The Journal of Laryngology and Otology
September 1993, Vol. **107**, pp. 840–842

Acute visual loss in association with sinusitis

YOUSRY EL-SAYED, F.R.C.S., HAMAD AL-MUHAIMEID, M.D. (Riyadh, Saudi Arabia)

Abstract

Acute visual loss may occur in association with sinusitis either as a complication of orbital cellulitis or, less frequently, as a part of the orbital apex syndrome. We describe two cases of temporary monocular visual loss caused by sinusitis. In one case the visual loss was due to orbital cellulitis; while in the other patient it was as due to incompletely developed orbital apex syndrome. This later mode of presentation is called 'partial orbital apex syndrome' by some authors and 'posterior orbital cellulitis' by others.

The relationship between sinusitis and blindness is discussed.

Key words: Vision disorders; Sinusitis; Orbital diseases

Introduction

Acute visual loss in association with sinusitis is a well recognized complication. Clinically, it may manifest itself in one of two forms:

(1) As a complication of orbital cellulitis and its subgroups (subperiosteal and orbital abscess). In this condition the visual loss occurs concomitantly or following the orbital soft tissue involvement: characterized by proptosis, eyelid oedema. chemosis and ophthalmoplegia (Chandler *et* al., 1970).

(2) As a component of orbital apex syndrome: which arises from involvement of those vessels and nerves passing through the superior orbital fissure and the optic foramen (Holt and de Routh. 1940: Kjoer, 1945; Abramovich and Smelt, 1982).

In the fully developed syndrome. the visual loss is accompanied by internal and external ophthalmoplegia (due to paresis of the IIIrd, IVth and VIth cranial nerves) and by changes in sensation (due to involvement of the three branches of ophthalmic nerve). These changes range from anaesthesia to neuralgia.

However, in some patients, visual loss may occur solely with minimal or no accompanying inflammatory orbital signs (Sanborn *et al.*, 1984; Slavin and Glaser, 1987: Tarazi and Shikani, 1991). This is attributed to involvement of the intraçanalicular or orbital apical segment of the optic nerve and. therefore. may be considered as partial orbital apex syndrome (Kronschnabel, 1974). However, this same pattern of presentation has been renamed 'posterior orbital cellulitis' by Slavin and Glaser in *1987.* They defined it as the clinical syndrome in which early severe visual loss overshadows or precedes accompanying inflammatory orbital signs.

In this report we describe two cases of acute visual loss caused by sinusiris: rhe first was due to orbital cellulitis and the second fulfilled the criteria of 'posterior orbital cellulitis'.

Case reports

Case *1*

A six-year-old girl presented with five days history of left mucopurulent nasal discharge. One day prior to admission, she developed a left periorbital swelling and her vision had decreased over the preceding twelve hours. On examination she looked ill and had a temperature of 39°C. There was swelling (reddish) of both eyelids partially closing the eyes. The eyeball was pushed laterally and anteriorly with moderate limitation of its movement. Vision was limited to hand movement. Fundoscopy showed congestion of the retinal vessels. Nasal examination revealed red mucosa with mucopurulent discharge in the left side. Plain sinus X-ray showed cloudiness of the left maxillary. ethmoid and sphenoid sinuses. A CT scan confirmed these findings and showed opacification between the left medial orbital wall and a displaced globe (Figure 1). Nasal and conjunctival swab grew *Streptococcus pneumoniae*. In view of the visual loss, it was felt that surgical exploration under antibiotic cover would be needed; therefore intravenous cefuroxime was started. However, 12 hours later there was a dramatic response with marked

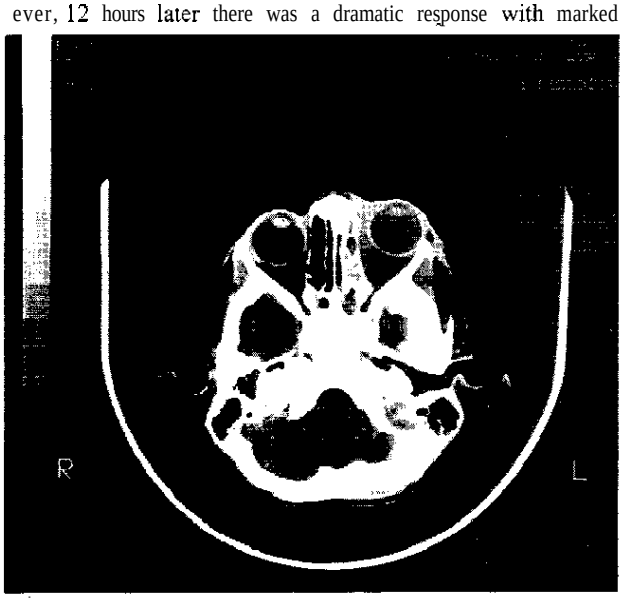

FIG. 1

An axial CT of patient 1 showing cloudiness of left ethmoid and sphenoid sinuses; with soft tissue opacity in the medial orbital wall pushing the eye globe anteriorly and laterally.

