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Case Report

UNILATERAL OPTIC NEUROPATHY AFTER COMPRESSIVE THORACIC INJURY: A CASE REPORT

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SUMMARY – Compressive thorax injury is mostly observed in road traffic accidents which lead to multiple traumas. Eye findings after thoracic injury can be Purtscher's retinopathy, Valsalva retinopathy, and even blindness. We report a rare case of unilateral optic neuropathy associated with compressive chest injury. In our case, a 31-year-old construction worker developed irreversible optic nerve damage after ground collapsed on him and buried him up to his neck. There was no evidence of head trauma and blood loss, but multislice computed tomography showed diffuse brain edema caused by prolonged hypoxia due to compression of the thorax.

Key words: *Brain edema; Compressive thorax injury; Hypoxia; Optic neuropathy*

Introduction

Compressive thoracic trauma is mostly sustained in motor vehicle collisions. Among other injuries, it can lead to pathological ocular findings such as Purtscher's retinopathy, Valsalva retinopathy, and even blindness.

Unilateral or bilateral blindness after trauma is mostly associated with direct or indirect head trauma[1-3]. Hasenbohler *et al*. have presented a case report of a man who had bilateral blindness following thoracoabdominal crush trauma[4]. They postulate massive increase in venous blood pressure with capillary stasis and consequent optic nerve damage to be the cause of blindness. Chandra *et al*. found Purtscher's retinopathy, traumatic optic neuropathy and Valsalva retinopathy in their case report[5]. However, due to uncertain exact history of trauma to their patient, concussive force could have led to indirect optic nerve injury. Raigal Cano *et al*. in their case report of a young man with total bilateral blindness after thoracoabdominal non-penetrating trauma speculate that profound hemorrhagic shock, prolonged arterial hypotension and sudden drop in hematocrit value caused ischemic optic neuropathy which led to blindness[6]. Fuller and Vote report on acute dysfunction of all orbital nerves due to marked hemifacial tissue edema which was associated with vascular compromise[7]. Unilateral visual impairment after backfilling is rarely found. In our case, construction worker developed irreversible optic nerve damage after the ground had collapsed on him and buried him up to his neck.

Case Report

We report a case of a 31-year-old male without relevant medical history who was transferred to hospital immediately after he had been buried up to his neck

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with soil while doing construction work. Eyewitnesses of the accident stated that his whole body, except for the head, was under ground with plastic pipe pressing upon his chest. He was conscious during rescuing, but had problems breathing due to chest compression. On admission, the patient was restless, had ecchymosis on his chest, signs of compression of superior vena cava, and signs of pulmonary edema. He had no signs of head trauma. His Glasgow Coma Scale score was 13. Laboratory findings were normal. Because of the life threatening condition, he was hospitalized at the intensive care unit and was on continuous mechanical ventilation for the first 24 hours. There were no signs of bone fractures on x-ray images. Abdominal and heart ultrasound was also normal. Multislice computed tomography (MSCT) of the head was performed promptly after admission, and it did not show hemorrhage or ischemia in the brain, or bone fractures, but there was generalized edema. During hospital stay, he complained of blurred vision on both eyes, more pronounced on the left eye. The pupil size was average with normal reactions to light and accommodation; relative afferent pupillary defect (RAPD) was negative. The best corrected visual acuity (BCVU) on Snellen's chart was 20/20 on both eyes with normal fundus findings. There was no evidence of direct trauma to the eye in the form of perforated globe, lens luxation, retinal tears or detachment, vitreal hemorrhage, or laceration of lids and conjunctiva. Eye movements were normal. The applanation pressure was 16 mm Hg on both eyes. After his vital parameters and overall condition stabilized, the patient was released for home care and scheduled for regular follow ups. On first follow up, he still complained of poor vision on his left eye with black spot in the center of his visual field. BCVU was still 20/20 on both eyes, but with eccentric fixation on the left eye. This time, RAPD was positive on the left. Detailed fundus examination revealed normal findings on the right eye and temporal paleness of disc on the left eye (Figs. 1 and 2). Visual field Octopus (Octopus 101, Haag Streit Diagnostic, TOP program) showed normal finding on the right eye and cecocentral scotoma with incomplete involvement of the lower temporal quadrant on the left eye (Fig. 3). Visual evoked potential revealed normal finding on the right eye and delayed latency on the left eye, which is consistent with optic nerve injury. The patient was diagnosed with optic neuropathy on the left eye. On 6-month follow up, optical coherence tomography of the left eye showed mild reduction of retinal nerve fiber layer thickness. Cecocentral scotoma and electrophysiological scar remained unchanged.

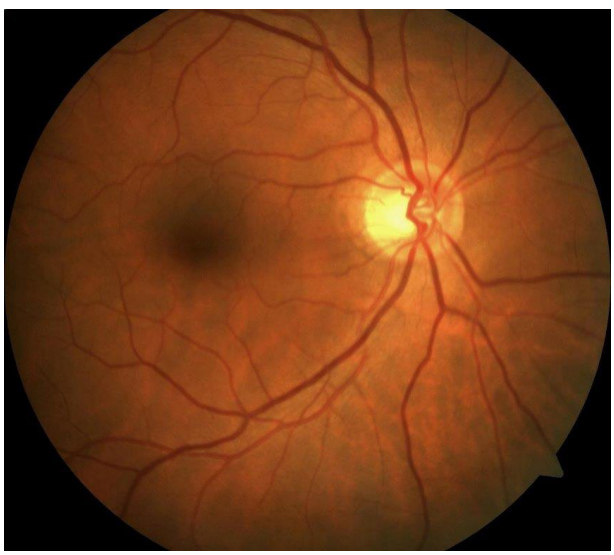

Fig. 1. Fundus of the right eye (normal findings).

