A Retrospective Study of 51 Pediatric Cases of Traumatic Asphyxia

luo huirong
Chongqing Medical University Pediatric College: Chongqing Medical University Affiliated Children's Hospital
https://orcid.org/0000-0002-2741-2238

xin jin (jinxincq@163.com)
Chongqing Medical University Pediatric College: Chongqing Medical University Affiliated Children's Hospital
https://orcid.org/0000-0001-9549-6704

Research Article

Keywords: Traumatic asphyxia, pediatric, thoracic trauma

DOI: https://doi.org/10.21203/rs.3.rs-357514/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License. Read Full License
Abstract

Background

traumatic asphyxia (TA) is a rarely reported disease characterized as thoraco-cervico-facial petechiae, facial edema and cyanosis, subconjunctival hemorrhage and neurological symptoms. This study aimed to report 51 children of TA at the pediatric medical center of west China.

Methods

scanned medical reports were reviewed and specific variables as age, sex, cause of injury, clinical manifestations and associated injuries were analyzed using SPSS 25.0.

Results

aged as 5.3±2.9 (1.3-13.2), 30 (58.8%) were boys and 21 (41.2%) were girls. Most TAs occurred during vehicle accident, object compression and stampede. All patients showed facial petechiae (100.0%, CI 93.0%-100.0%), 25 (49.0%, CI 34.8%-63.2%) out of 51 presented with facial edema, 29 (56.9%, CI 42.8%-70.9%) presented with subconjunctival hemorrhage, including bilateral 27 and unilateral 2. 6 patients had facial cyanosis (11.8%, CI 2.6%-20.9%). Other symptoms were also presented as epileptic seizure, vomiting, incontinence, paraplegia, etc.

The most frequent companion injury was pulmonary contusion (76.5%, CI 64.4%-88.5%). Other companion injuries included mediastinal emphysema, fracture, cerebral contusion and hemorrhage, hypoxic-ischemic brain injury, abdominal organ contusion, mastoid hemorrhage, hematocele of paranasal sinuses, spinal injury, hepatic insufficiency, myocardial injury and retinal hemorrhage and edema. Treatment was mainly supportive. No death occurred in our study. The prognosis is rather good if without damage of central nervous system.

Conclusion

TA could bring out multiple symptoms, among which retinal hemorrhage and edema, spinal injury and viscera impairment have been less observed. Comprehensive physical and auxiliary examination should be performed considering TA. Its prognosis is rather good with focus on life-threatening complications.

Introduction

Firstly described in 1837, traumatic asphyxia is characterized as thoraco-cervico-facial petechiae, facial edema and cyanosis, subconjunctival hemorrhage and neurological symptoms. \(^1\-^3\) TA could be triggered with or without external forces, so it also bears the name as compression asphyxia and crush asphyxia. \(^4\) The incidence of TA was unclear with possible underestimation. \(^5\) Usually caused by vehicle rolling over, object compression, it could also be presented from entrapment and uncontrolled crowd as Hillsborough tragedy. \(^6\) The treatment is supportive with focus on severe associated injuries. Here we reported 51
pediatric TA cases presented for treatment during May 2005 and November 2020 at the pediatric medical center of west China.

Methods

This retrospective study evaluated records of 51 children of TA between May 2005 and November 2020 at the pediatric medical center of west China. Inclusion criteria: 1. TA typically occurs as petechiae in the face, neck and chest or conjunctival hemorrhage or facial edema and cyanosis; 2. Diagnosed as ‘traumatic asphyxia’, ‘thoracic crush syndrome’ or ‘superior vena cava crush syndrome’ during hospitalization; 3. Admission within 10 days after trauma; Exclusion Criteria: 1. Incomplete medical records; 2. inconsistency between symptoms and examination results; 3. Outpatient medical records; 4. Lost to follow-up.

Data collection followed previously published guidelines on retrospective chart review. The electronic medical record browser of patients’ hospitalization and the platform of big data in our hospital were used to search for all TA patients. Without blindness, two already trained abstractors (HRL, XJ) abstracted data from the scanned medical records, including physician and nursing notes and auxiliary examinations.

