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The management of lateral sinus thrombosis

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Abstract

The standard recommended treatment for lateral sinus thrombosis is intravenous antibiotics. For those that fail, some authors recommend anticoagulant therapy, others internal jugular vein ligation. Despite these recommendations, there is still a significant mortality in all reported large series and the main reason this is that the cause of the failure has not been found or investigated.

Over a six-year-period, from January 1985 to December 1990, 36 patients were admitted with lateral sinus thrombosis. The initial treatment consisted of intravenous antibiotics and surgery. The surgery included cortical mastoidectomy for non-cholesteatomatous ears and modified radical mastoidectomy for cholesteatomatous ears and drainage of the intracranial collection of pus in patients with subdural empyema and brain abscess.

There were nine failures (25 per cent) with this treatment regimen. Blood cultures and pus swab of the ears were taken from these patients. The blood cultures were positive in four patients and the organisms isolated included β-haemolytic *Streptococcus sp.* in two, *Proteus mirabilis* and *Streptococcus pneumoniae* in one each. From the ear swab mixed cultures were found in eight and a single organism in one.

Gram-negative organisms were isolated in nine (100 per cent), *Proteus mirabilis* in eight (89 per cent) and *Enterobacter sp.* in two. Both these organisms were resistant to ampicillin and penicillin but were sensitive to amikacin.

Amikacin was then added to the treatment regimen and a dramatic response with a fall in the temperature, and improvement in the patients' general condition was noted within 24 hours, in all patients. There was not a single death, despite the fact that 42 per cent of the patients had other associated intracranial complications. The conclusion is that the treatment of choice for patients refractory to the conventional antibiotic therapy and surgery is amikacin therapy and not internal jugular vein ligation, or anticoagulant therapy.

Key words: Sinus thrombosis; Clinical protocols

Introduction

In the management of patients with lateral sinus thrombosis, there are two aspects to consider, the eradication of the predisposing disease and prevention of embolization.

Whilst the treatment of choice for eradicating the disease is intravenous antibiotics and mastoidectomy, that of embolization prevention remains unresolved. Two treatment modalities, anticoagulant therapy and internal jugular vein ligation have been recommended but they have not been proven to be entirely satisfactory because failures have been reported with both and it is for that reason that there are so many conflicting reports in the literature regarding both treatment modalities with Shambaugh and Glassock (1980) recommending anticoagulant therapy for all, Mawson and Ludman (1979) and Hawkins (1985) for patients with spreading thrombi only and Goycoolea and Jung (1991) not recommending it all.

A similar situation exists with internal jugular vein ligation. In the pre-antibiotic era it was recommended for all patients (Warren, 1929; Sutherland, 1938; Dawes, 1961). Later it was recommended for selected cases, Kimmick and Myers (1958), Jahrsdoerfer and Fitz-Hugh (1968), recommending it when thrombi are discovered at mastoidectomy, Proctor (1966), when the clot extends beyond the mastoid, Mawson and Lundman (1979), in the presence of embolic infarct, Shambaugh and Glassock (1980) when there is metastatic lung abscess and Seid and Sellars (1973), Teichgraeber *et al.* (1982), Samuel and Fernandes (1987), Mathews (1988) recommending it for patients in whom the septicaemia persisted despite intravenous antibiotic therapy and mastoidectomy. On the contrary Meltzer (1935) denies ligation prevents septicaemia and claims worse results with ligation.

Adding more uncertainty to the role of jugular vein ligation and anticoagulant therapy, deaths have still been reported in all large series (Seid and Sellars, 1973; Samuel and Fernandes, 1987; Mathews, 1988).

The purpose of this paper is therefore to determine the cause of the persistent septicaemia in those patients who fail to respond to the initial intravenous antibiotics and mastoidectomy, and thereafter recommend the appropriate treatment.

Materials and management

Over a six-year-period, from January 1985 to

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December 1990, 36 patients were admitted to King Edward VIII Hospital, with lateral sinus thrombosis. Their ages and sex are displayed in Figure 1. The signs and symptoms included neck pain in 22 (61 per cent), fever in 36 (100 per cent), rigors in seven (19 per cent), post-auricular abscess in 13 (36 per cent), neck abscess in three, labyrinthitis in one, hemiplegia in one, ophthalmoplegia in five, bilateral in two and unilateral in three. Other associated intracranial complications occurred in 15 (42 per cent), Table I. Chest pathology was present in three (8.3 per cent), bilateral staphylococcal type of pneumonia, bilateral bronchopneumonia and left-sided basal pneumonia in one each.