Accepted for pubhcation: 14 April 1993.

regression of the lid oedema and of the proptosis. Surgical intervention did not proceed but medical treatment continued. A rapid recovery occurred with resolution of the symptoms and a return of vision to normal.

Case 2

A 10-year-old girl was seen by the ophthalmologist because of sudden onset of visual loss in the left eye. The girl had been well until three days previously, when she had developed fever accompanied by left retro-orbital pain. Two **days** later, she had an episode of vomiting, diarrhoea and abdominal pain. She was seen by a primary care physician who gave her treatment for gastroenteritis. The following day she woke up with no vision in the left eye.

Physical examination revealed a temperature of 39°C with stable vital signs. Ophthalmic evaluation shoaed a normal right eye but no light perception in the left eye. The eye movements were normal and there was a very mild left proptosis. Fundoscopy showed normal fundi with no papilloedema or optic atrophy. The right visual field was normal, but the left shoaed a huge absolute scotoma involving both central and peripheral fields. Neurological examination was normal.

A CT scan (Figure 2) showed opacification of both sphenoids and the posterior ethmoids prompting referral for rhinologist consultation. In spite of a clear and clean nose. a diagnosis of acute sphenoethmoiditis was made and intravenous amoxycillin and metronidazole were started Immediately. Six hours later the sphenoids and the posterior ethmoids were explored and drained *via* trans-septal sphenoidostomy and bilateral intranasal ethmoidectomy. Both sphenoids were full of pus and rhe sphenoid septum was rudimentary allowing free connection between the two sides. The posterior ethmoids were lined by thick mucosa with no exudate. Pus was cultured to reveal *Hemophilus influenzae*: histological examination of the mucosa showed acute inflammatory reaction with no evidence of fungus or neoplasm.

Post-operatively, the girl showed remarkable improvement with complete recovery of vision occurring by the fifth day. The patient was followed-up for 18 months and remained asymptomatic.

Discussion

Acute visual loss may be associated with acute sinusitis either secondary to complicated orbital cellulitis or as a part of the orbital apex syndrome. Orbital cellulitis and its subgroups (periorbital cellulitis, subperiosteal and orbital abscess) are well known complications of sinusitis especially in children. Fearon

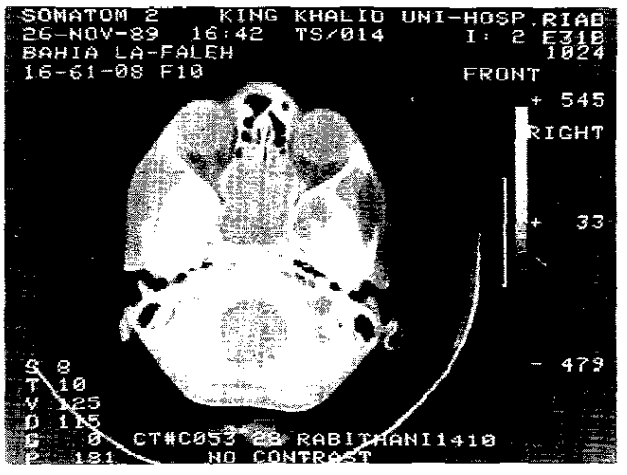

FIG. 2

A CT scan of patient 2 shoeing complete opacity of the sphenoids with cloudiness of both posterior ethmoids. The intersphenoid septum is not demonstrated. Mild left proptosis is noted.

et al, 1979) studied 6770 children with a diagnosis of sinusitis and found 159 (2.35 per cent) with orbital-facial complications. Orbital cellulitis may cause temporary or permanent visual loss characteristically occurring in association or as a sequela of orbital soft tissue involvement: fever, lid oedema, chemosis, proptosis and ophthalmoplegia (Core *1*). It is reported that approximately 10 per cent with orbital complications of sinusitis will have a temporary loss of vision which will resolve within two to six weeks (Schramm *et al*., 1978). On the other hand, from a total of 46 cases reported in the literature with a confirmed diagnosis of orbital and subperiosteal abscess in which visual results were recorded, permanent monocular blindness developed in seven cases (15 per cent, (Slavin and Glaser, 1987).

