Traumatic asphyxia: A rare syndrome in trauma children

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ABSTRACT

Abstract is not required for Clinical Images
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CASE REPORT

A 12-year-old boy, weight 31 kg, with no medical history, had falling from a horse-drawn carriage and was crashed by its wheels at the thorax and upper limbs for approximately 30 seconds. He was admitted to emergency department 45 minutes later. On arrival, he was lethargic, with a Glasgow Coma Score of 12 (E3V4M5); both pupils were equal and reactive to light. His blood pressure was 110/60 mmHg and his heart rate was 110/min. He had tachypnea with respiratory rate of 41/min, the pulsed oxygen saturation in ambient air was 94%.

The patient had facial purple congestion, diffuse head and neck edema and petechiae in the entire face, neck and upper chest (Figure 1). Ophthalmologic examination revealed the presence of sub-conjunctival hemorrhages without impact on visual acuity with a normal fundus (Figure 2). Abdominal examination showed epigastric abrasion without tenderness. Examination revealed a deformation of the right arm and ecchymotic bruises and abrasions on the right hip. The rest of physical examination was unremarkable.

Thoracic computed tomography (CT) scan showed bilateral pulmonary contusions, low abundance bilateral pneumothorax, and fractures of the 3rd and 4th left ribs. Cerebral CT scan was normal. A shoulder X-ray revealed...
right humeral fracture. The electrocardiogram showed a sinus tachycardia. Arterial blood gases on 3 L/min facial mask oxygen showed: pH 7.41, PaCO2 36 mmHg, PaO2 180 mmHg. The blood cell count and the coagulation test were normal. Renal and liver function tests and troponin Ic were unremarkable.

The clinical and radiological presentation of our case was in favor of traumatic asphyxia syndrome, because of the mechanism of the trauma which was the compression of the chest between the ground and a heavy object and because of the presence of classical triad of traumatic asphyxia in the head and neck region. The differential diagnosis in our case was obstruction of the superior vena cava and the skull base fracture; these diagnosis were ruled out by cerebral and thoracic CT scan.

The patient was hospitalized in the intensive care unit and was monitored continuously. Support was symptomatic including facial mask oxygen therapy at 6 L/min, fluid replacement, and multimodal analgesia including paracetamol 15 mg/kg/6H associated to morphine 20 µg/kg/H. The head of the bed was elevated to 30 degrees to help venous drainage of the head and the neck. Pneumothorax was minimal, it spontaneously regressed and there were no indication to chest drainage. Consciousness of the patient gradually improved and he became alert after six hours, although agitation and confusion that lasted for one day. Tachypnea regressed 48 hours later. Thoracic X-ray showed disappearance of pneumothorax and contusions. The humeral fracture was not displaced and was treated by plaster. The outcome was favorable, marked by the decline of the facial edema after three days and the progressive disappearance of petechiae and conjunctival hemorrhages three weeks later.

**DISCUSSION**

Traumatic asphyxia is a type of mechanical asphyxia, where external pressure on the body inhibits respiratory movements and compromise venous return from the head. The thoracic compression must be preceded by a Valsalva maneuver. It is a rare syndrome, first described in 1937 by Oliver d’Angers as the ecchymotic mask. Others names are also used to describe this syndrome: Traumatic cyanosis, compressive cyanosis, traumatic apnea, Oliver’s syndrome, and Perthes syndrome [1, 2]. Perthes syndrome is characterized by the association of edema and cyanosis of the head and neck, subconjunctival hemorrhage, and petechial hemorrhages of the face, neck and chest, secondary to a sudden compression of the thoracoabdominal region [3]. All of these findings were present in our patients. The weight and the duration and of compression affect the outcome. Important weight can be tolerated for a short period, whereas a lower weight associated to a longer period can result in severe consequences [4].

Our patient presented traumatic asphyxia due to a compression between the ground and a heavy object, this mechanism is common in reported cases. Other etiologies of Perthes syndrome are: motor vehicle crashes, crushing in a panicked crowd, entrapment beneath vehicles or falling down in a narrow space [5].

The combination of sudden increase in chest pressure and a deep breath with closed glottis leads to elevated pressure in the valveless head and neck venous system, which is responsible of venous stasis and breaking capillaries and veins [6]. The lower venous territory is protected by the presence of valves and by the obliteration or the compression of inferior vena cava after thoracic hyper pressure [4].

Perthes syndrome is frequently associated with other injuries: hemothorax, pneumothorax, pulmonary contusion, prolonged loss of consciousness, confusion and seizures, ophthalmic injuries such as retinal hemorrhages and visual loss [7].

Our patient presented pulmonary contusions, low abundance pneumothorax, and fractures of two ribs, with a minimal and transitory impact on respiratory function. This disparity between chest injuries and trauma mechanism is probably due to elastic chest cage in children.

Neurological involvement, which makes the severity of this syndrome, is common (90%). Its variable from confusion to coma, the frequency of neurological disorders contrast with the rarity of radiological findings [8]. The mechanism of neurological injury includes cerebral hypoxia, ischemia and venous hypertension, which lead to cortical dysfunction [2]. Usually, neurological events are reversible within 24–48 hours under early and adequate treatment [8]. Our patient had alteration of consciousness that lasted six hours, and was confused during 24 hours, but recovered a normal neurological status under symptomatic treatment.

Visual disturbances occur in some cases [9], secondary to the same mechanism as neurological involvement with multiple presentations: retinal hemorrhage, retrobulbar hemorrhage and vitreous exudates [2]. Therefore, ophthalmological follow-up is important. Our case had no ophthalmological abnormalities in fundoscopy.

The differential diagnosis of this syndrome includes obstruction of the superior vena cava, skull base fracture which clinical presentation contains: sub-conjunctival hemorrhage, periocular ecchymosis, epistaxis and otorrhagia. Tamponade can also induce cyanosis, respiratory distress, but more likely hemodynamic instability [10].

Traumatic asphyxia cases should be monitored after securing the airway. Oxygen therapy and fluid replacement need to be initiated and the patient shall be intubated and followed on mechanical ventilation as needed [1]. The management should include the elevation of the head at 30 degrees; and specific treatments may be needed for associated injuries.
The elasticity of children chest makes the difference of this syndrome in comparison with adults. Thus, in some pediatric cases, even with severe chest and abdominal compression, thoracic lesions were not associated with rib fracture [10]. In children, the prognosis is generally favorable in the absence of severe associated lesions, with the exception of possible visual sequelae, and the mortality rate is usually low [4].

CONCLUSION

Traumatic asphyxia should always be considered as a possible complication of injuries of the chest and abdomen. The prognosis of this syndrome depends on the nature and duration of the compressive force and the presence of others injuries. However, despite the dramatic appearance of Perthes syndrome, mortality remains low, especially in children, due to chest elasticity.

Keywords: Pediatrics, Perthes syndrome, Trauma, Traumatic asphyxia

Conflict of Interest
Authors declare no conflict of interest.

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