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Traumatic Asphyxia (Perthes's Syndrome)

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ABSTRACT

Introduction: Traumatic asphyxia or Perthes's syndrome is a powerful trauma or fatal injury related complication series usually seen in industrial machine or motorized vehicle accidents. We present a young patient suffocated with the compression of a powerful industrial compress machine.

Case presentation: A 19 year old male was found unconscious after an accidental compression of a press machine for ten minutes in a manufacturer's place by emergency staff. Immediate cardiopulmonary resuscitation was achieved as there was no pulse. Patient got spontaneous circulation after resuscitation. He was admitted to the emergency department (ED) and critical care unit respectively. Computer tomography scan showed 3-5 posterior rib fractures, right pneumothorax, contusion on right hemithorax. Bilateral chest tubes were inserted in ED. İnitial examination showed facial and periorbital ecchymosis, bilateral conjunctival hemorrhage. On follow up the patient needed high dose vasopressor support without any hemodynamic improvement and was lost in 6 days' time.

Conclusion: Diagnosis of Perthes's syndrome is easy when the impact is powerful enough. It is diagnosed clinically and is mortal when easily diagnosable.

INTRODUCTION

Traumatic asphyxia is a clinical phenomenon emerging in the presence of sudden increase in the thoracic pressure which leads to craniocervical cyanosis, subconjunctival hemorrhage, severe petechiae and neurologic symptoms. It is also known as Morestin's syndrome. It is more likely to be defined in children and can also be seen in adult patients [1]. Most important causes are asthma crisis, epileptogenic discharges, severevomiting which explains the high incidence in pediatric patients. Actual incidences are not clear as most of these patients are lacking diagnosis [2, 3].

CASE

A 19 year old male was found unconscious after compression of a press machine for ten minutes in a manufacturer's place by emergency staff. Immediate cardiopulmonary resuscitation was achieved as there was no pulse. Patient got spontaneous circulation after resuscitation. He was admitted to the emergency department (ED) and critical care unit respectively. Patient has no medical history. He works in an industrial fabric. Patient has no history of tobacco or alcohol use. During admission to the critical care he was sedated, unconscious, orotracheal intubated, right chest tube inserted and oscillating.

Cardiorespiratory examination revealed decreased respiratory sounds with ritmicritmic cardiac sounds without any murmur with a heart rate of 101 beats per min. His blood pressure was 123/98 mmHg and a peripheral pulse oximeter showed 98% saturation.

His electrocardiogram showed peaked T waves **(Figure 1)**. But his serum potassium levels were normal. There was no penetrated damage on thorax and abdomen. He had a well-definedecchymotic mask on his face **(Figure 2)**. Patient's eyelids werebothedematous. His temperature was 90°F. He was heated to 93°Fnd kept for 18 h according to therapeutic hypothermia protocol. Neurological examination revealedbilateral 3 mm pupil size with no pupillary light reflex and a Glasgow coma scale of 3. There were skin abrasions starting from left tragus to external meatus. Moreover examination of the abdomen and extremities showed no abnormalities. Computed tomography scan

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showed right pneumothorax so right chest tube were inserted in ED. Initial transthoracic echocardiography was performed in ED and visual ejection fraction was 55% and no pericardial fluid was detected. Cerebral computed tomography showed no bleeding or contusion. Parenchyma and bone structures were normal with no shift or hydrocephalus. Basal cisterns were open. Computed tomography (CT) scan showed right posterior 3-5 rib fracture and right pneumothorax and contusion in right hemithorax **(Figures 3 and 4)**. No abnormalities were found on abdomen CT interpretation.

Parenchyma and bone structures were normal in cranial CT. Mild edema was seen in his cranial CT at cerebellar level. His magnetic resonance imaging (MRI) showed bilateral globus pallidus diffusion weighted restrictions.

