Formosan Journal of Surgery (2015) 48, 168–171

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.e-fjs.com

CASE REPORT

Traumatic asphyxia combined with diffuse axonal injury

Chi-Yi Lin*, **Yu-Kun Chen**, **Chun-Hsiung Huang**

Division of General Surgery, Department of Surgery, Armed Forces Taoyuan General Hospital, Taoyuan, Taiwan

Received 10 September 2014; received in revised form 29 December 2014; accepted 13 May 2015
Available online 19 August 2015

KEYWORDS
diffuse axonal injury;
magnetic resonance imaging;
traumatic asphyxia

Abstract Traumatic asphyxia, a rare, blunt chest trauma-related condition, indicates severe injury and is characterized by subconjunctival hemorrhage, facial edema, cyanosis, and petechiae. This condition mostly appears on the upper chest and face. Rapid oxygen administration with effective ventilation is essential in the treatment of traumatic asphyxia. Prognosis depends on rescue time and associated injuries. Most neurologic symptoms resolve within 24–48 hours and have relatively satisfactory results over a long-term follow-up. We herein report the case of severe and complicated thoracoabdominal compression with a delayed change in consciousness. Susceptibility-weighted magnetic resonance imaging revealed diffuse axonal injury with multifocal microhemorrhages in the brain stem, basal ganglia, internal capsules, and the genu and splenium of the corpus callosum. The patient was in the intensive care unit for more than 21 days.

1. Introduction

Accidents resulting in blunt chest trauma incidences are increasingly common and can potentially result in death if diagnosis is incorrect or if treatment is delayed. Traffic-related accidents are the most common causes of blunt chest trauma.[1,2] Traumatic asphyxia, first mentioned by Ollivier in 1837,[3] is a rare, blunt chest trauma-related condition and an indicator of severe injury, including subconjunctival hemorrhage, facial edema, cyanosis, and petechiae on the upper chest and face,[4] with compression of the chest or upper abdomen being the causal factor. Associated injuries, such as pulmonary contusion, rib fractures, hemopneumothorax, diaphragmatic rupture, and myocardial injury, have also been described.[2,4–6]

Conflicts of interest: The authors declare no conflicts of interest.

* Corresponding author. Department of Surgery, Armed Forces Taoyuan General Hospital, Number 168, Zhongxing Road, Longtan Township, Taoyuan Country 32551, Taiwan.
E-mail address: thanksthegod@gmail.com (C.-Y. Lin).

http://dx.doi.org/10.1016/j.fjs.2015.05.002

2. Case Report

A healthy 25-year-old Vietnamese man working at an electronics factory was involved in an accident while cleaning the factory conveyor belts. This resulted in the compression of his thorax and limbs. He was extricated from the machine and was conscious and alert. The Glasgow Coma Scale (GCS) score upon arrival to hospital was 15 (E4M6V5). His vital signs were as follows: blood pressure, 151/94 mmHg; heart rate, 167 beats/min; respiratory rate, 18 breaths/min; and body temperature, 36.6°C. Physical appearance was characterized by facial cyanosis with bilateral subconjunctival hemorrhage, multiple abrasive wounds from the left chest to the left arm, and deformity of the left upper arm and thigh (Figure 1). Chest X-ray revealed a fracture in the left ribs (third to eighth ribs to be precise) with bilateral pulmonary hemorrhages (Figure 2). Additional X-rays showed a midshaft left humerus fracture, a midshaft left femur fracture, and proximal left tibia and fibula fractures. Chest-to-pelvis computed tomography (CT) revealed multiple rib fractures, left hemothorax, right pneumothorax, and pulmonary contusion (Figure 3). After 20 minutes, a sudden decline of O_2 saturation from 98% to 88% was observed, and an endotracheal tube intubation with mechanical ventilator support was arranged. Tube thoracostomy was performed because of decreased respiration on the left side, and the patient was admitted to the intensive care unit. After 2 days, the patient developed a change in consciousness and underwent a follow-up brain CT. There was no obvious hemorrhage or ischemic change. An electrolyte level analysis revealed an elevated serum creatine kinase concentration. Massive hydration with 3 L of isotonic saline/d and mannitol was administered. Because no change in the GCS score was observed, brain magnetic resonance imaging (MRI) was performed and extensive microhemorrhaging in the brain stem, basal ganglia, internal capsules, and the genu and splenium of the corpus callosum was observed (Figure 4). Therefore, a diffuse axonal injury was diagnosed. The patient's consciousness progressively improved after approximately 27 days of hospitalization, and he was transferred to the ward.

