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Professional paper

Indirect Traumatic Optic Neuropathy – Two Case Report

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A B S T R A C T

The aim of the study was to evaluate the treatment of indirect traumatic optic neuropathy (ITON). ITON is defined as traumatic loss of vision that occurs without external or initial ophthalmoscopic evidence of injury to the eye or its nerve. The optimal management of ITON remains controversial. History, clinical findings and treatment of two cases of ITON with high-dose corticosteroids are described. Improvement of visual acuity after treatment with high-dose corticosteroids was achieved in both cases. The treatment is evaluated in comparison to endorsed treatment modalities found in literature. We concluded that was clinically reasonable to decide to treat or not to treat the indirect optic neuropathy on an individual patient basis.

Introduction

Indirect traumatic optic neuropathy (ITON) is defined as traumatic loss of vision which occurs without external or initial ophthalmoscopic evidence of injury to the eye or its nerve[1]. It is an uncommon but often devastating cause of permanent loss of vision after blunt head trauma. The cause is blunt head trauma. Patients are usually young men, injured in motor vehicle or bicycle accidents[2]. Diagnosis is clinical: careful history is of great importance and unconscious patients are great problem. Visual acuity, color vision and visual field are often unavailable. Thus, the most important ocular sign is relative afferent pupillary block[1–4]. Computed tomography should be used to rule out a direct injury to the optic nerve (ON)[4]. Management of ITON remains controversial[2,5]. There is endorsement in literature for corticosteroids, surgical decompression of the optic canal, combination of corticosteroids and surgical decompression, and observation alone.

The aim of this study was to present and evaluate the treatment of two cases of ITON. History, clinical findings and the treatment are described and compared to the endorsed treatment modalities given in literature.

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Case report

Case 1

A 48-year-old patient was admitted to hospital after a fall from a roof. On arrival to the emergency room he was agitated. Few hours after injury his visual acuity was O.D: 1.0 and O.S: no light perception. Relative afferent pupillary block was present in the left eye. Left ocular motility was disturbed in all directions. Left conjunctiva was slightly chemotic in the lower part. Intraocular pressure was normal. Computed tomography, performed immediately after admission, showed fracture of the orbital roof and floor and fracture of the nasal bone. There was no sign of compression to the optic nerve in the optic canals, but edema of the ON was evident. Ultrasound investigation on the 1st day also showed optic nerve edema. Within 24 hours of injury the patient was given 1000mg of methylprednisolone and the same dose was administered for the next 4 days. On the 5th day the patient received 1mg/kg of methylprednisolone and the dose was slowly tapered over one month. On the 2nd day after injury visual acuity improved to hand movement and ultrasound examination showed regression of edema. On the 7th day ultrasound examination showed normal thickness of the ON. Final visual acuity 4 weeks after injury was count fingers at 10 cm. By that time slight optic atrophy in the lower temporal portion of the optic disc was noticed. Visual field examination was not performed.

Case 2

A 28-year-old patient was injured in a car accident. Upon arrival of the paramedics he was orientated. On the 2nd day he noticed poor vision in the left eye. The examination showed visual acuity O.D: 1.0 and O.S: no light perception. A relative afferent pupillary block was present in the left eye. There was mild hematoma on the upper and lower left lid. Ocular position and motility was normal. Intraocular pressure was normal. Computed tomography, performed immediately after admission, showed fracture of the left temporal and sphenoidal bone of the lateral orbital wall. There was no sign of compression to the ON in optic canals, but edema of the left ON was evident. Ultrasound examination on the same day also showed edema of the ON. Forty-eight hours after injury the patient was administered 1000 mg of methylprednisolone. The same dose was given for the next 4 days. On the 7th day the patient was given 1mg/ kg of methylprednisolone. The dose was tapered over one month. Two days after we started therapy his visual acuity improved and ultrasound examination showed regression of the ON edema. On the 7th day ultrasound examination was normal. Final visual acuity, 4 weeks after injury, was counting fingers at 1 meter. By that time we noticed slight optic atrophy in the lower temporal portion of the optic disc. Visual field examination was not performed. Both cases data are presented in Table 1.

