Otogenic Lateral Sinus Thrombosis—A Case Report

K C See,*, J L Leong,**MBBS, H K K Tan,***FAMS, MBBS (NSW), FRCS (Edin)

Abstract

***Introduction**: We portray and discuss a case of lateral sinus thrombosis following acute otitis media and mastoiditis. **Clinical Picture**: The patient presented with otorrhoea, otalgia, neck pain, fever and chills. **Treatment**: Cortical mastoidectomy was performed. Intravenous antibiotics and heparin were administered. **Outcome**: The patient had a complete recovery with no sequelae. **Conclusions**: Neurotologic complications of suppurative otitis media like meningitis, cerebral abscess, extradural abscess and dural sinus thrombosis are rare in the antibiotic era. Hence, doctors today have to maintain extra vigilance and a high index of suspicion for such complications.*

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Key words: Magnetic resonance imaging, Management, Mastoidectomy, Mastoiditis, Otitis media

Case Report

A 16-year-old boy, with a background history of beta-thalassaemia intermedia, splenectomy and hypogonadotrophic hypogonadism, was referred to our otolaryngological clinic for right otalgia following an episode of upper respiratory tract infection (URTI). He had hearing loss with tinnitus in the right ear, nasal obstruction with greenish nasal discharge and cough. Otoscopy revealed right acute otitis media. The nasal cavity was filled with mucopus. The left ear, throat and other head and neck regions were normal. He was diagnosed with right acute otitis media (AOM) and sinusitis. Oral cefuroxime was prescribed with rapid resolution of otalgia and nasal discharge within 2 days.

Two weeks later he returned with similar symptoms and signs. He was started on oral augmentin. Otitis media persisted 2 weeks later on follow-up. Right otalgia was severe and purulent effusion was seen behind the tympanic membrane. Myringotomy and grommet tube insertion were performed on the same day. A week later, the grommet tube was in place, patent and discharging pus. Culture of the pus showed moderate growth of *Klebsiella pneumoniae* sensitive to augmentin, cefuroxime, gentamicin and neomycin. The patient was maintained on augmentin and sofradex ear drops.

He returned 2 days later with additional symptoms of fever, right neck pain and yellow watery discharge from the right ear. On otoscopy, thick secretion or tissue was seen blocking the grommet tube. Neck palpation was negative for masses. A provisional diagnosis of acute mastoiditis and myositis associated with URTI was made. Oral metronidazole was added to the treatment regime. The patient was reviewed 4 days later. By then, the right ear was drying and the neck pain was resolving.

However, 9 days later he returned with recurrence of right neck pain and fetid discharge from his right ear. Urgent computed tomography (CT) scan of the temporal bone showed erosion and coalescence of the right mastoid air cells consistent with right mastoiditis. Full blood count revealed a raised total white of 16.5 x 10^9/L. The platelet count was 2 times normal at 700 to 800 x 10^9/L. Erythrocyte sedimentation rate (ESR) was raised at 80 mm/h. Cortical mastoidectomy was performed for him on the same night. Intraoperatively, pockets of pus at the mastoid tip and granulation tissue were seen. Culture of the pus again showed *K. pneumoniae* sensitive to cefuroxime. Intravenous ceftriaxone and metronidazole were administered with sofradex ear drops. After operation, his otalgia, neck pain and fever resolved. The patient was discharged on the fifth postoperative day. Outpatient review 3 days later noted healing of the mastoid wound.

A week later, 15 days after cortical mastoidectomy, he

* *Medical Student*
National University of Singapore
** *Trainee Medical Officer in Otolaryngology, Head & Neck Research Fellow, Hospital of the University of Pennsylvania, Philadelphia, USA*
*** *Associate Professor*
Department of Otolaryngology
National University Hospital
Address for Reprints: Associate Professor Henry Tan Kun Kiaang, Department of Otolaryngology, National University Hospital, 5 Lower Kent Ridge Road, Singapore 119074.

was readmitted with fever and discharge from the mastoid wound. Culture of the mastoid discharge again grew *K. pneumoniae* sensitive to cefuroxime. Intravenous augmentin was given for 72 hours. The patient was then discharged with oral augmentin. However, he was readmitted 5 days later for septic fever, chills and rigors. He did not have ear discharge, otalgia, papilloedema or neurological deficit. Blood culture revealed *K. pneumoniae*. Repeat CT scan of the temporal bone showed partial lateral sinus thrombosis (LST) (Fig.1). The thrombus extended from the proximal end of the sigmoid sinus intracranially to the upper end of the internal jugular vein in the neck. Intravenous ceftriaxone and heparin were started. His fever settled 6 days later. A further 3 weeks of intravenous ceftriaxone was given. The patient was then discharged and has been well since.