Tympanic membrane perforation was present in 29, central in 14 and posterior-superior in 15. Four patients had intact, but dull, tympanic membrane. In three patients, the tympanic membrane could not be visualized because of oedema of the external ear canal in one, and polyps in two. Only 14 (39 per cent) patients had evidence of middle ear disease in the contralateral ear, eight had central perforation and six posterior superior perforation. Otorrhoea was present in only five.

The diagnosis was made clinically in 31 (86 per cent) on the typical spiking temperature alone. The diagnosis was confirmed at surgery in all. In two patients, whilst CT scan of the brain was being performed to exclude other intracranial lesions, the base of the skull was scanned and this showed hypodense areas in the region of the internal jugular vein on the affected side. The radiologist felt that this was consistent with lateral sinus thrombosis. Unfortunately, blood culture was not performed on any patient at this stage.

The initial treatment consisted of intravenous antibiotics, ampicillin and metronidazole, in all patients except those with meningitis, brain abscess and subdural empyema. In these patients, penicillin, chloramphenicol and metronidazole were administered.

Exploratory mastoidectomy was performed in all patients, within 24 hours of admission; in the majority within 12 hours. The findings at surgery and the related ear pathology are displayed in Table II. In seven patients, the diagnosis was obvious, as the disease process had eroded the mastoid segment of the lateral sinus, leaving the inferior and superior thrombosed ends completely exposed. In the other 29 patients the diagnosis was confirmed by drilling the sinus plate and then puncturing the lateral sinus with an 18-gauge needle and aspirating with a 10 cc syringe. Once the diagnosis was confirmed, nothing further was done to the clot. Cortical mastoidectomy was performed in non-cholesteatomatous ears and modified radical mastoidectomy in cholesteatomatous ears. Incision and drainage of neck abscesses was performed in three patients.

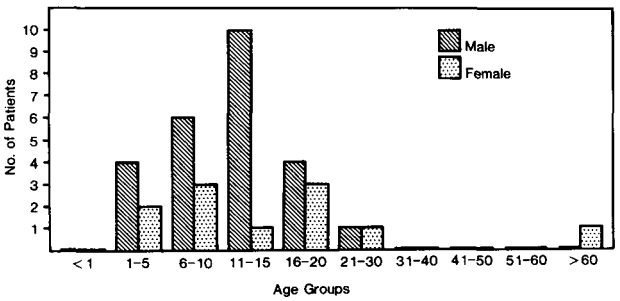

FIG. 1
Age and sex distribution of patients.

The intracranial collection of pus in patients with brain abscess and subdural empyema was drained through burr holes by the neurosurgical service.

The results of the initial treatment with conventional antibiotics and surgery are displayed in Table III.

In the nine failures, *i.e.* patients in whom the septicaemia persisted, despite intravenous antibiotic therapy and surgery, chest radiography, blood cultures and pus swabs of the ears were taken in all. The blood cultures were positive in four, β-haemolytic *Streptococci* were recovered in two, *Proteus mirabilis* and *Streptococcus pneumoniae* in one each. The results of the ear swabs are shown in Table IV.

The results of treating the resistant patients with amikacin are displayed in Table V. In none of the patients was the internal jugular vein ligated nor anticoagulant therapy administered. Two patients are described in greater detail below.

Case reports

Patient 1

A 17-year-old male patient was referred from a peripheral hospital, with a two-week history of neck stiffness, fever, headache and puffy eyes. On examination, he was fully conscious and cooperative with a temperature of 39°C; he had marked neck stiffness and tenderness in the frontal region. He had chemosis of the right eye, with limitation of movement, reactive pupil and intact vision. There was axial proptosis of the left eye, with marked chemosis, total ophthalmoplegia and a fixed and dilated pupil. The vision could not be assessed accurately but it seemed to be diminished.

Lumbar puncture performed at the peripheral hospital revealed turbid cerebrospinal fluid with 14 polymorphs, two lymphocytes and 18 red blood cells per high power field. The glucose was 3.3 mmol/l, protein 1.37 g/l, and the globulin was increased slightly (+). The full blood count revealed a white cell count of 23 400/ml and a haemoglobin of 10.8 g/dl.