His blood gas analysis revealed a pH of 7.32, PO_2: 160.4 mmHg, PCO_2: 32.2 mmHg, cHCO3st: 18.1, Lac: 2.61 mmol/L. Initial laboratory findings were creatinine: 0.98, sodium: 140 meq/L, WBC: 22.57 10^9/L, HGB: 17.8 g/dl, PLT: 239 10^9/L, CRP: <0.21,CPK: 2659 U/L, procalcitonin: 0.130, AST: 438.4, ALT: 453.1. CPK values peaked on the second day measured as 4010 U/L. Patient had decreased urine output being anuric on the 4th hour of admission. He was monitored the admission day. Continuous renal replacement therapy was initiated on the second day. Patient is sedated with propofol and fentanyl. Sedation could not be interrupted because of patient-ventilator dyssynchrony.

During follow up, patient had labile hemodynamics and did not make any cognitive or hemodynamic improvements with increasing vasopressor and inotropic demands. He was lost on the 6th day.

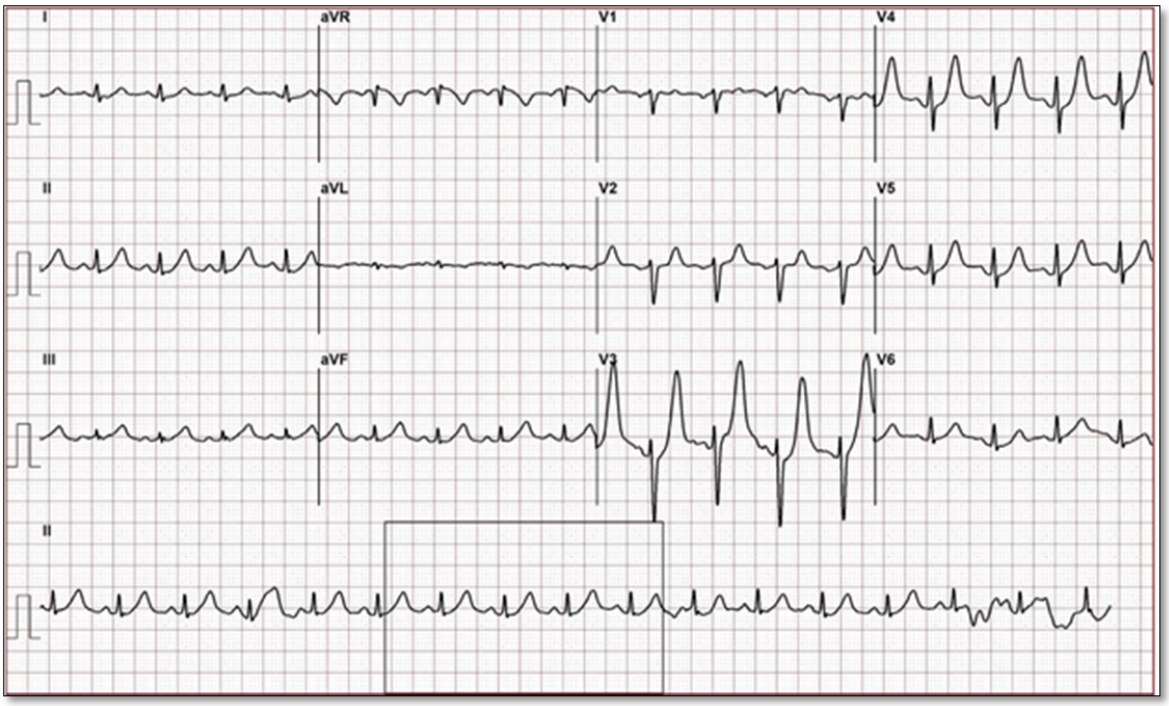

Figure 1. Electrocardiography of the patient.

DISCUSSION

Traumatic asphyxia is thought to be less reported then its actual incidence. Any situation that increases intrathoracic pressure or squeezes the thoracic cavity can lead to Perthes's syndrome. In pediatric patient group asthma crisis and trauma, in adult patients deep diving, heavy lifting, difficult delivery in women, epileptic discharges all suddenly interrupt venous return in upper extremity. Traffic and industrial accidents are included. Diagnosis is easily made with anamnesis, inspection and examination.

