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Cavernous sinus thrombosis following manipulation of fractured nasal bones

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Abstract
Septic cavernous sinus thrombosis is a serious infective condition with a high mortality and morbidity. We report the first case to our knowledge of septic cavernous sinus thrombosis following manipulation of fractured nasal bone under general anaesthesia. The patient later developed complications of pneumonia with pleural effusion, a vegetative lesion of the mitral valve, and blindness.

Key words: Cavernous sinus; Thrombosis; Nose deformities, acquired, surgery

Introduction

Septic cavernous sinus thrombosis is a rare condition which manifests with headache, severe toxaemia, fever, periorbital swelling, proptosis, chemosis, ptosis and ocular muscle palsies. The incidence of cavernous sinus thrombosis has greatly decreased since antibiotics began to be used but the mortality and morbidity are still considerable. Southwick *et al.* (1986) reported a number of other changes in cavernous sinus thrombosis since the development of antibiotics. Most patients with cavernous sinus thrombosis in the pre-antibiotic era had some form of infection of the medial third of the face, and in the early antibiotic era this figure dropped down to about 60 per cent of cases followed by otitis media as the second most common infection. At present, sphenoidal and ethmoidal sinusitis are the most frequent primary sources of infection (Sofferman, 1983; Southwick *et al.*, 1986). Dental infection, usually of the maxillary teeth, accounts for about 10 per cent of patients with septic cavernous sinus thrombosis (Oliver *et al.*, 1948) and are more often seen after dental extraction (Chow *et al.*, 1978; Palmersheim and Hamilton, 1982; Ogundiya *et al.*, 1989). A history of predisposing underlying illness was uncommon in the pre-antibiotic era, but now 28 per cent of septic cavernous sinus thromboses are associated with chronic illnesses like diabetes mellitus and chronic sinusitis (Southwick *et al.*, 1986).

Trauma and nasal surgery rarely lead to the development of septic cavernous sinus thrombosis. There are reports of septic cavernous sinus thrombosis following nose blowing (Orrell *et al.*, 1991) and head trauma (Coll *et al.*, 1994). Nasal surgical procedures mentioned in the literature to be complicated by cavernous sinus thrombosis have included rhinoplasty (Casaubon *et al.*, 1977; Marshal and Slattery, 1983), septoplasty (Haddad *et al.*, 1985) and nasal polypectomy (Nielsen *et al.*, 1992).

We report a case of a 28-year-old insulin-dependent diabetic man who developed septic cavernous sinus thrombosis after manipulation of fractured nasal bones. He subsequently developed septic embolic manifestations including pneumonia, pleural effusion, a vegetation on the mitral valve and blindness in one eye.

Case report

A 28-year-old insulin-dependent diabetic male, without any previous history of headache or sinusitis, had a manipulation of a fractured nose which he sustained as a result of an assault. Ten days later, he presented to the Accident and Emergency department with a history of severe frontal headache, pain on the left side of the nose, diplopia, swelling and redness of the left eye lids, right-sided chest pain and breathlessness all of which had developed in the previous 24 hours.

On examination he looked unwell and sweaty with a temperature of 39.8°C. There was a periorbital swelling, ptosis, chemosis and slight restriction of ocular movement in all directions in the left eye (Figure 1). The vision was normal but he had a diplopia on lateral gaze to the left.

Systemic examination revealed only a sinus tachycardia and tenderness on percussion of the anterolateral part of the right chest. The laboratory findings showed a leucocytosis (22 600/cu.mm) with normal urea and electrolyes, and blood gases. The random blood sugar level was 8.9 mmol/l. Plain radiograph of the chest showed no sign of cardiopulmonary disease. X-ray of the paranasal sinuses showed hazy maxillary antra.

A provisional diagnosis of cavernous sinus thrombosis was made. He was admitted and commenced on high doses

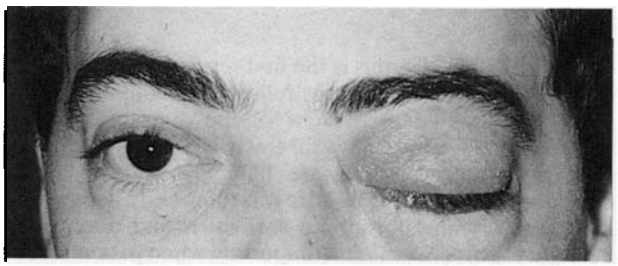

FIG. 1
The swelling of the left eye lids with chemosis during the early stages of presentation.

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of intravenous metronidazole, flucloxacillin and cefotaxime.

On the second day of admission he had swinging pyrexia, agitation and hypoxia (oxygen saturation of 78 per cent). The chest was clear and the heart sounds were normal. The vision in the left eye deteriorated to perception of light only, with normal vision on the right. There were restriction of eye movements in all directions in both eyes. A CT scan was arranged which showed extensive sinus disease bilaterally. No orbital abscess or any evidence of cavernous sinus thrombosis was noted. Bilateral antral washout at this stage revealed pus in both antra and the pus was sent for culture and sensitivity.

