TRAUMATIC OPTIC NEUROPATHY IN THE DIVISION OF NEURO-OPHTHALMOLOGY, DEPARTMENT OF OPHTHALMOLOGY, DR SOETOMO TEACHING HOSPITAL, SURABAYA

Siti Moesbadiany Yogiantoro

ABSTRACT

***Objective**: To identify the feature of patient's funduscopies, time between trauma to curing, treatment and results of Traumatic Optic Neuropathy (TON) outpatients at the Division of Neuro-ophthalmology, Dr. Soetomo Hospital, Surabaya, Indonesia. **Methods**: Data of outpatients with TON from January 2002 to October 2004 were collected and classified into several categories; age, time between trauma, curing, feature of funduscopy, treatment and result. **Results**: There were 44 patients, 10 female and 34 male. The age range was between 15 - 80 years old, and most of them 18 patients or 40.9% of all, was between 10 and 19 years old. They commonly came after fourth week post trauma (19 patients = 43.2%), only one patient (2.72%) came at second day post trauma with various Visual Acuity (VA) between no light perception and 6/10. The features of funduscopy: optic nerve head atrophy in 25 patients (56.82%), normal fundus, 16 patients (34.1%), vitreous bleeding, 2 patients (4.54%), papiledema only one patient (2.27%). Patients were treated with methylprednisolone in various dosages (16 - 100 mg). Neurotropic, NSAID and antioxydant were added, depending on their clinical manifestation. Twelve patients (27.27%) had increased VA, 3 patients (6.82%) decreased VA, 9 patients (20.45%) showed no change in VA and the others could not be evaluated because they did not come for follow up. **Conclusion**: Accurate diagnoses and therapy affect the results of therapy. High dose steroid systemic and optic canal decompression (if necessary) should be given in 48 hours after trauma.*

***Keywords:** ocular injury, optic neuropathy*

INTRODUCTION

The occurrence of head trauma may result in various disorders, one of which is the post-traumatic loss of vision due to damage in optic nerve. Diagnosis and therapy for various types of vision loss due to trauma and optic nerve damage is difficult. Hippocrates himself identified such condition from the phenomenon of vision loss, both acute or post-traumatic at or above the eyebrows. There are several types of Traumatic Optic Neuropathy, either due to direct trauma (resulting from penetrating projectile or object into the orbit) or indirect trauma (pressure is forwarded to optic nerve through the globe and the orbit) (Pless M, no year; Kerrison JB and Miller NR, 1998; Kline LB, 1996). The loss of vision may be intermittent or fixed, depending on the extent and type of damage in optic nerve, as well as the promptness and accuracy of diagnosis and therapy. The purpose of this study was to identify the feature of patient's funduscopies, time between trauma and curing, treatment and results of Traumatic Optic Neuropathy (TON) outpatients at the Division of Neuro-ophthalmology, Dr. Soetomo Hospital, Surabaya, Indonesia.

Department of Ophthalmology
Airlangga University School of Medicine
Dr Soetomo Teaching Hospital, Surabaya

METHODS

This was a qualitative retrospective study. Data were taken from outpatients diagnosed with Traumatic Optic Neuropathy (TON) who visited the Division of Neuro-Ophthalmology, Department of Ophthalmology, Dr Soetomo Teaching Hospital, Surabaya, from January 2002 - October 2004. Data obtained were categorized according to age, time between trauma and curing, the feature of patient's funduscopies, treatment and results of treatment.

RESULTS

A number of 44 patients were diagnosed with Traumatic Optic Neuropathy (TON) from January 2002 to October 2004, comprising 10 female (22.73%) and 34 male patients (85%) aged between 15 - 80 years. Most of the patients, comprising 18 individuals (40.9%), belonged to the age group 10 - 19 years, while age group 20 - 29 years comprised 10 patients (22.73%) ; 30-39 years 10 patients (22.73%); 40-49 years 5 patients (11.36%); 50-59 years 1 patients (2.27%) and 80-89 years 1 patients (2.27%). Time between the occurrence of trauma and the first contact with the patients was mostly after week IV (19 patients, or 43.2%), and the other patients came at day 2, 1 patient (2.27%) day 3 to week I, 7 patients (15.9%), week II, 10 patients (22.72%), week III, 7 patients (15.9%) and week IV or more, 19 patients

(43.2%). The vision at the first contact was varied between 6/10 and negative light perception.

Most of the patients showed papillary atrophy (in 25 patients, 56.82%), while other patients had normal fundus (16 patients, 34.1%), papillary edema in 1 patient (2.27%), and vitreus bleeding in 2 patients (4.54%). Drugs used for therapy, as well as the length of therapy, were varied from NSAID, antioxidants, and neurotropics. The length of therapy varied between 1 and 14 weeks. Results of therapy in 44 patients: 11 patients (25%) had visual improvement, 9 patients (20.45%) showed no vision change, 3 patients (6.8%) experienced visual loss, and 21 patients (47.73%) did not return after the first therapy.

