

Bilateral Retrobulbar Hemorrhage and Visual Loss Following Traumatic Asphyxia

Young Joo Choi¹, Sung Ju Lee¹, Hyung Jin Kim², Jin Ho Yim¹

¹Department of Ophthalmology, Ulsan University Hospital, Ulsan University College of Medicine, Ulsan, Korea

²Department of Radiology, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Korea

Retrobulbar hemorrhage and permanent visual loss are rare presentations following traumatic asphyxia. In this case, bilateral permanent visual disturbance developed in a woman after chest-crushing trauma without direct trauma to the orbits. A computed tomography scan confirmed bilateral retrobulbar hemorrhages. An ophthalmologic exam revealed bilateral subconjunctival hemorrhages and severe lid edema. Despite high-dose steroid therapy, visual recovery was limited, and optic nerve atrophy developed. Ischemia of the optic nerve associated with retrobulbar hemorrhage may be postulated as one of the causes of permanent visual impairment following traumatic asphyxia.

Key Words: Optic nerve atrophy, Retrobulbar hemorrhage, Traumatic asphyxia, Visual loss

Traumatic asphyxia is a rare syndrome caused by crushing injury to the chest. It is clinically characterized by cervicofacial cyanosis, edema, subconjunctival hemorrhage, and petechiae on the upper chest and face. Neurologic symptoms are often associated with traumatic asphyxia [1]. Severe subconjunctival hemorrhaging has been noted in almost all cases, and occasionally other ocular manifestations, including pupillary changes, exophthalmos, diplopia, retinal hemorrhage, disc edema, optic nerve atrophy, and transient or permanent visual disturbances, have been reported [2,3]. These symptoms and signs can occur because the high intrathoracic pressure generated by chest compression is transmitted directly to the ocular blood vessels. In the unsupported areas of the skin and conjunctivae, hemorrhage can result, but within the orbit, pressure is transmitted to and absorbed by the surrounding tissue. Therefore, retrobulbar hemorrhage is very rare.

Vision may be impaired or lost immediately in one or both eyes, although it promptly returns to normal in many patients [2,4]. The fundi are usually normal, but retinal edema, hemorrhage, cotton-wool spots, and disc edema have been found, and in the majority of instances, permanent visual loss or im-

pairment has been associated with retinal hemorrhages and edema [2]. To the best of our knowledge, this case is the first to report bilateral vision loss associated with ischemia of the optic nerve with bilateral retrobulbar hemorrhage in traumatic asphyxia.

Case Report

A 47-year-old woman working in a car manufacturing plant was pinned between a conveyer belt and the car body during assembly. Her left shoulder and chest were compressed. When released more than 20 minutes later, she was unconscious. Upon arriving at the emergency department, she was still unconscious, with facial cyanosis, severe edema, and petechiae. Radiographs and computed tomography (CT) scans of her chest, abdomen, and head revealed bilateral pneumothoraces, hemothoraces, multiple rib fractures, and a left scapular fracture. Her blood pressure was unstable. There were no other signs or history of head injury. A closed thoracotomy was immediately performed in the emergency room, and she was transferred to the intensive care unit for further management. In a bedside ophthalmologic examination, severe bilateral subconjunctival hemorrhages, chemosis, severe periorbital swelling, and mild exophthalmos were found (Fig. 1). Both pupils reacted sluggishly to light, and there was a relative afferent pupillary defect in the left eye. Intraocular pressure measured by a Tono-Pen was 17 mmHg in the right eye and 16 mmHg in the left eye. There were no signs of orbital compartment syndrome. Funduscopy

Received: July 16, 2009 Accepted: March 14, 2010

Reprint requests to Jin Ho Yim, Department of Ophthalmology, Ulsan University Hospital, Ulsan University College of Medicine, #290-3 Jeonha-dong, Dong-gu, Ulsan 682-714, Korea. Tel: 82-52-250-7170, Fax: 82-52-250-7174, E-mail: yimjinho@hanmail.net