3. Discussion

Traumatic asphyxia has characteristic pathophysiologic mechanisms, including elevation of thoracoabdominal pressure with closure of the glottis and the airway (Figure 5).[1,5,6]

Venous backflow and an increasing capillary pressure may cause inadequate tissue perfusion and capillary rupture. The most common clinical manifestations are craniocervical cyanosis, edema, and petechiae; in addition, neurologic symptoms are commonly associated with traumatic asphyxia and determine the patient outcome.[5–7] Brain edema and central nervous system ischemia have been described in various cases. In 1992, Jongewaard et al[8] reported 14 cases of sustained thoracic compression associated with varying degrees of neurologic symptoms, including loss of consciousness, brachial plexopathy, visual

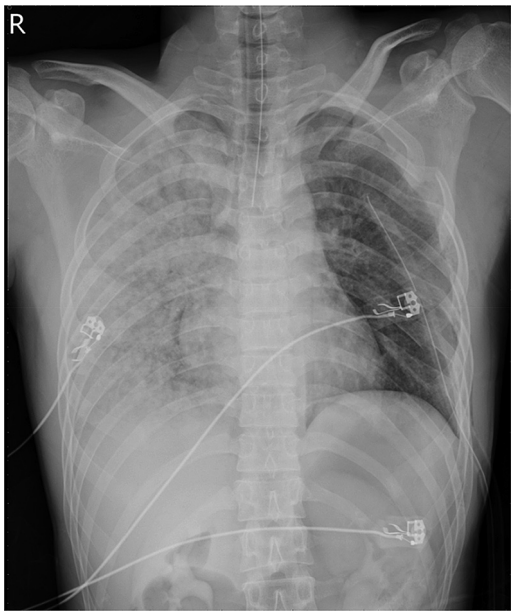

Figure 2 Chest radiography after tube thoracostomy was performed. Multiple rib fractures on the left side with bilateral pulmonary hemorrhages are clearly visible.

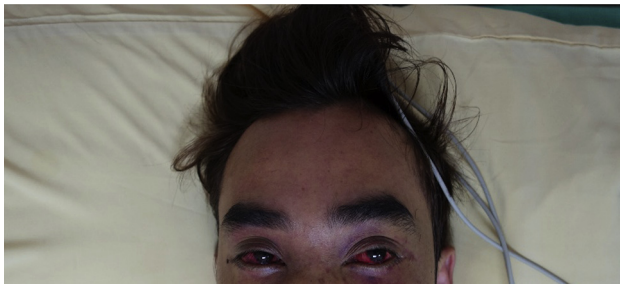

Figure 1 Patient with bilateral subconjunctival hemorrhage due to a crushing injury to the thorax.

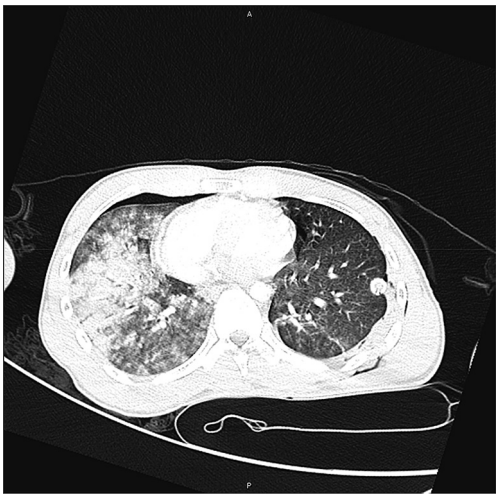

Figure 3 Computed tomography chest scan shows left hemothorax, right pneumothorax, and pulmonary contusion.

