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Transethmoidal decompression of the optic nerve in the case of craniocerebral trauma

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Abstract

Over a period of ten years, 39 patients who had suffered optic nerve compression after a craniocerebral trauma underwent transethmoidal decompression surgery. The operation was performed bilaterally on 5 patients. Fifty percent of patients involved suffered a blunt head or brain injury, the others brain compression or contusion. On the side of optic nerve compression, we found specific signs and symptoms of the compression such as negative or sluggish direct light reaction of the pupil, wounds on the lateral side of the eyebrow, bleeding from the nose, eyelid hematoma, skull fractures and intracranial hematomas. Since radiological and intraoperative findings were the same in only 67% of cases ophthalmological findings such as lack of direct pupil reaction occurring together with preserved consensual light reaction and progressive loss of vision after a traumatic incident are used as guideline for performing transethmoidal decompression of the optic nerve. Surgery produced restitution of visual function in about 10% more cases than conservative therapy reported in the literature.

Keywords: Decompression surgery, head and brain injuries, optic nerve injuries.

1 Introduction

According to several reports [5, 6, 11, 14] a lesion of the optic nerve is to be expected in 0,35% to 3,6% of all patients who suffer a craniocerebral trauma. As older surgical methods for treating optic nerve compression involved craniotomy and transfrontal osteoclastic decompression, they were dangerous in cases of severe brain injuries and ineffective compared to conservative treatment. The transethmoidal surgical approach to the optic canal was proposed in 1961 [8]. This technique allows the removal of up to 75% of the bony canal. Since the operation is carried out under local anesthesia, return of light reaction of the pupil or return of visual functions can direct the extent of the decompression [2, 8, 13]. A disadvantage is that the optic nerve cannot be decompressed at its entry to the canal.

Nevertheless, excellent results in a large number of patients were reported by SUGITA [13] and FUKADO [3]. European studies were done with smaller numbers of patients and their results were not as good [1, 4, 12]. Since 1976 we have performed decompression of the optic nerve on 37 patients with craniocerebral trauma using the transethmoidal approach. The diagnostic procedure was carried out in close cooperation between neurosurgeons and ophthalmologists, while the operation was performed by otolaryngologists.

2 Patients

All patients (39 cases) who had unilateral or bilateral traumatic optic nerve compression and underwent transethmoidal operative decompression at the University of Göttingen from September 1976 to June 1987 were included this study. Case histories, plain X-rays, and computer tomograms were evaluated according to:

- location and extent of visible bruises
- skull fractures and brain damage especially to the structures of the optic system
- post-traumatic and pre- as well as post-operative visual function
- clinical course
- other therapy and
- complications.

Since ophthalmological examinations differed in both extent and quality from patient to patient, a detailed grading of postoperative visual restitution was not possible the information available is listed in table I.

Patients with persisting reduction or spontaneous remission of visual function and those not receiving surgery were not included.

Table I. Results of transethmoidal optic nerve decompression

Postoperative results of visual function (n = 38)			
+++	Restitution	16% (n = 6)	visual field more than 3 quadrants, visus better than 0.8
++	Restricted restitution	21% (n = 8)	visual field more than 1 quadrant, visus better than 0.05
+	Highly restricted restitut.	16% (n = 6)	visual field less than 1 quadrant, visus less than 0.05
–	Persisting amaurosis	37% (n = 14)	
– –	Deterioration	10% (n = 4)	the three patients concerned became post-operatively amaurotic

3 Operative procedure

Operation was carried out under general anesthesia. A curved skin incision was made in the medial canthus, from the origin of the eyebrow up to the lateral nasal region, leaving trochlea and lacrimal sac intact.

The lateral nasal wall are ablated with a drill and the anterior ethmoid cells are opened and removed with Blakesly forceps. The retro-orbital ethmoid cells are removed and the anterior wall of the sphenoid sinus is opened. Where the retro-orbital cells meet the lateral sphenoid sinus wall, we usually find the optic canal.

