PATHOLOGY OF THE OPTIC NERVE INJURY

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PATHOLOGY OF THE OPTIC NERVE INJURY (Abstract): The optic nerve injury is produced by damaging the optic nerve within a craniocerebral trauma. The anatomo-pathology and the pathology of posttraumatic visual impairments are fundamental data for the therapeutic indications. The microscopic examination reveals only the particular cases of very serious or severe extensive lesions. Damage to the vessels of the optic nerve, damage and break of the optical fibers are responsible for immediate visual impairments and secondary hematomas and edemas involving. The factors for the late appearance of visual impairments are numerous and often confounding. Material and methods: In the present research, we have a female patient D.A., aged 29, diagnosed with closed fracture in the right frontal lobe, irradiated to the optic canal; blindness in the right eye. Results: Break of the optical fibers is a result of bone fragments or of traction and torsion. Damage to the vessels of the optic nerve represents the main cause of blindness for most authors. Traction and torsion of the vessels at the moment of injury may cause thrombosis and small hemorrhages which lead to severe ischemia of the optical fibers. Conclusions: The optic nerve injury is produced by damaging the optic nerve within a craniocerebral trauma, through an indirect mechanism most of the times. Keywords: CRANIOCEREBRAL TRAUMA, OPTIC NERVE, VISUAL IMPAIRMENTS
sequence of an injury to the optic canal or a direct injury. The direct injury to the optic nerve is visible; it can be a result of a foreign body or it can occur as a result of bone fractures. Optic nerve injury by fracturing the canal has a frequency which varies according to different authors: Rollet, Paufique, Barkan, Lazorthes (1, 3) consider them to be frequent injuries; Wagemann, Berlin, Strieff, Genet and Turner (7, 8) insist on the rare occurrence of this type of injury. This disagreement occurs because of insufficient radiologic examinations and due to the fact that some patients underwent a surgery while others were treated medically.

It is important to establish the cause-effect relationship between the bone fracture and the injury to the optic nerve. The opinions are different. In some cases, the cause effect relationship is obvious: the nerve is compressed by bone fracture in the canal area, the canal and the nerve are displaced, and bone fragments penetrate the nerve while the sheaths are breaking down – a fracture without an evident displacement (6, 7, 8).

According to other authors (9, 10), the optic canal frequently has a very small and reversible deformation due to a flattened diameter. These deformations can also be permanent as some radiographies show it – they can injure the optic nerve. In rare cases, the bone lesions accompany the optic nerve injuries without being the causal factor.

**MATERIAL AND METHODS**

The female patient D.A., aged 29, has the following diagnosis: - closed fracture in the right frontal lobe, irradiated to the optic canal; blindness in the right eye; right frontal flap, disease of the scalp skin; bone flap with lateral pedicle; lumbar puncture with convenient intracranial hypotension. Dura mater is separated from the roof to the little wing along which a fracture is found parallel to the posterior border unfolding into numerous fragments together with the inferior, superior and external fragment. It follows frontal ablation of the orbital roof and bone fragment compression and 75% of the circumference of the optic canal is liberated. Inside, an ethmoid cell is opened and many bone fragments are found inside; these fragments are ablated.

**RESULTS AND DISCUSSION**

The ethmoid cell is obstructed. Haemostasis is assured. The dura is suspended and the anatomical plans are restored. The diagnosis is established intraoperative; interruption of the right optic nerve syndrome; frontal vault fractures irradiated to the optic canal.

Break of the optical fibers are a result of bone fragments or of traction and torsion. Minassian (4) reported a case of injury to the optic nerve without fracturing the canal. Nucci et al. (6) makes the assumption that there may be optic nerve contusions with interrupted nerve fibers caused by the relative motion of the encephalon in the skull, producing tractions and torsions at the level of optic canal and junction areas. Nevertheless, this hypothesis cannot be demonstrated surgically.