This study recorded the following variables: age, sex, cause of injury, department of admission, way of admission, vital signs in admission, past medical history, initial Glasgow Coma Score (GCS), loss of consciousness, initial CT results of head, chest, abdomen and extremities, MRI results of head and spine, testing results of myocardial markers and liver function, routine urine test. Symptoms as facial edema and cyanosis, petechiae on the face, neck and chest, subconjunctival hemorrhage, hemorrhage of ear, nose and oral mucosa, vomiting, seizure, incontinence, retinal hemorrhage and edema were also recorded. Pediatric intensive care unit (PICU) stay and hospital length of stay (LOS) were also recorded. Other records were viewed without a standardized form, such as companion injuries, treatment with drugs, surgeries, blood infusion and other interventions.

SPSS 25.0 (IBM SPSS Statistics for Windows, Version 25.0, Armonk, NY) was applied for data analysis to calculate 95% confidence interval (CI). Normal approximation interval (standard CI) were calculated for proportions if $n \geq 30$ and $np>5$ and $n(1-p)>5$. Exact 95% CIs (Clopper-Pearson Confidence Interval) were used for smaller sample. Mean ± standard deviation was used to describe age. Range (Min-Max, Median) was used for length of stay (LOS) and length of stay in PICU. Categorical data were analyzed with Fisher's exact test in cases of small cell size. A Kruskal-Wallis test was used to compare quantitative data which were not normally distributed. P<0.05 was deemed statistically significant.

This retrospective study has been conducted with approval of the Ethics Committee of the Children's Hospital of Chongqing Medical University (Approval Number: (2021) Institutional Review Board (IRB) (STUDY) No.44).

Results
Characteristics

Through rough research, we collected data of 87 patients with TA diagnosis, including 20 children from outpatient, 8 repeated data of patient, 3 patients of inconsistent symptoms with TA diagnosis, 2 patients with incomplete hospitalized data, 2 patients admitted in 10 days after trauma and 1 patient discharged automatically without proper treatment. Finally, 51 children were included in our study. They were admitted with TA between May 2005 and November 2020 at the pediatric medical center of west China, 30 (58.8%) were boys and 21 (41.2%) were girls. 41 cases were transferred in from inferior hospitals, 5 were admitted from outpatient department and 5 were from emergency department. Their average age was 5.3±2.9 (1-13) years old. The causes of TA were traffic accident in 34 cases, object compression in 10, stampede in 5, knife stab in 1 and falling from high and compressed by an adult in 1. The descriptive features of the causes were presented in Table 1. Grouped by cause of injury, only ages were statistically different. 35 out of 51 (68.63%) had vital signs that were within normal limits. During admission, apart from a patient by knife stab with vital signs as unattainable temperature and blood pressure, pulse of 25 bpm, respiration of 20 under respiratory machinery, and oxygen saturation as 25%, 4 children got fever over 37.3 °C, 11 children got respiration more than 30 times per minute with highest as 45, and 5 children got heart rate over 130 beats per minute with the highest as 170. All patients got a clean past medical history.

Symptoms

All patients (100.0%, CI 93.0%-100.0%) showed petechiae on the upper body, especially on face. 25 (49.0%, CI 34.8%-63.2%) out of 51 presented with facial edema. As for subconjunctival hemorrhage, 29 (56.9%, CI 42.8%-70.9%) presented with subconjunctival hemorrhage, including bilateral 27 and unilateral 2. 6 patients had facial cyanosis (11.8%, CI 2.6%-20.9%). The photo of 1 patient was shown as figure 1. 22 children went through loss of consciousness within minutes or days, 3 children got confusion within days, and the other 26 remained conscious after injury. The change of neurological states with time was shown in figure 2. during admission, 82.4% (42/51) of patients were of GCS 15, 7.8% (4/51) were of GCS 13-14, 7.8% (4/51) were of GCS 4-7. 6 patients went through epileptic seizure, 5 vomited, 11 patients got incontinence after injury and 2 presented with paraplegia. Patients also showed nasal hemorrhage in 7, ear hemorrhage in 5, oral mucosa hemorrhage in 2 and hemoptysis in 1. Other symptoms included 2 cardiac arrest within minutes after injury, exophthalmos in 1 and blurred vision in 1. All symptoms were presented in table 2 with numbers and percentages.