Reports of sinusitis causing orbital apex syndrome are relatively rare (Kjoer, 1945; Jarrett and Gutman, 1969; Abramovich and Smelt. 1982) The term orbital apex syndrome is classically applied when all the structures at the optic canal and superior orbital fissure are affected by the disease process (Holt and de Roth. 1940). Kroschnabel (1974), however, believes that the term should also include various subdivisions where only some of these structures are involved. In this context, Slavin and Glaser (1987) described three cases of sphenoethmoiditis causing irreversible visual loss associated with minimal signs of orbital inflammation and renamed the entity 'posterior orbital cellulitis'. They defined it as a clinical syndrome in which early severe visual loss overshadows or precedes accompanying inflammatory orbital signs. Tarazi and Shikani (1991) described a similar case. Case 2 reported here fulfils rhe criteria of 'posterior orbital cellulitis' described by the authors mentioned above However, in our case the vision recovered completely following early recognition and treatment.

Meanwhile. under the term 'optic neuritis' many authors reported cases of isolated visual loss and attributed them to sinusitis (Rothstein *et* al., 1984; Sanborn *et* al., 1984). Although some of these cases may be considered as partial orbital apex syndrome with the disease process affecting only the nerve, an etiological relationship is rarely demonstrated in most cases (Sanborn *et al*., 1984).

Since there is a faucity of lymph&s in the orbit (Chandler *et* al., 1970); and the general circulation is an unlikely route for local spread: infection can pass from the sinuses to the orbit either *via* veins or by direct spread. The *thrombophlebitis* route is considered to be the most common route due to the direct connection of their valveless veins (Chandler *et* al., 1970).

Infection may, also, spread from the paranasal sinuses to the orbit directly through their separating bony walls. This is most likely to occur anteriorly through the thin lamina papyracea; but also may occur posteriorly directly to the optic nerve from the sphenoid and the posterior ethmoid due to their intimate anatomical relationship. Bansberg *et al*. (1987) in a study of 80 normal high resolution CT scans. found 88 per cent of the sphenoid sinuses and 48 per cent of the posterior ethmoid cells were intimately related to the ipsilateral optic canal. Moreover, fen per cent of rhe sphenoid sinuses were in contact with both canals due to rudimentary development "fthe sphenoid septum. The width of the optic canal wall that separated the optic nerve from the sinuses cavity was 0.9-I mm. Direct spread can also occur through the neurovascular foramina or via acquired and congenital dehiscence.

Weille and Vang (1953) described dehiscence of the sphenoid sinuses near the optic nerve in two out of 50 specimens and near the optic chiasma in another four. They, to", found two out of 171 ethmoid labyrinths dehiscent near the optic nerve.

The dreadful complication of visual loss should be avoided if sinusitis is diagnosed and treated early and appropriately. The physician should have a high index of suspicion and be aware that sphenoethmoiditis patients, in particular, are at a high risk of developing visual loss. The unreliability of plain X-rays and the importance of CT scan for diagnosis is emphasized by many authors (Abramovich and Smelt, 1982: Rothstein et al., 1984; Sanborn *et* al., 1984). Meanwhile, early drainage of the subper-

iosteal abscess rather than observation of the response to antibiotics therapy over several days is stressed by Harris (1983) to prevent development of blindness.

The occurrence of visual loss in association with sinusitis is a rhinological emergency. It entails prompt use of intravenous antibiotics in combination with early surgical decompression and drainage of the sinuses.

Early treatment reduced the threat of the visual loss occurring in the fellow eye and may result in recovery of vision as it occurred in our case. Similar rewarding outcomes have been reported by other authors (Jarrett and Gutman, 1969; Fearon *et al.*, 1979; Rothstein *et al.*, 1984; Sanborn *et al.*, 1984).

In conclusion, paranasal sinus infection may extend to the orbit causing visual loss which may be the presenting symptom. It should be diagnosed and treated early. If suspected a high index of suspicion should be kept up and diagnosis established by CT scan. Early aggressive treatment is imperative to avert irreversible blindness.

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Address for correspondence:
Dr Yousry El-Sayed,
Department of ORL,
King Abdul Aziz University Hospital,
PO Box 245, Riyadh 11411,
Saudi Arabia.
Fax: 477-5748.