Peripheral scotoma associated with chronic ethmoidal sinusitis. A case report and review of the literature*

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SUMMARY

Diseases of the paranasal sinuses may cause visual disturbances, especially diminished visual acuity and visual field defects, by affecting the optic nerve. We report the unusual case of a female patient with unilateral peripheral, quadrantic scotoma and concomitant chronic ethmoidal sinusitis. Visual acuity was not diminished. Despite extensive diagnostic examinations no other cause of the scotoma could be evaluated. As conservative therapy had been unsuccessful, endonasal pansinus operation was performed. Upon surgery, nearly all ethmoidal cells appeared to be filled with polypous mucosa. The sphenoid sinuses, however, contained air. In contrast, pre-operative CT scans had shown only a slight opacity of the ethmoid bone. Already two weeks after surgery a reduction in size of the scotoma could be noted. In addition to this case report, possible causes of visual field defects due to inflammatory diseases of the paranasal sinuses are discussed. In cases of unilateral visual field reduction associated with symptoms of chronic paranasal sinusitis, early operative exploration of the paranasal sinuses should be considered after exclusion of other possible causes, even if radiological findings do not warrant such a procedure.

Key words: scotoma, chronic sinusitis, sinus surgery

INTRODUCTION

A frequent complication of inflammatory diseases of the paranasal sinuses is their spread into the orbit. Thus, there are many reports on orbital complications associated with acute and acutely-exacerbated chronic sinusitis and with formation of muco- and pyocoeles (Welsh and Welsh, 1974; Smelt and Migdal, 1983; Slavin and Glaser, 1987; Patt and Manning, 1991). They have their origin mostly in the ethmoidal cell system and the frontal sinus, less frequently in the maxillary sinus of the affected side. Depending on the severity of symptoms, these complications are being classified under pathological and anatomical aspects such as lid oedema, orbital periostitis, subperiostal abscess and orbital phlegmone, respectively abscess (Chandler et al., 1970; Kronschnabel, 1974; Kastenbauer, 1992). Functional disorders include diplopia, reduction in visual acuity and visual field defects.

Visual disorders caused by clinically-occult chronic inflammation of the paranasal sinuses are rather rare by comparison. This is why there are only occasional case reports in the literature (Rothstein et al., 1984; Simpson and Moser, 1988; Sato et al., 1994). We report here on a female patient with bilateral chronically-polypous ethmoidal sinusitis and unilateral peripheral quadrantic scotoma on the left side, which diminished after curative paranasal sinus operation.

CASE REPORT

A 25-year-old female patient presented for the first time at the Department of Ophthalmology (University of Göttingen) on June 3, 1996. She complained about a ”black spot” in her left eye that had persisted for about three weeks. Anamnesis did not reveal previous ophthalmological diseases. She suffered from chronic bronchitis since birth. When specifically questioned the patient reported recurrent nasal infections, but denied headache. Visual acuity was not diminished on either side. Inspection of the visual field (Goldmann-type dynamic perimetry) revealed no pathological findings, but on the left side we found a peripheral quadrantic scotoma in the temporal superior visual field, extending up to 15° towards central (Figure 1A). The anterior segments of both eyes were normal for her age; as was intraocular pressure with 12 mm Hg, which is within the normal

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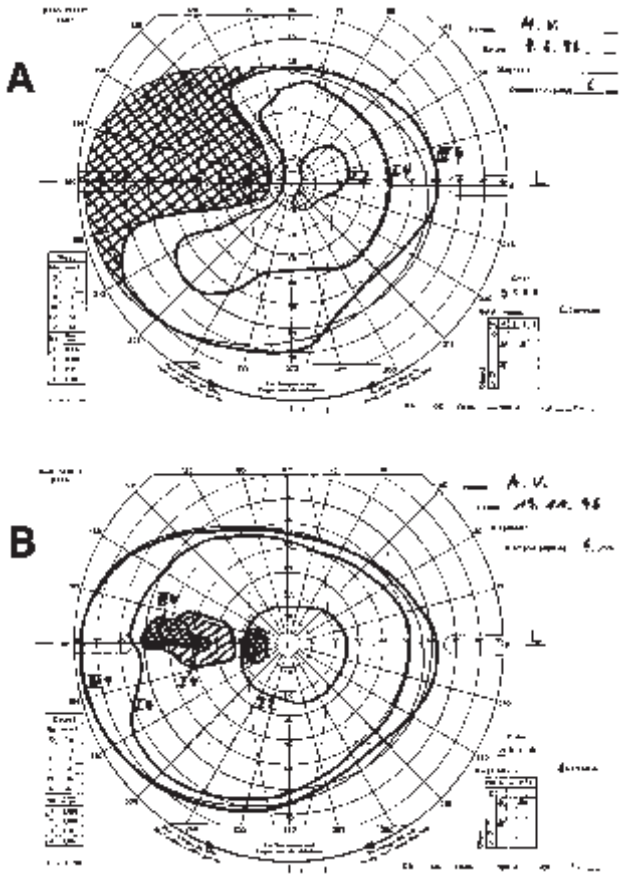