CT scan of the brain revealed no intracranial pathology, whilst CT scan of the paranasal sinuses showed mucosal thickening in the ethmoid sinus, on the right (Figure 2). The provisional diagnosis at this stage was left orbital cellulitis secondary to ethmoiditis. The patient was put on intravenous ampicillin and metronidazole and then underwent a left external frontoethmoidectomy with bilateral antral washout the same evening. No pus was found in the ethmoid sinus nor the maxillary antra.

TABLE I
ASSOCIATED INTRACRANIAL COMPLICATIONS

Intracranial lesion	No. of patients	Percentage
Meningitis	1	3
Brain abscess	3	8
TLA 2		
PFA 1		
Subdural empyema	1	3
Extradural empyema	5	14
Cavernous sinus thrombosis	5	14
Total	15	42

TLA = temporal lobe abscess; PFA = posterior fossa abscess.

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TABLE II
FINDINGS AT SURGERY

Sinus pathology	Cholesteamatous ears (%)	Non-cholesteamatous ears (%)	Total
Lateral sinus thrombosis	9 (50)	9 (50)	18
Lateral sinus thrombosis and perisinus abscess	3 (27)	8 (73)	11
Lateral sinus erosion	7 (100)	–	7
Total	19 (53)	17 (47)	36

The next day his condition had remained unchanged and the temperature was still swinging. On examination he had marked tenderness of the mastoid tip and profuse otorrhoea on the left. After aural toilet a posterior-superior perforation with cholesteatoma was noted. The right ear drum was intact and normal. At mastoidectomy pus was found to be under pressure because as the lateral wall was drilled the pus gushed out from the mastoid cavity. Drilling of the sinus plate revealed perisinus abscess and lateral sinus thrombosis; this was confirmed by puncturing the sinus with an 18-gauge needle and aspirating with a 10 cc syringe. Modified radical mastoidectomy was performed with complete excision of the cholesteatoma from the attic and antrum. Twenty-four hours after the surgery, the temperature returned to normal. The proptosis and chemosis settled after two weeks. The eye movement and vision began returning after four weeks. At a three-month follow-up, he was completely well and without any sequelae.

The definitive diagnosis in this patient was left otitis media with lateral and cavernous sinus thrombosis.

Patient 2

A 12-year-old male patient was referred from a peripheral hospital with persistent pyrexia and neck stiffness and essentially normal cerebrospinal fluid (clear colourless fluid with six polymorphs, no lymphocytes or red blood cells: glucose 3.4 mmol/l, protein 0.11 g/l, and no organisms).

On examination he was fully conscious and cooperative. He was pyrexial with a temperature of 39°C. There was marked neck stiffness with tenderness, particularly along the anterior border of the left sternocleidomastoid muscle. There was marked percussion tenderness of the mastoid tip, on the left. There was a posterior-superior perforation with a retraction pocket on the left, the otorrhoea was minimal. The right ear drum was mildly inflamed but intact.

CT scan of the brain showed no intracranial pathology whilst CT scan of the base of the skull revealed two hypodense areas in the region of the internal jugular vein on the left (Figure 3). The radiologist felt this was consistent with lateral sinus thrombosis. Full blood count revealed a white cell count of 7200/ml and a haemoglobin of 10.1 g/dl.

Intravenous ampicillin and metronidazole were commenced and the mastoid cavity explored. Pus was oozing from the lateral sinus into the mastoid cavity through a small defect in the sinus plate. Drilling of the sinus plate revealed perisinus abscess and lateral sinus thrombosis. This was confirmed by puncturing the sinus with an 18-gauge needle and aspirating with a 10 cc syringe. Pus was also oozing through the dural plate into the mastoid cavity. On drilling of this plate, about 5 cc of pus was evacuated. The cholesteatoma from the attic and mastoid antrum was completely excised by a modified radical mastoidectomy. The next morning he had rigors lasting for one to two minutes, coinciding with a temperature spike and suggesting septicaemia. The chest radiograph was normal, on the blood culture β-haemolytic *Streptoccus* was isolated and was sensitive to penicillin. The swab of the ear revealed β-haemolytic *Streptococcus* sensitive to ampicillin, and *Proteus mirabilis* resistant to ampicillin, tetracycline, erythromycin, cotrimoxazole and cefril but sensitive to amikacin.