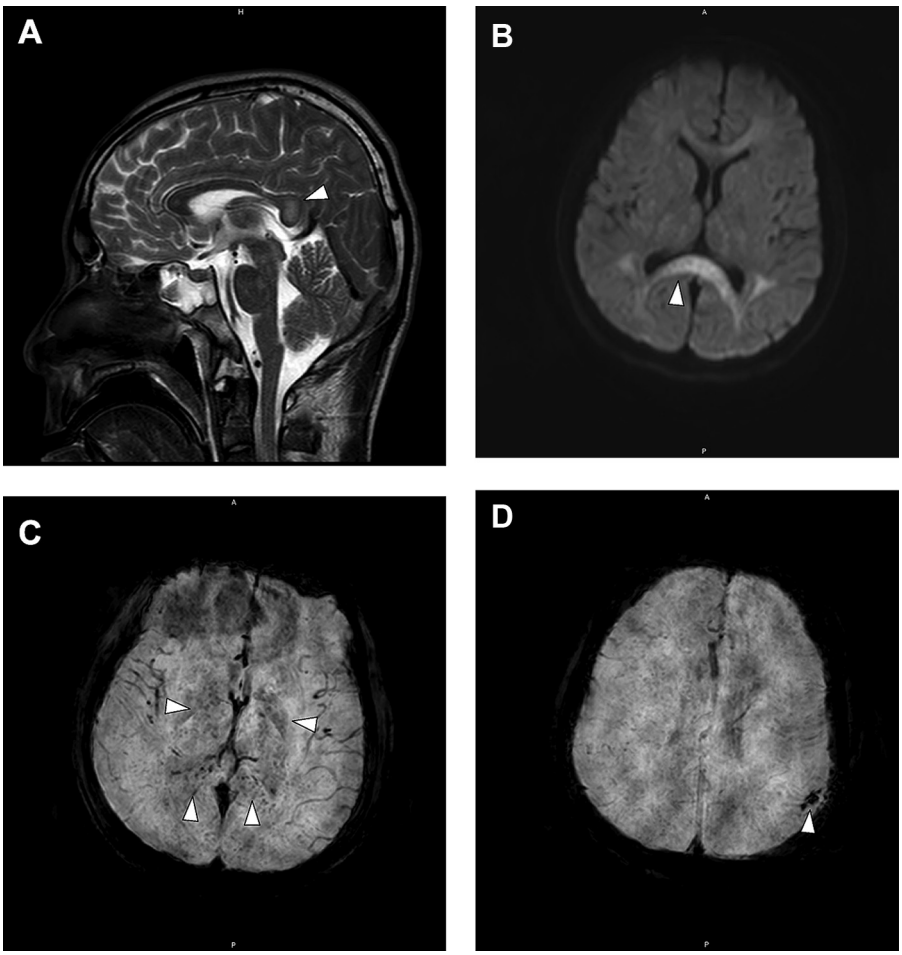

Figure 4 (A) Sagittal T2-weighted magnetic resonance image (MRI) of this patient shows a hyperintense axonal injury lesion involving the corpus callosum (arrowhead); (B) axial diffusion-weighted MRI shows a hyperintense injury in the corpus callosum splenium (arrowhead); (C) axial susceptibility-weighted MRI shows multifocal microhemorrhages of the basal ganglion and corpus callosum (arrowhead); and (D) axial susceptibility-weighted MRI shows a cortical microhemorrhage over the left parietal lobe (arrowheads).

disturbance, and paralysis. In 2008, Senoglu et al[9] reported Perthes syndrome associated with an intramedullary spinal cord hemorrhage in a 4-year-old girl who sustained thoracic compression. In the present case, the patient experienced a temporary head contusion when he was roughly extricated from the machine by his colleague but was conscious and alert. The patient showed a delayed change in consciousness and MRI revealed brain lesions.

