Pathology of the optic nerve injury

Ledia Balla¹, N. Ianovic², D. Costin³

¹PhD Student in Neurosurgery, “Gr.T. Popa” UMPh, Iasi
²Neurosurgery, “N. Oblu” Clinical Emergency Hospital, Iasi, Romania
³“Gr.T. Popa” UMPh, Iasi, 2nd Ophthalmological Clinic, Clinical Emergency Neurosurgical Hospital

Abstract

The optic nerve injury is produced by damaging the optic nerve within a craniocerebral trauma. The anatomopathology and the pathology of posttraumatic visual impairments are fundamental data for the therapeutic indications. However, most of the times, these are only hypothesis and consequently, many errors occur. The microscopic examination reveals only the particular cases of very serious or extensive lesions; the anatomo-clinic examination is usually missing. Only the neurosurgical intervention undergone in order to explore the nerve can provide information macroscopically. The macroscopic examination finds specific relations by highlighting cause and effect, but sometimes the examination undergone during the surgical intervention is incomplete; moreover, opening the optic nerve sheath is rarely found in the literature.

There are several causes distinguished of immediate and late visual impairment. A. Causes of immediate visual impairment

According to Juge’s classification, optic nerve injuries can be divided into:
1. Immediate blindness
2. Partial vision impairment
3. Secondary impairments
4. Late impairments

The anatomopathology and the pathology of posttraumatic visual impairments are fundamental data for the therapeutic indications.
b) Optic nerve injury by fracturing the canal has a frequency which varies according to different authors: Rollet, Lazorthes consider them to be frequent injuries; Wagemann, Strieff, 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 nerve are displaced, and bone fragments penetrate the nerve while the sheaths are breaking down – a fracture without an evident displacement.

According to Streiff and other authors 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.

Observation: 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.

The ethmoid cell is obstructed. Haemostasis is assured. The dura is suspended and the anatomical plans are restored.

The diagnosis is established intraoperatively; interruption of the right optic nerve syndrome; frontal vault fractures irradiated to the optic canal.

<table>
<thead>
<tr>
<th>No.</th>
<th>Case</th>
<th>Fracture</th>
<th>Hematoma</th>
<th>Sheath</th>
<th>Nerve</th>
<th>Other obs.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>DA</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td></td>
<td>Bone fragment compressing the optic nerve</td>
</tr>
<tr>
<td>2.</td>
<td>CA</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Bone fragment compression resulting from fracturing 1/3 of the right orbital roof</td>
</tr>
<tr>
<td>3.</td>
<td>AI</td>
<td>2 older hematomas near the optic nerve</td>
<td></td>
<td></td>
<td></td>
<td>Bone fragments reaching the exterior wall of optic canal</td>
</tr>
<tr>
<td>4.</td>
<td>SP</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>compressed</td>
<td>Triangular bone fragment from the orbital roof which compresses the optic nerve close to the top of the orbit.</td>
</tr>
</tbody>
</table>
2. Break of the optical fibers – are a result of bone fragments or of traction and torsion.

Lazorthes and Anduze reported a case of injury to the optic nerve without fracturing the canal.

Hadjean makes the assumption that there may be optic nerve concussions 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.

Damaging the optic nerve through this mechanism is exceptional.

3. Damage to the vessels of the optic nerve – for most authors, this represents the main cause of blindness (Rollet, Calendoli, Guillaumat, Streiff, Turner).

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.

By analogy with the head injuries which produce encephalic damages, Brihaye says that this theory was plausible due to the lack of axial vascularization at the level of the optic canal. At this level, the vascularization is made through the pia mater vascular network. A dissecting hematoma of the intra nervous area can be produced due to this phenomenon.

4. Functional disturbances of the optic nerve – they 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 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. He sustains that the main cause is represented by the molecular perturbations caused by the intraocular pressure.

B. Secondary appearance of impairments or blindness

- are a consequence of the initial perturbations, but which lack clinical expression or the examination does not reveal it. Evolving, these initial perturbations lead to obvious clinical examination. Consequently, clinically speaking, it is difficult to differentiate late impairments from the immediate ones.

There are two main causes of secondary impairment occurrence:

- hematomas
- edemas

1. Hematomas – can lie inside 3 levels: inside the dura mater and extra-dural, 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 pia mater;
- the arterioles break in the vaginal space may produce intra-nervous hematomas; they can organize themselves in order to
produce fibrotic gliac tissue and adjacent necrosis.

These intravenous hematomas can explain the unsystematized amputations of the visual field.

The cadaver studies showed numerous hematic collections in the nerve sheath.
- Holder – 77% of the cases
- Glauning – 90% of the cases
- Muller – 87% of the cases

The hemorrhage center was studied in 84 necropsies: 90% of the cases presented hemorrhages, 77% showed hematic lesions to the dura mater, 83% subdural hematoma, and 36% injuries to the optic nerve. In terms of topography, the hemorrhages predominated in the optic canal and in Zinn’s ring.

2. Edemas – it seems that these are early lesions and not immediate ones. Juge 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 oedematous forms, the postoperative treatment with corticosteroids is compulsory.

C. Late impairments

They 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.

The causes are numerous and often confounding

1. Gliosis scar tissue – they are 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.

2. Peri-optic arachnoiditis – were described by Marinescu and Cazaban. 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 chiasma. Sometimes incisures can be seen as a result of the strangulation of the optic nerve or chiasma. 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.

3. Chronic swelling of the nerve – it was reported in many observations.

4. Progressive development of a bone callus – is an exceptional lesion. Streff 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.

5. Functional disturbances of the optic fibers or of the vascularization of the nerve – it seems that they play an important role.

6. Cavernous carotid aneurysms – (according to Brihaye) or ophthalmic artery aneurysm.

In 1966, Walsh made a much simpler but artificial classification after 140 necropsies. He distinguished two types of lesions: primary and secondary. This classification has a fundamental importance.
in terms of surgical indications.

There are 3 types of primary lesions:
- hemorrhages (inside the dura mater, inside the subdural space, and in the optic nerve)
- nerve injuries
- necrosis

There are 3 types of secondary lesions:
- edemas and nerve swelling
- necrosis secondary to the circulatory insufficiency
- infarction lesions

Conclusions

The optic nerve injury is produced by damaging the optic nerve within a craniocerebral 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 the immediate appearance of a bilateral or unilateral blindness, stationary or slightly regressive. The fracture trajectory may be visible through imagistic diagnosis or not at the level of the canal or the optical hole, and it is not compulsory to have dilacerate or compressive bone fragments.

References