

# Traumatic Asphyxia

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**Abstract:** Traumatic asphyxia is a rare condition in children that usually occurs after severe compression to the chest or abdomen. We report 3 cases in patients 18, 20, and 36 months of age who presented signs and symptoms of traumatic asphyxia after car accidents. Two clinical features were consistent in all 3 patients: multiple petechiae on the face and bulbar conjunctival hemorrhage; 2 patients had facial cyanosis, and 1 had facial edema.

In children, the number of clinical manifestations that should be evident to diagnose traumatic asphyxia has not been ascertained. However, in any history of trauma with compression of the chest or abdomen and signs of increased intravenous craniocervical pressure, traumatic asphyxia should be suspected.

**Key Words:** traumatic asphyxia

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**T**raumatic asphyxia is a rare disease manifested by facial edema, cyanosis, conjunctival hemorrhage, and petechiae on the face and chest; periorbital edema, respiratory distress, and altered mental status have also been described.<sup>1–4</sup>

This condition was initially reported as ecchymotic mask (*masque ecchymotique*) by Ollivier D'Angers in 1837. The clinical features described were craniocervical cyanosis, subconjunctival hemorrhage, and cerebral vascular congestion. These findings were observed in autopsies of patients who were crushed by a mob in Paris.<sup>4</sup> Later, in 1900, Perthes added neurological symptoms to this syndrome. Traumatic asphyxia occurs as a result of severe compression of the chest, upper abdomen, or both. During the period from January 2010 to December 2011, 3 cases of traumatic asphyxia were diagnosed in the pediatric emergency department.

## CASES

Patient 1 is a 36-month-old female patient who was hit by a truck when riding on a motorcycle; she was ejected and had a thoracoabdominal trauma when she hit the ground. She was admitted to the pediatric emergency department with a heart rate of 141 beats/min, a respiratory rate of 29 breaths/min, and oxygen saturation (SatO<sub>2</sub>) of 99%. Her Glasgow Coma Scale score (GCS) was 15, and her Pediatric Trauma Score (PTS) was 10. Cyanosis was seen on the face and multiple petechiae on the face and neck, with hemorrhage of the bulbar conjunctiva bilaterally. The thorax was swollen, and she had a deformity of the distal one third of the left clavicle with no evidence of respiratory distress. She also

presented a deformity and limitation of movement in her left leg. X-rays showed a fracture of the left clavicle and the proximal third of the left femur. Computed tomography (CT) of the skull and chest and abdomen was reported normal.

During hospitalization, her clavicular fracture was immobilized, and the femoral fracture was reduced with placement of a thoracopelvic cast. She was discharged 7 days after admission with a follow-up at 6 months showing consolidated fractures after rehabilitation.

Patient 2 is an 18-month-old male patient who was run over by a truck, with a crush injury in the region of the left abdomen. He entered the emergency department with a heart rate of 156 beats/min, a respiratory rate of 35 breaths/min, a SatO<sub>2</sub> of 99%, a GCS score of 14, and a PTS of 11. Cyanosis and facial edema were noted together with multiple petechiae on the face and oral mucosa and hemorrhage in the bulbar conjunctiva of the right eye. A CT scan of the chest and abdomen showed a contusion of the left lung. The patient was discharged after 24 hours with a follow-up 3 months later that showed no alterations.

Patient 3 is a 20-month-old male patient. He received a thoracoabdominal trauma caused by a car. He was admitted to the emergency department with a heart rate of 134 beats/min, a respiratory rate of 26 breaths/min, and a SatO<sub>2</sub> of 99%, a GCS score of 14, and a PTS of 10. Multiple petechiae were seen on the face and oral mucosa, as well as hemorrhage of the bulbar conjunctiva of the right eye and an abrasion of the nasolabial area (Fig. 1). A thoracoabdominal CT scan was performed and reported as normal. The patient was discharged 24 hours after admission. Follow-up at 4 months showed no alterations.

The clinical characteristics of the 3 patients are summarized in Table 1.

## DISCUSSION

The most common cause of traumatic asphyxia is motor vehicle accidents; other causes are episodes of asthma, seizures, persistent vomiting, and even sexual abuse.<sup>5,6</sup> The incidence in children has not been described but is thought to be a rare event in this age; however, in adults, the incidence is 1 case per 18,500 accidents.<sup>1,3,4,7–9</sup> The 3 cases we report had a thoracoabdominal trauma secondary to a motor vehicle accident.

