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Lateral sinus pathology
(22 cases managed at Groote Schuur Hospital)

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Abstract

One hundred and thirty patients with acute and acute on chronic mastoiditis were managed at Groote Schuur Hospital between 1980 and 1984. Twenty-two (16.9 per cent) had pathology of the lateral (sigmoid) sinus and 19 of these patients had cholesteatomas. Nine patients (40.9 per cent) had concomitant intradural sepsis and there were two deaths.

The modern literature is reviewed and the pathology, presentation and management of lateral sinus pathology is discussed. This condition is rare and clinical features may range from subtle signs to gross toxaemia, torticollis and evidence of septic embolization. The otolaryngologist must be competent in diagnosing and treating this condition in all forms of its wide spectrum of presentation.

Introduction

Pathology of the lateral sinus secondary to mastoid sepsis is a well recognized complication with a significant mortality. The use of antibiotics has not only resulted in a dramatic decline in the incidence of this disease but in many cases changed the clinical presentation making the diagnosis very difficult. It has been claimed, however, that the mortality has not changed since the pre-antibiotic era (Teichgraeber *et al.*, 1982)—probably due to the high association with other more sinister intracranial sepsis.

A comprehensive description is given by Shambaugh and Glassock (1980) in Surgery of the Ear, in which they detail the progression of the pathology, the clinical presentation and discuss the change in management since the pre-antibiotic era.

It is important for modern otolaryngologists to appreciate the difficulties our predecessors experienced and how current management philosophy evolved. The substantial experience at Groote Schuur Hospital and the rarity of lateral sinus pathology in many parts of the world prompted this report.

Case material

In the 5 years between 1980 and 1984, 130 patients had surgery for acute and acute-on-chronic mastoiditis at Groote Schuur Hospital. Twenty-two patients (16.9 per cent) had pathology of the lateral sinus. Nineteen of these patients (86.4 per cent) had cholesteatomas and Table I illustrates that 25.7 per cent of patients with cholesteatomas operated on in the acute phase had lateral sinus pathology.

The sinus was thrombosed in 12 patients, contained frank pus in 9 patients and was fibrosed and obliterated in one patient. There was a male predominance of 15 to 7 females and the ages ranged from 2 to 40 years (Fig. 1). In this series there was a peak incidence in the 11 to 15 year age group, all of whom had cholesteatomas. The 3 patients without cholesteatomas were all under 10 years old.

There was perisinus granulation tissue and perisinus pus in 4 and 14 patients respectively. Lateral sinus pathology was present in 4 patients without documented perisinus disease.

All patients presented with pyrexia and otalgia and 11 had neck stiffness. Of these, 9 had definite unilateral neck tenderness. Seven patients had neurological signs: 2 had seventh nerve palsies, 2 had hemipareses, one had ataxia, one patient had a generalized fit and two had depressed levels of consciousness. One patient had a temporary sixth nerve palsy which may have been due to apical petrositis (Gradenigo's syndrome) or raised intracranial pressure. Positive evidence of embolic phenomena was evident on Chest X-ray in 4 patients and definite hydrocephalus was demonstrated by computerized tomography in another 4 patients.

Lumbar punctures are not done as a routine but were performed in 6 patients, of whom 3 had active meningitis; one had a neighbourhood syndrome with 11 lymphocytes and no polymorphonuclearcytes and two patients had normal cerebrospinal fluid. If lumbar puncture is done, a Queckenstedt or Toby-Ayer test is often helpful but possible false results and the dangers of sudden increase in the intracranial pressure must be appreciated.

