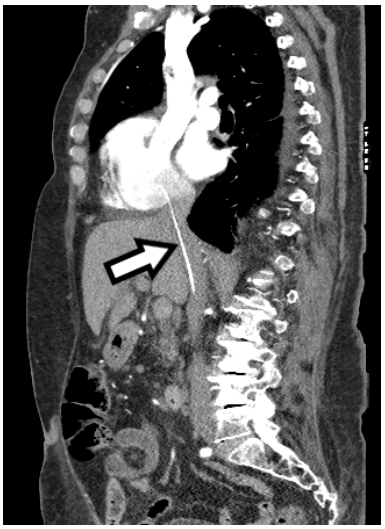

Figure 3: A tubular dense structure (arrow) extending from the left paravertebral venous tract to the inferior vena cava and right atrium on CTPA imaging.

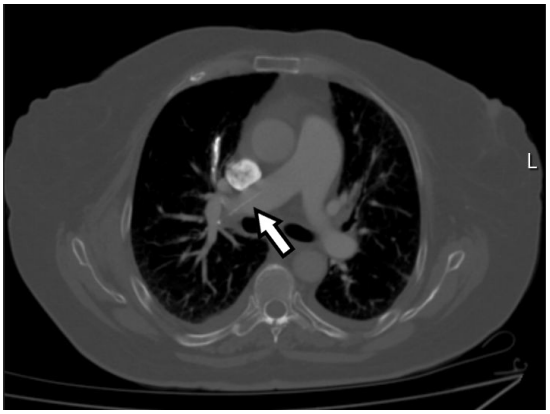

Figure 4: A tubular dense structure (arrow) in the right main pulmonary artery on CTPA imaging.

References

1.For the CSA Fracture Working Group of the International Osteoporosis Foundation, Boonen S, Wahl DA, Nauroy L, Brandi ML, Bouxsein ML, vd. Balloon kyphoplasty and vertebroplasty in the management of vertebral compression fractures. Osteoporos Int. Aralık 2011;22(12):2915-34.
2.Burton AW, Rhines LD, Mendel E. Vertebroplasty and kyphoplasty: a comprehensive review. FOC. Mart 2005;18(3):1-9.
3.Ünlü N. Vertebroplasti Sonrası Semente Bağlı Pulmoner Emboli: BT Bulguları. Ankara Üniversitesi Tıp Fakültesi Mecmuası. 2012;
4.Kim YJ, Lee JW, Park KW, Yeom JS, Jeong HS, Park JM, vd. Pulmonary Cement Embolism after Percutaneous Vertebroplasty in Osteoporotic Vertebral Compression Fractures: Incidence, Characteristics, and Risk Factors. Radiology. Nisan 2009;251(1):250-9.

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A Rare and Easily Overlooked Diagnosis in a Pediatric Trauma Case: Traumatic Asphyxia

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Introductıon:

Traumatic asphyxia is a clinical syndrome characterized by cervicofacial cyanosis, edema, petechiae, subconjunctival hemorrhage, and neurological symptoms following sudden, severe, compressive blunt thoracoabdominal trauma (1). This syndrome is also referred to as acute thoracic compression syndrome, ecchymotic mask, or Perthes syndrome (2). It was first described in 1837 by Oliver d'Angers based on autopsy findings, and its exact incidence remains unknown (3).

The clinical findings associated with traumatic asphyxia can involve the skin, eyes, brain, gastrointestinal system, airway, and chest. The hallmark of the syndrome, cervicofacial cyanosis, is typically accompanied by petechiae, which are generally observed in the head, neck, and upper chest. Morbidity and mortality are related to concurrent cardiovascular, neurological, and pulmonary injuries and their severity (4).

Blunt trauma is particularly common in forensic cases and is a primary cause of crushing injuries to the upper chest. Following trau-

ma, diffuse petechiae may appear in the face and neck regions, giving the patient's face a livid appearance. This condition is terminologically known as an "ecchymotic mask." Although patients with an ecchymotic mask may present with a visually alarming appearance, their clinical condition is often not as severe as it seems (5).

This article presents a case of traumatic asphyxia developing as a result of severe thoracoabdominal trauma.

Case Report:

A previously healthy 9-year-old male patient was brought to the emergency department (ED) after falling from the trailer of a tractor. The patient landed face-down, striking his chest against the ground. Upon arrival at the ED, his vital signs were within normal limits. He was conscious, cooperative, and oriented, with a Glasgow Coma Scale (GCS) score of 15. Numerous petechiae were observed on the scalp, face, neck, and upper chest wall. Bilateral eyelid edema, bulbar conjunctival hemorrhage, and edema in both eyes were noted. No petechiae, purpura, or ecchymosis were found on other parts of the body (Image).