Patients undergoing a compressive force to the chest or upper abdomen tend to hold their breath and close the glottis, which increases intrathoracic and mediastinal pressure (Valsalva maneuver). This increase in intrathoracic pressure causes blood from the right atrium to flow through the innominate and jugular veins, leading to a pressure increase and stasis in venules and capillaries of the cervicofacial region, causing petechial hemorrhage and hydrostatic edema.<sup>2–6,8,10–14</sup> The glottis closure and chest compression are essential to produce a significant increase in venous pressure, delayed venous flow, and capillary rupture.<sup>7–9</sup>

In pediatric patients, traumatic asphyxia is rare because of the greater elasticity of the rib cage, and it is thought that the mechanism differs from that in adult patients.

In our report, 2 clinical manifestations occurred in the 3 cases, multiple petechiae on the face, and bulbar conjunctival

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hemorrhage, but facial cyanosis was evident in only 2 patients, and facial edema in only 1.

Petechiae may be more prominent in the conjunctiva and oral mucosa, most frequently 2 to 3 hours after the accident.<sup>11,15</sup> Craniocervical manifestations occur because the tissues of the scalp and neck drain into the external jugular veins that despite having valves (2 pairs) are not competent to prevent flow reversal. However, the brain and airway structures that drain into the internal jugular vein are protected by more competent valves, but at pressures greater than 45 mm Hg, these can fail and cause neurological symptoms. The severity of the manifestations is proportional to the time and intensity of the compression.<sup>16</sup>

Otorrhea, hemotympanum, tinnitus, and hearing loss can also be found. In adults, there are abnormalities during ophthalmic examination in up to 50% of cases with pupillary changes and exophthalmos that is present in up to 20%, and transient visual changes and blindness, which can be temporary or permanent.<sup>1,7,8,10,11,14–18</sup>

Neurological manifestations have been reported in up to 90% of cases and may present as agitation, loss of consciousness, confusion, or seizures. Those are caused by indirect damage due to anoxic injury, ischemia, or cerebral edema secondary to venous obstruction and increased vascular pressures. Mild neurological manifestations usually resolve in 24 to 48 hours.<sup>10,12,13</sup>

One of our patients had a lung contusion that was related to the mechanism of injury and that had a favorable outcome with the patient being managed with supplemental oxygen; he was discharged without complications. Other lung manifestations described are hemothorax and pneumothorax.<sup>2,11</sup>

The differential diagnosis includes the superior vena cava syndrome and skull base fracture where conjunctival hemorrhage and periorbital ecchymosis can exist.<sup>11,16</sup>



**FIGURE 1.** Patient 3 with multiple petechiae on the face; arrow points to hemorrhage of the right bulbar conjunctiva.

**TABLE 1.** Clinical Features

Manifestations	Patient 1	Patient 2	Patient 3
Facial cyanosis	+	+	—
Petechiae on the face	+	+	+
Oral mucosal petechiae	—	+	+
Petechiae on the neck	+	—	—
Facial edema	—	+	—
Bulbar conjunctiva hemorrhage	RE-LE	RE	RE
Lung contusion	—	+	—
Fracture	Clavicle, femur	—	—

LE indicates left eye; RE, right eye.

Despite the severe appearance, none of the patients had significant lesions and evolved with a favorable outcome, an early discharge, and no complications at follow-up. The vast majority of cases of traumatic asphyxia have a good prognosis. All injuries tend to progressively resolve, including the neurological symptoms.<sup>7,8,18</sup>

Treatment of traumatic asphyxia is directed at associated lesions. Management includes adequate oxygen supplementation and maintaining good perfusion. The prognosis is generally good, but prolonged compression may leave neurological sequelae. Recovery is related to the severity and duration of the injury and the accompanying injuries.

The diagnosis of traumatic asphyxia is made by the clinical manifestations of increased craniocervical pressure trauma associated with chest or abdominal compression or both. The true incidence of traumatic asphyxia is unknown in children because of underreporting because attention is given to concomitant lesions that can be serious, and the clinical manifestations or a diagnosis of traumatic asphyxia is not recorded. Another factor that influences incidence is that there is no consensus on how many manifestations should be present to diagnose this entity in children. Therefore, research in pediatric patients is needed to determine which or how many manifestations are necessary for diagnosis, especially because of the differences in the physiology of pediatric patients compared with adults.

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