function.[8] We have performed lateral canthotomy in the emergency department for our first patient and his visual function was normal at discharge.

Since the presentation is noticeably serious, long term follow-up of patients who have survived traumatic asphyxia is usually good without significant mortality and morbidity with effective treatment. Management of these patients is supportive, and treatment is aimed at associated injuries. The mainstay of treatment is oxygenation, and elevation of the head of the bed to 30º once the spine has been cleared of injury. Patients should be preferably admitted to an intensive care unit for observation because intubation and mechanical ventilation may be required. If a significant crush injury has been sustained, treatment with fluids, mannitol and bicarbonate must be given as necessary to prevent renal failure secondary to rhabdomyolysis.[5] If there was a prolonged cerebral anoxia due to prolonged compression, undesirable neurologic sequelae can occur. Fatal cases often involved patients who had been compressed for a prolonged period, whereas others had survived large compressive forces for very brief periods of time.[9,10] In fatal cases where prolonged compression was not involved, deaths were caused by associated injuries. All three cases we presented were discharged from hospital with good recovery. They did not require any major surgery except orthopaedic operations.

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