The high association of lateral sinus thrombosis with other intracranial sepsis is well documented and in this series; apart from those with perisinus disease, there

TABLE I

	Number of patients	Lateral sinus pathology	%
Non-cholesteatoma patients	56	3	5.4
Cholesteatoma patients	74	19	25.7
Total	130	22	16.9

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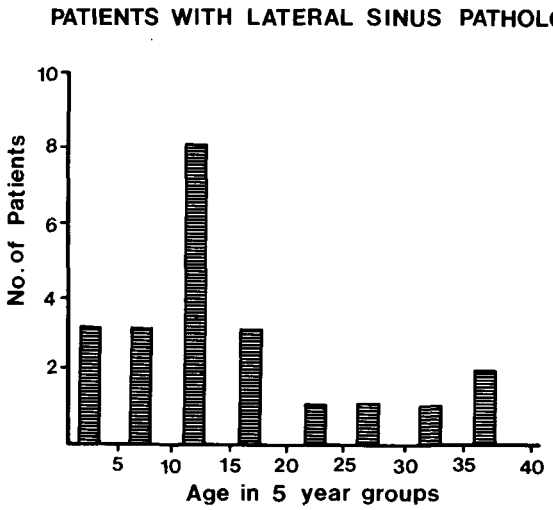


FIG. 1

were 3 patients with middle cranial fossa extradural pus, 1 with a supratentorial subdural empyaema, 4 with temporal lobe abscesses and 4 with cerebellar abscesses—i.e. 9 patients (40.9 per cent) had associated intradural sepsis.

The internal jugular vein was ligated in 3 patients and incised and drained in another 2 patients.

Positive cultures were obtained in 17 patients, 12 of whom had Proteus mirabilis, 5 had Bacteroides, 3 had β-haemolytic Streptococci and 3 had Staphylococcus aureus. (7 patients had mixed growths). Intravenous ampicillin and metronidazole have proved the most effective and are used until the culture and sensitivity of the organisms may indicate a change.

There were two deaths—a mortality of 9.1 per cent. A 3-year-old girl and a 15-year-old boy, both with cholesteatomas, had been treated at peripheral hospitals for meningitis before deterioration prompted referral to Groote Schuur Hospital. The girl had an associated temporal lobe abscess and an internal jugular vein empyema and the boy had a cerebellar abscess. Prompt surgery was performed on both patients but they succumbed to overwhelming septicaemia.

Discussion

Lateral sinus pathology is rare, often resulting in delayed recognition and treatment. A recent survey of otolaryngology clinics of North America (Goldenburg, 1985) in which 88 responded, showed only 34 per cent as having encountered the condition in the preceeding 5 years.

The widespread use of antibiotics has not only dramatically lowered the incidence of this disease, but in many cases has changed the clinical presentation to a subacute form, often with vague symptoms and signs. The clinical picture of an ill patient with a high spiking temperature, anaemia, headache, otalgia, neck stiffness and tenderness under the sternocleidomastoid muscle is rarely seen and most cases present with an intermittent low grade temperature, vague headaches and possibly mild discomfort on moving the head.

The spectrum of the pathology includes perisinus granulations and pus, mural thrombus formation, occlusive thrombophlebitis, intrasinus empyaema and finally organization of the thrombus with recanalization or obliteration. The thrombophlebitic process may progress to the superior sagittal sinus, *via* the superior petrosal sinus, to the cavernous sinus and inferiorly to the internal jugular vein. Showering of septic emboli may produce multiple pyaemic abscesses, peritonitis, or suppurative arthritis. Extension to the mastoid emissary vein may cause a localized painful mass, called Griesinger's sign.

Aseptic and septic forms of lateral sinus pathology occur with different clinical presentations and requiring different treatment. Aseptic thrombosis is often found incidentally or may present insidiously with signs and symptoms of raised intracranial pressure. Headaches become more severe and may be associated with visual disturbances, papilloedema and unilateral or bilateral sixth nerve palsies. The exact mechanism of raised intracranial pressure is still unclear (O'Connor and Moffat, 1978) and more sinister pathologies such as brain abscesses and intracranial tumours must be excluded. It is in this respect that computerized tomographic scanning is most helpful (Schønsted-Madsen *et al.*, 1984). Symonds (1931) first used the term 'otitic hydrocephalus' to describe the well-known clinical picture of raised intracranial pressure complicating otitis media and with a normal cerebrospinal fluid. However, the ventricular system is normal and there is no evidence of hydrocephalus. This condition is a variant of benign intracranial hypertension or pseudotumour cerebri and led Horowitz in 1949 to introduce the more accurate term of 'otogenic intracranial hypertension'.

