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OPTIC NERVE CHANGES IN CHRONIC SINUSITIS PATIENTS: CORRELATION WITH DISEASE SEVERITY AND RELEVANT SINUS LOCATION

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ASBTRACT

Background: Optic neuritis is an acute inflammatory disorder of the optic nerve that is clinically manifested by a temporary but severe unilateral visual loss. In a typical case, impaired color vision, visual field defects and a relative afferent pupillary defect can also be observed in the affected eye. Optic neuritis may be a complication of many diseases. The most common is demyelination secondary to multiple sclerosis or neuromyelitis optica spectrum disorders. However, optic neuritis has also been reported in association with hypertension and hyperthyroidism, infectious diseases such as rubella, mumps, measles and mononucleosis, systemic neoplasia, lead, arsenic, and methanol poisoning and paranasal sinus diseases. **Materials and methods:** Data were collected from eighty-eight eyes of 46 chronic sinusitis patients and 93 eyes of 57 normal controls. Visual sensitivity using standard automated perimetry (SAP) and inner retinal thickness using optical coherence tomography (OCT) were measured. The LundMackay system was used to quantify radiographic findings on the ostiomeatal unit CT scan with a numerical score representing the severity of sinusitis. **Result:** There was a significant positive correlation between the pattern standard deviation (dB) and Lund-Mackay score ($P = 0.031$). Nasal retinal nerve fiber layer (RNFL) thickness, average, minimum, superotemporal, superior, superonasal, and inferonasal ganglion cell-inner plexiform layer (GCIPL) thickness were negatively correlated significantly with Lund-Mackay score (all, $P < 0.05$). Eyes with grade 2 opacification of the posterior ethmoid sinus showed a significantly lower mean deviation (dB) and higher pattern standard deviation (dB) than those with clear respective sinuses ($P = 0.007$ and <0.001, respectively). **Conclusion:** Though rhinogenic optic neuropathy as a complication of sinusitis is uncommon, this report reaffirms the importance of

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considering and recognizing sinusitis as a possible cause of visual loss and the need for the prompt initiation of medical and surgical treatment of the underlying disease.

Keywords: Sinusitis, Optic neuropathy, Optic neuritis, Onodi cell, Sphenoethmoiditis

INTRODUCTION

Optic neuritis is an acute inflammatory disorder of the optic nerve that is clinically manifested by a temporary but severe unilateral visual loss. In a typical case, impaired color vision, visual field defects and a relative afferent pupillary defect can also be observed in the affected eye. [1] In approximately 90% of all cases, the patients have pain, particularly during eyeball movements, for about 3-5 days. Vision recovery lasts over the subsequent 3-6 weeks. [2] Optic neuritis may be a complication of many diseases. The most common is demyelination secondary to multiple sclerosis or neuromyelitis optica spectrum disorders. [3] However, optic neuritis has also been reported in association with hypertension and hyperthyroidism, infectious diseases such as rubella, mumps, measles and mononucleosis, systemic neoplasia, lead, arsenic, and methanol poisoning and paranasal sinus diseases. [4,5]

The mechanisms of optic nerve damage due to sinusitis include occlusive vasculitis and bone loss in the sinus wall. [6] Acute isolated sphenoid sinusitis occurs in 3-7% of cases of all paranasal sinus infections. [7] The most common sign of ON related to sphenoid sinusitis is headache, which can be retrobulbar (different from discomfort acceptable as a feature of demyelinating ON), parietal, or frontal. In addition, systemic symptoms such as nausea/vomiting, dizziness, fever, or ophthalmoplegia may be reported. The pathological diagnosis usually involves bacterial infection, but it may also be fungal infections such as aspergillosis, sinus tumor, or polyps. [8]

In the beginning, the optic disc was elevated with normal average RNFL thickness on OCT. However, with subsequent attacks of inflammation, we observed progressive RNFL reduction in the affected eye, which suggested degeneration of the optic nerve fibers. [9] After surgical drainage, the patient reported having occasional slight headaches, but neuropathy of the left optic nerve resulted in permanent vision loss. A causal relationship between sphenoid sinusitis and optic neuritis has been reported several times in the available literature. [10] On examination, visual acuity of the affected eye decreased to light perception, and the optic nerve was grey with the neuroretinal rim almost completely absent. [11] Polyps were noted on nasendoscopy, and computed tomography identified an extensive paranasal sinus disease causing osteomyelitis of the sphenoid sinus wall and involving the orbital apex and the chiasm on the affected side. [12]

The optic nerve heads appeared normal in both eyes, but VEPs showed increased latency and reduced amplitudes on both sides, more prominent on the right side. The fundus was normal in both eyes, but VEPs showed absent response on the left side. [13] Radiological imaging revealed optic neuropathy caused by an isolated mucocele in a sphenoethmoidal air cell, commonly

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known as the Onodi air cell. The patient did not agree to rhinological surgery and was treated conservatively with antibiotics, mucolytics, and decongestants. [14]

MATERIALS AND METHODS

In this prospective study conducted over a 1-year period in a tertiary eye care center and department of Ophthalmology, consecutive subjects (40 chronic sinusitis patients and 40 normal controls) were recruited. This study received approval from the institutional review board and was conducted in accordance with the Declaration of Helsinki. Patients agreed and participated in this study by their own free will.