The airway was patent, and there were no signs of respiratory distress. Laboratory test results were within normal limits. Physical examination of other systems was unremarkable. Radiographs of the extremities, along with computed tomography (CT) scans of the cranium, chest, and abdomen performed due to the high-energy trauma, were also reported as normal.

During observation in the ED, the patient's vital signs and clinical condition remained stable. Ophthalmology consultation ruled out any acute pathology. The patient was monitored in the ED for 24 hours and discharged with recommendations after no worsening of symptoms was observed.

Dıscussıon:

The most common cause of traumatic asphyxia in children is motor vehicle accidents (e.g., traffic collisions). Traumatic asphyxia can also occur in situations involving being crushed under heavy machinery or furniture and/or being trapped between two objects (6). Non-traumatic conditions such as epileptic seizures, severe vomiting, whooping cough, and asthma exacerbations may result in similar clinical presentations (7). In these cases, the underlying mechanism is not trauma-related but rather asphyxia induced by the disease, leading to comparable clinical outcomes.

In adults, the frequency of traumatic asphyxia is approximately 1 case per 18,500 accidents. It is exceedingly rare in children, and its actual incidence remains unknown. The true frequency in pediatric cases is uncertain, likely due to underrecognition or underreporting by pediatricians (5).

The treatment process for traumatic asphyxia depends on the severity and duration of compression. While heavy loads may be tolerated for short durations, prolonged exposure to moderate loads can result in fatal outcomes (7). The characteristic features of traumatic asphyxia include petechiae, cranio-cervical cyanosis, subconjunctival hemorrhage, and facial edema involving the upper body, neck, and face. Petechiae usually appear 2-3 hours after the incident and are more prominent in the conjunctiva and oral mucosa. This results in a livid appearance of the patient's face (8). This clinical condition, known as an "ecchymotic mask," was consistent with the diagnosis in our case.

The underlying mechanism of these clinical findings is the increased blood flow from the right atrium to valveless brachiocephalic and jugular veins due to positive pressure generated in the mediastinum following blunt thoracic and upper abdominal trauma. The sudden rise in venous pressure results in petechiae. Petechiae are absent in the lower body because venous valves in the lower extremities control the increased venous pressure. Additionally, if the inferior vena cava is compressed or obstructed, elevated airway pressure may further protect the lower body (5).

Depending on the severity of the trauma, rib fractures, pulmonary contusions, pneumothorax, hemothorax, solid organ lacerations, and neurological sequelae may occur. A study conducted in adults recommended cranial, abdominal, and pelvic CT scans, along with Doppler ultrasonography of the neck and upper extremity vessels and echocardiography, to identify these injuries. However, there are no specific recommendations for children (8). Considering the anatomical and physiological differences between children and adults, performing all these tests in pediatric patients may be unnecessary. Diagnostic workups should be planned based on the severity of the trauma, the patient's GCS, and pediatric trauma score (5).

The prognosis of traumatic asphyxia is generally favorable with effective and timely treatment. Prolonged thoracic compression can lead to cerebral anoxia and neurological sequelae. Therefore, in patients with blunt thoracoabdominal trauma, a detailed trauma history should be obtained, and appropriate diagnostic and therapeutic plans should be implemented (5).

Treatment is generally conservative and recovery is closely related to associated injuries. In uncomplicated cases, the head should be elevated to 30 degrees, and oxygen therapy should be administered to reduce intracranial pressure. In cases with complicated organ injuries, advanced life support measures should be applied according to the patient's clinical condition, and cervical spine immobilization should be ensured (9). In our case, the patient was monitored in the emergency department for 24 hours and discharged with outpatient follow-up recommendations after no worsening of symptoms was observed.

Conclusıon:

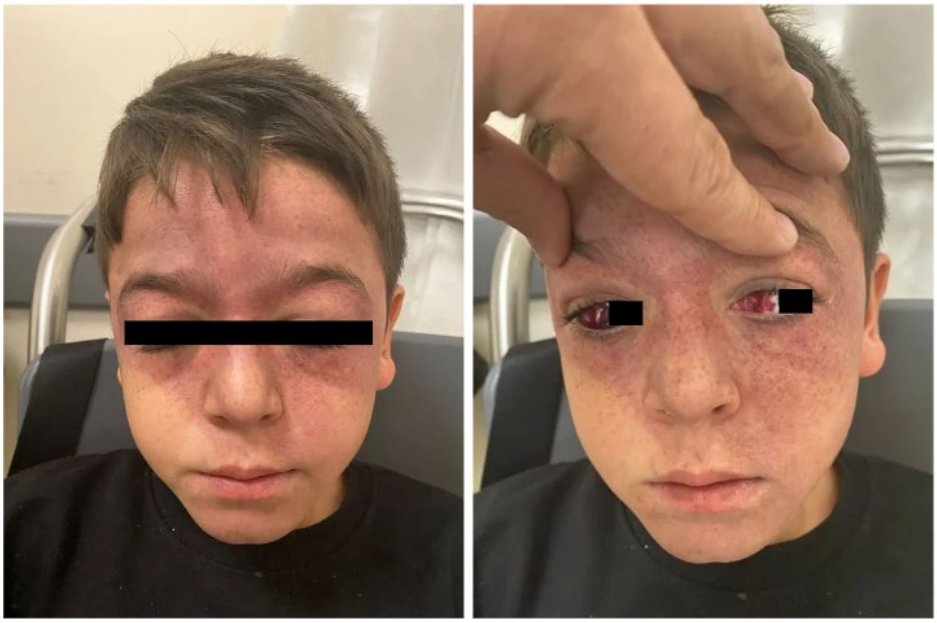
In conclusion, the diagnosis of traumatic asphyxia should be considered in pediatric and adult patients with blunt thoracoabdominal trauma. Despite its catastrophic appearance, it is typically a benign condition.

