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The evaluation of patients with optic disc edema: A retrospective study

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ABSTRACT

OBJECTIVE: Optic disc edema is among major problems that neuro-ophthalmology clinics encounter. We intended to analyze patients with optic disc edema in this article.

METHODS: Data related to the main complaint, associated systemic disease, visual acuity, characteristics of optic disc swelling, other ocular findings, topical or systemic drugs, treatment methods, follow-up examination, and related data of the patients were obtained retrospectively.

RESULTS: There were 77 female and 23 male patients in the study. Optic disc edema was detected bilaterally in 65 patients, unilaterally in 35 patients. The duration of the symptoms until the first application was 19.82±17.18 (0–90) days. There were no systemic disorders in 74 patients but diabetes mellitus in 11 patients, hypertension in four patients, coronary artery disease in three patients, urticaria in two patients, lymphoma in one, multiple sclerosis in one patient, mastoiditis in one patient, scleroderma in one, and pregnancy in two patients were detected. While 93 patients had no additional ocular findings, 2 had uveitis, 1 had corneal dystrophy, 1 had keratoconus, 1 had cataract, 1 had previous cataract surgery, and 1 had peripheral retinal degenerations. The major etiology of the optic disc edema was idiopathic intracranial hypertension, which was detected in 44 patients. In all these patients, bilateral optic disc edema was observed and 43 patients were given oral acetazolamide and one patient oral topiramate.

CONCLUSION: The presence of optic nerve edema should be absolutely evaluated in patients presenting with symptoms of vision loss and increased intracranial pressure. The early diagnosis with fundoscopic examination may increase visual acuity in these patients.

Keywords: Etiology; ocular findings; optic disc edema.

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Optic disc edema is swelling of intraocular portion of the optic nerve. The axons of retinal ganglion cell which forms the nerve exit the eye through scleral lamina cribrosa and convey the visual signal to the occipital cortex. The compression of the fibers in the lamina cribrosa leads to tissue edema and increases intercellular matrix pressure [1]. Optic disc edema may present with optic nerve head bulging, hyperemia, loss of optic disc boundaries, vascular congestion and peripapillary hemorrhages [2]. A binocular indirect fundoscopy is essential to document optic disc findings. Especially, central venous pulsation loss is an important finding in the examination. Although the disc edema is isolated generally, sometimes retinal edema may accompany the picture and may give rise to neuroretinitis.

These patients may demonstrate symptoms of visual loss, headache, nausea, vomiting, pain on ocular motility, decreased color vision, constriction of visu-

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al field, and diplopia. The visual loss may range from mild to profound degree and an important cause for patient morbidity.

Optic disc edema arises from the blockage of retrograde and orthograde axoplasmic transport in the optic nerve [3]. Inflammatory, infectious, and other factors may impede the flow and various factors should be considered in evaluation of such patients including age, systemic disorders, duration of symptoms, visual loss, and unilaterality or bilaterality of the disease. Optic disc edema is among major problems that neuro-ophthalmology clinics encounter. Therefore, although not very valid in common practice, a comprehensive examination accompanying the diagnosis should be performed and patients should be searched ophthalmologically and systemically. In this article, we intended to analyze patients with optic disc edema.

MATERIALS AND METHODS

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. The study was conducted after getting the approval of the Kahramanmaras Sutcu Imam University Clinical Research Ethics Committee (approval number-date: 452-07.11.2018) and the patients who had the diagnosis of optic disc edema between January 2014 and November 2018 were determined from hospital electronic database retrospectively.

The data regarding the basic complaint, onset of symptoms, associated systemic disease, visual acuity, characteristics of optic disc swelling, other ocular findings, topical or systemic medications, radiological investigations including magnetic resonance imaging (MRI) and computerized tomography, treatment modalities, follow-up examination, and related data were obtained from the files of the patients.

In all patients, full ophthalmological examination including dilated fundus examination and visual acuity on a standard Snellen visual acuity chart had been earlier performed. Neuroradiological and other related consultations had been done and the final diagnosis of the patients had been ensured according to these results. The management of the patient according to the presumed diagnosis was recorded as well.

Highlight key points

- Optic disc edema may develop due to many ocular and systemic diseases.
- In patients with optic disc edema, detailed clinical and radiological examinations should be performed to determine the underlying cause.
- Treatment based on the cause can help prevent vision loss in these patients.

Statistical Analysis

The comparison of the data was done with a statistical software program (SPSS 20, IBM, Chicago, ILL). Numerical variables were given as mean±standard deviation, frequency, and percentage, and Wilcoxon signed-rank test was used for comparison of the means.

