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Review Article

Management of lateral sinus thrombosis: update and literature review

Eng Hooi Ooi, Malcolm Hilton, Garrett Hunter

Abstract

The management of four cases of lateral sinus thrombosis (LST) over a four-year period at the Royal Darwin Hospital is presented in this retrospective review. The patients were aboriginal and presented with otalgia, otorrhoea and sepsis. Two cases had an associated complication of an otitic hydrocephalus and a subperiosteal abscess. Cholesteatoma was found in three cases. Computed tomography (CT) scan confirmed the LST in three cases. Three patients were anaemic and thrombocytopenic. All patients had positive blood cultures. The organisms were predominantly mixed anaerobes and *Bacteroides* species. Three patients were managed surgically as a two-stage procedure. One patient was managed as a single-stage procedure with a modified radical mastoidectomy. Therapeutic anticoagulation was utilized in one case. There were no deaths. The prognosis of LST is good if treatment is instituted early with broad-spectrum intravenous antibiotics and surgery. The role of clot removal at surgery and the use of anticoagulation are discussed in this paper.

Key words: Lateral Sinus Thrombosis, Cholesteatoma; Surgical Procedures, Operative; Treatment Outcome

Introduction

Intracranial complications of suppurative ear disease are rarely seen today.[1–3] Lateral sinus thrombosis (LST) is a well-known otogenic complication with serious consequences if left untreated. A combination of surgical intervention and antibiotics has reduced the reported mortality but it can still be as high as 27 per cent.[4] There is still controversy regarding surgical treatment of the sinus and the role of anticoagulation.

Large series of LST have been reported from South Africa[5,6] and Iran,[7] where access to health care is limited, but LST is rarely seen in Western developed countries.[8–12] The dramatic drop in the incidence of lateral sinus thrombosis can be attributable to the introduction of antibiotics, earlier diagnosis and prompt effective treatment.[13]

The Royal Darwin Hospital is the major tertiary referral centre for the population of the Northern Territory in Australia. A recent case of LST seen at our institution, the fourth case in a four-year period, prompted a review of the literature concerning the modern management of LST. Medline was used to search the English literature from 1966 to the present on LST. The aim of this article is to present our experience with the diagnosis and treatment of LST secondary to otogenic disease. We also aim to highlight the bacteriology and clinical pattern of this disease. An awareness of this particular complication by today's otolaryngologist is essential if a satisfactory outcome is to be ensured.

Materials and methods

The medical records of all cases with a discharge diagnosis of lateral sinus thrombosis at the Royal Darwin Hospital from 1999 to 2002 were reviewed retrospectively. Only cases with surgically or radiologically proven lateral sinus thrombosis were included. The data collected from the case notes included the patient's demographic details, presenting symptoms and signs, bacteriology results, relevant investigations, treatment, complications, duration of the pre-admission illness and outcomes.

Case reports

Case 1

The patient is a 14-year-old aboriginal male who was previously treated at a peripheral hospital with penicillin and Sofradex® eardrops for three days. He was transferred to the Royal Darwin Hospital because of worsening sepsis following a five-day

From the Department of Otolaryngology, Royal Darwin Hospital, Northern Territory, Australia.
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history of right otalgia, otorrhoea, and fevers. On examination he had a tender mastoid, and purulent ear canal with a temperature of 39° Celsius. Haemoglobin was 86 g/L and the platelet count was 31×10^9/L. CT scan of his mastoid with contrast demonstrated opacification of his right mastoid and middle-ear space, and absence of normal enhancement with gas tracking over the right sigmoid sinus. CT scan of the brain was normal. A diagnosis of acute mastoiditis with lateral sinus thrombosis was made. Treatment was started with intravenous ceftriaxone, metronidazole, gentamicin, and benzyl penicillin, and correction of the haematological parameters. Blood and ear cultures both grew mixed anaerobes and *Enterococcus avium*. A right cortical mastoidectomy was performed 24 hours later where granulation tissue was found in the attic. The mastoid cavity was filled with pus and granulation tissue extending into the attic. All the infected tissue was removed down to the mastoid tip and the lateral sinus. The lateral sinus plate was skeletonized with the drill but the sinus was not incised nor needled. The patient had a stormy post-operative course requiring a re-exploration of his mastoid seven days later because of ongoing sepsis. The mastoid cavity contained thrombus that was not infected. He developed pneumonia and an adult respiratory distress syndrome in intensive care but eventually made a good recovery after antibiotic treatment. He was discharged home 26 days later.