Companion injuries

All patients had CT scan of head, thorax and abdomen within 24h of injury. Some also got results of MRI, ultrasonic inspection and fundus. Based on all those images and laboratory tests, only 3 patients got no companion injuries except skin abrasion. Companion injuries were presented as thoracic, craniocerebral, abdominal, craniocerebral, and other injuries as shown in Table 3. 39 (76.5%, CI 64.4%-88.5%) patients
got pulmonary contusion (bilateral in 31 and unilateral in 8) with varying degrees of hemothorax and pneumothorax. 5 patients (9.8%, CI 1.4%-18.3%) got mediastinal emphysema. 7 patients (13.5%, CI 3.9%-23.1%) got multiple rib fracture, 2 patients (3.9%, CI 1.4%-13.6%) got single rib fracture. 6 patients (11.8%, CI 2.6%-20.9%) got clavicle fracture. 8 patients (15.7%, CI 5.4%-26.0%) showed cranial fracture and 6 patients (11.8%, CI 2.6%-20.9%) got cerebral contusion or hemorrhage, including 1 cerebral contusion and subarachnoid hemorrhage, 1 cerebral contusion and subdural hemorrhage, 2 cerebral contusion and 1 epidural hemorrhage. 10 patients (19.6%, CI 8.3%-30.9%) got abdominal injury, including liver contusion in 6 (11.8% CI 2.6%-20.9%), kidney contusion in 5 (9.8%, CI 1.4%-18.3%), spleen contusion in 2 (3.9%, CI 1.4%-13.6%), pancreas injury in 1 (2.0%, CI 0.0%-10.5%) and adrenal injury in 1 (2.0%, CI 0.0%-10.5%).

22 patients (43.1%, CI 29.1%-57.2%) got other kinds of fractures including fracture in extremities, pelvis, scapula and vertebrae. 7 patients (13.5%, CI 3.9%-23.1%) got mastoid hemorrhage and 10 (19.6%, CI 8.3%-30.9%) got hematocoele of paranasal sinuses. 3 patients (5.9%, CI 1.2%-16.2%) got straingthing or reversal of cervical lordosis. 3 patients (5.9%, CI 1.2%-16.2%) got atlantoaxial subluxation. There showed 3 cases of spinal injury with spinal MRI characterized as abnormal signal as edema and hemorrhage between or below specific thoracic fragments, among whom paraplegia was also noted as temporary or possibly permanent. There was also evidence of hematuria in 6 patients (11.8%, CI 2.6%-20.9%). With insufficiency of specific examinations, we found 26 out of 35 (74.3%, CI 59.1-89.5%) presenting with myocardial injury defined as elevation of creatine kinase isoenzyme-MB (CK-MB) and cardiac troponin (cTn) within 3 days after hospitalization and 26 out of 49 (53.1%, CI 38.6%-67.5%) showing hepatic insufficiency defined as elevation of glutamic-pyruvic transaminase, glutamic oxaloacetic transaminase and lactate dehydrogenase within 2 days after hospitalization without considering abnormal hepatic function caused by definitive liver contusion.

We also found 3 retinal hemorrhage and edema from 15 fundus (20.0%, CI 4.3%-48.1%), among which the optical coherent tomography (OCT), fundus and thickness map was presented in Figure 3.

**Treatment and prognosis**

Patients were hospitalized into different departments, including 19 in neurosurgery department, 15 in cardiothoracic surgery, 6 in orthopedics, 5 in hepatobiliary surgery, 4 in critical medicine, 1 in traumatology and 1 in burn and plastic surgery, among whom 3 patients were transferred into rehabilitation department for further treatment because of spinal injury in 2 and severe cerebral ischemia in 1. The hospital length of stay (LOS) of all patients was 3-73 days (Median 15 days). 10 patients went into Pediatric Intensive Care Unit for 1-43 days (Median 4 days). All patients got supportive treatment as hemostatic drugs, oxygen uptake and stay in bed with raised bed head by 15-30°. The 2 arrested patients received successful cardiopulmonary resuscitation. 2 patients received noninvasive ventilator assisted ventilation, 7 patients received invasive ventilator assisted ventilation and positive end expiratory pressure (PEEP) was applied for all with pressure between 4-10 cmH2O. Patients with multiple rib fracture
accepted external rib fixation. 3 received closed thoracic drainage because of pneumothorax or hemopneumothorax. 8 patients received surgeries, including 5 debridement suture and 3 open reduction internal fixation for extremity fracture. 6 patients received hormone treatment, including 2 hormone bolus for spinal injury, 3 low-dose drip and 1 nebulization for pulmonary injuries. 14 patients got blood infusion due to loss of blood. Other treatment included external fixation of fracture or atlantoaxial subluxation, bronchofiberscope lavage, rehabilitation treatment, reduction of intracranial pressure by mannitol, and sedation.