Amikacin was commenced on the fourth day and there was a dramatic response (Figure 4) not only with regard to the temperature, but also the patient's overall general condition. After five days of amikacin therapy, the patient was completely well and discharged to the peripheral hospital. At a two-week follow-up he remained well.

TABLE III
RESULTS OF INITIAL TREATMENT WITH CONVENTIONAL ANTIBIOTICS AND MASTOIDECTOMY

Antibiotics used	Success	(%)	Failure	(%)	Total
Ampi + Metro	15	(71)	6	(29)	21
Pen + Metro + Chlor	12	(80)	3	(20)	15
Total	27	(75)	9	(25)	36

Ampi = ampicillin; Metro = metronidazole; Chlor = chloramphenicol; Pen = penicillin.

Discussion

The incidence of lateral sinus thrombosis has decreased world-wide since the introduction of antibiotics but, unfortunately, in South Africa it has remained unchanged. Of the 104 reported cases in the literature from 1980, 73 (70 per cent) have been from South Africa (Samuel and Fernandes, 1987; Mathews, 1988; Yaniv and Pocock, 1988). The incidence seems to be particularly high in the province of Natal because of the 73 patients 45 (40 per cent) have been from our ENT service (Samuel and Fernandes, 1987).

Like other otogenic intracranial complications, lateral

TABLE IV
ORGANISMS CULTURED FROM PUS SWAB OF THE EARS IN RESISTANT PATIENTS

Organisms	No. of patients
Proteus mirabilis	8
Enterobacter sp.	2
β-*haemolytic Streptococcus*	2
Pseudomonas aeroginosa	2
Streptococcus pyogenes	1
Streptococcus pneumoniae	1
Streptococcus faecalis	1
Staphylococcus aureus	1

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TABLE V
RESULTS OF TREATING RESISTANT PATIENTS WITH AMIKACIN

Antibiotics used	Success	(%)	Failure
Ampi + Metro + Amikacin	6	(100)	Nil
Pen + Metro + Chlor + Amikacin	3	(100)	Nil

Ampi = ampicillin; Metro = metronidazole; Pen = penicillin; Chlor = chloramphenicol.

sinus thrombosis predominantly occurs in males (Rosenwasser, 1945; Samuel and Fernandes, 1987; Mathews, 1988) and peaks in the first and second decade of life (Rosenwasser, 1945; Seid and Sellars 1973; Samuel and Fernandes, 1987; Mathews, 1988). A similar sex and age predominance was noted in the present series (Figure 1).

The patients in this series and all those reported by Samuel and Fernandes (1987) from the same ENT service between 1978 and 1984, presented with the typical spiking temperature similar to the 'picket-fence' temperature recorded in the pre-antibiotic era (Shambaugh and Glasscock, 1980). This differs from the reports by Jahrsdoerfer and Fitz-Hugh (1968), Teichgraeber *et al.* (1982) and Mathews (1988) in which they mentioned that because of the widespread use of antibiotics, the typical spiking temperature does not occur and instead patients present with intermittent low-grade fever, thus making the diagnosis of lateral sinus thrombosis difficult. This is contrary to our findings in that the patients reported in the present series and those by Samuel and Fernandes (1987) presented with the typical spiking temperature, despite the fact that the majority of them were on intravenous antibiotics at the peripheral hospitals, prior to admission.

Twenty-two patients (64 per cent) presented with neck pain. The neck pain in lateral sinus thrombosis can be easily mistaken for neck stiffness due to meningitis on first impression but closer examination will reveal that the pain and tenderness is along the anterior border of sternocleidomastoid muscle and there is also percussion tenderness of the mastoid tip. Kernig's and Brudzinski reflexes are absent, except in patients with associated brain abscess, subdural empyema and meningitis in which case there is true neck stiffness. A similar observation was made by Mathews (1988); of the 11 patients presenting with neck stiffness, it was unilateral in nine.