Table 1. Patients distribution according to age

Age groups	Female	Male
10 - 19 years	4	14
20 – 29 years	2	8
30 – 39 years	2	7
40 – 49 years	2	3
50 – 59 years	-	1
60 – 69 years	-	-
70 – 79 years	-	-
80 – 89 years	-	1
Total	10	34

Table 2. Time between trauma and the first contact

Time between trauma and the first contact	Total
2 x 24 hours I	1
> 2 x 24 hours I – Week I	7
Week II	10
Week III	7
Week IV and thereafter	19
Total	44

Table 3. Funduscopic features

Funduscopic features	Total
In normal range	16
Papillary Edema	1
Papillary Atrophy	25
Vitreus Bleeding	2
Total	44

Table 4. Patients with visual improvement after therapy

Patients' number	Age (Year)	F/M	Trauma-Contact I (day-)	Visus		Therapy			Length of therapy (Weeks)
				First	Last	Steroid	Neurot-ropic	Others	
1	25	M	2	LP +	5/7.5 ph 5/6	MP 3x32 mg	+	Antioxidant	3
4	15	M	18	1/300	3/60	-	+	NSAID	2
7	18	M	6	1/300	2/60	MP 4x16 mg	-	-	5
11	22	M	> 4 Week	1/60	5/60	-	-	Antioxidant	1
12	38	M	> 4 Week	5/60	5/60 ph 5/30	-	-	Antioxidant	1
14	18	M	7	1/300	1/60	MP 3x16 mg	+	-	3
21	32	M	15	3/60	6/6	-	+	+	3
31	18	F	21	3/60	4/60	-	+	NSAID	2
32	26	F	21	5/20	5/10	-	+	Antioxidant	2
34	16	M	7	6/40	5/6	-	+	-	4
44	17	M	21	1/300	1/60	MP 3x8 mg	+	-	1

Table 5. Patients without visual changes during therapy

Patients' number	Age (Year)	F/M	Trauma-Contact I (day-)	Visus		Therapies			Length of Therapy (Weeks)
				First	Last	Steroid	Neurotrop ic	Others	
2	17	M	150	1/60	1/60	-	+	-	3
3	17	M	150	LP -	LP –MG + RC -	-	+	-	4
9	15	M	> Week 4	LP +	LP +	MP 3x16 mg	+	Antioxidant	4
10	34	M	> Week 4	LP -	LP -	Prednisone 3x8 mg	+	-	6
13	32	F	II	1/60	1/60	MP 3x16 mg	-	-	1
15	17	M	II	LP -	LP -	-	+	-	14
19	15	F	30	LP -	LP -	-	+	Antioxidant	3
27	22	M	194	5/60	5/60	-	+	-	1
42	40	M	5	LP -	LP -	-	+	-	2

Table 6. Patients with visual reduction during therapy

Patients' number	Age (Year)	F/M	Trauma-Contact I (day-)	Visus		Therapies			Length of Therapy (Weeks)
				First	Last	Steroid	Neurot-ropic	Others	
25	31	F	12	LP +	LP -	MP 3x16 mg	+	-	1
28	26	F	30	5/60	1/60	-	+	Antioxidant	3
33	17	M	8	1/60	1/300	MP 1x32 mg	+	Antioxidant	3

Table 7. Patients loss to follow-up

Patients' number	Age (Year)	F/M	Trauma-Contact I (day-)	Visus	Therapies		
				First	Steroid	Neurotropic	Others
5	38	M	3	LP -	MP 3x8 mg	+	-
6	15	M	150	LP -	-	+	-
8	20	M	14	LP -	-	-	Antioxidant
16	17	F	Week 4	6/6	MP 4x16 mg	+	-
17	52	M	10	LP -	-	-	Antioxidant
18	15	M	16	LP -	MP 4x16 mg	+	-
20	32	M	30	1/60	MP 1x16 mg	+	-
22	27	M	3	LP -	MP 3x16 mg	+	-
23	18	M	3	LP -	MP 3x16 mg	+	-
24	80	M	14	LP -	-	-	Antioxidant
26	25	M	4	LP -	MP 1x32 mg	+	-
29	45	M	58	1/60	-	+	-
30	42	F	> Week 4	LP -	-	-	Antioxidant
35	18	F	> Week 4	LP -	-	-	Antioxidant
36	46	F	20 years	5/40	-	+	Antioxidant
37	22	M	10	LP -	-	-	Antioxidant
38	27	M	73	LP -	-	+	Antioxidant
39	30	M	87	1/300	-	+	Antioxidant
40	41	M	16	6/8.5	-	-	Antioxidant
41	30	M	14	1/60	-	-	NSAID Lyteers Polineomycin
43	18	M	9	LP -	MP 3x16	+	-