No death occurred in our study. 47 (92.2%) patients were discharged without severe sequelae. The other 4 patients with sequelae included 3 patients of spinal injury and 1 patient of severe hypoxic-ischemic brain injury.

**Discussion**

Traumatic asphyxia (TA), namely, perthes syndrome, thoracic compression syndrome, superior cava vena compression syndrome, has been rarely reported in literature, but its incidence is likely to be underestimated.\(^8\) It was initially reported by Ollivier d'Angers in an entrapment accident in France, and it was further illustrated by perthes with focus on neurological symptoms.\(^1-2\) Later, TA was found in car accident, object compression, entrapment or even without a large external force, such as crying, asthma, epilepsy.\(^3\)

The recognized mechanism of TA was the counterforce between respiration and thorax, and increase of central venous system by thoracic compression and fear response\(^9\), leading to superior vena cava (SVC) obstruction with incompetent valves to inhibit flowing back, arterial low perfusion and hypoxia, which could cause facial edema and cyanosis, neurological symptoms, petechiae of upper body and hemorrhage of conjunctiva, retina, ear, nose, oral mucosa, bronchi, etc.\(^3\)

In our study, all patients showed petechiae on the upper body, especially on face. 25 patients (49.0% CI 34.8%-63.2%) out of 51 presented with facial edema. As for subconjunctival hemorrhage, 29 patients (56.9%, CI 42.8%-70.9%) presented with subconjunctival hemorrhage. It is consistent with previous literature except that subconjunctival hemorrhage was of less percentage\(^10\). Compared to other existing literature were subconjunctival hemorrhage was seldomly absent\(^11\), we got a much lower occurrence as 56.86%. This is possibly due to observation negligence or lacked medical recording by doctors who were not familiar with TA manifestations. Also, the incidence of facial petechiae was higher than previously reported, the reason for which might be that doctors are more possible to consider diagnosis of TA when encountering such characteristic appearance.

Hemorrhages of nose and ear were common in appearance and auxiliary examinations in this series. There were 7 mastoid hemorrhage and 10 hematocoele of paranasal sinuses. Patients also showed nasal hemorrhage in 7, ear hemorrhage in 5, oral mucosa hemorrhage in 2 and hemoptysis in 1. Hemorrhage of mastoid and paranasal sinuses is possibly related to venous stasis and fear response through
Eustachian tube and imbalanced pressure between middle ear and thorax with possible barotrauma.\textsuperscript{12} As both brain and other region of head have veins belonging to SVC, it is reasonable to assume that hemorrhages out of brain could reduce the cerebral impairment as a protective reaction.\textsuperscript{12}

There are differing neurological symptoms as loss of consciousness, confusion, epileptic seizure.\textsuperscript{13} In our study, 22 children underwent loss of consciousness within minutes or days, 3 children got confusion within days, and 25 remained conscious after injury. 6 patients went through epileptic seizure, 5 vomited, and 11 got incontinence after injury, which may be correlated to both TA itself by circulatory abnormality and companion injuries as head bump. These symptoms revolved within days without surgical intervention except debridement suture. Only a patient with severe hypoxic-ischemic brain injury remained intellectual disability. Both respiratory inhibition and hemodynamic disturbances contribute to cerebral impairment in TA.\textsuperscript{13-14} However, their respective role is yet to be determined with not striking pathologic changes except congested vessel and petechial perivascular hemorrhage.\textsuperscript{15}

We want to highlight the evidence in viscera impairment of TA. There were autopsy evidence of subepicardial and subpleural petechiae.\textsuperscript{12} Children got a less rigid thorax with intolerance to compression. Thus, the inner organs could suffer more from the compression with danger in cardiac and pulmonary impairment.\textsuperscript{16} The potential mechanism of myocardial injury is the compression itself, the anoxia, and the cardiac capillary rupture due to venous hypertension as cardiac petechiae was found in TA victims.\textsuperscript{12} We found a high percentage as 74.3\%, indicating necessity in evaluation and intervention of cardia for TA patients. The mechanism of fear response, including deep inspiration, closure of the glottis, splinting of the thoracic and abdominal musculature, and chest compression, might provide protection to heart with elevated intra-thoracic pressure.\textsuperscript{17} In addition, we found a percentage of hepatic insufficiency as 53.1\% while the role of inferior vena cava (IVC) change in TA has been less focused in literature, even although there are evidence of increased pressure in IVC and visceral changes as “nutmeg” liver or liver petechiae during autopsy.\textsuperscript{12,18}