Patients and control groups

Patients with chronic sinusitis, who had symptoms for at least 12 weeks and whose symptoms persisted despite adequate medication, were enrolled. Their chronic sinusitis was confirmed by endoscopy (mucopurulent nasal discharge and/or nasal polyposis) and computed tomography. On the other hand, those with negative endoscopic and CT findings for sinusitis and without symptoms of chronic sinusitis were enrolled as controls. We excluded patients with an unstable systemic disease and pregnant or lactating

Ophthalmologic examinations

The ophthalmologic examination was performed within one month before and after the ENT examination. Ophthalmologic eligibility criteria were determined based on a complete ophthalmologic examination, which included a review of the patient's medical history, bestcorrected visual acuity measurements through manifest refraction, Goldmann applanation tonometry, slit-lamp examination of the anterior segment, gonioscopy, dilated fundus examination, red-free fundus photography, Humphrey standard automated perimetry visual test, and Cirrus OCT. Eyes were recruited from subjects with no history or evidence of intraocular surgery, no media opacity on slit-lamp examination, no history of glaucoma (personally or in a first-degree relative), no smoking and alcoholic habits, no family history of optic neuropathy, and no retinal pathologic features.

A VF test was performed by automated static perimetry. The results of VF testing were considered reliable when fixation loss was < 20%, and false-positive and false-negative errors were both < 15%

In all participants, a Cirrus HD-OCT was used to acquire one optic disc cube protocol and one macular cube protocol in each qualifying eye. The optic disc cube protocol was designed to position the cube scan on the optic nerve head (ONH). This protocol generated a cube of data through a 6-mm-square grid by acquiring a series of 200 horizontal scan lines, each composed of 200 A-scans (40,000 points). The retinal nerve fiber layer (RNFL) thickness at each pixel was measured and an RNFL thickness map was generated. A calculation circle 3.46 mm in diameter

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and consisting of 256 A-scans was automatically positioned around the optic disc and the mean and sectoral (temporal, superior, nasal, and inferior) RNFL thicknesses were subsequently measured.

Classification according to the relationship between the paranasal sinuses and optic nerve

The patients who had a sphenoid sinus and/or posterior ethmoid sinus involvement on CT scan were categorized into four groups based on the classification by Delano,[16] which associates the relationship between the sphenoid sinus, posterior ethmoid sinus, and the optic nerve. The optic nerve was categorized as (1) Type 1 when it was found to be lying adjacent to the superior and lateral walls of the sphenoid sinus, (2) Type 2 when it was found to make an indentation on the sphenoid sinus, (3) Type 3 when it traversed the sphenoid sinus, and (4) Type 4 when it was adjacent to the sphenoid and posterior ethmoid sinuses and covered by aerated cells.[16]

Statistics

When both eyes of a patient were eligible, both were included in the analysis. Visual sensitivity parameters and retinal thickness parameters were compared between the chronic sinusitis group and normal control group using the generalized estimating equation (GEE) adjusted for inter-eye correlation. Ophthalmic features among the four groups that were classified by the anatomic relationship between the paranasal sinuses and optic nerve were compared using GEE adjusted for age, sex, spherical equivalent, and inter-eye correlation, and the comparisons were done by post-hoc analysis. Data analysis was carried out using the Statistical Package for Social Sciences (SPSS) version 27.0 (SPSS, Inc., Chicago, IL, USA).

RESULTS

There were 80 eyes of 40 patients in the chronic sinusitis group and 100 eyes of 50 subjects in the normal control group. The time difference between OMU CT study and the visual field and OCT was 51.75 ± 30.55 days. The mean age of the chronic sinusitis and normal control group was 49.18 ± 14.58 and 52.98 ± 15.55 years (mean ± SD) (P = 0.148) (Table 1).

The mean intraocular pressure (IOP) for patients with chronic sinusitis and normal controls was 16.28 ± 0.57 and 17.05 ± 0.52 (mean ± SE), respectively (P = 0.288). The corredted visual acuity for patients with chronic sinusitis and normal controls was 0.07 ± 0.18 and 0.08 ± 0.18 (LogMAR), respectivlely (P = 0.655). The visual field mean deviation of the chronic sinusitis and normal control groups were -1.48 ± 0.33 dB and -0.68 ± 0.29 (mean ± SE) (P = 0.048). The average and sectoral RNFL and the average and sectoral GCIPL thickness measured using OCT did not differ significantly between those with chronic sinusitis and the normal controls (Table 2).

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Table 1. Baseline characteristics of study population.