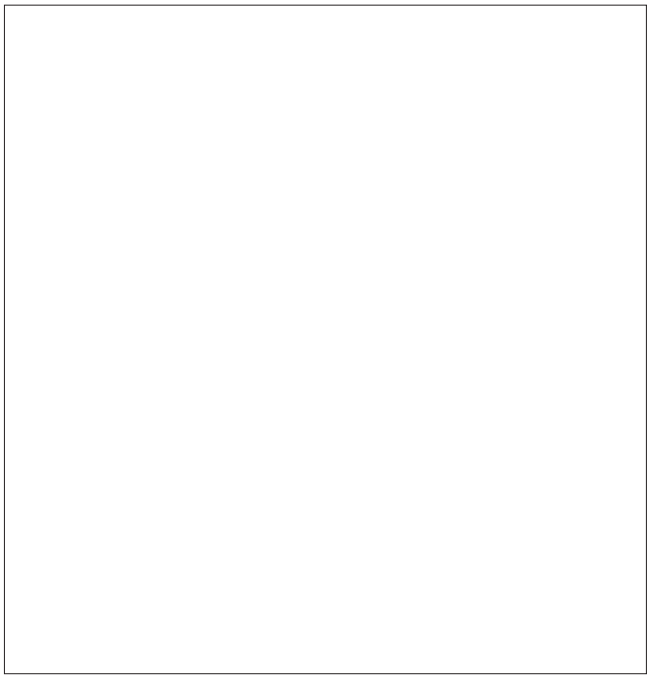


Fig. 1. Axial CT scan image demonstrating partial thrombosis of the upper end of the right internal jugular vein.

Discussion

Otitis media (OM) is the most common childhood disease, accounting for 20% to 40% of clinic visits in the first 5 years of life.[1] It is a relatively benign, often self-limiting infection of the middle ear cavity.[2] Nonetheless, a small percentage (<0.5%) of cases result in serious complications, compared to >40% early this century.[3]

In 1883, Politzer[4] described dural sinus thrombosis secondary to OM and ascribed this to two pathogenic mechanisms: direct spread of infection from the mastoid created by erosive osteitis and retrograde thrombophlebitis via emissary veins and other venous communications. Thrombophlebitis starts at the lateral sinus, which abuts the posterior aspect of the mastoid. Anterograde propagation of thrombus occludes the internal jugular vein and may rarely extend to the superior vena cava, giving the superior vena cava syndrome.[5] Retrograde extension of thrombus through the transverse sinus and the superior sagittal sinus may impair cerebrospinal fluid (CSF) drainage through the arachnoid granulations. This elevates CSF pressure and results in otitic hydrocephalus.[6,7] Medial spread via the superior and inferior petrosal sinuses can result in cavernous sinus thrombosis.[8] Mural thrombosis can also lead to septic embolisation, bacteraemia and septicaemia.

Non-infectious causes of LST also exist: traumatic or non-traumatic intracranial haemorrhage in neonates;[9,10] dural sinus vasculitis in Behcet's disease.[11]

In our patient, splenectomy (as a form of immune impairment) had heightened his risk of otitis media,[12] especially from infections by encapsulated organisms like *Streptococcus pneumoniae, Haemophilus influenzae* and *K. pneumoniae*. Even then, neurotologic complications like LST are exceedingly rare (0.15%[13]) at present. This compares with an incidence of less than 0.001% in the general population with otitis media.[14,15] Diagnosis requires a high index of suspicion.

Data from Garcia et al,[16] Debruyne,[17] Cohen et al,[18] Kelly et al[19] and Bharath[20] are combined to yield a case series of 55 paediatric patients with lateral sinus thrombosis in the antibiotics era (1930s and beyond). The top 3 symptoms are headache in 42 patients (76%), otalgia in 27 patients (49%) and vomiting in 16 patients (29%). Other symptoms include vertigo, diplopia, chills, neck pain, photophobia and cough. The top 3 signs are fever in 36 patients (65%), otorrhoea in 31 patients (56%) and papilloedema (secondary to otitic hydrocephalus[6]) in 23 patients (42%). Other signs include abducens palsy and neck rigidity. Abducens palsy is due to reactive perineural inflammation of the abducens nerve, which runs in close relation to the inferior petrosal sinus, the latter being involved by thrombophlebitis.[21]

These observations are in congruence with the early signs and symptoms of neurotologic complications in general, the top 3 being purulent/malodorous drainage, headache (from dural involvement)/deep ear pain and fever.[22] Our case presented with otorrhoea, otalgia, neck pain, fever and chills which agree with the above findings. "Picket fence" fever is rare, which is probably suppressed by antibiotics. Griesinger's sign, which is mastoid process oedema secondary to mastoid emissary vein thrombosis, was not observed.

In comparison, for AOM, otalgia, fever and otorrhoea are present >75%, 23% and <3% of the time respectively.[12] As such, while otalgia does not distinguish uncomplicated AOM (>75%) and LST (49%) well, fever (23% for AOM

versus 65% for LST) and otorrhoea (<3% for AOM versus 56% for LST) may.

Moreover, for uncomplicated AOM, symptoms begin to abate within 2 to 3 days,[23] and most children are well within 1 week. Prolongation of symptoms for more than 2 weeks before hospitalisation[16] should again alert the doctor to more serious complications of AOM like acute mastoiditis and neurotologic conditions.