The management of aseptic lateral sinus thrombosis is controversial, with some authors (Pennybacker, 1961; Goldenberg, 1985) proposing a 'wait and see' approach for those with minimal symptoms. If therapy is required this is essentially medical with surgery reserved for rapidly failing vision, deterioration on medical treatment and the presence of mastoid pathology. The sinus is initially needled and if a thrombus is demonstrated, the sinus is incised but obtaining free flow of blood is not necessary. Once otogenic intracranial hypertension is established and vision becomes jeopardized, various manoeuvres aimed at lowering the pressure can be utilized and include repeated lumbar puncture, steroids, acetazolamide, glycerol dehydration and finally subtemporal decompression or orbital optic nerve decompression (O'Connor and Moffat, 1978).

Septic lateral sinus thrombophlebitis is a more florid condition requiring urgent mastoid surgery and antibiotics. The symptoms and signs are related to the infection, the raised intracranial pressure and possible effects of septic embolization. In these cases, the sinus should be opened widely, as much of the contents evacuated as possible and a drain inserted. It may be possible to 'milk' the pus out by massage over the internal jugular vein. An empyema of the internal jugular vein should be treated by early incision and drainage but thrombophlebitic extension need only be treated by ligation with uncontrolled pathology; for example, the presence of septic emboli after definitive mastoid and lateral sinus pathology and high dose intravenous antibiotics. This procedure was indicated in 3 of the patients, with dramatic results (2 others required incision and drainage of the internal jugular vein).

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The exact place of ligation of the internal jugular vein has been controversial. Some authors claimed worse results (Meltzer, 1935) and others cited the danger of air embolism and damage to local structures (Jensen, 1962). In the pre-antibiotic era, some surgeons ligated the vein routinely, above the level of the common facial vein to prevent propagation along this vessel. An excellent description of this historical controversy is given by Teichgraeber *et al.* (1982) but reasonable consensus has now been reached. There is no place for ligation as a routine but its value in some selected cases should not be underestimated.

Anticoagulant therapy to prevent further propagation of the thrombus in patients with septic embolization is advocated by Shambaugh and Glasscock (1980) but its use is highly controversial. Heparin was used in one patient with septic pulmonary embolism from lateral sinus thrombosis reported by Hawkins (1985). He states that anticoagulants may be beneficial with extending thrombus, especially with impending cavernous sinus thrombosis. In the Groote Schuur Hospital series, anticoagulants were not used at all.

The mortality of lateral sinus thrombosis is quoted as being between 18 and 36 per cent (Jackson and Dickens, 1979; Teichgraeber *et al.* 1982). Meltzer (1935) reported 45 deaths in 161 patients (27.9 per cent) and the statement is often made that, although antibiotics have decreased the incidence of this disease, the mortality remains similar to the pre-antibiotic era. It is important to appreciate that lateral sinus pathology has a high association with intracranial sepsis and it is really in these patients that the high mortality applies.

Jensen (1962) reported 3 deaths in 12 patients (25 per cent) occurring in Denmark in 5 years; two had cerebellar abscesses and one had a temporal lobe abscess. In Seid and Sellars' review of 13 cases in 1973, there were 3 deaths (23.07 per cent); one had a temporal lobe abscess, another had cavernous sinus thrombosis and the third an overwhelming septicaemia.

In this series, there were 2 deaths in 22 patients (9.09 per cent) and both of these patients presented late and had intracerebral abscesses.

With early diagnosis and prompt aggressive management, the prognosis for patients with lateral sinus pathology, even with internal jugular vein thrombophlebitis and septic embolization, should be much better than previously accepted.

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