RESULTS

A total of 100 patients were included into the study finally after excluding patients with missing data and incomplete follow-up. The mean age of the patients was 38.58±15.11 (12–76) years. There were 77 (77%) female and 23 (23%) male patients in the study.

Sixtythree patients presented due to visual loss, 33 headaches, two with diplopia, two with headache, and one with photophobia. One patient was diagnosed with optic disc edema on a regular examination without any complaint.

Optic disc edema was detected bilaterally in 65 patients, unilaterally in 35 patients. The duration of the symptoms until the first application was 19.82±17.18 (0–90) days. There were no systemic disorders in 74 patients but diabetes mellitus in 11 patients, hypertension in four patients, coronary artery disease in three patients, urticaria in two patients, lymphoma in one, multiple sclerosis in one patient, mastoiditis in one patient, scleroderma in one, and pregnancy in two patients were detected. While 93 patients had no additional ocular findings, 2 had uveitis, 1 had corneal dystrophy, 1 had keratoconus, 1 had cataract, 1 had previous cataract surgery, and 1 had peripheral retinal degenerations.

While the medication history revealed that 82% of the patients used no medication, 10% of the patients used antidiabetics, 4% of the patients antihypertensives, 3% immunosuppressives, and 1% antibiotics.

In 13 of 65 patients with bilaterally edema and 13 of 35 patients with unilaterally edema had concomitant sys-

Table 1. Age, gender, and systemic disease relations in unilateral and bilateral disc edema

	Unilateral (n=35)	Bilateral (n=65)	p
Age (Mean±SD) (y)	46.74±16.91	34.18±12.06	<**0.001***
Additional systemic disorders (+/-)	13/22	13/52	0.065
Gender (F/M)	21/14	56/9	**0.005***
Patient with diagnosis	29	60	0.150

SD: Standard deviation; *: P≤0.05, the difference between the groups was statistically significant; F: Female; M: Male.

temic disorders. The 60 of 65 patients were diagnosed as having bilaterally edema and 29 of 35 patients unilaterally edema (Table 1).

Neuroradiological investigations demonstrated that no findings in 79 patients, while 21 patients showed pathological manifestations. As radiologically, ischemic optic neuropathy in 5 patients, optic neuritis in 4 patients, signs of idiopathic intracranial hypertension (IIH) in 4 patients, optic disc drusen in 3 patients, cavernous sinus thrombosis in 2 patients, intracranial mass in 2 patients, and diabetic papillopathy in 1 patient were detected.

Based on these evaluations, the diagnosis of patients were as follows: 44 patients IIH, 22 patients optic neuritis, seven patients non-arteritic ischemic optic neuropathy, three patients optic disc drusen, three patients hypertensive papillopathy, two patients cavernous sinus thrombosis, two patients diabetic papillopathy, two patients retinal vein occlusions, two patients intracranial mass, one patients Harada syndrome, and one patient sarcoidosis. Investigations yielded no possible diagnosis in 11 patients. Age, sex, laterality, the number of patients treated according to diagnoses, and visual acuities at initial application and at the last follow-up are presented in Table 2.

The management of the patients consisted of acetazolamide use in 43 patients, intravenous and oral steroids in 26 patients, acetylsalicylic acid (ASA) and clopidogrel in six patients, antihypertensives in three patients, coumadin in two patients, subtenon corticosteroid in two patients, immunosuppressives in two patients, intravitreal anti-vascular endotelial growth factor (anti-VEGF) therapy in two patients, and topiramate in one patient. Furthermore, the surgical intervention was made in two patients. The eleven patients were not received any treatment.

The mean follow-up duration was 14.83±14.50 (0–60) months. Treatment period was 32.22±22.04 (0–180) days. When the visual acuities evaluated with Snellen chart were examined, it was observed that visual acuity of 70 patients increased after treatment and visual acuity of 15 patients did not change. Furthermore, it was seen that visual acuity was decreased in four of the undiagnosed patients.

The major etiology of the optic disc edema was idiopathic IIH, which was detected in 44 patients. The mean age in this group was 33.38±10.49 (14–58) years and there were 42 female and two male patients. It was observed that bilateral optic disc edema was observed in all these patients, and 43 patients received oral acetazolamide and one patient received oral topiramate. The follow-up period for this group was 19.97±16.76 months.