	Control(n=50)	Sinusitis(n=40)	P
Age(y)	52.98±15.55	49.18±14.58	0.148
M: F	32:18	25:15	0.058
DM(Yes)	2(4%)	6 (15%)	0.088*
Hypertension(Yes)	6 (12%)	5 (12.5%)	0.868

Table 2. Ophthalmic characteristics according to the presence or absence of chronic sinusitis

	Control eyes(n=50)	Sinusitis eyes(n=40)	P
Intraocular pressure(mmHg)	17.05±0.52	16.28±0.57	0.288
Spherical equivalent(Diopter)	-1.00±0.36	-0.59 ±0.28	0.295
Corrected visual acuity (LogMAR)	0.08±0.18	0.07±0.18	0.655
VF			
Mean deviation	-0.68±0.29	-1.48 ±0.33	**0.048**
Pattern standard deviation	1.72±0.18	1.72±0.18	0.342
OCTRNFL parameter(μm)			
Average thickness	98.65±2.23	97.98±2.65	0.741
Temporal thickness	72.78±2.64	71.44±2.54	0.543
Superior thickness	124.45±2.85	122.45±4.48	0.515
Nasal thickness	71.82±2.28	72.81±2.48	0.598
Inferior thickness	125.48±2.99	126.23±3.93	0.841
OCTGCIPL parameter(μm)			
Average thickness	84.85±0.82	85.07±0.97	0.845
Minimum thickness	81.82±0.94	82.48±2.07	0.635
Superotemporal thickness	84.33±0.85	84.33±0.98	0.999
Superior thickness	85.41±0.88	86.00±1.08	0.635
Superonasal thickness	86.81±0.93	87.42±1.15	0.668
Inferonasal thickness	84.49±0.88	85.23±1.07	0.588
Inferior thickness	82.45±0.88	82.74±0.99	0.831
Inferotemporal thickness	85.35±0.82	85.78±0.98	0.736

DISCUSSION

Optic neuritis is an inflammatory neuropathy affecting the optic nerve that presents with visual loss. The patient may also complain of pain with eye movement. An afferent pupillary defect is present and visual field loss may be demonstrated. Optic neuritis secondary to sinus disease is an infrequent but documented association. [15] If unabated, as the second case described here illustrates, optic neuropathy or damage to the optic nerve may result.

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There are various means by which sinusitis is postulated to give rise to optic neuritis and/or neuropathy. Direct spread of the sinus infection to the optic nerve is probably the most common way in which this occurs. [16] Osteomyelitis affecting the sinus walls secondary to chronic infection may also be in direct contact with the nerve. [17]
Compressive optic neuropathy may be caused by ethmoid and/or sphenoid sinus mucoceles or mucopyoceles and/or associated edema and thickening of the sinus walls. [18] Polyps involving the mucosa of the sphenoid sinus also cause compression of the nerve. [19]

The local anatomy of the venous circulation in the orbitalapical region may also play a role in the pathogenesis of optic neuropathy associated with sinus disease. Optic neuropathy could be related to the spread of cytokines and/or immune mediators from the sinuses to the orbital apical portion of the optic nerve through the local venous circulation, or to local vasomotor changes for example. That the area of the most prominent inflammatory changes observed in the optic nerves of those patients with optic neuropathy on a background of sinus disease in previous studies was found where the superior and inferior ophthalmic veins traversed the orbit close to the nerve adds some weight to this theory. [20] Secondary inflammatory occlusive vasculitis causing optic neuritis would explain the rapid resolution which occurs in some cases following antibiotic therapy.

Acute sphenoid sinusitis can be fatal. It may be associated with serious complications including permanent cranial nerve deficits. The incidence of vision loss and third, fourth, fifth and sixth cranial nerve palsies for inflammatory lesions have been reported to be 12% and 12%, respectively. [21,22]

Visual loss in association with sphenoid sinusitis should be considered a rhinological emergency. [23] Parenteral antimicrobial therapy is recommended as the first-line treatment. The initial presentations of impaired vision and/or afferent pupillary defect associated with ipsilateral sinusitis have been cited as indications for surgical exploration of the involved sinuses. [24] The first case we describe here illustrates that this may not always be necessary. Drainage and sinus decompression should however be performed if the symptoms worsen or continue for 24 - 48 h or if there are signs of complications.

Complication rates of endoscopic sinus surgery range from 2% to 17% depending on the study reviewed. [25] In experienced hands, the overall complication rate is around 9% with those most common being adhesion formation, orbital hematoma and antrostomy closure. More serious complications such as cerebrospinal fluid leak with meningitis, diplopia and blindness are very rare. Overall, endoscopic sphenoethmoidectomy is a safe procedure in experienced hands. [26]

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CONCLUSION

Though rhinogenic optic neuropathy as a complication of sinusitis is uncommon, this report reaffirms the importance of considering and recognizing sinusitis as a possible cause of visual loss and the need for the prompt initiation of medical and surgical treatment of the underlying disease. The presence of sinus opacity and particularly of the sphenoid and/or ethmoidal sinuses in patients with optic neuritis may well be significant and should not be dismissed as an incidental finding. The first case we describe here adds further evidence to previous reports in the literature that optic neuritis can occur with sinusitis secondary to non-compressive mechanisms. The second case we describe here confirms that chronic sinusitis is an indolent inflammatory process that can cause visual loss.

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