Case 2

The patient is 13-year-old aboriginal female who presented with a five-day history of right otalgia, headache, painful neck and fevers. She was initially admitted at a peripheral hospital where treatment was started with amoxycillin. She was transferred three days later to the Royal Darwin Hospital under the care of the paediatricians with a presumed diagnosis of meningitis. The significant examination findings were a tender mastoid and torticollis with a temperature of 38.8° Celsius. Haemoglobin was 101 g/L and the platelet count was 16×10^9/L. Lumbar puncture was normal. There was no clinical improvement after 48 hours of treatment with intravenous ceftriaxone. A CT scan of her neck was performed because of a concern about a neck abscess. It demonstrated opacification of her right mastoid and middle-ear space with thrombosis of the right internal jugular vein and sigmoid sinus. An otolaryngological opinion was then sought and a diagnosis of lateral sinus thrombosis was made. Treatment was changed to intravenous gentamicin, metronidazole, and timentin and a right cortical mastoidectomy and thrombectomy was performed six hours later. The significant findings were a cholesteatoma sac, a sclerotic mastoid with infected granulation tissue, and a thrombosed lateral sinus with exposed posterior fossa dura. Blood and mastoid cultures grew mixed anaerobes. A combined approach tympanoplasty and removal of all cholesteatoma was performed 10 days later after the patient had recovered from the septic episode. Her post-operative progress was complicated by pneumonia that responded to antibiotic treatment. She made a good recovery and was discharged home 22 days later. She was initially lost to follow up so her second-look tympanoplasty occurred two years later. There was no recurrent cholesteatoma found at that procedure.

Case 3

The patient is a 23-year-old aboriginal female who was initially admitted under the care of the neurologists with the symptoms of left otalgia, headache, dizziness, diplopia and vomiting. Examination revealed a tender mastoid, temperature of 39.5° Celsius, and a left VIth cranial nerve palsy. Fundoscopy revealed bilateral papilloedema, haemorrhages at the disc margin and dilated retinal veins. Haemoglobin was 79 g/L and blood cultures grew *Staphylococcus aureus*. CT and magnetic resonance imaging (MRI) demonstrated bilateral sclerotic mastoid cavities, normal venous flow and normal brain. Lumbar puncture showed a high cerebrospinal fluid pressure and no organisms. She was treated with intravenous timentin for a total of seven days. The symptoms resolved and she was discharged home with a diagnosis of benign intracranial hypertension. She had two further treatments of lumbar puncture at a peripheral hospital. The patient was admitted 25 days later with severe headache, left otalgia and otorrhoea and treated with intravenous timentin. An otolaryngological opinion was sought three days later where an attic cholesteatoma was seen. The patient underwent a left modified radical mastoidectomy where a large cholesteatoma sac filling the attic, antrum and mastoid cavity to the mastoid tip was found and removed. The sigmoid sinus was covered with granulation tissue. It was needled and no blood was aspirated. She made a good recovery and was discharged home three days later and remains disease free at follow up two years later.

Case 4

A 21-year-old aboriginal female had presented to a peripheral hospital with an 11-day history of right otalgia and fevers. She was treated with oral amoxycillin for an acute otitis media. However the symptoms persisted and she became progressively unwell with a headache and right otorrhoea. She was then transferred to our institution where examination revealed a stiff and tender neck, tender mastoid with a right tympanic membrane perforation and a temperature of 38.5° Celsius. The platelet count was 26×10^9g/L and lumbar puncture was normal. CT scan demonstrated opacification of her right mastoid cavity and middle-ear space, a subperiosteal abscess over the petrous temporal bone, and a right lateral sinus thrombosis. Treatment was started with meropenem, gentamicin, and benzyl penicillin. Right cortical mastoidectomy was performed three hours later. The antrum was filled with granulation tissue and pus. This was all removed. A thrombosed lateral sinus was incised and the clot evacuated. Intravenous

