

Amaurosis by Traumatic Optic Neuropathy direct secondary to the “transorbitario” foreign body: Report of an atypical case

Amaurose por neuropatia óptica traumática direta secundária à corpo estranho transorbitário: relato de um caso atípico

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ABSTRACT

The purpose of this research is to describe a clinical study of a male patient who experienced late-blindness due to traumatic optic neuropathy after getting into a bicycle accident. It is stressed the importance of performing pupillary/reflexes examination and computed tomography scan in order to diagnose “transorbitario” foreign body. It is also cover its surgical removal as well as an interdisciplinary approach and clinical evolution.

Keywords: Eye foreign bodies; Eye injuries; Wounds penetrating; Optic nerve diseases; Blindness

RESUMO

Neste trabalho descreve-se o caso clínico de um paciente do sexo masculino que evoluiu para cegueira tardia por neuropatia óptica traumática após queda de bicicleta. Enfatiza-se a importância do exame oftalmológico/reflexos pupilares e da tomografia computadorizada no diagnóstico de corpo estranho orbitário, sua remoção cirúrgica, abordagem interdisciplinar e evolução.

Descritores: Corpos estranhos no olho; Traumatismos oculares; Ferimentos penetrantes; Doenças do nervo óptico; Cegueira

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INTRODUCTION

The optic pathway is injured in only approximately 5% of patients with traumatic brain injury (TBI). However, initial ophthalmologic assessment in the trauma room is often impaired by the patient's altered level of consciousness or severe palpebral edema.⁽¹⁾ As the eyeball occupies only 1/4 of the entire orbital cavity, fragments of orbital foreign body (FB) can be accommodated, and they are mainly made of wood, metal and glass,^(1,2) being more common in adolescents and middle-aged men.⁽²⁾

Diagnosis is made with ectoscopy in most cases, allowing for relatively simple surgical removal. However, when its direct visualization is not possible, imaging methods such as radiography, ultrasonography, computed tomography (CT) and magnetic resonance imaging (MRI) may aid the diagnosis.⁽²⁾ Therefore, optic nerve (ON) lesions should always be investigated at the patient's hospitalization whenever possible, since the treatment may vary from clinical to surgical, and depending on the severity of the lesion it may progress to traumatic optic neuropathy (TON).^(1,3)

TON is a severe uni or bilateral condition that can occur through direct or indirect mechanisms. Trauma by direct mechanism usually results from penetrating lesions in the orbit.⁽⁴⁻⁶⁾ It is not uncommon closed traumatism with extensive face or orbital fracture have ON injury by bone fragments. There may even be a total ON section. In addition, a significant part of the cases is related to peri-orbital hematoma.^(1,3,7,8)

The present study describes a case of late blindness by TON after trauma, the relevance of CT in the diagnosis of orbital FB, its treatment and evolution at the Teaching Hospital Luiz Gioseffi Jannuzzi de Faculdade de Medicina de Valença (HELGJ-FMV). For this purpose, the patient was requested to sign a free and informed consent form, submitted and approved in the Ethics and Human Research Committee of Centro de Ensino Superior de Valença through the platform Brazil.

CASE REPORT

A 39-year-old man had infraorbital short-blunt wound on the right, and body abrasions after a bicycle fall on a steep slope, with initial care at Hospital Geral de Rio das Flores - RJ, where the wound was sutured. After initial stabilization, the patient was referred to a private hospital contracted by the local Mayor to provide emergency imaging exams, returning immediately. The CT of the skull and face made at this time showed orbital fractures, and the medical report did not suggest the presence of FB.

After 24 hours of trauma, he presented edema and evident phlogistic signs in the sutured facial region, being then transferred to HELGJ-FMV. At the ophthalmological examination in the bed, he presented in the right eye: probable orbital cellulitis manifested by ophthalmoplegia, conjunctival hyperemia, ecchymosis and hyposphagma, with normal visual acuity (VA) in the bed, according to information collected (SIC) and normal funduscopy. No microbiological exam was carried out. As for the left eye, it presented medium mydriasis, relative afferent pupillary defect (Marcus-Gunn pupil), VA decrease / visual haze, (SIC) and normal funduscopy.