On the third day of admission bronchial breath sounds were heard over the left mid- and lower zone of the chest. The temperature continued to fluctuate and on the fourth day, a systolic murmur at the apex was noted. Echocardiogram showed a small vegetation on the anterior mitral valve leaflet. Fucidin 500 mg eight-hourly and erythromycin 1 g six-hourly was now added to his antibiotic regimen. The sinus washout showed a growth of *Staphylococcus aureus* and group B *Streptococcus* sensitive to flucloxacillin and erythromycin. Repeated blood culture during this period did not grow any microorganisms.

On the sixth day, the right eye developed abductor palsy with swelling of the eyelids and chemosis. The visual acuity remained normal on the right but the left eye became completely blind with complete ocular muscle palsy. The corneal reflex was also impaired in the left eye. The antibiotic regimen was altered to flucloxacillin 3 g four-hourly, rifampicin 600 mg 12-hourly, cefotaxime 1 g eight-hourly and metronidazole 500 mg eight-hourly.

Repeat CT scan failed to show any evidence of cavernous sinus thrombosis but this remained the clinical diagnosis. Serial chest X-rays performed over the third, fourth and fifth day showed a gradual development of hazy opacities in both side of the chest consistent with bilateral basal effusion. The patient's temperature settled on the new antibiotic regimen but when three days later the flucloxacillin was reduced from 3 g four-hourly to 3 g six-hourly the temperature rose again. The flucloxacillin had to be continued four-hourly. After six weeks of intravenous antibiotics the patient was well and allowed home. However, during this period he had developed a persistent right pleural effusion which had become loculated and difficult to aspirate. He underwent right thoracotomy and total pleurectomy three months later. A repeat echocardiogram before his discharge showed that the anterior mitral valve leaflet had become thinned in the area which previously had a small vegetation. During the whole period of his stay in the hospital the blood sugar level was monitored closely and a good control was maintained with insulin.

Discussion

To our knowledge this is the first reported case of septic cavernous sinus thrombosis following manipulation of fractured nasal bone. In view of the poor outcome of established cavernous sinus thrombosis it is important the primary infections which are liable to spread to the cavernous sinus should be recognized as early as possible and treated with appropriate antibiotics and drainage if necessary. This is even more important when patients have an underlying chronic systemic condition like diabetes mellitus.

The diagnosis of septic cavernous sinus thrombosis is primarily clinical but radiology may help to confirm the diagnosis. It is important to note in our case that the eye signs were unilateral at presentation with bilateral involvement at a later stage (see Figure 1). Orbital venography has been considered as the definitive radiological investigation (Brismar, 1975; Fiandaca *et al.*, 1986). However with the increasing availability of magnetic resonance imaging this will become the investigation of first choice (Dyken *et al.*, 1990; Savino *et al.*, 1986). CT scan is not very sensitive in demonstrating cavernous sinus thrombosis.

Staphylococcus aureus is isolated in two-thirds of cases of septic cavernous sinus thrombosis, with pneumococci, streptococci, gram-negative bacteria and anaerobes accounting for most of the remaining cases (DiNubiles, 1988). Fungal infections are rare (Dyken *et al.*, 1990). The pathogen depends on the primary source of infection (Miller, 1991), and the presence of any other underlying condition. John *et al.* (1990) found that diabetes mellitus was the most common predisposing factor in 101 cases of staphylococcal bacteraemia. Blood cultures are positive in most cases of septic cavernous sinus thromboses (Miller, 1991), but systemic septic embolization are uncommon. Southwick *et al.* (1986) reported the findings at autopsy and surgery of 23 cases of septic cavernous sinus thrombosis and found septic infarcts of other organs in four cases. Organs reported to be affected by septic emboli have included the lungs, kidneys, liver, spleen, brain and orbits (Bassey and Elebute, 1968; Clune, 1963; Karlin and Robinson, 1984; Wolf, 1944).

The thrombus from the cavernous sinus can extend to other dural venous sinuses and cortical veins resulting in cerebral oedema and infarcts. Direct spread of infection from the cavernous sinus can cause meningitis, brain abscess and subdural empyema. Visual loss can result from ischaemic oculopathy caused by occlusion of either internal carotid, ophthalmic or central retinal arteries, or due to ischaemic optic neuropathy. The IIIrd, IVth, VIth and the ophthalmic and maxillary division of the Vth cranial nerves can be damaged as they pass through the infected cavernous sinus.

The mortality in septic cavernous sinus thrombosis has improved with the introduction of antibiotics from between 88 per cent and 100 per cent in the pre-antibiotic era to 30 per cent. At present full recovery is expected in fewer than 40 per cent of cases, and most of those who recover have residual complications which include blindness, cranial nerve palsies, hemiparesis, facial numbness, paraesthesia, or pain, pituitary insufficiency and other embolic manifestations.

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