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TABLE I
DEMOGRAPHICS AND CLINICAL FINDINGS

	Case 1	*Case 2*	*Case 3*	*Case 4*
Age	14	13	24	21
Sex	M	F	F	F
Duration of pre-admission symptoms	5 days	5 days	38 days	8 days
Otalgia	+	+	+	+
Otorrhoea	+	+	+	+
Headache	–	+	+	+
Fevers	+	+	–	+
Painful neck	–	+	–	+
Tender mastoid	+	+	–	+
VIth palsy	–	–	+	–

heparin anticoagulation was instituted 24 hours post-operatively because of concerns of raised intracranial pressure from the thrombosis. Blood cultures grew *Prevotella intermedia* and the ear cultures grew mixed anaerobes. Headache and increased drowsiness four days later prompted a repeat CT brain scan that was normal. She continued to make a steady recovery with resolution of her symptoms and was discharged home 20 days later. A modified radical mastoidectomy six weeks later found and removed cholesteatoma disease from the right attic and antrum.

Results

Demographics (Table I)

There were four cases of lateral sinus thrombosis during the study period. There was one male and three females and all the patients were aboriginal. The ages ranged from 13 to 23 years. Two of the patients had had antibiotic treatment elsewhere prior to admission. The duration of symptoms preceding admission ranged from five days to 38 days.

Otological findings (Table I)

All the patients had otalgia and otorrhoea. The other common presenting symptoms and signs were headache, fevers, a tender mastoid and a painful neck. Otoscopic findings were abnormal in all the cases. There were three cases of cholesteatoma and the other case was due to acute middle-ear disease. The right side was involved in three cases and one in the left side. Two of the patients had abnormal neurological signs. One had a depressed level of consciousness and another had temporary VIth cranial nerve palsy.

Radiological and laboratory investigations

All patients underwent CT of the temporal bones and brain with intravenous contrast and had a demonstrable radiographic abnormality of the mastoid. CT scan demonstrated the lateral sinus thrombosis in three of the cases. Additionally two of the patients underwent MRI, which confirmed the CT findings. A post-operative CT scan was performed in one patient (*Case 3*) because of a suspected intracranial complication. Three out of the four patients were anaemic (*Cases 1, 2* and *3*), and thrombocytopenic (*Cases 1, 2,* and *4*). There was a leucocytosis in all the patients.

Bacteriology (Table II)

The blood cultures were positive in all the cases. Ear cultures were positive in 75 per cent of the cases. Lumbar puncture was performed in three cases with no growth. The most common organisms cultured were mixed anaerobes and *Bacteroides* species. Other organisms cultured included *Staphylococcus aureus* (25 per cent), *Prevotella intermedia* (25 per cent), and *Enterococcus* species (25 per cent). *Enterocuccus* sp was resistant to ampicillin and the other organisms were sensitive to all tested antibiotics.

Treatment (Table III)

Medical treatment. The patients were all initially treated with intravenous antibiotics upon hospital admission and abnormal haematological and biochemical parameters were appropriately corrected. Broad-spectrum antibiotics were used in all the patients. The most commonly used was gentamicin (three patients), followed by timentin (two patients), metronidazole (two patients), and benzyl penicillin

TABLE II
BACTERIOLOGY AND ANTIBIOTIC TREATMENT

	Case 1	*Case 2*	*Case 3*	*Case 4*
Mixed anaerobes	BC + EC	BC + EC	–	EC
Prevotella intermedia	–	–	–	BC
Enterococcus sp	BC + EC	–	–	–
Staphylococcus aureus	–	–	BC	–
Antibiotic treatment	Ceftriaxone, Gentamicin, Metronidazole, Benzylpenicillin	Timentin, Gentamicin, Metronidazole	Timentin	Meropenem, Gentamicin, Benzylpenicillin

BC = Blood cultures; EC = ear cultures

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TABLE III
SURGICAL FINDINGS AND TREATMENT

	Case 1	*Case 2*	*Case 3*	*Case 4*
Cholesteatoma	–	+	+	+
Side of disease	R	R	L	R
Initial ear surgery	CM	CM	MRM	CM
Definitive ear surgery	–	CAT	–	MRM
Other surgery	Wound explored	Tympanoplasty (2 years later)	–	–
Managing the sinus surgically	Sinus left intact	Clot evacuated	Sinus needled	Clot evacuated
Therapeutic anticoagulation	–	–	–	+

CM = Cortical mastoidectomy; CAT = combined approach tympanoplasty; MRM = modified radical mastoidectomy

(two patients). The other antibiotics used were meropenem, vancomycin, ceftriaxone, and flucloxacillin. The antibiotics were adjusted accordingly once the culture results were known.