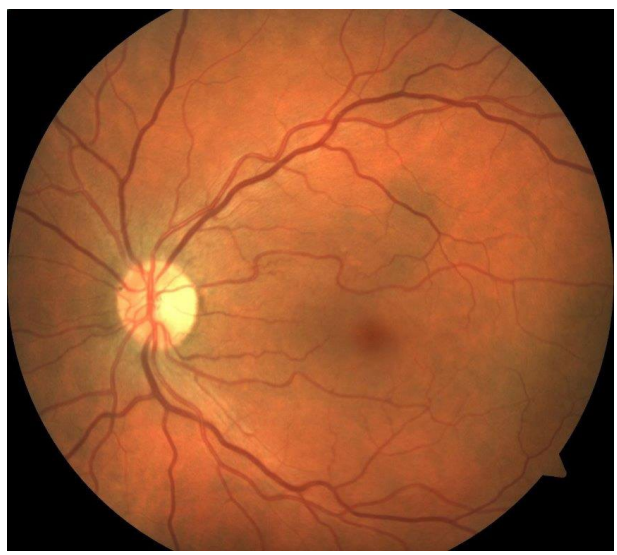

Fig. 2. Fundus of the left eye (temporal paleness of the optic disc).

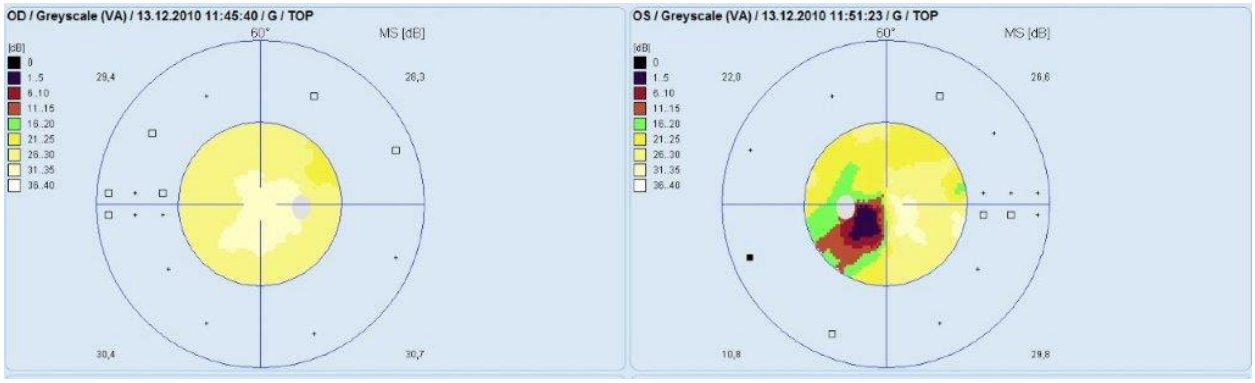

Fig. 3. Visual field Octopus 101: normal finding on the right eye, cecocentral scotoma with incomplete involvement of the lower temporal quadrant on the left eye.

Discussion

There could be several causes of blurred vision after the accident in our patient, such as Purtscher's retinopathy, Valsalva retinopathy, traumatic optic neuropathy, or ischemic optic neuropathy due to the loss of blood. According to eyewitnesses and physical examination, the patient had no direct head trauma that could have led to traumatic optic neuropathy. Laboratory findings and abdominal ultrasound ruled out internal bleeding as the possible cause of hypotension and consequently loss of vision, as described in several cases[8,9]. We ruled out Purtscher's and Valsalva retinopathy because ocular fundus finding would not be normal initially or on the first follow up examination. It is well known that, while backfilling, there is high pressure on the chest and therefore impairment of breathing function. This resulted in development of pulmonary edema, hypoxia and brain edema, as found in MSCT and x-ray images. Diffuse brain edema could have led to vision damage, but because of visual evoked potential findings of optic nerve injury, we suggest a different scenario. Hypoxia subsequent to pulmonary edema also led to optic nerve edema, which was the cause of optic nerve damage.

The presented case demonstrates that hypoxia after thoracic compression injury can cause diffuse brain edema, as well as unilateral optic neuropathy and irreversible visual impairment.

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Sažetak

JEDNOSTRANA OPTIČKA NEUROPATIJA NAKON KOMPRESIJSKE OZLJEDE PRSIŠTA: PRIKAZ SLUČAJA

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Kompresivna ozljeda prsnog koša najčešće se primjećuje u prometnim nesrećama koje dovode do politraume. Nalazi na očima nakon torakalne ozljede mogu biti Purtscherova retinopatija, Valsalvina retinopatija pa čak i sljepoća. U ovom radu prikazujemo rijedak slučaj jednostrane optičke neuropatije povezane s kompresijskom ozljedom prsišta. U 31-godišnjeg građevinskog radnika razvilo se nepovratno oštećenje vidnog živca nakon što se zemlja srušila na njega i zakopala ga do vrata. Nije bilo dokaza o traumi glave i gubitku krvi, ali je MSCT pokazao difuzni edem mozga uzrokovan produljenom hipoksijom zbog kompresije toraksa.

Ključne riječi: *Edem mozga; Kompresijska ozljeda prsišta; Hipoksija; Optička neuropatija*