Also, the evaluation of retina through fundus is necessary. We found an incidence of 20.0\% (CI 4.3\%-48.1\%) from 15 patients in retinal hemorrhage, edema and exudates. Based on the result of 1 patient (\textit{Figure 3}), the retina was hemorrhagic with edema and exudates around the optic disc in the left eye. Moreover, the optic disc was pushed temporally, thus with optic nerve compression. The low-dosage dexamethasone was used as treatment for 3 days. However, in the 1-month and 1.5-month follow up, the retina still turned atrophy and optic disc was drafted nasally. It is reasonable to assume that the impairment is irreversible and could lead to lifelong amblyopia. Based on that, this evaluation is especially important for children as they are not capable to express feelings clearly and optic impairment could make its influence throughout their life.\textsuperscript{19}

As for other companion injuries, they are the main factors determining the prognosis and pulmonary contusion was the most frequent concomitant injury both in our study and other literature.\textsuperscript{20} Pulmonary parenchymal injury results from several mechanisms, including direct compression, counter-coup
compression, shearing forces and laceration by fractured ribs.\textsuperscript{21} Interestingly, flail chest has not been found in our study. Accordingly, apart from suffocation, associated injuries are more causal in death instead of TA itself.\textsuperscript{22}

In addition, based on our study, there are 3 spinal injury. Spinal injury deserves more attention as a severe injury correlating intimately with prognosis. Spinal injury was seldom reported with assumed injury mechanism as spinal anoxia and unstable blood flow.\textsuperscript{23} However, we considered spinal injury as concomitant injury because spinal fracture often accompanies and spinal MRI usually showed spinal edema and abnormal signal from a specific segment, especially from the thorax.

No death occurred in our study, which is consistent with previous literature that TA got a rather good prognosis. This might be due to their thoracic wall elasticity.\textsuperscript{12} Death of traumatic asphyxia is mostly caused by large compression force, compression overtime, and associated injuries.\textsuperscript{12} The prognosis of TA depends on the severity of prognostic-related injuries.

**Limitations and strengths**

As a retrospective study, the medical record might be imperfect because facing the rarely encountered TA by doctors from different departments, some symptoms might be neglected or misjudged. We failed to collect enough data about the forces causing traumatic asphyxia. There lacked accurate description of accident details as mechanism, time and patient’s state after injury. The examinations were not complete for myocardial markers, hepatic function, fundus and urine routine. CT scan was not completed immediately after injury or admission, which not only interferes our analysis with possibly lower positive rates in statistics but also probably inhibit proper and timely treatment for concomitant injury.

Meanwhile, as a large and highly ranked medical center for children, we have collected 51 pediatric cases in TA, which is, from the acknowledgement of authors, of the largest sample ever. We reported also the rare OCT, fundus and thickness map results with comparison between admission and follow-up, thus drawing attention to the importance of retinal evaluation of TA.

**Conclusions**

We reported 51 pediatric cases of TA which were mostly caused by traffic accident, object compression and stampede. Petechiae in facial, cervical and thoracic region, facial edema and cyanosis, subconjunctival hemorrhage and neurological symptoms were commonly found while the injury of heart, lung, liver, spine and retina should not be neglected. Comprehensive physical and auxiliary examination should be performed after considering TA. No specific treatment other than supportive one was required for TA itself. The prognosis is rather good with timely intervention for companion injuries if central nervous system was not severely damaged.
Declarations

Ethics approval and consent to participate: this study has been conducted with approval of the Ethics Committee of the Children's Hospital of Chongqing Medical University (Approval Number: (2021) Institutional Review Board (IRB) (STUDY) No.44). Figure 1 has been presented with approval of patient's legal guardian.

Consent for publication: publication has been approved by all authors in this article.

Availability of data and materials: statistical data are available from all authors.

Competing interests: none.

Funding: none.