Examination should be kept comprehensive as serious complications like hemothorax, pulmonary contusion, flail chest, laryngeal edema, tongue welling, difficult intubation is expected in such excessive thoracic trauma patients. Spinal, sternal and rib fractures are commonly seen in these patient groups as well [1, 2, 4]. Cardiopulmonary compression duration in these cases are reported to be usually 2-5 min [5, 6]. Our case had a compression time of ten minutes. There is no information about the arrest length of the patient. The longer the pressure on thorax, the longer the lack of venous return to thorax and the higher the mortality is [4, 7]. Chest contusion can also lead to cardiac injury or stagnancy and pericardial fluid leading to decreased cardiac output.

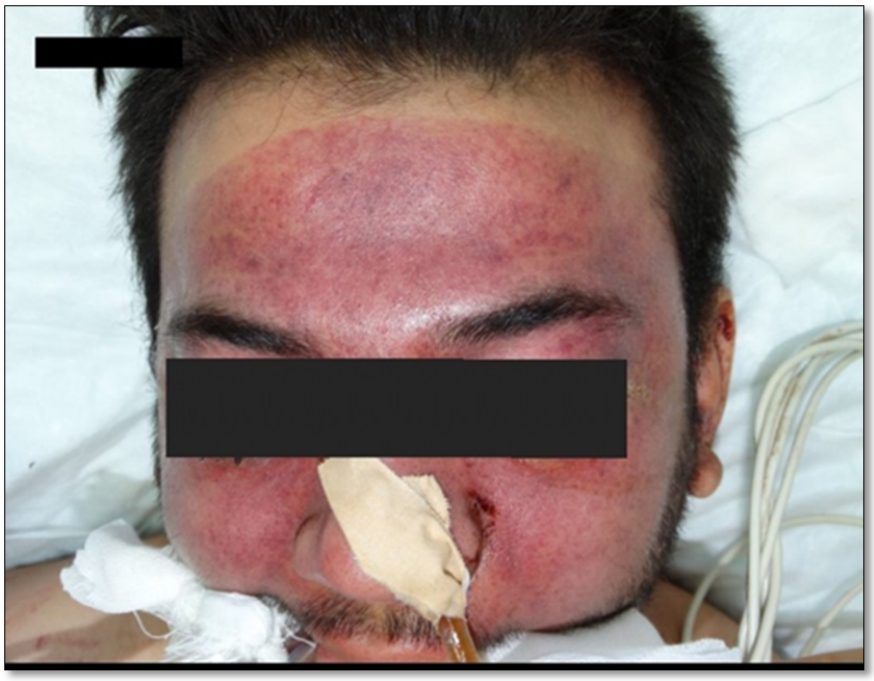

Figure 2. Facial photo of the patient.