Figure 1. Left visual field before (A: June 4, 1996) and after (B: November 19, 1996) paranasal sinus operation. The reduction in size of the temporo-parietal scotoma is clearly visible.

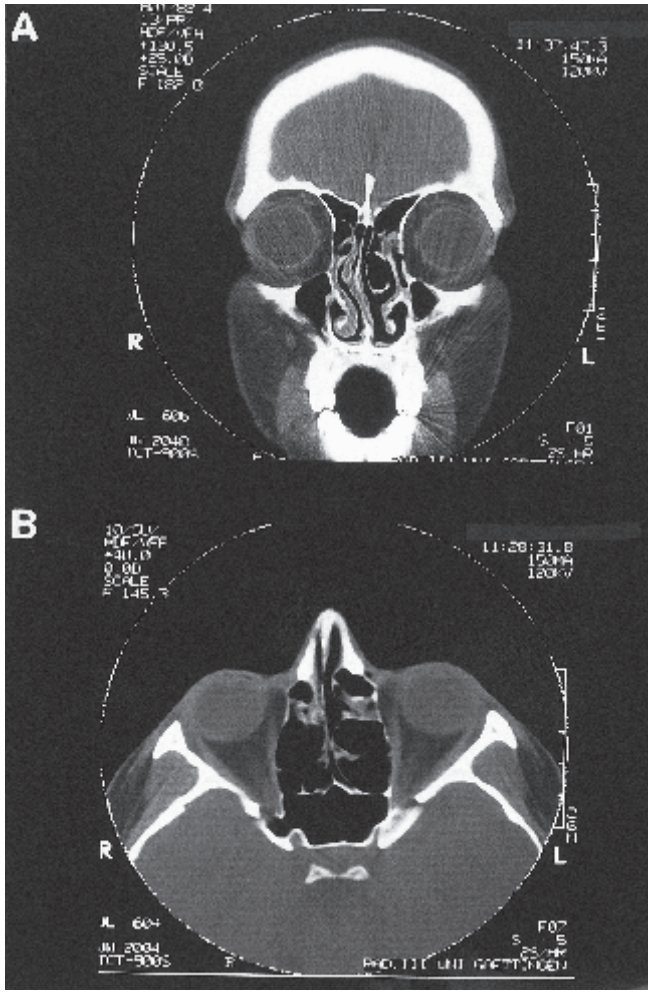

Figure 2. Pre-operative paranasal sinus CT scans (November 5, 1996): coronal (A) and axial (B) sections. Circumscribed opacity is found only in single anterior ethmoidal cells. Upon surgery (November 6, 1996), however, nearly all ethmoidal cells appeared to be filled with polypous mucosa. The sphenoid sinuses on both sides seem to be free, with relatively extended pneumatization. The right optic nerve seems to run partially free through the sphenoid sinus (B).

range. Funduscopy showed no pathological changes of the optic nerve, macula, blood vessels and peripheral retina. There were no clinical signs of papillitis or retrobulbar neuritis, and visually evoked potentials gave no indication of pathologic changes. CT scans of the orbit showed a normal image of the optic nerve up to the optic canal on both sides, contrast CT-scans and MRI excluded displacement by tumour, bleeding and ischaemic or inflammatory cerebral lesions. Diagnostic serology was negative with respect to acute infection or autoimmune diseases. Neurological examination was unsuspicious, as was Doppler sonography of both carotid arteries. Suspecting a posterior ischaemic neuropathy of the optic nerve, rheologic therapy was applied consisting in 500 ml/day of 6% Haes and 600 mg/day of pentoxifyllin (i.v.) for 7 days, but subjective discomfort and objective findings remained unchanged.