The medial bony border of the optic canal is ablated with a microdrill, exposing the nerve up to the chiasm so that the periostal envelope can be split.

4 Results

4.1 Pattern of injuries

60% of the patients were between 20 and 30 years of age, most of them between 15 and 25 years of age (Figure 1). Traffic accidents were the most common cause of optic nerve injury (60%). This was followed by occupational accidents (16%). Polytrauma was frequent (40%). More than half of the patients had suffered a blunt head trauma or a brain concussion only (Figure 2). The rest had brain contusion, in five cases combined with considerable space-occupying intracerebral hemorrhage. Additionally, we found extracerebral hematomas in five patients. Thirteen patients had to undergo neurosurgery, mainly an evacuation of hematomas.

Brain contusion was usually localized in the frontal lobe (75%); the rest were in the temporo – parietal region. They were always located on the same side as the optic nerve compression as were the extracerebral hematomas, with one exception: a seven year old girl whose primary contusion site and all visible injuries were seen contralaterally since the main fracture line crossed the other optic canal. Fractures of the orbita were seen most frequently, followed by fractures of the skull base (Figure 3). Fractures of the facial skull outside the bony canal of the optic nerve were found in 84% of the cases. These fractures exclusively or predominantly affected the side of optic nerve compression. Visible

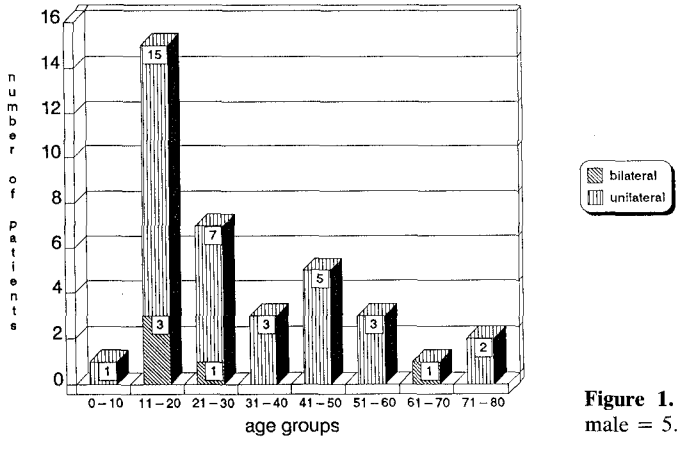

Figure 1. Age distribution of patients, male = 32, female = 5.

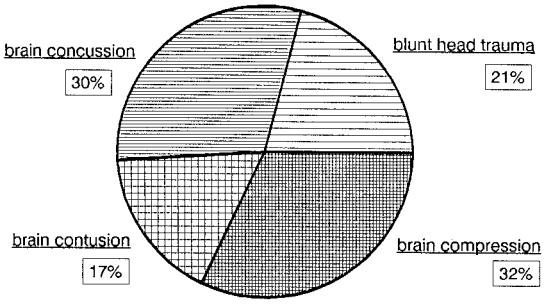

Figure 2. Brain trauma (n = 37): light 51%, severe = 49%.

bruises or injuries of the face and galea were also always found on the side of the optic nerve compression except in the one case mentioned. They proved to be more frequent in the supra- as infraorbital areas; the majority were situated at the fronto – temporal border.

4.2 Symptoms

Two thirds of the patients showed the three symptoms reported by FUKADO [2]: "loss or sluggishness of the direct light reaction of the pupil on the affected side, a wound on the lateral side of the eyebrow and bleeding from the nose".