Surgical treatment. There was a total of eight procedures performed. Three patients had a two-stage procedure that involved a cortical mastoidectomy initially followed by definitive surgery after they had recovered from their initial sepsis. These were a combined approach tympanoplasty (*Case 2*) and modified radical mastoidectomy (*Case 4*). One patient was managed as a single-stage procedure with a modified radical mastoidectomy. Re-operation was required in one case for exploration of mastoid wound.

The surgical management of the lateral sinus involved several different approaches: The clot was evacuated in *Cases 2* and *4*, needled in *Case 3*, and exposed but not incised or needled in *Case 1*. Therapeutic anticoagulation with intravenous heparin followed by oral warfarin was employed in *Case 4*. The internal jugular vein was not ligated in any of the cases.

Complications and outcomes

There was one intracranial complication of otitic hydrocephalus and one extracranial complication of a subperiosteal abscess. There were no meningitis or cerebral abscesses in our series. Post-operative complications included two cases of pneumonia and one case of adult respiratory distress syndrome. There were no deaths in this series. The total number of days spent in hospital averaged 20 days and ranged from 13 days to 26 days. Three patients required intensive care post-operatively.

Discussion

The reported mortality from lateral sinus thrombosis has fallen dramatically due to various reasons (Table IV). This downward trend is shown in the results from South Africa[6] and Iran.[7] The mortality of LST was 100 per cent prior to operative treatment. Successful surgery and then the introduction of sulphonamides and penicillins further reduced the mortality of LST to below 30 per cent.[14] The improvement in mortality can also be attributed to a combination of earlier diagnosis and treatment.[13,15,16] There were no deaths in our series of patients with LST. It is felt that a combination of broad-spectrum antibiotics and early surgery is important in achieving this outcome. The improvement in post-operative care was a contributing factor with three of our patients receiving intensive care. The association of LST with other intracranial complications is well recognized.[6,17] The mortality is highest in patients with brain abscesses, meningitis or multiple intracranial complications but low in

TABLE IV
REPORTED SERIES OF LST

Authors	Year	Cases	Study period (years)	Acute cases	Chronic cases	Mortality (%)
Meltzer[41]	1935	161	10	116	55	39
Pennybacker[16]	1961	97	16	29	68	18
Proctor[14]	1966	51	16	19	32	27
Jahrsdoefer & Fitz-Hugh[4]	1968	11	22	1	10	27
Seid and Sellars[5]	1973	13	4	4	9	23
Teichgraber *et al.*[13]	1982	6	10	1	5	16
Gower *et al.*[50]	1983	5	19	4	1	0
Samuel and Fernandes[6]	1987	45	6	27	18	10
Mathews[17]	1988	22	4	3	19	9.1
Yaniv and Pocock[19]	1988	6	3	1	5	0
Amirmajdi[7]	1988	16	7	1	15	0
O'Connell[9]	1990	3	–	2	1	0
Singh[26]	1993	36	6	17	19	0
Syms *et al.*[20]	1999	6	5	0	5	0
Kaplan *et al.*[42]	1999	13	15	6	7	8
Bradley *et al.*[45]	2002	9	6	7	2	0

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those with only a single complication.[16] This might explain the low mortality.

The use of antibiotics has been associated with a reduction in the development of LST.[14] The pattern of intracranial complications from middle-ear disease has changed with the near-universal application of antibiotics. Complications are now more likely to arise from chronic ear disease or cholesteatoma rather than acute otitis media.[7,13,14,17–20] Acute otitis media can still play a role in the development of LST.[6] Patients with cholesteatoma presenting acutely are likely to have an intracranial complication.[18] In our study there were three cases of a first presentation with cholesteatoma and an intracranial complication. Our patient population could explain this finding. A large proportion of the patient population is aboriginals living in remote areas where there is limited access to health care and often a reluctance to seek medical treatment for their ear disease until a serious intracranial complication occurs.