Damage to the vessels of the optic nerve represents the main cause of blindness for most authors (7, 8). Traction and torsion of the vessels at the moment of injury may cause thrombosis and small hemorrhages which lead to severe ischemia of the optical fibers.

Functional disturbances of the optic nerve generate general physico-pathological reactions and some spontaneous remissions.
of immediate blindness can be explained. These functional disorders are in connection with the blocking of subdural and subarachnoid space; this way, the passing of fluid along the nerve is prevented; the flow of the fluid elements can be restored only after the decompression of these inextensible areas – the optic canal and Zinn’s ligament, accordingly.

Castros (6) claims that immediate visual impairments can also occur within the traumatic retinal angiopathy through venous reflux (Purtscher’s retinitis) and due to retinal posttraumatic ischemic syndrome, through central retinal artery thrombosis.

Secondary appearance of impairments or blindness is a consequence of the initial perturbations, but which lack clinical expression or the examination does not reveal it. Hematomas can lie inside three levels: inside the duramater and extradural, inside the subdural space and in the optic nerve. These hematomas are frequent inside the subdural space; they do not have specific limits and are easily evacuated. Subdural hematoma or sheath hematoma can stretch along the walls of the nerve, and this may cause severe compromised vascularization; hematic compressive lesions may lead to necrosis of nerve substance; it may also lead to thrombosis of small vessels of piamater. The arterioles break in the vaginal space may produce intra-nervous hematomas; they can organize themselves in order to produce fibrotic glial tissue and adjacent necrosis. These intravenous hematomas can explain the unsystematised amputations of the visual field (6, 7).

Edemas apparently seem that are early lesions and not immediate ones. Juge (4) finds cases in which edemas appear 3 months after the initial trauma. Due to the rigidity of the bone canal, the edema causes a veritable nerve strangulation, so that during the decompression it can be seen the trace left on the nerve sheath. In edematous forms, the postoperative treatment with corticosteroids is compulsory.

Late impairments are often unknown due to the fact that they evolve gradually and they are clinically latent. These forms have a certain importance because they get good results after the surgical intervention.

Gliosis scar tissue is consecutive to the attrition of the optical fibers and nerves’ vessels and they are represented, at least at the beginning, by histological repairs of the damage. New lesions are produced due to the fact that these scarring processes can extend.

Perioptic arachnoiditis were described by Marinescu and Cazaban (3). They can occur in nervous system disorders, traumas, tumors and infections, and the general conditions of the body (intoxications, infections, especially facial cavities infections). Arachnoid neoformations form a construction of the nervous tissue and they tend to comprise the optic nerve and chiasm. Sometimes incisures can be seen as a result of the strangulation of the optic nerve or chiasm. So, the adhesions press the nervous tissue transacting it. At the same time, the circulation is disturbed: the arteries appear to be atrophic, the flow in the arterial circulation is diminished; the veins are dilated and congested, so the return flow is not normal. The mechanic and vascular elements contribute to the anatomic and functional alteration of the nervous cells.

Chronic swelling of the nerve – it was reported in other observations (8, 10).

Progressive development of a bone cal- lus is an exceptional lesion. Streiff (4) cited a case where the diagnosis was established
radiologically, 5 years after the trauma took place, even if the patient had lost his vision immediately after the injury. Functional disturbances of the optic fibers or of the vascularisation of the nerve – it seems that they play an important role. Also cavernous carotid aneurysms, according to Brihaye (6) or ophthalmic artery aneurysm were detected.

CONCLUSIONS
The optic nerve injury is produced by damaging the optic nerve within a cranio-cerebral trauma, through an indirect mechanism most of the times. In these cases, the fracture of the skull base, with or without the involvement of the optic canal, determines immediate bilateral or unilateral blindness, stationary or slightly regressive. The fracture trajectory may or may be not visible through imagistic diagnosis at the level of the canal or the optical hole, and it is not compulsory to have dilacerated or compressive bone fragments.

REFERENCES