DISCUSSION

Traumatic Optic Neuropathy (TON) is a disorder in optic nerve resulting from trauma, both directly onto the eyes or indirectly affecting the optic nerve itself. Data available in this study revelaed no information on the mechanism of trauma, except the presence of previous history of traffic accidents before the reduction of visual acuity. The reduction is varied from normal to the negative perception of light. Patients with TON treated at Dr Soetomo Hospital had variable visual acuity at the first visit, from 6/10 to negative perception of light. Abnormalities commonly found in TON, besides the reduction of visual acuity, are the reduction of visual field, either central, paracentral, arcuate or altitudinal; the reduction of relative brightness, red desaturation and positive result of Marcus gunn dyschromatopsia test with negative direct light reflex. Funduscopic features in patients with TON are also varied. It may present as normal funduscopic feature, the presence of papillary edema, vitreus bleeding, retinal bleeding, and papillary atrophy, depending on the location and duration of TON in optic nerve. Papillary optic nerve will be athrophied 4 - 5 weeks after trauma. Funduscopic feature most commonly found in patients at Dr Soetomo Hospital was papillary atrophy (25 patients = 56.82%). This might be due to delayed first contact. Most of them came for treatment 4 weeks or more after trauma (19 patients = 43.2%).

According to age group, most of the patients belonged to age group of 10 - 19 years, followed with 20 - 29 years and 30 - 39 years. Individuals in these ages are those with high productivity who often have outdoor

activities, increasing the possibility of trauma during activity. According to sex, most of the patients were male. This confirmed the statement found in the Handbook of Ocular Disease Management, which stated that most of the patients were male aged from 10 to 30 years.

Diagnosis establishment and provision of treatment modalities required not only physical examinations, but also additional examinations, such as CT scan or MRI, to identify the presence of foreign bodies in the eyes, optic canal fracture, bleeding in optic nerve, superiosteal hematoma that suppresses optic nerve as well as optic nerve avulsion. In United States, 5% of Traumatic Optic Neuropathy (TON) cases were found in head injury, while 2.5% in those with midfacial fracture. These examinations, however, have not been routinely conducted in the Outpatient Clinic of Dr Soetomo Hospital for patients with TON.

The management of Traumatic Optic Neuropathy (TON) consists of strict observation, optic canal decompression, and the administration of high dose systemic corticosteroid. The success of treatment is determined by initial therapy, diagnostic accuracy, the extent of damage in optic nerve. Optic canal decompression and the administration of high dose systemic steroid may provide satisfactory results if they are given in the first 2 x 24 hours. This is because optic neuropathy may occur 48 hours post-trauma. The recommended steroid dose is intravenous methylprednisiolone of 30 mg/kg BB iv loading dose, followed with 5.4 mg/kg/day in drips for 2 - 3 days. Therapy given in Dr Soetomo Hospital was the administration of methylprednisolone in varied doses (8 - 100 mg/days) for patients admitted less than 4 weeks post-trauma with varied outcomes, ranging from improved to deteriorated visual acuity.

CONCLUSION AND SUGGESTION

Prompt and accurate diagnosis and therapy have an influence on the outcome of patients with Traumatic Optic Neuropathy (TON). The examination of visual acuity, visual field, red denaturation, relative brightness, color perception, funduscopy, and CT Scan or MRI should be routinely carried out to establish accurate diagnosis and procedures. Better outcome may be obtained if high dose systemic steroid and optic canal decompression (if needed) are given in the first 48 hours.

REFERENCES

Cook MW et al, 2003. Traumatic Optic Neuropathy. In: Danny M, et al (eds). Neuro-Opthalmology, Basic and Clinical Science Course section 5. San Fransisco American Academy of Ophthalmology, pp 154-5

James FA, Michael DS, 1997. Traumatic Optic Neuropathy. In: James FA, Michael DS (eds). Common Problems in Neuroophthalmology. London: WB Saunders Company, pp 110-1

Kerrison JB and Miller NR, 1998. Disorder of the optic Nerve and Afferent Visual System. In: MacCumber MW (ed). Management of Ocular Injury and Emergencies. Philadelpia-New York: Lippincot-Raven, pp 353-55

Kline LB, 1996. Traumatic Optic Neuropathy. In: Kline LB (ed). Optic Nerve Disorders, Ophthalmology Monograph section 10. San Fransisco: American Academy of Ophthalmology, pp 179-197

Kline LB and Bajandas FJ, 2001. The Pale Optic Disc: Optic Atrophy. In: Kline LB, Bajandas FJ (eds). Neuro-Ophthalmology, Review Manual. 5th ed. New York: Slack Incorporated, pp. 168-9