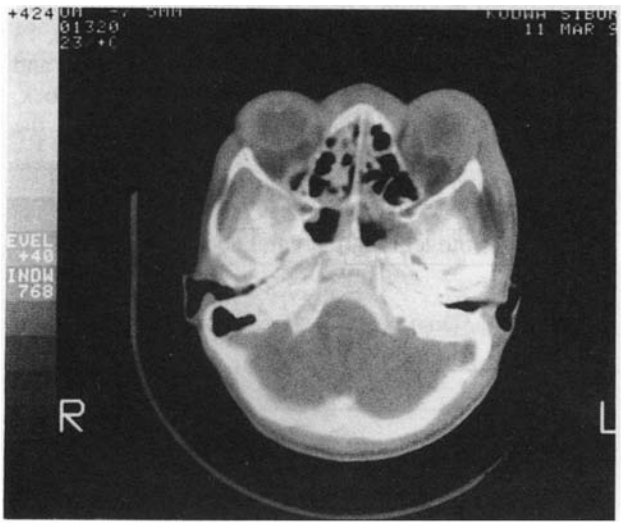

FIG. 2
CT scan of the paranasal sinus, showing mucosal thickening of the ethmoid air cells on the right.

True embolic phenomena were observed in the pre-antibiotic era, when there was embolization to the joints (hip, shoulder, elbow, sternoclavicular), foot, lung and spinal column (Rosenwasser, 1945). Since the introduction of antibiotics embolization to the other regions of the body, except the lung, have been rare. There has only been one reported case of true embolization in the post-antibiotic era and this was by Wilfowitz (1972) who reported gangrene of the big toe in a patient with lateral sinus thrombosis.

Even pulmonary embolization is rare as there have only been a few reported cases: Samuel and Fernandes (1987) reported two (4.4 per cent), Mathews (1988) four (18 per cent) and Yaniv and Pocock two (33.3 per cent).

In the present series, there were three patients (8.3 per cent) with pneumonic changes, one had typical features of staphylococcal pneumonia in both lung fields; the other bilateral bronchopneumonia and the third a left basal pneumonia. These are not typical features of pulmonary embolization as Rosenwasser (1945) states that pulmonary manifestations of embolization often simulate ordinary pneumonic infection.

Lateral sinus thrombosis is closely associated with other intracranial complications. In the present series there were 15 patients (42 per cent) with other intracranial lesions (Table I). A similar high incidence was noted by Dawes (1961) 43 per cent, Teichgraeber *et al.* (1982) 50 per cent, Samuel and Fernandes (1987) 31 per cent and Mathews (1988) 41 per cent. With such high association it is imperative to perform a CT scan of the brain on all patients suspected of having lateral sinus thrombosis to exclude other associated intracranial lesions. The diagnosis can usually be made on clinical grounds, supplemented by CT scanning. More invasive tests, such as digital subtraction angiography, retrograde jugulography and carotid arteriography for diagnostic purposes, as mentioned by Teichgraeber *et al.* (1982), are unnecessary.

The diagnosis of lateral sinus thrombosis was made clinically in 31 patients (86 per cent) on the basis of the typical spiking temperature alone. In two patients the diagnosis was missed on the initial examination despite

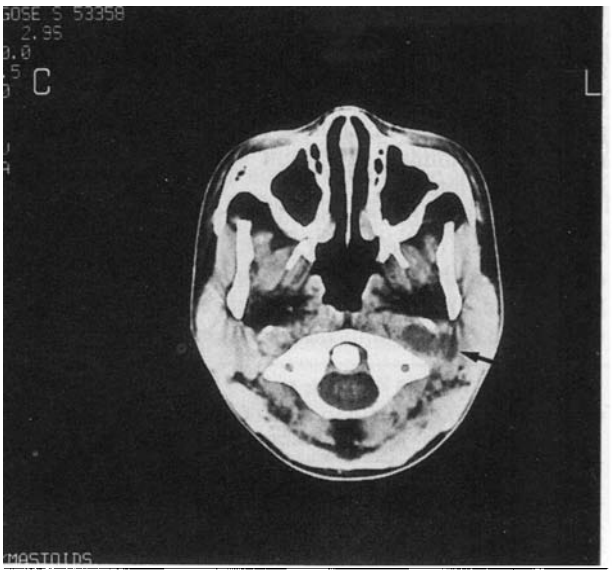

FIG. 3
CT scan of the neck displaying two hypodense areas in the region of the left internal jugular vein.