Authors' contributions: concept and design of Study: HRL, XJ; Acquisition of Data: HRL, XJ; Analysis of Data: HRL; Drafting of Manuscript: HRL, XJ; Revision of manuscript critically for important intellectual content: XJ. Approval of final manuscript: XJ, HRL. Acknowledgements: We thank the department of surgical emergency in CQMU to provide the availability of those rare cases in our study.

References

1. D. A. Ollivier. Relation medicale des événement survenus au Champs-de-Mars le 14 juin, 1837. Ann d’Hyg 1837;18:485-89.
2. Georg Perthes. Ueber „Druckstauung“. Deutsche Zeitschrift für Chirurgie 1900;55(3):384-92. doi: 10.1007/BF028161331900-01-01.
3. F. Montes-Tapia, I. Barreto-Arroyo, I. Cura-Esquível, A. Rodríguez-Tamez, M. de la O-Cavazos. Traumatic asphyxia. PEDIATR EMERG CARE 2014;30(2):114-16. doi: 10.1097/PEC.0000000000000067 pmid:244881622014-02-01.
4. Guy N. Rutty. Traumatic, Crush and Compression Asphyxia Including 'Burking'. In: Burkhard Madea, ed. Asphyxiation, Suffocation, and Neck Pressure Deaths: CRC Press, 2021:222.
5. Y. A. Karamustafaoglu, I. Yavasman, S. Tiryaki, Y. Yoruk. Traumatic asphyxia. Int J Emerg Med 2010;3(4):379-80. doi: 10.1007/s12245-010-0204-x pmid:21373082010-08-25.
6. J. Wardrope, F. Ryan, G. Clark, et al. The Hillsborough tragedy. BMJ 1991;303(6814):1381-85. doi: 10.1136/bmj.303.6814.1381 pmid:17606071991-11-30.
7. A. Worster, R. D. Bledsoe, P. Cleve, et al. Reassessing the methods of medical record review studies in emergency medicine research. ANN EMERG MED 2005;45(4):448-51. doi: 10.1016/j.annemergmed.2004.11.021 pmid:157957292005-04-01.
8. J. R. Dunne, G. Shaked, M. Golocovsky. Traumatic asphyxia: an indicator of potentially severe injury in trauma. INJURY 1996;27(10):746-49. doi: 10.1016/s0020-1383(96)00113-1 pmid:91357581996-12-01.
9. J. S. Williams, S. L. Minken, J. T. Adams. Traumatic asphyxia—reappraised. *ANN SURG* 1968;167(3):384-92. doi: 10.1097/00000658-196803000-00012 pmid:56385231968-03-01.

10. R. W. Byard, R. Wick, E. Simpson, J. D. Gilbert. The pathological features and circumstances of death of lethal crush/traumatic asphyxia in adults—a 25-year study. *FORENSIC SCI INT* 2006;159(2-3):200-05. doi: 10.1016/j.forsciint.2005.08.003 pmid:161832292006-06-02.

11. M. C. Lee, S. S. Wong, J. J. Chu, et al. Traumatic asphyxia. *ANN THORAC SURG* 1991;51(1):86-88. doi: 10.1016/0003-4975(91)90456-z pmid:19855831991-01-01.

12. M. N. Arslan, C. Kertmen, Melez I. Esen, D. O. Melez. Comparison of autopsy findings and injury severity scores in deaths due to traumatic asphyxia (perthes syndrome). *J FORENSIC LEG MED* 2018;56:42-47. doi: 10.1016/j.jfml.2018.03.002 pmid:29533203.[Copyright (c) 2018 Elsevier Ltd and Faculty of Forensic and Legal Medicine. All rights reserved.:*2018-05-01*].

13. W. R. Jongewaard, T. H. Cogbill, J. Landercasper. Neurologic consequences of traumatic asphyxia. *J Trauma* 1992;32(1):28-31. doi: 10.1097/00005373-199201000-00006 pmid:17325701992-01-01.

14. Koraichi A. El, R. Benafitou, J. Tadili, et al. [Traumatic asphyxia or Perthe's syndrome. About two paediatric cases]. *Ann Fr Anesth Reanim* 2012;31(3):259-61. doi: 10.1016/j.annfar.2011.12.011 pmid:22305399.[Copyright A(c) 2011 Societe francaise d’anesthesie et de reanimation (Sfar). Published by Elsevier SAS. All rights reserved.:*2012-03-01*].