The most frequent presenting symptoms were headache, otalgia, fevers, nausea, and vomiting.[13,21] Abnormal otoscopic findings, otorrhoea, posterior auricular swelling and tenderness, a painful neck, papilloedema and abducens nerve palsy are common findings.[21] Our findings concur with these results. Furthermore the use of antibiotics does not appear to prevent the development of an otogenic intracranial complication.[22] The presence of a persistent fever, with an ear infection and sepsis despite the administration of appropriate antibiotics should alert the clinician to the possibility of LST.

One study found that only 22 per cent of patients with an intracranial complication from middle-ear disease eventually had an otolaryngological evaluation.[23] The clinical picture of LST may be unclear due to previous antibiotic use and the overlap of signs and symptoms with other diseases. The condition of LST was not initially recognized upon admission in two of our cases resulting in a delay in obtaining an otolaryngological opinion. Today's otolaryngologist will rarely see this complication as a questionnaire survey revealed that 66 per cent of otolaryngology clinics in North America in 1985 had not reported any cases of LST in the preceding five years.[24] Delay in presentation for treatment or misdiagnosis is associated with a higher mortality.[16] A high index of suspicion is therefore essential for the diagnosis of LST because its clinical features can be similar to otitis media or mastoiditis.

Bacteriology

The bacteriology of LST has also changed with the use of antibiotics. *Pneumococcus* and β haemolytic *Streptococcus* used to be frequently cultured in 90 per cent of blood cultures.[14] *Pseudomonas* sp and *Proteus* sp were other common findings.[5] Treatment failures in acute otitis media due to drug-resistant pathogens are also commonly encountered now.[25] Amakacin has been used with good success in patients with persistent sepsis despite conventional antibiotic therapy.[26] Cultures now characteristically yield a mixed flora reflecting the bacteriology of chronic ear disease: *Proteus* sp, *Staphylococcus aureus*, *Escherichia coli*, *Haemophilus influenzae* and anaerobes.[13,17,19,26,27] Frequent use of antibiotics has resulted in most cultures being negative.[9,21,28] Contrary to other series, all of our cases had a positive culture. Mixed anaerobes were the most common organisms cultured. The only patient with a Gram positive organism, *Staphylococcus aureus*, was a patient with cholesteatoma (*Case 3*). It was found that both blood and ear cultures were frequently helpful. Our findings support the use of broad-spectrum antibiotics with anaerobic cover, until culture results and sensitivities are known, since septic LST consists of mixed organisms.

Septic LST develops from the direct spread of infection in the mastoid air cells through neighbouring eroded bone or via the mastoid emissary veins to the lateral sinus.[21,29] A mural thrombus develops as the walls of the sinus become infected. The sinus on the right side is more frequently involved than the left side for unknown reasons. Propagation of the thrombus may occur proximally, to involve the internal jugular vein, or distally to involve the dural venous sinuses and prevent normal CSF drainage leading to venous hypertension and raised intracranial pressure.[30]

Otitic hydrocephalus

There is some uncertainty in the literature regarding the coexistence of otitic hydrocephalus (OH) and LST. The pathophysiology and association between the two conditions are poorly understood.[31] OH is seen most often with LST, however, most cases of LST do not cause OH. It presents insidiously with symptoms and signs of raised intracranial pressure (ICP).[24] Severe headaches, diplopia, papilloedema and abducens nerve palsy are prominent features. It was originally described as the clinical occurrence of intracranial hypertension with normal cerebrospinal fluid complicating acute or chronic otitis media.[32] It is thought to be propagation of a lateral sinus thrombus with blockage of the arachnoid villi and subsequent decrease in CSF resorption.[30] The management of OH is controversial. The condition can resolve without treatment presumably due to opening of anastomotic channels.[14] Repeated lumbar punctures are only useful for a short-term basis. Treatment is mainly medical and this includes antibiotic therapy, acetazolamide and glycerol dehydration.[15,32] Surgery is required if visual acuity deteriorates from optic atrophy.[32] This may involve mastoidectomy with subtemporal decompression, orbital nerve decompression or ventricular shunt.[14,20,33] LST raising the ICP by increasing venous pressure should be considered a separate disease entity from otitic hydrocephalus.[23] The clinical features of LST are related to the sepsis that requires urgent mastoid surgery and antibiotics.