© 2010 The Korean Ophthalmological Society

This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<http://creativecommons.org/licenses/by-nc/3.0/>) which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

examination showed bilateral mild optic disc edema. Admission axial CT scans of the orbit demonstrated bilateral retrobulbar hemorrhages, mild proptosis, and severe eyelid swelling (Fig. 2). Follow-up CT scans obtained 3 days later showed reduced exophthalmos and retrobulbar hemorrhages. On the third day after the accident, the patient recovered consciousness and her corrected visual acuity was finger counting at 50 cm in the right eye, and hand motion in the left eye. Visual evoked potentials revealed bilateral delayed latency, decreased amplitude in the right eye, and a flat wave in the left eye. High-dose steroid therapy was begun with the intravenous injection of 1.0 g methylprednisone daily for five

days. Seven days after course of steroid treatment, corrected visual acuity improved to 0.1 in the right eye, but there was a marked visual field defect, and the corrected visual acuity of



Fig. 1. Severe bilateral subconjunctival hemorrhages, chemosis, severe eyelid swelling, and mild exophthalmos were found.

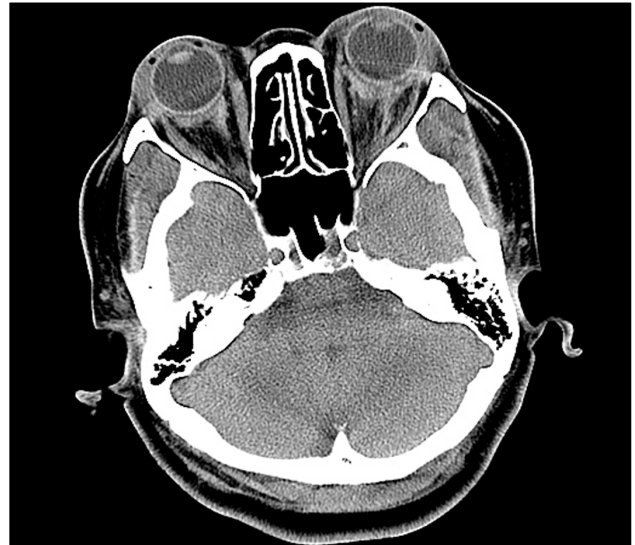


Fig. 2. Admission axial computed tomography scans of the orbit demonstrated bilateral retrobulbar hemorrhages, mild proptosis, and severe eyelid swelling. Note the orbital fat interposed between the globe and medial wall of the orbit.