15. S. Al-Sarraj, R. Laxton, B. Swift, et al. Neuropathology and brain weight in traumatic-crush asphyxia. *J FORENSIC LEG MED* 2017;52:110-15. doi: 10.1016/j.jfml.2017.08.009 pmid:28892750.[Copyright (c) 2017 Elsevier Ltd and Faculty of Forensic and Legal Medicine. All rights reserved.:*2017-11-01*].

16. J. L. Meller, A. G. Little, D. W. Shermeta. Thoracic trauma in children. *PEDIATRICS* 1984;74(5):813-19. pmid:64938751984-11-01.

17. L. Gorenstein, G. K. Blair, B. Shandling. The prognosis of traumatic asphyxia in childhood. *J PEDIATR SURG* 1986;21(9):753-56. doi: 10.1016/s0022-3468(86)80358-x pmid:37726971986-09-01.

18. J. R. Gill, K. Landi. Traumatic asphyxial deaths due to an uncontrolled crowd. *Am J Forensic Med Pathol* 2004;25(4):358-61. doi: 10.1097/01.paf.0000147316.62883.8b pmid:155775302004-12-01.

19. T. Kantor, B. Grigorescu, G. Popescu, et al. [Traumatic asphyxia with permanent visual loss. Case report]. *Orv Hetil* 2017;158(22):864-68. doi: 10.1556/650.2017.30750 pmid:285616322017-06-01.

20. A. E. Balci, A. Kazez, S. Eren, et al. Blunt thoracic trauma in children: review of 137 cases. *Eur J Cardiothorac Surg* 2004;26(2):387-92. doi: 10.1016/j.ejcts.2004.04.024 pmid:152969022004-08-01.

21. R. B. Wagner, WO Jr Crawford, P. P. Schimpf. Classification of parenchymal injuries of the lung. *RADIOLOGY* 1988;167(1):77-82. doi: 10.1148/radiology.167.1.3347751 pmid:33477511988-04-01.

22. G. Campbell-Hewson, C. V. Egleston, A. R. Cope. Traumatic asphyxia in children. *J Accid Emerg Med* 1997;14(1):47-49. doi: 10.1136/emj.14.1.47 pmid:90236271997-01-01.

23. M. C. Plewa, A. B. Peitzman, R. D. Stewart. Benign cervical prevertebral soft tissue swelling in traumatic asphyxia. *J Trauma* 1995;38(6):937-40. doi: 10.1097/00005373-199506000-00021
Tables

Due to technical limitations, table 1, 2 and 3 is only available as a download in the Supplemental Files section.

Figures
Figure 1

Photo of one TA patient. This patient suffered from vehicle roll-over when playing outside. Taken within 1 day after trauma, there showed obvious petechiae all around the face, especially around the mouth and eyes. There presented also petechiae on the cervical and thoracic region, but the thoracic petechiae might be also related to direct vehicle roll-over with tyre marks. His face showed severe congestion and edema. Both of his eyes presented with severe subconjunctival hemorrhage. No other symptoms or change of
consciousness occurred, but there accompanied unilateral pulmonary contusion and mastoid hemorrhage. This patient received supportive treatment and non-invasive mechanic ventilation with PEEP. He was discharged after 12 days without sequelae except pulmonary changes.

**Figure 2**

State of consciousness after injury. With time, unconscious patients decreased from 22 right after injury to 0 in 10 days after injury, conscious patients increased from 26 right after injury to 50 in 10 days after injury. A transitional period of confusion was also observed.
optical coherent tomography (OCT), fundus and thickness map results of left eye of a TA patient during hospitalization (a), left eye in 1-month follow-up (b), left eye 1.5-month follow-up (c) and right eye in 1.5-month follow-up (d). Shown in figure 3a, the retina was hemorrhagic with edema, thickening and exudates around the optic disc in the left eye. The optic disc was pushed temporally with possible optic nerve compression. However, this child did not report about visual change at that time. In the 1-month and 1.5-month follow up as shown in Figure 3b and 3c, the retina turned atrophy and the optic disc was drafted. These retinal changes are irreversible and responsible for lifelong amblyopia. Figure 3d presented the right eye in 1.5-month follow-up without obvious retinal changes.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- table1.pdf
- table2.pdf
- table3.pdf