

# Traumatic asphyxia in children

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Eight children with traumatic asphyxia were evaluated retrospectively. There were five boys and three girls. The mechanism of injuries was motor vehicle accidents in six children. A fall in one patient and compression by lift in one patient. Clinical features of traumatic asphyxia developed in all patients. Five patients were disoriented and consciousness. Associated injuries were noted in all patients often involving thorax and head. Cerebral seizures complicated head injury in one patient. No mortality was recorded.

KEY WORDS: Asphyxia, traumatic - Blunt injuries - Child.

The clinical syndrome characterized by subconjunctival hemorrhages, facial edema, and cyanosis, combined with ecchymotic petechial hemorrhages on the upper chest, neck, face and subconjunctival is called traumatic asphyxia (TA).<sup>1-4</sup> These were first observed by Ollivier in 1837; he termed the syndrome «masque échyмотique» when noting these characteristic features in a patient trampled to death by crowds in Paris.<sup>5</sup> Perthes in 1900 fully described the clinical syndrome.<sup>2</sup> Many other phrases have been used to describe this syndrome: traumatic cyanosis, compression cyanosis, Perthes symptom complex, cervico-facial cutaneous asphyxia, and cervicofacial static cyanosis.<sup>6</sup> The most common causes of TA are direct compression of the chest wall or upper abdomen due to blunt trauma. Other etiological factors are epileptic seizures, vomiting, whooping cough, bronchial asthma, deep-sea diving and difficult obstetric deliveries.<sup>6</sup>

Although the incidence of TA is difficult to ascer-

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tain, it is a rare pathology. The TA is usually self-limited and resolves over a period of several weeks without complications.<sup>6</sup> However, the patients with TA have most commonly associated injuries and morbidity and mortality of these patients related to the severity of these associated injuries.<sup>2</sup> In this study, we retrospectively evaluated eight patients with TA and clinical signs, severity of injury, associated injuries, neurologic status, morbidity, mortality and long-term follow-up are discussed.

## Materials and methods

We reviewed the medical records of eight patients consecutively evaluated at the Faculty of Medicine, Department of Pediatric Surgery, Trabzon, with traumatic asphyxia between June 1992 and October 1995. All diagnoses were confirmed clinically.

After patients were stabilized in the emergency room including intubation if required, intravenous fluids and blood transfusion, chest radiography, and ultrasonographic and computed tomographic examinations were performed. If necessary chest drainage was performed in the emergency room. After strict examinations all patients were admitted to the intensive care unit.

Charts were carefully reviewed for age, sex, spe-

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Fig. 1.—Typical appearance of traumatic asphyxia in a nine year old girl.

cific details of the accident, clinical status, associated injuries, duration of hospital stay, management, complications and mortality.

### Results

The patients ranged in age from 2.5 to 12 years (mean, 6.9 years); five were males, and three were girls. Motor-vehicle accidents caused the injuries in six children (four was compressed by a wheel, and two were run down). One patient fell from the fifth floor and one patient was compressed by lift.

Clinical features such as cervicofacial cyanosis and petechiae of the head and neck regions, facial edema, and subconjunctival hemorrhages developed in all patients in varying degrees (Fig. 1). Tachypnea and dyspnea were noted in five patients. Five patients were disoriented and consciousness. Four patients were in shock because two had hemothorax and two had associated abdominal trauma. Fundoscopic examination was performed in all patients and two patients had disc hemorrhages, two patient had venous congestion, an four other patients had normal fundi.

Associated injuries were noted in eight patients. Seven patients had pulmonary contusion, six patients had head injuries, five patients had rib fractures. Pneumomediastinum and extensive subcutaneous emphysema were noted in three patients. Other associated injuries included muscu-

loskeletal in four, hemothorax in two, pneumothorax in two, and hepatic and splenic injuries in two patients.

Cerebral CT examinations were performed in all patients and cerebral edema and/or hemorrhages were noted in six patients.

All patients were successfully treated conservatively including broad spectrum antibiotics oxygen mask, steroids and bed rest. One patient required intubation and mechanical ventilation. Chest drainage was performed in three patients due to hemothorax and/or pneumothorax. Two patients with intra-abdominal bleeding were also treated nonoperatively.