The patient was initially treated with eyedrops tobramycin 0.3%, venous antibiotic therapy with amoxicillin 1g + clavulanate 200mg, and oral corticosteroid therapy based on prednisone 60mg/day. A new CT scan of the skull and face was requested to exclude FB. This suggested elongated, low-density artifact in the right infra-orbital region with an oblique path through the soft parts, extending to the contralateral retro-orbital region, repelling medial straight muscles, completely transfixing the nasal cavity and ending in comminuted fractures of the orbital laminae and ethmoid cells (Figures 1 and 2). It was possible to evidence fractures of the medial walls (Figures 1 and 2) and probable compression of the left

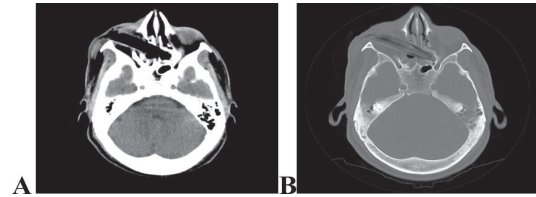


Figure 1: Axial cuts of skull CT in tissue window showing transorbital FB in tissue window (A) and bone window (B).

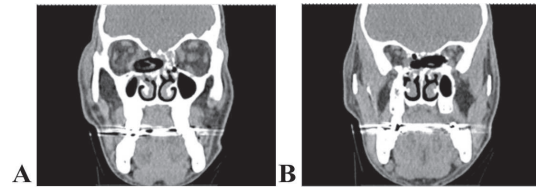


Figure 2: Coronal CT scans of the face on the tissue window showing transorbital FB and comminuted fractures in the medial wall of right (A) and left (B) orbits.

ON by direct trauma of FB and bone spicules (Figures 1A and B).

After the second imaging exam on the 8th post-trauma day, the patient underwent exploratory surgery by the Ophthalmology / Orbit and General Surgery team of HELGJ-FMV under local anesthesia, sedation and additional dose of venous antibiotic / cephalothin 1g. A cylindrical wood FB with dimensions of 7.6 cm x 1.3 cm was removed through the trauma wound (Figures 3A and B) and a Penrose drain was used (Figure 4). The patient maintained the venous antibiotic therapy for 13 days and progressive weaning from corticosteroid therapy, progressing satisfactorily. He was discharged on the 15th day of hospitalization, with referral to the HELGJ-FMV Ophthalmology ambulatory and prescription of the antibiotic used for 10 days. After evaluation, the oral and maxillofacial surgeon suggested conservative treatment.

Ambulatory ophthalmologic evaluation made 36 days after trauma and 28 days after surgery revealed normal endotropy, funduscopy and AV (20/20) on the right, and persistent mydriasis, Marcus-Gunn pupil, preserved MOE, funduscopy still normal and left amaurosis on the left. The patient received general guidance and had the stitches removed. After this first appointment, he did not return to the follow up.

Despite the late amaurosis, it can be said that the TON had an early onset, since Marcus-Gunn's pupil was observed since the beginning of the condition, even with normal funduscopy and appearance of the ON. Funduscopy imaging record was not possible due to the unavailability of equipment for this purpose.

DISCUSSION

The ON lesions lead to several types of focal defects, and may also occur with diffuse atrophy of the fibers, especially in the chronic phase. Thus, optic neuropathies represent axonal death due to insult, which may be of inflammatory, demyelinating, ischemic, hereditary, toxic, deficient, compressive, and traumatic causes.⁽⁴⁾

In this context, traumatic causes of high kinetic energy or related to fractures of the floor, orbital ceiling or optic foramen are suggestive of TON.^(1,2) Besides, the TON may be a uni or bilateral condition, and may occur through direct or indirect mechanisms. Trauma by direct mechanism usually results from penetrating lesions in the orbit.^(4-6,9)

It is not uncommon that closed traumatism with extensive face or orbital fracture have ON injury by bone fragments. In addition, a significant part of the cases is related to peri-orbital hematoma, as in the present clinical case.^(1,4,7-9)

In this case, CT also detected a direct trauma of the left ON by the FB and the bone fragments of the orbital apex (Figure 1A and B). Note that, although the FB has penetrated the right side,

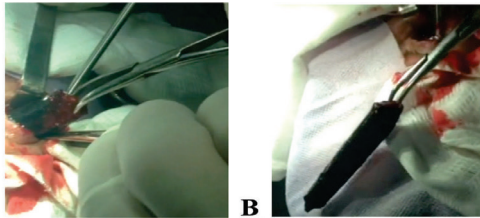


Figure 3: Surgical act (A) with removal of cylindrical FB of wood (B).