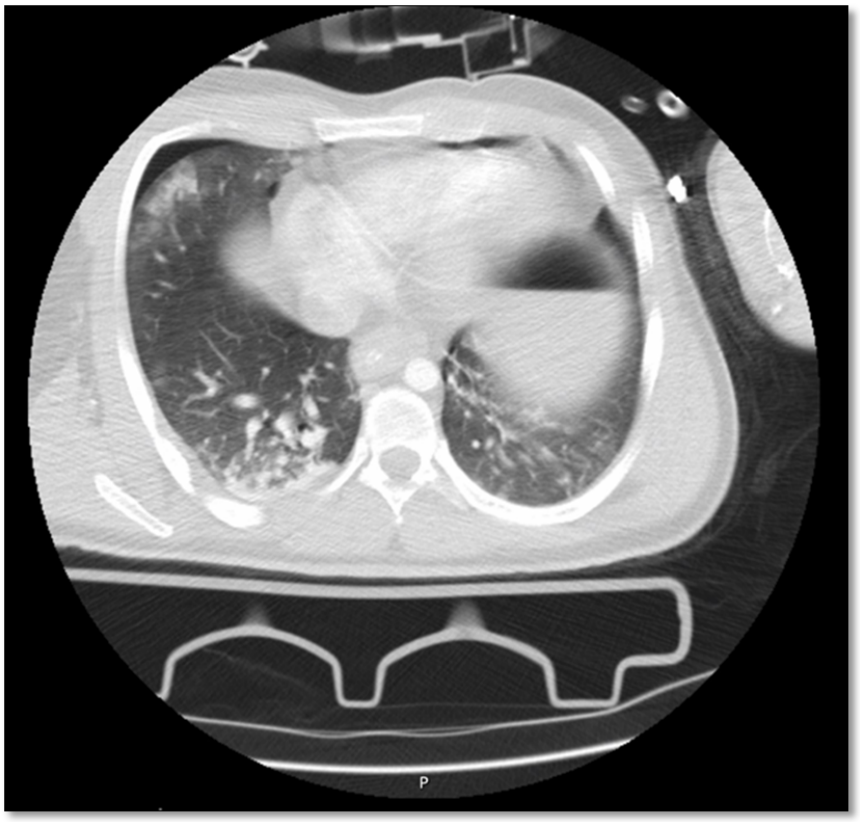

Figure 3. Thorax CT of the patient (pneumothorax).

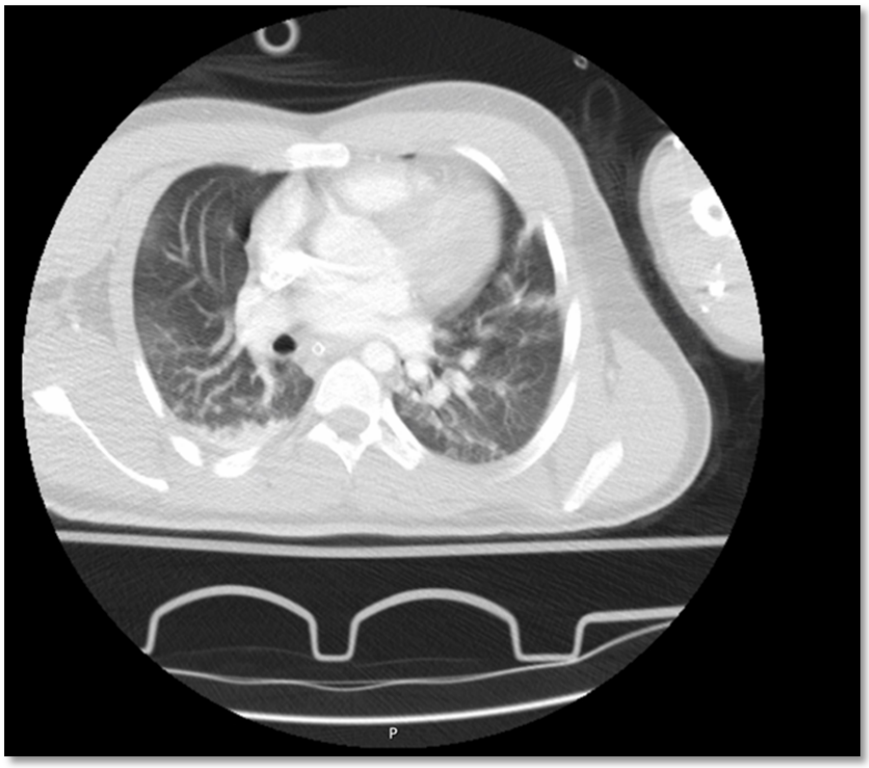

Figure 4. Thorax CT of the patient (contusion).