References:

1. Çelik, B., Öztürk, D., & Gürz, S. (2012). Travmatik asfiksi. Journal of Experimental and Clinical Medicine, 29(2), 163-165.
2. Şenoğlu, N., Öksüz, H., Zincirci, B., Ezberci, M., Yasım, A., 2006. Ağır travmatik asfiksi: İki olgu sunumu eşliğinde literatürün gözden geçirilmesi. Türk Göğüs Kalp Damar Cer. Derg.14, 78-81.
3. Oliver, D.A., 1837. Relation medicale des evenements survenus au Champ-de-Mars le 14 juin. Ann.Occup. Hyg. 18, 485-489.

4. Ibarra, P., Capan, L.M., Wahlander, S., Sutin, K.M., 1997. Difficult airway management in a patient with traumatic asphyxia. Anesth. Analg. 85, 216-218.
5. Güleryüz, O. D., Akar, T., & Atmaca, Y. M. (2020). Four traumatic asphyxial cases with an “masque ecchymotique” in pediatric emergency department
6. Lowe L, Rapini RP, Johnson TM. Traumatic Asphxia. J Are Acad Dermatol. 1990; 23:972-4
7. Montes-Tapia F, Barreto-Arroyo I, Cura-Esquivel I, Rodríguez-Taméz A, de la O-Cavazos M. Traumatic asphyxia. Pediatr Emerg Care. 2014;30:114-6.
8. Hurtado TR, Della-Giustina DA. Traumatic asphyxia in a 6-year-old boy. Pediatr Emerg Care. 2003;19:167-8
9. Sarihan H, Abes M, Akyazici R, Cay A, Imamoglu M, et al. Traumatic Asphyxia in children. J Cardiovasc Surg (Torino). 1997;38:93-5.

Image. Petechiaes on the face and bilateral eyelid edema, bulbar conjunctival hemorrhage.



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Old Enemy At New Address: From Lıver Cyst Hydatıd To Pulmonary Localızatıon

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Introductıon

Hydatid disease is a zoonotic parasitic disease caused by Echinococcus granulosus. It is more common in people who have close contact with animals in endemic areas and is most frequently located in the liver (65%) and lung (25%). The liver is the most commonly affected organ because it is the first barrier through the portal vein; however, in some cases, the parasite can progress to the lungs and cause pulmonary hydatid cyst formation. The cyst usually grows slowly and patients may remain asymptomatic for a long time (1). The most common symptoms in pulmonary hydatid cysts are cough, dyspnea, chest pain and hemoptysis. The enlarged cyst may open into the bronchi and develop a complicated pulmonary hydatid cyst characterized by air-fluid levels. Ruptured or infected cysts may cause severe inflammation and suppurative complications (2). Imaging methods and serological tests are of great importance in the diagnosis of hydatid cyst. Medical (albendazole) and surgical (cystectomy, capitonnage) methods are preferred in treatment according to the patient's clinic (3). Especially in patients with a history of hepatic hydatid cyst, if there is new chest pain or respiratory complaints, pulmonary hydatid cyst should be included in the differential diagnosis.

Case Report

A 33-year-old female patient applied to the emergency department with a complaint of stabbing pain under her left breast. The patient stated that the pain had been worsening for a few days and was occasionally accompanied by dyspnea. She had a known history of asthma and had undergone surgery for a hepatic hydatid cyst approximately three years ago. It was learned that the patient did not complain of cough or sputum production, but occasionally felt mild chest tightness. On physical examination, the patient's general condition was good and he was conscious. Body temperature was 37.8°C, pulse 95/min, blood pressure 120/80 mmHg and respiratory rate 22/min. Auscultation revealed a significant decrease in breath sounds in the lower zone of the left lung. Laboratory tests revealed high infection markers. C-reactive protein level was 201 mg/L and white blood cell count was 17,000/mm³. First, the