The patient first presented at our ENT department on June 7, 1996. Except for a septum deviation all findings were normal. CT of the paranasal sinuses revealed circumscribed opacification of single ethmoidal cells. Both sphenoid sinuses were well pneumatized and contained air. The left maxillary sinus showed a moderate, marginal opacification. In view of these apparently only slight inflammatory sinusoidal changes our treatment consisted, for the time being, in a 10-day antibiotic and decongestive therapy with amoxycillin tablets and decongestive nose drops.

Further ophthalmological control examinations were performed on July 5, August 15 and November 5, 1996. The scotoma on the left side was found to be unaltered. Another CT scan of the paranasal sinuses conducted on November 5, 1996, did not differ from the first (Figure 2A-B). When the patient presented again at the ENT department for another consultation, we decided to perform an endonasal pansinus operation, including correction of the septum deviation, on the following day (November 6, 1996). During surgery it became apparent that, different from the CT findings, all ethmoidal cells were nearly completely filled with polypous mucosa. The sphenoid sinuses on both sides, however, contained air and were free of secretion and inflammatory mucosal changes. Furthermore, there was no evidence of bony dehiscences of the lateral wall. To provide optimum ventilation of the sphenoid sinuses, their anterior walls were opened wide. Histological examination of the mucosa removed from the paranasal sinuses showed the typical signs of chronic sinusitis, without any indications of malignancy.

The post-operative course was free of complications, and the nasal plug was removed on the second post-operative day.

Already then, the patient subjectively noted a decrease in scotoma size. Thirteen days after the operation, she was examined again at the Department of Ophthalmology. Goldmann perimetry did not show anymore absolute quadrantanopia (Figure 1B), while the other findings were unchanged and full visual acuity was preserved. Besides a slightly enlarged blind spot, merely an absolute scotoma of the dimension 20°x10° was demonstrable temporally in the middle periphery, adjacent to a relative scotoma of about equal size, and projecting centrally. The outer borders were free. Findings by perimetry were unchanged at another control examination conducted on March 6, 1997. Nasal endoscopy revealed a normal ethmoidal mucosa and the openings of the sphenoid sinuses appeared wide and free.

DISCUSSION

Several possible causes for the involvement of the orbit, respectively the optic nerve, in inflammatory diseases of the paranasal sinuses have been suggested, such as the spread of the infection by dehiscences in the orbital walls or in the optic nerve canal, spread of the infection via lymphatic or venous vessels, and inflammation-induced pressure on the optic nerve (Chandler et al., 1970; Kronschnabel, 1974; Welsh and Welsh, 1974; Rothstein et al., 1984; Slavin and Glaser, 1987; Simpson and Moser, 1988; Shimo-Oku et al., 1989; Patt and Manning, 1991; Sato et al., 1994; Postma et al., 1995). Ischaemic lesions of the optic nerve and retina by thrombophlebitis of the orbital veins or by vasculitis of the orbital vessels is considered to be still another cause of rhino- or sinusogenic visual disorders. Inflammation-induced involvement of the optic nerve into focal processes is a matter of controversy.

Inflammation of the optic nerve (*neuritis nervi optici*) is commonly accompanied by a reduction of visual acuity and a central, respectively centrocoecal, visual field loss. The majority of cases shows involvement of the more posterior portions, and the optic disc appears normal on funduscopy (retrobulbar neuritis). The acute stage of optic nerve inflammation is usually followed by spontaneous partial remission. Optic neuritis is in most cases associated with multiple sclerosis. Further causes are, besides infectious diseases and intoxications, also inflammatory diseases of the paranasal sinuses. In approximately 25% of all patients with optic neuritis signs of sinusitis are found (Bossy et al., 1961; Sanborn et al., 1984).

The connection between visual disorders, on the one hand, and chronic sinusitis and missing signs of acute inflammatory orbit involvement, on the other hand, is controversial. There are reports in the literature describing improvement of the clinical picture after curative operation for inflammatory processes not directly adjacent to orbit or optic canal (Philippe et al., 1991). Cases are known in which the signs of optic neuritis following paranasal sinus surgery improved, although intra-operatively no inflammatory mucosal changes had been found (Rothstein et al., 1984). It seems possible that at least some of these cases were connected with a clinically still-occult multiple sclerosis, suggesting the need for further follow-up (Chawla et al., 1991; Kastenbauer, 1992), especially since chronically-inflammatory changes of the paranasal sinuses are said to occur more frequently in multiple sclerosis patients (Gay et al., 1986).

