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Lateral sinus thrombosis
(A review of 45 cases)

by

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Introduction

There has been a decline in the rate of occurrence of otogenic intracranial complications since the discovery of antibiotics. The problem of lateral sinus thrombosis, however, continues to be formidable, with a substantial incidence of mortality, amounting to 36 per cent in one of the series (Teichgraeber *et al.*, 1982).

The present article will review our experience with 45 patients suffering from lateral sinus thrombosis in the Black population, where otitis media is neglected and patients are reluctant to seek medical treatment.

This paper is a reminder of the problem which is prevalent in many parts of the world in the lower socio-economic groups.

Clinical material

Lateral sinus thrombosis occurred in 45 patients admitted to King Edward VIII Hospital during the period 1978–1984; 25 were male and 20 were female. The age of the patients ranged from 1 year to 40 years; 37 (82 per cent) were under the age of 15 years (Table I).

All patients presented with a retroauricular swelling and a history of discharging ears which varied from some days to years; 15 (30 per cent) of them had had the initial episode within 3 weeks prior to admission.

The presenting symptoms of lateral sinus thrombosis in our patients were postauricular oedema and otorrhoea, headache for 1–2 weeks prior to admission, spiking temperature, nausea and vomiting.

Fourteen patients presented with neck stiffness. All patients had a CT scan. A lumbar puncture was performed in 9 patients who had a normal CT scan and showed bacterial meningitis. In the remaining 5 patients CT scan revealed a posterior fossa abscess (Table II). All patients with posterior fossa abscess were operated simultaneously by a neurosurgeon and an ENT surgeon.

Four patients presented with facial palsy.

Chest X-ray showed pneumonic changes in 2 cases and pulmonary tuberculosis in 1 case. In the latter case, the diagnosis of disseminated T.B. was confirmed by histological examination of the mastoid granulations.

At mastoidectomy granulations were found in 27 (60 per cent) cases out of 45, and cholesteatoma in 18 (40 per cent) cases out of 42.

Perisinus abscess was recorded in 4 cases. The sinus was needled in all cases and, if bleeding did not occur, then the sinus were incised and the clot removed.

The facial nerve was found exposed in 7 patients but only 4 of them presented clinically with facial palsy.

Ligation of the internal jugular vein was performed in 5 cases in whom the jugular vein was indurated and chest X-ray showed pulmonary changes: these patients presented with the features of septicaemia, i.e., spiking of temperature to 40°C, deterioration of general condition and patchy changes on chest x-ray,

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TABLE I
LATERAL SINUS THROMBOSIS: AGE DISTRIBUTION

Age	No. of Patients
1–5 yrs	6
6–10 yrs	20
11–15 yrs	11
16–20 yrs	5
21–30 yrs	1
31–40 yrs	2
Total:	45

suggesting septic emboli. Torticollis with tenderness of the ipsilateral sternomastoid area was present.

Four deaths were recorded in our series. All of them had other intracranial complications. Medical treatment consisted of Ampicillin and Metronidozole intravenously.

Discussion

Lateral sinus thrombosis and abscess formation may result either from direct spread of the infection from the mastoid or from thrombophlebitis of small veins of the mastoid which communicate with the lateral sinus. The presenting symptoms of lateral sinus thrombosis in our patients were earache and otorrhoea, headache for 1–2 weeks prior to admission, spiking fever, nausea, vomiting, cervical tenderness or lymphadenopathy and occasionally signs of septicaemia. Papilloedema was not present.

CT scan was of great assistance in diagnosis and in excluding intracranial complications. The Tober-Ayer test was not considered necessary and is only of historical importance in our opinion since the advent of the CT scan.

The diagnosis was confirmed by surgical exploration of the sinus plate. (The sinus plate is examined in all patients with mastoid sepsis and suspected intracranial complications and therefore prior diagnosis with Tober-Ayer test is not necessary).

The surgical protocol consists of needling the sinus on completion of the mastoidectomy and, if passage of blood does not occur, then the sinus is incised and the thrombus removed as much as possible. We do not insist on free bleeding. Exploration of the sinus beyond the limits of the mastoid is not performed.

