Traumatic asphyxia
創傷性窒息

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Traumatic asphyxia is a clinical syndrome related to cervicofascial cyanosis, petechiae, subconjunctival haemorrhages and neurological symptoms. This syndrome occurs after a transient, severe and compressive blunt thoracic trauma. Here, we presented two cases of traumatic asphyxia. They both had prominent petechiae on upper parts of their bodies. The prognosis was excellent in the boy. Contrary, second case was dead in emergency department due to subarachnoid haemorrhage and intracerebral petechial haemorrhages. Traumatic asphyxia alone does not predict morbidity and mortality. It is the concomitant cardiovascular, pulmonary and neurologic injuries that affect the outcome. (Hong Kong j.emerg.med. 2011;18:339-342)

創傷性窒息是一種臨床綜合徵，表現為頭和面紫紺、瘀斑、結膜下出血和神經症狀。這種綜合徵的發生，源於一短暫的嚴重擠壓性胸廓創傷。在這裡，我們報告兩例創傷性窒息。他們都有明顯的身體上部瘀斑。男孩的預後較優。相反，第二例病人由於蛛網膜下腔出血和腦瘀斑性出血，在急診部宣告死亡。創傷性窒息本身並不能預測併發症和死亡發生率。影響結果的是伴隨的心血管、肺及神經創傷。

Keywords: Cyanosis, fatal outcome, Perthe’s syndrome, purpura, thoracic injury

關鍵詞：紫紺、致命的結果、Perthe’s綜合徵、紫癜、胸外傷

Introduction

Trauma related death is the third leading cause among all age groups after cardiovascular and cancer related death.1 About 33% of blunt trauma patients have a thoracic injury and it is estimated that 25% of traumatic death are secondary to chest trauma.2 After blunt chest trauma, pulmonary and chest wall injuries may occur. Traumatic asphyxia was first described after autopsy studies by Oliver d’Angers in 1837. This syndrome has various other names: acute thoracic compression syndrome, Oliver’s or Perthes syndrome, and ecchymotic mask. It is a rare entity presenting with cervicofacial cyanosis and oedema, subconjunctival haemorrhage, and petechial haemorrhages of the face, neck, and upper chest as a result of a compressive force to the thoraco-abdominal region.3,4 We presented two patients suffering from traumatic asphyxia with one of them being certified dead in the emergency department (ED).

Case 1

A 5-year-old boy was found immobile and stuck for a few hours to part of a parked tractor while he was
playing outdoor. He was brought to our ED by ambulance. His initial Glasgow Coma Scale (GCS) was 15. He was oriented and cooperated with no neurological deficit. His initial examination showed bilateral subconjunctival haemorrhages and petechiae on face (ecchymotic mask) and neck (Figure 1). There was no hearing nor visual loss. Trachea was on the midline and there was no cervical palpable lymph node. Vital signs were stable (BP=100/60 mmHg, HR=90 beats/min, RR=19/min, axillary temperature=36.7°C, Height=110 cm, Weight=19 kg). Urgent jugular venous and carotid Doppler ultrasounds were normal. His chest, cervical and head X-rays as well as his cranial and thoracoabdominal computerised tomography (CT) examinations were all normal. He was hospitalised in thoracic surgery clinic for three days and then he was discharged without any sequel.

Case 2

Our second patient was a 21-year-old man who was brought to our ED by ambulance because of pedestrian injury during traffic accident. He was in asystole on arrival and GCS was 3. Immediate cardiopulmonary resuscitation (CPR) was started but not successful. His initial examination revealed bilateral subconjunctival haemorrhages and petechiae on his face, neck, shoulders and upper chest (Figure 2). His autopsy findings included two occipital linear fractures,

Figure 1. Petechial haemorrhages on face, conjunctivas and neck.

Figure 2. Petechial haemorrhages on conjunctivas and chest.
subarachnoid haemorrhage (SAH) and cortical petechial haemorrhages on frontal lobes. In the thoracic region, there were mild bilateral haemothorax and diffuse superficial petechial haemorrhages on both lungs. Autopsy examination revealed that the lungs and trachea having excessive bloody and foamy liquid. Abdominal and extremity findings were unremarkable.

**Discussion**

Traumatic asphyxia is generally caused by severe chest compression but has also been reported to relate to asthma, paroxysmal coughing, protracted vomiting, and jugular venous occlusion. There are two possible pathophysiologic mechanisms for traumatic asphyxia. Firstly, cervical venous drainage is provided by the internal and external jugular veins. The tissues and structures below the deep cervical fascia (i.e., larynx, trachea, oropharynx) channel through the internal jugular vein while those superficial to it (subcutaneous tissue of the scalp and neck) drain into the external jugular vein. Though the external jugular vein has two pairs of valves, they are not adequate to block flow reversal. As a result, abrupt accelerations in pressures of venules and capillaries cause petechial haemorrhages. The brain, the deep soft tissues of the neck and the airway are partially protected against reversal of blood flow by the venous valves and are thus less likely to be affected.

The second mechanism in the pathophysiology is thought to be glottis closure. Closure of glottis is believed to occur as a response to fear of accident. Such a response may elevate intra-thoracic pressure causing further tissue damage. Animal studies showed that intra-jugular pressure raised more when the glottis was closed. In traumatic asphyxia with open glottis, no craniofacial finding was reported. We believe that in our first patient sudden elevation of external jugular vein pressure caused craniofacial findings; however, in the second patient, accelerations of internal jugular vein pressure lead to petechial haemorrhage in the frontal lobe of the brain.

The following injuries have been reported in patients with traumatic asphyxia: pulmonary contusion, haemothorax, pneumothorax, prolonged loss of consciousness, confusion and seizures, ophthalmic injuries such as exophthalmoses, retinal haemorrhages and visual loss, and abdominal injuries such as liver and splenic lacerations and gastrointestinal haemorrhage. Pathophysiologic features of traumatic asphyxia in children differ from adults due to the greater elasticity of the chest in children. As a result, fewer chest fractures and pulmonary pathologies have been reported in children in contrast to adults. We did not find any significant additional injury in our first patient; however, in the second patient, autopsy findings revealed petechial haemorrhages on pulmonary and frontal lobe and subarachnoid haemorrhage.

In patients with traumatic asphyxia, age and severity of trauma directly affected the outcome including morbidity and mortality. It was reported that patients could recover without any sequel if they were diagnosed and treated early. Patients without additional injury as in our first patient could be managed supportively such as oxygenation and elevation of the head 30 degree upward. However, specific treatments would be needed for the associated injuries.

**Conclusion**

Traumatic asphyxia alone does not predict morbidity and mortality. It is the concomitant cardiovascular, pulmonary and neurologic injuries that affect the outcome.

**References**