Discussion

The mechanism of ON injury has been described as a compartment syndrome with bleeding, edema and inflammation leading to necrosis and infarction[5]. ITON is defined as traumatic loss of vision occuring without external or initial ophthalmoscopic evidence of injury to the eye or its nerve after a blunt head trauma[1]. While the diagnosis of the condition can usually be made with the aid of a careful history and examination, its optimal management is far less well defined. In literature we lack large randomized controlled trials of management because of the low incidence of this condition. There are different ways of treatment of ITON: corticosteroids, surgical decompression, which

TABLE 1
CLINICAL COURSE OF EXAMINATED PATIENTS

Age and gender	48 years male	28 years male
Follow-up period	1 month	1 month
Cause of trauma	Falling accident	Car accident
State of mind	Agitated	Orientated
Computed tomography	No compression of the optic canals, fracture of orbital roof and floor, fracture of maxilla, edema of the optic nerve	No compression of the optic canals, fracture of lateral orbital wall and nasal bones, edema of the optic nerve
Ultrasound examination	Edema of the optic nerve	Edema of the optic nerve
Treatment	High-dose corticosteroids	High-dose corticosteroids
Final visual acuity	CF 10 cm	CF 1 m
Course	Improvement from NLP to CF in 5 days	Improvement from NLP to CF in 8 days

can be combined with corticosteroid treatment, and observation alone. Corticosteroid therapy was categorized according to the initial daily dose of methylprednisolone as (I) mega-dose, for ≥ 5400 mg, (II) very high dose, for 2000–5399 mg, (III) high-dose, for 500–1999 mg, (IV) moderate-dose, for 100–499 mg and (V) low-dose, for < 100 mg[2].

There are many reports that favor the use of mega-dose corticosteroids[6,7,13]. All of them were retrospective studies and some of them excluded patients who had optic canal surgery after not improving from corticosteroids. Some authors report a benefit of transethmoidal optic canal decompression treatment in cases of ITON[11–13]. Anderson et all, however, report poor visual results with this treatment in patients who completely lost vision immediately after accident. In these cases high-dose corticosteroids were reported to be successful[14]. On the other hand, there are data in literature that advocate surgical therapy after unsuccessful treatment with corticosteroids or in cases where visual acuity decreases after initial improvement[1,2,13].

Hughes studied a spontaneous improvement and reported an improvement rate of 48%, but in other studies, all retrospective, the rate of improvement was lower[6–10].

The recent International Optic Nerve Trauma Study[2], which included patients from 16 different countries, was designed as a comparative nonrandomized study with concurrent treatment groups. The primary outcome measure was visual acuity. The study showed no clear benefit of either corticosteroid therapy or/and optic canal decompression, or observation alone. There was no indication that the dosage of corticosteroid treatment was important, because the outcome was not significantly different whether low-dose, medium-dose, high-dose or mega-dose was prescribed. The conclusion of this study is that there is clinically reasonable to decide to treat or not to treat on an individual patient basis[2].

In our cases the compression of the ON in the optic canals was ruled out by computed tomography. CT and ultrasound examination revealed edema of the ON. The initial visual acuity was no light perception in both cases. One conclusion that can be drawn from different studies is that the prognosis for visual improvement is worse when there is no light per-

ception following the optic nerve injury[2,14]. It was on these bases that we decided for the high-dose corticosteroid treatment. Visual acuity of the first patient improved on the 2nd day of treatment to hand movement. Ultrasound examination showed regression of the edema two days after treatment. Edema totally disappeared on the 7th day of treatment. Final visual acuity was counting fingers at 10 cm.

Visual acuity of the second patient improved on the 2nd day of treatment to hand movement. Ultrasound examination showed regression of the ON edema on the 4th day of treatment. Edema totally regressed seven days after treatment and final visual acuity was counting fingers at one meter. Because of the visual acuity improvement and regression of edema we didn't go for surgical decompression since the literature suggests to proceed with surgical decompression only in cases when visual acuity does not improve or in cases where visual acuity decreases after initial improvement[1,2,13]. Four weeks after injury we noticed the initial signs of the ON atrophy in both cases, but visual acuity remained stable.

In cases of ITON we never decide for observation alone. According to our experience initial high-dose corticosteroid treatment showed benefit in such devastating conditions. Only if there was no improvement of visual acuity and clinical picture after the initial corticosteroid treatment, we would consider other treatment modalities. The decision on the treatment of ITON should strictly be made on an individual patient basis.

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INDIREKTNA TRAUMATSKA OPTIČKA NEUROPATIJA – PRIKAZ DVA SLUČAJA I PREGLED LITERATURE

S A Ž E T A K

Cilj rada bio je prikazati i procijeniti liječenje indirektne traumatske optičke neuropatije (ITON). ITON se definira kao traumatski gubitak vida do kojeg dolazi bez vanjskih ili inicijalno oftalmoskopski prisutnih znakova povrede oka, odnosno njegovog živca. Optimalno liječenje ITON-a i dalje je kontroverzno. Prikazane su anamneze, klinički nalazi i liječenje dvaju slučaja ITON-a visokim dozama kortikosteroida kojima je postignuto poboljšanje vidne oštrine u oba slučaja. Liječenje je procijenjeno u usporedbi s prihvaćenim načinima liječenja navedenim u literaturi. Odluku o načinu liječenja indirektne traumatske optičke neuropatije potrebno je donijeti za svakog pacijenta individualno.