DISCUSSION

In our study, we retrospectively investigated the patients having optic disc edema who had admitted to our eye clinic. It was found that optic disc edema was most frequently associated with IIH, but also it was determined that it was seen less frequently with other central nervous system pathologies and systemic diseases. It can be stated that the patients often applied with loss of vision and lesser symptoms of increased intracranial pressure. Furthermore, it was observed that visual acuity can improve with treatment that may change depending on etiological factors.

Optic disc edema may arise due to many factors. At the first place, any space occupying lesion in the central nervous system should be excluded, which may impose a lethal threat to the life. Other reasons for disc edema are include idiopathic IIH, ischemic, inflammatory, infiltrative lesions of optic nerve, toxic causes, and hereditary optic neuropathies [4].

The IIH was the most common etiology causing optic disc edema in our study. In the study, there were 44 IIH patients, of which 42 were female and two were male. Optic disc edema due to IIH is specifically called papilledema, though these two terms may be mistakenly used interchangeably sometimes [2]. IIHs are presumed to be generated due to decreased absorption of cerebrospinal fluid (CSF) through arachnoid villi [5]. Having an annual incidence of 0.9 in 100.000 people, it affects frequently obese fertile women aged between 20 and 44 years [6]. Obesity may increase CSF pressure by increased intra-abdominal pressure pressing on medulla spinalis or decreas-

TABLE 2. Age, sex, laterality, and the number of patients treated according to diagnoses and visual acuities at initial application and at the end of the follow-up

	Unilateral	Bilateral	Age	Male	Female	Initial visual acuity	Last visual acuity	Treatment
Idiopathic intracranial hypertension	0	44	33.38±10.50	2	42	0.85±0.30 (0.1–1.0)	0.92±1.82 (0.1–1.0)	44
Optic neuritis	17	5	40.30±16.69	10	12	0.47±0.24 (0.1–1.0)	0.62±0.33 (0.1–1.0)	22
Non-arteritic ischemic optic neuropathy	7	0	64.86±9.23	4	3	0.41±0.28 (0.1–1.0)	0.43±0.31 (0.1–1.0)	7
Optic disc drusen	1	2	27.33±11.01	0	3	1.0	1.0	–
Hypertensive papillopathy	0	3	42.00±5.66	2	1	0.80±0.28 (0.6–1.0)	0.80±0.28 (0.6–1.0)	3
Diabetic papillopathy	0	2	46.50±17.67	0	2	0.80±0.14 (0.7–0.9)	0.80±0.14 (0.7–0.9)	1
Cavernous sinus thrombosis	0	2	35.00±5.66	0	2	1.0	1.0	2
Retinal vein occlusions	2	0	59.5±9.19	0	2	0.30±0.28 (0.1–0.5)	0.60±0.00 (0.6–0.6)	2
Intracranial mass	1	1	46.50±2.12	1	1	1.0	1.0	2
Harada syndrome	0	1	19	0	1	0.9	1.0	1
Sarcoidosis	1	0	31	1	0	0.7	1.0	1
No diagnosis	6	5	37.91±15.60	3	8	0.61±0.36 (0.1–1.0)	0.53±0.36 (0.1–1.0)	4
Total	35	65		25	75			89

ing venous return to the heart from the brain [7]. Patients generally apply to physician due to headache, blurred vision, photophobia, tinnitus, or diplopia although asymptomatic cases have also been reported [8, 9]. Diagnosis can be achieved with a high CSF pressure, radiological methods, and normal CSF biochemistry in addition to the presence of a optic disc edema [10]. Treatment relies on decreasing the production of CSF by acetazolamide, topiramate, furosemide, or in some cases minimal corticosteroids [11, 12]. Furthermore, there are some researches reporting that weight loss could help improve the prognosis [13]. Our study is in congruity with the literature that IIHs were seen mostly in young female patients who responded well to acetazolamide therapy. Eating habits and nutritional preferences depending on the geographical area are seen major factors causing expansion of obesity in our country and worldwide. In our study, IIH may be related to high obesity frequency encountered in this region.

In our study, 22 patients were diagnosed as optic neuritis, which was the second most common cause of optic nerve head edema. It is non-infectious inflammation of the optic nerve and can be classified as papillitis, retrobulbar neuritis, or typical/atypical optic neuritis. Typical optic neuritis is generally associated with multiple sclerosis, which is acute, inflammatory demyelinating disease with relatively milder prognosis. Atypical optic neuritis is a kind of optic neuritis developed due to infectious, inflammatory reasons other than multiple sclerosis or autoimmune causes [14, 15]. The optic neuritis has an incidence of 1–2/100.000 people and is generally common among young, white, female population [14].