Laboratory findings

Laboratory tests may be supportive in the diagnosis of LST. Anaemia and leucocytosis are common

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features but thrombocytopenia is not usually a feature. However, severe thrombocytopenia was a prominent finding in three patients. This was presumably due to a consumptive coagulopathy related to overwhelming sepsis. Lumbar puncture was performed in three of the cases because they were initially misdiagnosed as meningitis. All the CSF cultures were negative. Culture may have been negative because of previous antibiotic use but this is unlikely given that positive blood cultures were obtained in all cases.

Radiological investigations

The diagnosis of LST is usually suspected on clinical grounds but radiology is essential to confirm it. CT is particularly important for demonstrating pathology in the mastoid and cranial cavity and excluding coexisting intracranial complications.[34] CT scan with contrast can demonstrate a filling defect in the thrombosed sinus, and ring enhancement or 'delta sign' around a thrombosed sigmoid sinus.[21,34,35] CT can be non-diagnosed because of bone-related artifacts.[36] A review of paediatric cases found that the CT was positive in only 42 per cent of cases with LST.[28] Angiography and venography are said to be the most definitive methods of demonstrating LST.[21,37] They are both invasive procedures and carry a risk of stroke or dislodging the thrombus.[34] MR imaging is more definitive in confirming thrombus. It may show both an abnormal signal and lack of flow that is likely to be due to venous sinus occlusion by thrombus.[28,38] MR angiography now can be performed rapidly; free of bone-related artifacts, does not use ionizing radiation, and is non-invasive.[36] The use of MRI should be considered in cases where the CT is non-diagnostic but clinical suspicion of LST remains high.

Medical treatment

There is universal agreement that treatment of LST with a combination of antibiotics and surgery is required. The patients often benefit from antibiotic treatment, hydration, and correction of abnormal haematological parameters before surgery. Patients presenting acutely were operated on at least within 24 hours after diagnosis. Medical therapy alone with intravenous antibiotics in selected cases of LST may be successful.[28] Associated intracranial complications should prompt immediate neurosurgical consultation.

Surgical treatment

Controversy still arises regarding the extent of mastoid surgery and management of the thrombosed sinus. Cholesteatoma is a persistent source of infection and is unresponsive to antibiotics. The early removal of this source reduces the possibility of further intracranial extension, shortens the duration of illness and provides definitive treatment. A modified radical mastoidectomy has been used successfully in the treatment of cholesteatomatous ears presenting acutely with LST.[14,15,18,20,26,39] It has the advantage of providing definitive treatment for the patient while avoiding the need for a second procedure. The evidence from the literature illustrates that early surgery in these patients ensures a better prognosis.[18,19] A modified radical mastoidectomy, however, does not carry any advantage in an ear with chronic mucosal disease and an intracranial complication.[40] A cortical mastoidectomy is sufficient treatment for non-cholesteatoma ear disease.[15,18] It allows drainage of the initiating infection and confirms the diagnosis of LST. Perisinus disease can be found despite a normal appearing sinus plate.[5] It is recommended that the removal of the sinus plate overlying the sinus should always be performed. Ideally, patients who are medially stable should be managed with single-stage surgery. Three of our patients had severe coagulopathy and septicaemia and it was felt that they would not tolerate a prolonged radical mastoidectomy. A cortical mastoidectomy was performed initially to obtain surgical drainage of the infection. Patients with cholesteatoma then proceeded to have definitive surgery after treatment of their initial sepsis. One patient had a modified radical mastoidectomy and another patient had a combined approach tympanoplasty. The use of a combined approach tympanoplasty in those ears with cholesteatoma and an intracranial complication is controversial. There is a risk of residual cholesteatoma with the intact canal wall technique and it should only be considered in cases where regular follow up is ensured.[15]

The appropriate management of the thrombus in the sinus is uncertain. The wall of the sinus can appear normal but still contain thrombus so the sinus should be needled for diagnosis.[41] If free blood is aspirated then no further intervention is required. If there is no return of blood the diagnosis is confirmed. Most studies support incision of the sinus and evacuation of the clot.[6,7,13,17,42,43] It is not necessary to obtain free bleeding from the sinus. The sinus can then be packed with an antibiotic impregnated pack.[7] The prognosis does not appear to be improved by exploring the sinus or removing the thrombus.[20,21,26,30] Two patients in our series had a thrombectomy whereas the clot was untouched in the other two patients. There did not appear to be any significant difference in their overall outcome. Conservative surgical treatment of the sinus does not seem to affect overall prognosis.