Interestingly, LST can occur even after middle ear disease has resolved. This is because blockage of the aditus by mucosal changes (e.g. oedema) divides the mastoid antrum and the middle ear into separate compartments. Middle ear disease then remits relatively quickly with antibiotics and the return of Eustachian tube function, while the mastoid infection worsens and extends intracranially. This is the basis for "silent otitis media".[24]

Historically, LST was usually confirmed during mastoidectomy. Currently, CT and magnetic resonance imaging (MRI) provide valuable non-invasive diagnostic alternatives for LST and other neurotologic complications of OM like meningitis, extradural abscess and intracranial abscess. On plain CT, the "cord sign", which represents a high-density clot in a dural venous sinus, is pathognomic of sinus thrombosis but is seldom seen.[25] On CT with contrast, the delta sign, a low density central region surrounded by a high intensity rim, may be seen.[26] This is due to contrast-enhanced dura surrounding a space-filling clot. Our case incidentally showed the delta sign on contrast-enhanced CT (Fig. 1). MRI with contrast gives a similar picture.[27] MR angiography can provide further information like direction of flow within the dural sinuses.[28] Evidence from Garcia et al[16] showed that CT was only 42% predictive of surgically confirmed LST while MRI was 100% predictive. A further 13 cases studied by McMurdo et al,[29] McArdle et al,[30] Harris et al,[27] Kelly et al[19] and Giuseppe et al[31] made a stronger case for MRI over CT; MRI detected the thrombi in 100% of the cases, while CT missed them all. In addition, LST and other intracranial complications frequently occur in tandem, with rates of association ranging from 31% to 50%.[20,32-35] Therefore, one should be looking out for these other complications when interpreting CT/MRI scans. Cerebral angiography, with its risks of increasing intracranial pressure or dislodging thrombus, should no longer be used. The Tobey-Ayer test is only of historical importance as CT and MRI are much safer diagnostic methods of LST today.

According to Garcia et al's review of paediatric patients,[16] complications of LST include otitic hydrocephalus in 26% of patients. Other LST complications like meningitis, new-onset seizures, transient hemiparesis, brain abscess, septic emboli and deep neck abscess occurred sporadically.

Treatment of otogenic LST requires prolonged intravenous antibiotics,[35] guided by culture and sensitivity results from ear discharge, intraoperative pus specimen and venepuncture. Anticoagulation is clearly recommended in LST only in the following four instances: presence of embolic phenomena, progressive anterograde thrombophlebitis down the internal jugular vein (IJV),[36] retrograde extension to the superior sagittal sinus,[7] and medial extension of LST to the cavernous sinus.[37] Otherwise, no studies on anticoagulation have documented improved survival in uncomplicated otogenic LST. Moreover, anticoagulation is not without its risks of unwanted and uncontrollable haemorrhage. Nonetheless, we employed intravenous heparin to limit intracranial extension of the clot and to preclude serious thromboembolic complications like pulmonary embolism.

The most common surgical procedure for LST was mastoidectomy with subtemporal decompression, done in 84% of patients, while thrombus evacuation was attempted in only 21% of patients.[16] From our point of view, the latter should be abandoned as only partial clearing of thrombus can be achieved, with no real advantage. Mastoidectomy should be done urgently, as the mastoid is the nidus of infection. Removing the contaminative source reduces subsequent bacterial seeding of the meninges and dural sinuses, thereby speeding up recovery.[38] Cholesteatoma, if present, requires modified or radical mastoidectomy, otherwise a cortical mastoidectomy is enough. Internal jugular vein ligation is rarely done, and only for progressive IJV thrombophlebitis, sepsis and metastatic abscess formation.[39,40] Furthermore, IJV ligation may ironically increase the incidence of embolisation.[41] This procedure has thus been dropped from modern practice.

Permanent morbidity from LST is around 8% to 10%, consisting mainly of hearing loss of varying severity.[15,16,21,33,34,38] Other morbidity like papilloedema should resolve within 6 months after discharge.[6,36,38,42] Mortality from LST is 5% for paediatric patients[16] and approximately 10% for the general population.[21,33,34,38]

Conclusions

Diagnosis of lateral sinus thrombosis requires a high index of suspicion. Headache, otalgia, vomiting, fever, otorrhoea and papilloedema should alert one to the possibility of neurotologic complications like LST, particularly if symptoms are prolonged beyond 2 weeks. Although CT confirmed the diagnosis of LST in this case, CT in general is relatively insensitive for detecting LST, though it can readily detect other intracranial diseases like cerebral abscess. MRI overcomes CT's deficit and should be used for picking up LST, in addition to screening for associated neurotologic complications of OM. We had not used MRI for our case as we were considering conditions

like intracranial abscesses rather than LST.

The treatment of LST involves prolonged intravenous antibiotics and urgent mastoidectomy. Anticoagulation is still controversial. Permanent morbidity from LST is about 10% for both children and adults. Mortality from LST is about 5% for children and 10% for the general population.

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