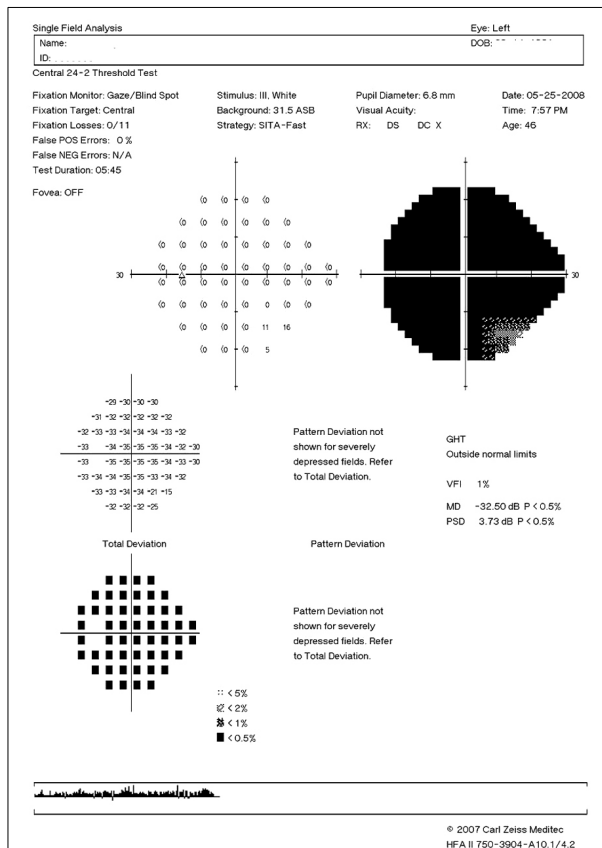
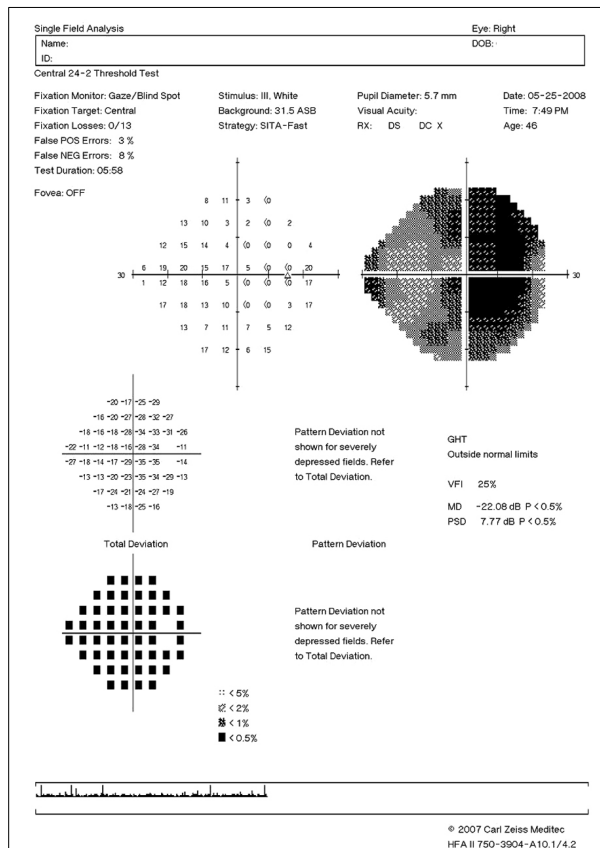


Fig. 3. Visual field test shows the marked field defects in both eyes 6 weeks after injury.

her left eye remained hand motion without recovery (Fig. 3).

Three months later, the patient experienced no interval change in vision, but the visual field defect was worse, and fundus examination revealed bilateral optic nerve atrophy, especially in the left eye. Optical coherence tomography demonstrated that the retinal nerve fiber layer (RNFL) thickness had significantly decreased in the right eye, and there was a near total loss of RNFL in the left eye.

Discussion

The underlying pathophysiology producing cervicofacial cyanosis, petechiae, and subconjunctival hemorrhaging in traumatic asphyxia is sudden elevation of venous pressure [2]. After violent compression of the thorax or abdomen, positive pressure is transmitted to the mediastinum, and blood is forced out of the right atrium, through the valveless innominate and jugular veins into the head and neck. Victims who anticipate trauma tend to hold their breath and close the glottis, further increasing the intrathoracic pressure, and this sudden marked increase in pressure in the small veins and capillaries causes rapid dilatation and minute hemorrhages, resulting in petechiae [2]. Where the vessels are not supported by the surrounding tissue, as in the conjunctival and buccal mucosa, or when the retrograde pressure is excessive, there is actual extravasation of erythrocytes from the vessels, with production of petechiae and ecchymoses [2,5]. Intracranial and retinal hemorrhages have been surprisingly infrequent in traumatic asphyxia. The only explanation for this offered so far, is that intracranial and intraocular pressures oppose the pressure in the blood vessels, thus preventing their rupture [2]. Majority of neurologic symptoms seen in traumatic asphyxia are caused by the indirect injury, which results in hypoxia. Reports have postulated that the pathogenesis of neurologic manifestations is related to ischemia of the brain or cord secondary to venous obstruction and elevated pressures [6-8].

The mild proptosis seen in this patient might have been secondary to medial displacement of orbital fat, together with retrobulbar hemorrhages. This disruption of orbital architecture may have been caused by the high intrathoracic pressure [9].