In the management of lateral sinus thrombosis, Proctor (1966) recorded that ligation of the internal jugular vein was performed routinely in the pre-antibiotic era. He also found lateral sinus thrombosis to be the most common intracranial complication before 1943, where 48 cases were found, against the 3 cases reported between 1953–1962.

Pennybacker *et al.* (1961) reviewed 97 cases of lateral sinus thrombosis and found 42 cases with multiple cranial complications in the cerebrum, cerebellum, meninges or subdural space, and in three cases the thrombus extended into the superior petrosal sinus or cavernous sinus.

Four deaths (10 per cent) were recorded from lateral sinus thrombosis. The mortality rate from lateral sinus thrombosis in some modern series ranges from 18–36 per cent (Teichgraeber *et al.*, 1982).

Our mortality rate was only 10 per cent as opposed to the above-quoted figure. Teichgraeber *et al.* (1982) found the lateral sinus thrombosis more commonly associated with chronic ear disease but Gower and McQuirt (1983) reported 83 per cent lateral sinus thrombosis with acute ear disease. Our experience confirms that of Teichgraeber and not that of Gower and McQuirt.

We do not use anticoagulants because we fear: (a) that, with a septic thrombophlebitis, septic emboli may be released by clot breakdown; (b) that uncontrollable haemorrhage may result from clot breakdown in the mastoid. Mawson and Ludman (1979) suggested anticoagulant therapy if the clinical condition shows a spreading thrombosis, i.e., jugular vein thrombosis or signs of cavernous sinus thrombosis, but this view is not shared by

TABLE II
LATERAL SINUS THROMBOSIS: ASSOCIATED COMPLICATIONS

Meningitis	9
Posterior fossa abscess	5
Facial palsy	4
T.B. mastoiditis	1

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other authors who no longer recommended anticoagulant therapy.

Ligation of the internal jugular vein was performed in five cases (11 per cent) in which the internal jugular vein had an indurated and cord-like appearance under anaesthesia. Pulmonary complication with continuous spiking fever was a mandatory indication for exploration of the neck.

The controversy about the indications for ligation of the jugular vein still exists in the literature. There are those who advocate ligation on all occasions (Warren, 1929; Sutherland, 1938), and at the other extreme those who deny that ligation prevents the spread of septic emboli (Meltzer, 1935).

Kimmick and Myers (1958) believe that the jugular vein should be ligated in the course of the surgery if thrombosis is discovered at operation.

A number of authors feel that the ligation should be done in certain circumstances; when the clot extends beyond the mastoid area (Proctor, 1966) or when the sepsis persists (Seid & Sellars, 1973). We agree with this protocol.

No internal jugular vein ligation was performed after the introduction of routine use of Metronidazole in addition to Ampicillin.

We feel that this is a very important finding. In a previous series from our Department, anaerobes were present in all cases of intracranial complications (Maharaj, 1986). The use of Metronidazole may, in fact, make extension of lateral sinus thrombosis needing internal jugular vein ligation a thing of the past.

Warren (1929) found cholesteatoma producing the majority of cases of lateral sinus thrombosis. We, however, found that infective granulations (60 per cent) were more common than cholesteatoma.

Summary

Forty-five cases of lateral sinus thrombosis were recorded in King Edward VIII Hospital from 1978 to 1984. Eighty-two per cent of the patients were under the age of 15 years.

Associated complications recorded were: 9 patients with meningitis, 4 with posterior-fossa abscess, 1 patient suffering from posterior fossa empyema; 4 presented with facial palsy and 1 with tuberculous mastoiditis. Four deaths were recorded.

Findings at mastoidectomy showed a prevalence of infective granulations (60 per cent) over cholesteatoma (40 per cent) as a cause of the extension of infection.

The surgical protocol for the management of lateral sinus thrombosis was mastoidectomy, needling the sinus and removal of the thrombus, if necessary. Medical treatment consisted of Ampicillin and Metronidozol.

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