After the initial drainage of the infection, a high index of suspicion must be maintained to ensure complete resolution of the sepsis. Re-operation may be required if an intracranial infection develops.[20] Prolonged treatment with antibiotics is often required for at least two weeks post-operatively. Patients with successfully treated LST are not at any more risk of developing long-term complications provided the ear disease is under control.[42]

Internal jugular vein ligation

The role of internal jugular vein (IJV) ligation is controversial. Historically the most common complication of LST was septic emboli with hip, ankle, knee

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and shoulder joint involvement.[41] In the pre-antibiotic era ligation of the IJV was commonly performed to prevent septic emboli.[14] Metastatic emboli actually occurred four times more commonly after ligation of the IJV. There was a high neck wound infection rate after IJV ligation.[41] The procedure also adds potential risk of damage to the vagus, accessory and hypoglossal nerves in the neck.[44] The routine use of this procedure is not recommended since the introduction of antibiotics has made the complication of septic emboli rare. Today the procedure is only indicated for specific reasons: (1) when the clot extends beyond the mastoid area; (2) persisting septicaemia and pulmonary complications despite initial treatment with surgery and antibiotics; (3) infection or thrombosis of the IJV.[5,6,13,14,42,44] We had two cases of pneumonia that may have been secondary to septic emboli. Both those cases recovered with antibiotic treatment.

- **This is a review article on the management of lateral sinus thrombosis arising from chronic middle-ear disease**
- **The paper is based on four clinical cases, three of whom had cholesteatoma**
- **The literature on the diagnosis and management of such cases is comprehensively reviewed**

Anticoagulation

The role of anticoagulation in septic LST remains unclear.[45] Anticoagulation has been advocated to prevent extension of the thrombus to distal sinuses.[42,45] However, it is rarely used now as most infections can be controlled with antibiotics and surgery, and this tends to prevent the thrombus propagating.[5] Risks of anticoagulation include releasing septic emboli from clot breakdown, and uncontrollable haemorrhage at the surgical site.[6] Anticoagulation may be indicated in select cases with evidence of thrombus progression, embolic events and neurological changes.[45] Anticoagulation was used in one patient (*Case 4*) to prevent further intracranial extension of thrombus. She had papilloedema and symptoms of raised intracranial pressure post-operatively. The patient was non-compliant with warfarin but recovered without any complications.

Non-otological causes of LST can be due to hypercoagulable states, scalp abscess, or following a radical neck dissection.[8,33,46,47] The non-septic type of LST (NSLT), that is not associated with ear infections, is now increasingly diagnosed.[48] Mastoid changes are frequently detected on CT or MRI in cases of NSLT. An otolaryngology evaluation is usually required to exclude coexisting mastoiditis. The mastoid changes are likely to be due to venous congestion as a consequence of the thrombosis, rather than mastoiditis.[49] Treatment for NSLT is anticoagulation.

Conclusion

Lateral sinus thrombosis remains a rare otogenic intracranial complication. A diagnosis of LST should be considered in patients with persistent fever, otalgia and otorrhoea despite receiving antibiotic treatment. Cholesteatoma disease is the most common cause of LST. Blood cultures are frequently helpful in identifying the organism(s) responsible. The bacterial aetiology of LST is often polymicrobial with a predominance of anaerobes. Antibiotic treatment should initially be broad spectrum and have good blood brain barrier penetration. Imaging with CT scan or MRI is essential to evaluate the mastoid cavity, lateral sinus, and exclude associated intracranial complications. The role of clot removal at surgery and anticoagulation is controversial. Mortality from LST remains low if treatment with antibiotics and surgery is instituted early.

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Address for correspondence:
Dr Eng Ooi,
C/o 3/98 Fisher Street,
Fullarton,
SA 5063, Australia.

E-mail: eooi@ozemail.com.au

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