The prognosis for vision after traumatic asphyxia is usually good or temporary vision loss. But there have been a few cases of immediate or late blindness caused by retinal hemorrhages and cotton wool exudates in the fundus, which is known as traumatic retinal angiopathy. If there is no traumatic retinal angiopathy, the return of vision is usually prompt and complete [2,3]. Thus, the presence or absence of retinal angiopathy is a prognostic factor in the recovery of visual acuity. Out of 100 survivors of traumatic asphyxia, 16 patients experienced an immediate loss of vision, and only 8 among these 16 patients had no return or only a partial return of vision [2]. Most of these cases were reported before CT scans were routinely performed. From among these, there was the second case reported by Baldwin et al. [10], in whom

the resulting visual acuity in the left eye was permanent blindness and optic nerve atrophy. But in that patient there was a head injury and widespread patches of retinal edema surrounding the left macula, so this may be a case of Purtscher's retinopathy [10]. In the case of permanent impairment where no hemorrhage involving the macula and/or optic disc can be found, some other explanation is necessary, as that in neurologic symptoms [11]. The high intrathoracic pressure is transmitted directly to the retinal vessels and may cause capillary rupture, reflex vasospasm, and subsequent tissue hypoxia within the retina. Initial vasospasm and vascular stasis with resultant ischemia may presumably lead to degenerative retinal changes [4]. Above all, both the duration and weight of compression are the most important factors in the prognosis of vision after traumatic asphyxia. Considerable weight can be tolerated for a short period, whereas a comparatively modest weight applied for a longer period may result in death [12]. Patient in this report was compressed for more than 20 minutes, and additional ischemic injury due to unstable blood pressure and depressed respiration in the early stage of trauma may have aggravated ischemia in the retina.

This case demonstrates that ischemia of the optic nerve associated with retrobulbar hemorrhage may be postulated as one of the causes of permanent visual impairment following traumatic asphyxia. Even though the amount of hemorrhage was small, optic nerve ischemia could have been aggravated by a combination of the relatively long duration of compression, inadequate ventilation, arterial hypotension, and venous hypertension. In managing traumatic asphyxia, a complete ophthalmologic examination should be performed and documented. This is particularly important in patients who may inaccurately report visual impairment, such as children and patients with prolonged unconsciousness, especially intubated patients with retrobulbar hemorrhage.

Conflict of Interest

No potential conflict of interest relevant to this article was reported.

References

1. Williams JS, Minken SL, Adams JT. Traumatic asphyxia--reappraised. *Ann Surg* 1968;167:384-92.
2. Heuer GJ. Traumatic asphyxia with especial reference to its ocular and visual disturbances. *Surg Gynecol Obstet* 1923;36: 686-96.
3. Parker FJ. Optic atrophy from traumatic asphyxia with report of a case. *Arch Ophthalmol* 1911;11:159-62.
4. Purdy RH, Cogbill TH, Landercasper J, Ryan DK. Temporary blindness associated with traumatic asphyxia. *J Emerg Med* 1988;6:373-6.
5. Shamblin JR, Mcgoon DC. Acute thoracic compression with traumatic asphyxia. Report of six cases. *Arch Surg* 1963;87: 967-75.

6. Reichert FL, Martin JW. Traumatic asphyxia: experimental and clinical observations with a report of a case with concomitant paraplegia. *Ann Surg* 1951;134:361-8.
7. Sandiford JA, Sickler D. Traumatic asphyxia with severe neurological sequelae. *J Trauma* 1974;14:805-10.
8. Jongewaard WR, Cogbill TH, Landercasper J. Neurologic consequences of traumatic asphyxia. *J Trauma* 1992;32:28-31.
9. Macnab AJ, Baldwin GA, McCormick AQ, Flodmark O. Proptosis and diplopia following traumatic asphyxia. *Ann Emerg Med* 1987;16:1289-90.
10. Baldwin GA, Macnab AJ, McCormick AQ. Visual loss following traumatic asphyxia in children. *J Trauma* 1988;28:557-8.
11. Schabdach DG, Goldberg SH, Breton ME, et al. An animal model of visual loss from orbital hemorrhage. *Ophthal Plast Reconstr Surg* 1994;10:200-5.
12. Furuya Y. Experimental traumatic asphyxia (1)--grades of thoracic compression and mortality. *Igaku Kenkyu* 1981;51: 117-9.