The average period of hospitalization was 12.3 days, with a range of 2 to 42 days. Cutaneous lesions and subconjunctival hemorrhage were lost between eight and twenty days. Complications during hospitalization occurred in one patient with cerebral seizures due to head injury. Other seven patients were followed up for six months and two years. There was no persisting physical or mental disabilities resulting from or attributable to their injuries. No mortality was noted in our cases.

### Discussion and conclusions

Traumatic asphyxia is, for the most part, seen in children with their elastic walls. It occurs only with sudden severe crushing forces. Most reported cases of TA result from anterior-posterior compression of the victim's chest and/or upper abdomen. The usual cause of TA includes motor vehicle accidents, in which the patient is compressed by the wheel.<sup>1,5</sup> The amount of force required and duration of application to produce the typical manifestation of TA is not know. Weights ranging from 3000 pounds to several tons have been recorded and the typical range of compression is between two and five minutes.<sup>3,11</sup>

The pathophysiology of TA involves four factors: 1) deep inspiration, 2) closure of the glottis, 3) thoracoabdominal effort, eg, splinting of thoracic and abdominal musculature, and 4) thoracic or abdominal compression.<sup>5</sup> Following compression of the chest or upper abdomen, pressure is transmitted to the mediastinum and then to valveless superior vena cava into the head and neck with



subsequent disruption of superficial capillaries. Chest compression produces varying amounts of apnea and hypoxia, which may contribute to contusion of the abdominal sympathetic nerves causing vasodilatation of the head and neck region and atony of the small vessels.<sup>1-5</sup> Experimental studies demonstrated that occlusion of the superior vena cava alone could cause a considerable congestion of the upper extremities and head regions without the clinical features of TA.<sup>7</sup> Williams *et al.*<sup>12</sup> demonstrated that chest compression for 30 seconds at full inspiration with a closed glottis produced an acute rise in jugular venous pressure, 8.8 times greater than the control group, however when chest compression occurred with an open glottis jugular venous pressure rose only 1.5 times over the control group in dogs. They proposed that the patients glottis with TA is closed due to fear reflex or panic during trauma.<sup>12</sup>

Usual clinical findings of TA are petechiae covering the face, neck, and upper chest as well as subconjunctival and retinal hemorrhages. The face and neck are usually blue and swollen. Below the level of impact the skin is normal.<sup>1-5</sup> At fundoscopy retinal hemorrhage may be evident.<sup>10</sup> Immediate impairment or loss of vision has also been reported as a result of retinal edema, but may promptly recover within hours or days.<sup>10</sup> Pulmonary and/or great vessels injuries are evident, a pneumothorax and/or hemothorax, mediastinal air, subcutaneous emphysema and hemoptysis may be seen.<sup>2 8 11</sup> Sometimes, venous pressure is transmitted into the intra-abdominal viscera via the inferior vena cava and hematemesis, rectal mucosal bleeding, hematuria and albuminuria may also occur.<sup>2 4</sup>

Neurological symptoms are often associated with TA. The patients are usually disoriented, agitated and restless. Loss of consciousness has been reported in 33% of patients.<sup>9</sup> These neurological signs may be associated with minor intracranial hemorrhage or cerebral edema.<sup>2</sup> Brachial plexus lesions, quadriplegia and coma without evidence of skull or spinal cord injury have also been documented.<sup>6</sup>

Management of TA is symptomatic and there is

no specific therapy. The patients are observed in an intensive care unit initially. When pulmonary contusion and/or cerebral edema are noted steroid treatment should be performed. If there is hemothorax and/or pneumothorax chest drainage is performed. Associated abdominal injuries may require surgical intervention.<sup>1-5</sup>

The prognosis for TA is very good despite the alarming initial physical appearance.<sup>2 3 8</sup> The lesion is usually self limited and resolved over a period of several weeks.<sup>2</sup> Neurological lesions are transient and most recover within 48 hours.<sup>2 5 6</sup> Neurological sequelae are rare and are related to associated cerebral injury.<sup>9</sup> In our series neurological sequelae developed in one patient due to associated cerebral injury, and no long-term disability was noted at a mean of 2.5 years follow-up in other seven patients. Mortality is rarely related to TA. In fatal cases, compression had been prolonged.<sup>2</sup> It is proposed that prolonged compression restricts chest wall motion producing apnea, hypoxia, and death.<sup>2</sup> Mortality in our series was nil.

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