Unilateral central acute visual loss is a commonly encountered symptom and relative afferent pupillary defect and color vision disturbance generally accompany the disease at early period. MRI and CSF analyses should be undertaken essentially to rule out multiple sclerosis (MS) in isolated optic neuritis cases [16, 17].

Optic neuritis treatment trial demonstrated that a patient with a diagnosis of optic neuritis had a risk of MS development of 40% in 10 years. For optic neuritis patients with normal MRI, this risk is 22% while patients having 3 mm plaques on MRI have a risk of 56% [16, 18].

Therapeutic corticosteroids may improve visual prognosis in early period but have no effect at the end of 3 years. Immunomodulatory interferons could be suggested since they reduce relapse frequency [18]. In atypical optic neuritis, therapy should be oriented toward etiology and steroids along with other immunosuppressive drugs may improve prognosis substantially in neuromyelitis optica [14, 18]. In our study, investigation to reveal the underlying etiology of the cases was carried out but still many cases remained undiagnosed and only one optic neuritis case who had been diagnosed with MS before was detected.

Non-arteritic ischemic optic neuropathy is another possible cause of optic disc edema, which is characterized with painless acute visual loss in patients over 50 years of age [19] and was detected in seven patients in our study. It has a reported incidence of 2–10 in 100.000 people and does not show any gender tendency [20]. Diabetes mellitus, hypertension, smoking, acute hemorrhage, anemia, and hypotension are among the risk factors reported [21]. There is not any certain treatment modality for the disease but anticoagulants, subcutaneous vasodilators, and thrombolytics have been tried with limited success [22]. The role of corticosteroids is controversial and not free from side effects, especially in diabetic, hypertensive, and elderly people, in spite of some probable positive effects, its use is limited [23]. ASA has no role in the treatment of non-arteritic ischemic optic neuropathy but its use may have a protective effect on the fellow eye [24]. Our study found similar clinical features with the literature with regard to this patient group.

Uncontrolled diabetes and hypertension may lead to diabetic papillopathy bilaterally or unilaterally and we had two patients with this clinical entity [25, 26]. The disc edema in diabetic papillopathy is caused by microvascular circulation disorder of the optic disc, and pronounced telangiectatic vessels may be difficult to distinguish from neovascularization. The moderate vision loss may occur in these. Diabetic papillopathy can occur in the settings with no diabetic retinopathy. The treatment of associated systemic disorders may help alleviate disc edema. Systemic steroid should be avoided not to worsen blood sugar and hypertension control but the use of intravitreal triamcinolone was reported in some research [27, 28]. In our study, it was found that these patients with uncontrolled diabetes and hypertension were treated with endocrinology and nephrology clinics through consultation.

Pseudopapilledema is swollen appearance of optic disc in conditions without any disease. This condition should be distinguished before any treatment is supplemented. Optic disc drusen, myelinated nerve fibers, and high hypermetropia may give rise to a pseudopapilledema appearance. Optic disc drusen could be differentiated easily with autofluorescence, ultrasonography, computerized tomography, and optic coherence tomography [29, 30]. These drusen materials are calcified hyaline bodies and do not require any treatment.

Posterior uveitis, retinal vein occlusion, posterior scleritis, and other systemic infections (tuberculosis, lepra, etc.) and autoimmune disorders (Harada disease, sarcoidosis, etc.) can lead to optic disc edema [1]. The treatment should be tailored according to the etiology in these cases.

There are some limitations in our study. First, there were some patients who did not have enough data in their files, which required the exclusion of those patients. Furthermore, despite clinical and radiological research, no reason for optical disc edema was found in some. There could be other patients whom we were unable detect and missed due to some mistakes at diagnosis entry since we searched our database for predetermined diagnostic terms such as optic neuritis or optic disc edema.

Conclusion

Optic disc edema is a manifestation of several disorders rather than a diagnosis and should be detected at an early level. A thorough clinical investigation should aid the physician to make decisions and a detailed approach should accompany the management. The presence of optic nerve edema should be absolutely evaluated in patients presenting with symptoms of vision loss and increased intracranial pressure. As well as early diagnosis with fundoscopic examination before neuroradiological examinations, a treatment can be arranged that according to etiological factors may increase visual acuity in these patients.

Ethics Committee Approval: The Kahramanmaras Sutcu Imam University Clinical Research Ethics Committee granted approval for this study (date: 07.11.2018, number: 452).

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