If an inflammation of the paranasal sinuses is accompanied by reduced vision, with radiologically demonstrable orbital infiltration or clinical symptoms of orbital complications, the conclusion of a rhinogenic or sinusogenic cause of optic neuritis seems justified. This applies also to the rare cases in which visual loss and visual field defects predominate and present as initial symptoms, while other signs of acute orbital involvement remain in the background. Pathological courses like these have been described as “posterior orbital cellulitis” by Slavin and Glaser (1987), and the inflammatory process is supposed to encompass mainly the posterior orbital parts. As the symptoms of chronic inflammation of the ethmoid bone or the sphenoid sinus are often only weakly pronounced, sometimes only a CT scan may demonstrate the sinusogenic origin (Sanbom et al., 1984; Simpson and Moser, 1988). Besides unspecific inflammatory processes, differential diagnosis must also pay attention to, and possibly exclude, neoplasia, fibrous dysplasia, formation of mucocoeles and mycoses, particularly *Aspergillus* infection, of the paranasal sinuses (Yumoto et al., 1985; Rifai and Kenawi, 1990).

Data vary on the prognosis of sinusogenic visual loss (Kronschnabel, 1974; Postma et al., 1974). Irreversible loss occurs particularly in cases of intraorbital abscess formation, which is not always clearly visible by CT scans (Patt and Manning, 1991). Reductions in visual field are described for these cases as concentric restriction of the visual field as well as central scotoma (Kronschnabel, 1974; Rothstein et al., 1984; Simpson and Moser, 1988).

In the case of our patient with chronically-polypous pansinusitis there has been a unilateral, peripheral quadrantanopsia in the temporal superior visual field of the left eye, without reduction of central visual acuity. As far as we know, this constellation has not yet been described in the literature. The fully preserved visual acuity and the type of visual field loss do not support the assumption of an inflammation of the optic nerve as this is usually accompanied by a sharp decline in central visual acuity and by the occurrence of a central or centrocoecal scotoma.

In our opinion, the sector-shaped configuration of the scotoma and its rapid decline in size after surgery can be explained by a circumscribed affection of the left optic nerve. The loss in the temporal upper part of the visual field indicates a lesion of the medial and caudal fibres of the optic nerve (Lachenmayr and Vivell, 1992). In the optic canal these fibres run very close to the sphenoid sinus cavity and, thus, seem especially exposed to damage by mechanical or inflammatory sphenoid processes, particularly if there are bony dehiscences (Renn and Rhoton, 1975), or if the wall of the canal is very thin. In our case the aetiology of a possible optic nerve alteration is unclear, but a plausible hypothesis may be a pressure-induced nerve lesion caused by insufficient ventilation of the sphenoid sinus due to the observed inflammatory polypous mucosal changes of the adjacent ethmoidal cells, resulting in alternating changes between supra- and sub-atmospheric pressure within the sphenoid sinus. However, during surgery no endoscopy was

performed to evaluate the patency of the sphenoid sinus ostium. A reversible lesion of the optic nerve due to pressure caused by disorders in sinus ventilation has also been suggested as the cause of a transient, recurrent ipsilateral visual loss in connection with a pneumosinus dilatans of the sphenoid sinus (Bachor et al., 1994). Here, too, the symptoms disappeared after wide surgical opening of the sinus. A pressure-induced retrobulbar lesion of the optic nerve outside its canal is not likely in the case of our patient, since both orbit CT scans and MRI were unsuspicious. A further explanation may lie in circumscribed circulatory disorders in the region of the optic nerve, induced by inflammatory changes of the ethmoid cells, as assumed by Ayaki et al. (1994) in a patient with fluctuating visual disorders and simultaneous ethmoidal sinusitis. Visual acuity as well as visual field showed distinct diurnal fluctuations. These symptoms disappeared after paranasal sinus surgery.

As this case report shows, sinus involvement should be taken into account as a possible aetiological factor in the development of unclear visual disorders accompanied by atypical perimetric findings. Early operative exploration, respectively curative surgery, of the paranasal sinus system ought to be considered, even if CT scans indicate only moderate or circumscribed inflammatory sinus affection. Since the incidence of clinically-occult chronic sinusitis is rather high, a long-term post-operative control of the clinical course seems necessary to exclude multiple sclerosis manifestation, particularly in cases in which clinical, respectively radiological, examination does not reveal orbital infiltration.

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