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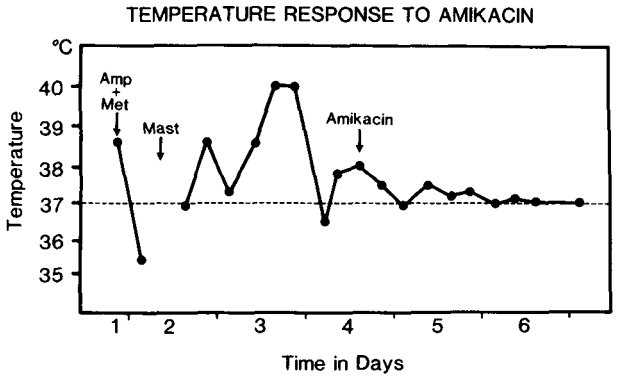

FIG. 4

Temperature chart displaying the typical swinging temperature of patients with lateral sinus thrombosis and the dramatic response to amikacin therapy.

their presenting with the typical swinging temperature. This was because the patients had signs and symptoms referrable to cavernous sinus thrombosis (chemosis, proptosis, ophthalmoplegia with diminished vision). In these patients, an erroneous diagnosis of acute ethmoiditis with orbital cellulitis was made because the CT scan showed mucosal thickening of the ethmoid sinus. The diagnosis of cavernous sinus thrombosis, secondary to lateral sinus thrombosis, was not entertained for two reasons, firstly the patients did not have a post-auricular abscess to alert the examiner that the otitis media was the cause of the patient's signs and symptoms and secondly the commonest cause of orbital cellulitis and cavernous sinus thrombosis in our region is acute ethmoiditis.

The reason why the septicaemia persists in a certain group of patients has remained unanswered. According to the present study, the causative organism is resistant to the antibiotics (ampicillin and penicillin) normally used to treat these patients. This is illustrated by the fact that from all patients, in whom the septicaemia persisted despite antibiotic therapy and mastoidectomy, gram-negative organisms resistant to ampicillin and penicillin were cultured from the ear swabs and as soon as amikacin was added to the treatment regimen, there was a dramatic response not only in the temperature pattern, but also the patients' general condition as clearly demonstrated in the case report of Patient 2. It is obvious from this that the cause of the septicaemia is a gram-negative organism but, unfortunately, it was proven in only one patient on blood culture. This is not surprising because often cultures are negative in septicaemic patients (Rosenwasser, 1945; Seid and Sellars, 1973), and the reason may be that they are not taken at the actual time of the bactercaemia.

Lateral sinus thrombosis is not without mortality as deaths have been reported in all large series from the pre-antibiotic era to the present day. The mortality in the pre-antibiotic era was 100 per cent, but after the introduction of surgery it decreased to 50 per cent (Jensen, 1962). By 1920 the surgical techniques had improved and the mortality decreased further to between 25 and 35 per cent (Boies, 1932; Lyman, 1935; Behrman, 1937). Proctor (1966) in the pre-antibiotic period between 1934 and 1937 reported 16 cases with six deaths (38 per cent); in the period between 1938 and 1947 reported 32 cases with seven deaths (22 per cent); whilst in the post-antibiotic period between 1953 and 1962 reported three cases with one death (33.3 per cent). Thereafter the mortality in all large series has ranged between 18 to 36 per cent (Jahrsdoerfer and Fitz-Hugh, 1968; Wolfowitz, 1972; Seid and Sellars, 1973). Recently a further decrease in mortality has been noted by Teichgraeber *et al.* (1982) reporting a 16.6 per cent mortality, Samuel and Fernandes (1987) 10 per cent and Mathews (1988) 4.1 per cent. In the present series, there were no deaths despite it being a very large series with 36 patients and 42 per cent having other associated intracranial complications. This has largely been due to the use of amikacin in patients resistant to the conventional antibiotics.

The advantageous effect of amikacin over internal jugular vein ligation is demonstrated when the mortality from the present series is compared to a previously reported series from the same neurological service by Samuel and Fernandes (1987), when this type of patient was managed with internal jugular vein ligation, and the mortality was 10 per cent (four deaths).

Conclusion

In the management of lateral sinus thrombosis, the treatment of choice for resistant patients *i.e.* patients in whom the septicaemia persists, despite conventional antibiotic therapy and mastoidectomy, is amikacin therapy and not internal jugular vein ligation or anticoagulant therapy.

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