We also found an ipsilateral eyelid hematoma in 90% of these patients. However, when hematoma is extensive this can make diagnosis more difficult. Contusion of the ocular bulb can also make diagnosis more difficult (Figure 4). Hyposphagma was found in 65% of the eyes concerned. In such case a sensitive examination method which shows bleeding inside the ocular bulb or effects of contusion on the iris or eyes' fundus was necessary for diagnosis [7]. Correct diagnosis was also impeded by lack of cooperation in nearly half of the patients, posttraumatic space-occupying complications (compressio cerebri) in one third of the patients, and a primary traumatic injury of the oculomotor nerve. We diagnosed this injury ipsilateral to the optic nerve compression in five patients. A simultaneous lesion of the oculomotor nerve delayed the diagnosis for about ten hours. The average delay between traumatic insult and diagnosis was 35 hours. Unexpectedly we found that this interval was about twenty hours longer with conscious, cooperative patients than with unconscious patients after a severe brain injury. This was probably due to the more frequent primary defect of the reaction of the pupil and to more intensive primary care with patients after a severe brain damage. While only 20% of the unconcious patients showed secondary compression of the optic nerve, 55% of the conscious patients development amaurosis caused by secondary compression.

4.3 Pathogenetic factors

Preoperative radiological diagnosis was confirmed by intra-operative findings in only 67% of cases (Figure 5). This might be partly due to the fact that during operation the lateral part of the optic nerve canal cannot be seen so that a fracture in this area is not verifiable. Additionally, compression inside the canal is not easily identified when the transethmoidal approach is used. In five cases decompression of the nerve was only indicated by clinical and neuro-ophthalmological findings. Plain X-rays and CT-scans revealed periorbital fractures, but no signs of optic nerve compression or optic canal fracture. In two of these cases no decompression was found.

4.4 Operative decompression results

As three patients died during the first two weeks after traumatic insult, the results of the transethmoidal decompression could be followed in 38 decompressed optic nerves only (Table I).

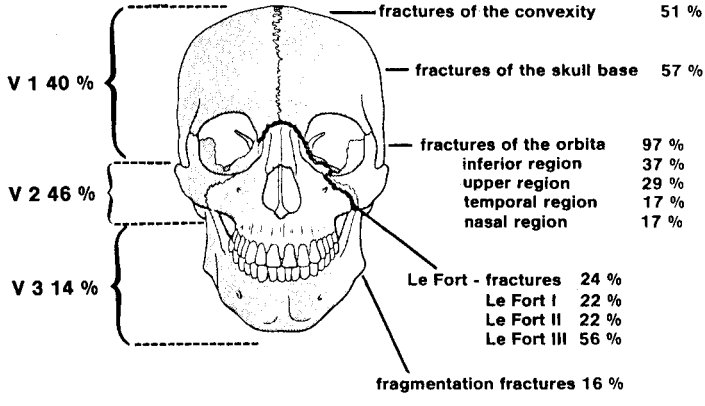

Figure 3. Fractures of the skull accompanying traumatic optic nerve compression (n = 37). The percentages on the left side of the figure demonstrate the relative quantity of skull fractures according in the various trigeminal nerve areas.

Eyelid haematoma	90%
Hyposphagma (eyeball contusion)	65%
Lack of patient's cooperation	47%
Brain compression	32%
Oculomotor nerve lesion	14%
Light reaction absent bilaterally	14%

Figure 4. Factors hindering diagnosis (n = 37)

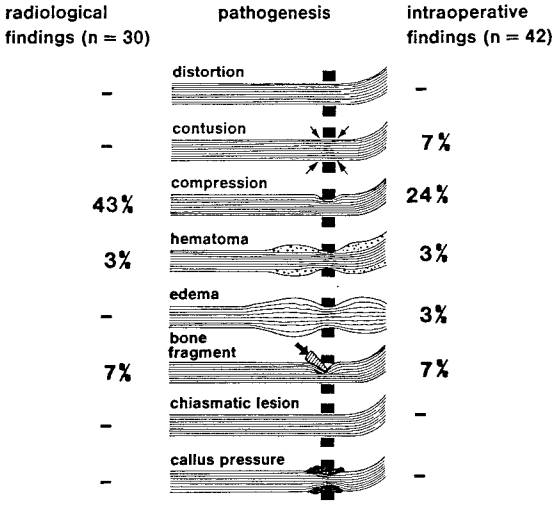

Figure 5. Radiological and intraoperative findings of pathogenetic factors leading to an optic nerve lesion in sequence of their frequency according to HAGER [4].