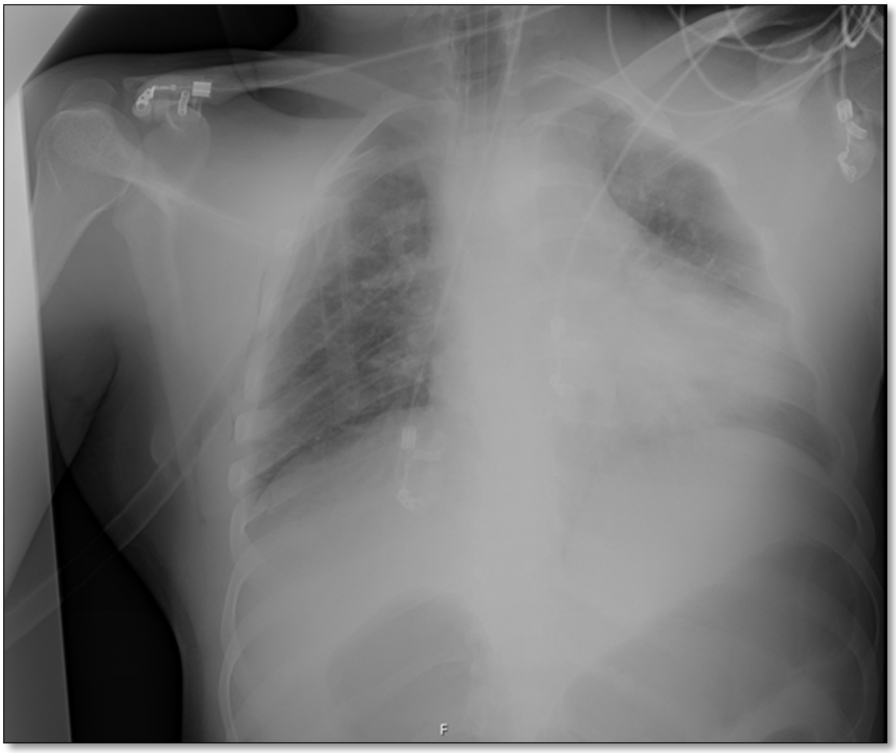

Figure 5. Anteroposterior (AP) chest graphy of the patient.

Sequential electrocardiography follow and detailed echocardiography should be performed in these patients. Our patient only had sinus tachycardia with normal echo findings with an EF of 55%. The Typical appearance seen in these patient group is because of the ruptured cranial veins, venules and capillaries [8]. Petechial hemorrhages and hypoxia are also expected in Perthes's syndrome. Our patient had edematous eyes, subconjunctival hemorrhage with a very prominent ecchymotic mask, a swelled face. Veins lacking valves like external jugular vein are the reason for facial ecchymosis. Valves veins are shown to be effective of preventing regurgitation up to 45 mmHg [9]. Our patient did not show severe hypoxia. It may be due to the compression time, ageor pressure power. Due to sudden venous regurgitation and excessive pressure, traumatic asphyxia patients show signs of cerebral edema clinically and radiologically [6]. Our patient had a mild cerebral edema seen in cranial CT. However, most of these patients get chest compressions before or at the time of emergency admission, hence we are not sure if the edema was a result cardiopulmonary resuscitation or trauma itself. Cerebral congestion may be superimposed with cerebral anoxia. Agitation and disorientation are expected to be seen in mild cases. Neurological outcome is variable due to the severity of trauma. Treatment is conservative. Supportive therapy includes airway management, treating hypoxia and fluid resuscitation. Patients generally need mechanical ventilation [10]. In order to let intracranial pressuredecrease, patient's head may be elevated to 30°. Cerebral damage may be mild to severe and should be treated. There are patients discharged with cerebral sequelae, sent to home care [11, 7].

Oxygenation should be monitored with blood gas analysis and renal functions with biochemistry laboratory. Our patient had acute renal failure requiring renal replacement therapy. Crush syndrome should also be considered in these patient group where large tissue is damaged and squeezed [6]. Our patient was monitored for possible crush syndrome. CPK levels peaked on the second day and decreased gradually. Our case is a severe traumatic asphyxia patient who just after the trauma, got sudden arrest and never woke up. Supportive therapy did not make any improvements and patient lost his life in less than a week time. Clinicians should be careful about Perthes's syndrome while evaluating all trauma patients.

CONCLUSION

Traumatic asphyxia patients should all be recognized in emergency wards and critical care units considering the severity of the trauma. Possible hypoxia should be promptly treated. Care should also be taken while resuscitating the patient as there may be possible rib or sternum fractures and pneumothorax.

ETHICS APPROVAL & CONSENT TO PARTICIPATE

Patient was unconscious for the whole hospital stay. Approved consent for the use of medical data was taken from his parents.

CONSENT FOR PUBLICATION

Written informed consent was obtained from the father of the patient as legal guardian for publication of this case report. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

AVAILABILITY OF DATA AND MATERIAL

Some of the data analyzed during this study are included in this published article. The remaining datasets generated during and analyzed during the current case report are available from the corresponding author on reasonable request.

COMPETING INTERESTS

The authors declare that they have no competing interests in this case report.

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AUTHORS' CONTRIBUTIONS

FSY wrote the manuscript. FSY acquired patient data. FSY and AE reviewed the case notes and were major contributors in writing the manuscript. All authors read and approved the final manuscript.

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