Figure 4: Final appearance with Penrose drain.

there was TON only on the left.

In the case of posterior trauma in the orbit or at the level of the optic canal, the optic disc looks normal during the acute phase. Progressive atrophy of the nerve fibers usually appears in a few weeks, and can be demonstrated clinically or by means of techniques such as laser scanning polarimeter and optical coherence tomography.^(4,6)

Literature reports that although axonal degeneration is completed within about 7 days after injury, the cell body and the axonal portion attached to it maintains normal appearance for up to 4 weeks. Thereafter, the entire remaining structure degenerates rapidly, to the point that there is no viable affected ganglion cell after 6 to 8 weeks of severe ON injury.⁽⁴⁾

In a recent study where 3 patients with ON trauma were observed, all of them evolved with a gradual reduction of the retinal nerve fiber layer starting in the second week after the trauma and increased from the fourth week on, with a maximum accentuation after 12 weeks.^(4,6) In the present report, the trauma was direct posterior, and only the funduscopy exam was performed. Up to 4-5 weeks after trauma there was still no funduscopy manifestation of ON atrophy.

The optic disc may become pale, noted 3 to 4 weeks after the trauma. This phenomenon is observed in the most severe cases, and progresses to complete atrophy. This pallor may be more late and discrete in less severe cases.⁽⁴⁾

The clinical course for optic atrophy, in this case, was not observed until approximately 5 weeks. It is not possible to prove the exact age of the onset of papillary pallor due to the non-return of the patient.

Despite not being the best method for evaluating ON, skull, orbit and face CT provides information on the presence of fractures of the orbital canal, as well as the presence of bone fragments in the interior, with bilateral lesions or chiasmatic lesions in 10% of the cases, generally asymmetric, with severe unilateral involvement associated to contralateral temporal hemianopsia. By definition, it can be said that TON is a post-trauma visual loss occurring in the presence of an afferent pupillary defect, with no evidence of eye trauma or ON.^(1,2) In this report, CT was of special value in the diagnosis of the presence of FB, whose wound was already sutured. It also helped the surgical prescription and, along with the pupillary examination, the diagnosis of probable TON contralateral to the penetrating lesion.

Regarding the treatment of TON, both the use of corticosteroids and a surgical approach in order to decompress the ON can be used. It is a consensus that either one, combined or not, offers better results to the patient than none of the approaches.^(2,10) In our case, intravenous corticosteroid therapy was performed, but the patient presented only favorable development of the infectious condition after surgery for the removal of the transorbitary FB.

FINAL CONSIDERATIONS

The incidence of damage to the optic pathway after TBI described is relatively low. However, initial ophthalmologic assessment in the trauma room is often impaired by altered level of consciousness or severe palpebral edema, and we have to consider that they may be underdiagnosed. Therefore, ON injury should always be considered in the hospitalization of patients with TBI, since the treatment may vary from clinical to surgical, especially when orbital fractures and high kinetic energy traumas occur.^(1,2,9)

The present study describes an atypical case of orbital FB after penetrating facial trauma undiagnosed during first care, with gender and age group according to the literature reports.⁽²⁾ The suspicion of TON and the presence of the FB was only made after ophthalmologic evaluation by direct funduscopy, pupillary reflex investigation and skull and face CT at HELGJ-FMV. It also provided a concise and non-invasive surgical approach. The CT scan performed as a matter of urgency at the first appointment was not seen by the team of the Teaching Hospital, and its report only showed orbital fractures, not diagnosing the presence of the FB. There is no records of ophthalmologic examination / pupillary reflexes in the referral to HELGJ-FMV.

The local infection evolved well only after surgery to remove the wood fragment from the transorbitary region. Despite this, there was worsening of the contralateral VA to amaurosis, probably due to both direct damage of the ON by the FB and by the bone fragments of the fractures (Figure 1A and B). Even with the FB removal and corticosteroid use, there was progression of atrophy of ON fibers. It could be reversible if the FB had been withdrawn earlier.

Recently, a similar report was described in the literature, also evolving to contralateral blindness. In addition, gender, age group, and FB composition in our case coincide with most cases described in the literature.⁽²⁾

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