Traumatic asphyxia combined with diffuse axonal injury has not been reported previously. Cervical venous drainage in the head and neck is divided by internal and external jugular veins. The external jugular vein has two pairs of valves, including the lower pair at its entrance into the subclavian vein and the upper pair approximately 4 cm above the clavicle. The internal jugular vein contains a pair of valves, which are bilaterally placed 2.5 cm above the termination of the vessel. They are protected against venous backflow to the face and brain when the vascular pressure is less than 45 mmHg.[1,9] Venous hypertension and capillary rupture may result in tissue or organ hemorrhages, including bulbar conjunctiva hemorrhage and facial and cerebral petechiae.[1,7,8] The definite mechanism of diffuse axonal injury is the acceleration and deceleration of inertial forces, and the microscopic features include axonal stretch injury and Wallerian-type axonal degeneration. The image-specific findings of diffuse axonal injury show multifocal puncture hemorrhages at the corticomedullary junction, corpus callosum, deep gray matter, and upper brain stem. Brain CT findings may often be normal but MRI may show obvious injury. A T2-weighted MRI demonstrated multifocal hyperintense foci at characteristic locations, diffusion-weighted MRI demonstrated hyperintense foci of restricted diffusion, and susceptibility-weighted MRI depicted more than T2* gradient-echo sequence. Clinicians should be aware of the fact that diffuse axonal injury can accompany traumatic asphyxia.

4. Conclusion

Traumatic asphyxia combined with diffuse axonal injury is extremely rare. Early neurological and ophthalmic examinations can detect neurologic defects and prevent further complications. However, favorable neurologic outcomes are determined by a short rescue time and airway maintenance. In the present case, a delayed change in consciousness occurred after normal brain CT. Brain MRI at an

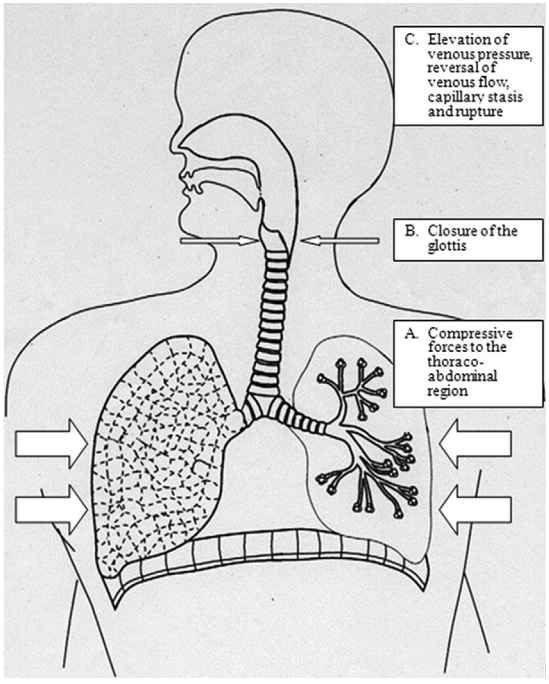

Figure 5 Pathophysiological mechanism of traumatic asphyxia.

early stage may have provided sensitive findings and influenced the therapeutic strategy.

References

1. 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—116.
2. Kamali S, Kesici S, Gunduz I, Kesici U. A case of traumatic asphyxia due to motorcycle accident. *Case Rep Emerg Med*. 2013;2013:857131.
3. Ollivier DA. Relation médicale des événements survenus au Champs-de-Mars le 14 Juin, 1837. *Ann Hyg*. 1837;18:485—494.
4. Dunne JR, Shaked G, Golocovsky M. Traumatic asphyxia: an indicator of potentially severe injury in trauma. *Injury*. 1996;27:746—749.
5. Sertaridou E, Papaioannou V, Kouliatsis G, Theodorou V, Pneumatikos I. Traumatic asphyxia due to blunt chest trauma: a case report and literature review. *J Med Case Rep*. 2012;6:257.
6. Jobé J, Ghuysen A, Hartstein G, D'orio V. A fatal case of Perthes syndrome. *J Emerg Trauma Shock*. 2013;6:296—297.
7. Landercasper J, Cogbill TH. Long-term followup after traumatic asphyxia. *J Trauma*. 1985;25:838—841.
8. Jongewaard WR, Cogbill TH, Landercasper J. Neurologic consequences of traumatic asphyxia. *J Trauma*. 1992;32:28—31.
9. Senoglu M, Senoglu N, Oksuz H, Ispir G. Perthes syndrome associated with intramedullary spinal cord hemorrhage in a 4-year-old child: a case report. *Cases J*. 2008;1:17.