We produced a restitution or a restricted restitution of visual function in 37% of cases. All of these cases showed recovery of the ability of the pupil to react by the forth day. In up to 16% of cases postoperative visual function remained highly restricted; some were only left a recognition of hand movements or light gleams. In cases in which radiodiagnosis had shown fracture and compression of the optic canal restitution was restricted.

4.5 Bilateral optic nerve compression

All five patients who underwent bilateral decompression had suffered polytrauma and severe central fractures of the facial skull. All of them had fractures of a Le Fort II and III type; three had fragmentation fractures. These five patients could be divided into two groups. In the first one (two patients) they had suffered blunt craniocerebral trauma. A limited direct reaction of the pupils was seen on both sides. In one patient ophthalmoscopy showed streaky bleedings on both fundus of the eye, in the other it revealed a blurredness of both papillas. After maxillary surgery, loss of the pupils reaction developed and remained unchanged despite decompressive surgery carried out on second or third day. No further injuries were found during operation. Later we found a lesion of the chiasmatic region in one of the cases and a bilateral occlusion of the a. centralis retinae in the other. The three patients in the second group had suffered severe craniocerebral trauma. Because of loss of the pupils' reaction to light, decompression was performed during the first twelve hours after brain compression had been excluded. Intraoperatively fragmentation fractures of the ethmoidal structures and the optic canal were seen. One of these patients died after three days; one kept half his visual capacity and field in one of his eyes; and the third kept limited vision in the upper nasal field.

4.6 Complications

In the 42 operations that we performed, one purulent secondary infection developed at the operation site five years later. There were two cases of postoperative hemorrhage. In one of these cases, hemorrhage might have been responsible for postoperative deterioration of vision.

5 Discussion

Extensive statistics on the results of conservative therapy in the literature [6, 9, 10, 14] report improvment in 40% to 75% of patients. However a restitution of visual function (visual field more than 3 quadrants, visus better than 0.8, dimensionless number, compare Table I) was seen only in an average of about 20% of successfully treated cases, while we achieved restitution in 30% of our cases showing an improvement of visual function after optic nerve decompression. For this reason we think, transethmoidal optic nerve decompression should be performed when ophthalmological examination and radiodiagnostic evidence indicate optic nerve compression. We do have to admit however, postoperative deterioration in 10% of cases.

It should be emphasized that although we perform a thorough radiological examination when we suspect optic nerve compression, for us ophthalmological findings are of greater relevance for deciding whether to operate or not. The two major indications of compression are:

- lack of direkt reaction of the pupil to light and preserved consensual light-reaction combined with normal eye fundus
- progressive loss of vision after a craniocerebral trauma.

Our results demonstrate that patients with a radiologically proven optic nerve compression

reached, at best, a restricted visual function. We therefore conclude that the prevalence of opthalmological rather than radiological symptoms seems to be an important indication of possible successful operative decompression. We think, as do many other authors, that decompression of the optic nerve should be performed as quickly as possible, even though this could not be proven by significant statistics.

6 Conclusions

Manifestation of a typical pattern of injuries to the orbita requires close control of direct and consensual light reaction of the pupils, even when examination is hindered by an eyelid hematoma.

In the case of severe bilateral mid-face fractures of a Le Fort II or Le Fort III type, we advise a conservative approach consisting of external fixation without mobilisation of the fractures since a secondary amaurosis might be induced as it was in two of our patients. This was also the conclusion of a poll among plastic surgeons in the USA in 1984 [15]. We propose that a detailed layered computerized tomography of the orbita be performed immediately when tomography does not show brain compression and the pupil shows no reaction to light.

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