



## OTOGENIC LATERAL SINUS THROMBOPHLEBITIS : A CASE REPORT WITH REVIEW OF LITERATURE

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**ABSTRACT** Otogenic lateral sinus thrombophlebitis is a dangerous but rare intracranial complication of Squamosal type of otitis media in this modern era of antibiotics. The patient had longstanding foul smelling ear discharge with reduced hearing in the affected ear. He was a 22 year old male and labourer by occupation. He presented with severe headache and Picket Fence fever. After undergoing routine ENT examination, Audiology, CT, MRI, MR Venogram, he was diagnosed as Lateral Sinus Thrombophlebitis. He was given broad spectrum antibiotics including both aerobic and anaerobic cover. No thrombolytic agents were used. Canal wall down Mastoidectomy with evacuation of clot was done. He had a positive outcome without the use of thrombolytic agents. Lateral sinus thrombophlebitis though a rare complication can still occur and we need to have a high degree of suspicion especially in developing countries like India. Several studies imply that the use of thrombolytic drugs is important, while others argue that it is not necessary.

**KEYWORDS :** Lateral Sinus Thrombophlebitis, Squamosal Otitis Media, Mastoiditis, Picket Fence Fever

### INTRODUCTION

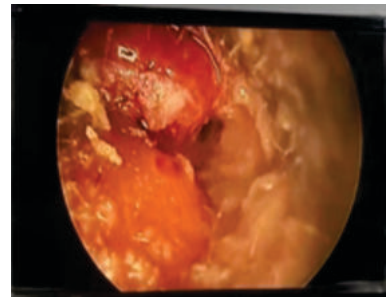
Thrombosis occurring in sigmoid and transverse sinuses are called lateral sinus thrombophlebitis (LST) which is a potentially dangerous complication of otitis media. The rate of occurrence has decreased drastically since the introduction of antibiotics. The incidence rate of LST ranges from 6% to 19% and ranked third or fourth most common intracranial complication of otitis media.<sup>[3,8,9]</sup> Now it is rare, but it poses a danger that require immediate medical and surgical treatment.

The anatomical location of the middle ear and mastoid air cells in the cerebral venous sinuses makes them vulnerable to thrombosis secondary to middle ear infection and inflammation.<sup>[6]</sup> Infection from the mastoid air cell system reaches the peri sinus area, resulting in peri sinus abscess, which then spreads to the dura and intimal layer of the sinuses, causing mural thrombus. Unless effective treatment is promptly started, the mural thrombus grows and necrotizes, forming an intramural abscess. The mural thrombus within the lumen of the sinus propagates proximally to other cerebral venous sinuses, and extends caudally to the internal jugular and subclavian veins. Embolization of the propagating infected thrombus into the systemic circulation causes septicemia. LST usually develops as a complication of chronic suppurative otitis media (CSOM) caused by the direct spread of the disease through the adjacent eroded bone, or as a complication of acute suppurative otitis media (ASOM) caused by thrombophlebitic spread through the emissary veins without bony erosion. The mastoidectomy surgeries, intravenous antibiotics, and radiological investigations, such as computed tomography (CT) scans and magnetic resonance imaging (MRI), have all contributed greatly to the current management protocol. Nowadays, the antibiotic therapy masks the typical presentation. The untreated clinical picture is marked by spiking fevers (the so-called picket fence fever curve, due to the periodic release of septic emboli from the infected sinus thrombus), headache, malaise, and progressive anaemia.

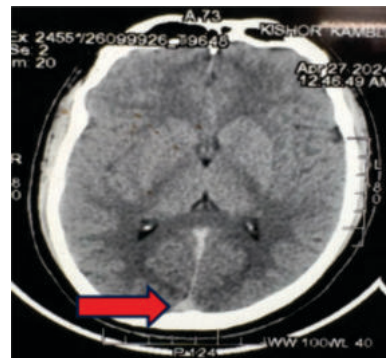
### Case Report

The 22 year old patient presented with excruciating headache. He had history of right ear discharge and right sided reduced hearing since 1 year. He was treated conservatively with Oral amoxicillin and Clavulanic acid 625 mg for a week. He was having high grade fever with temperature peaking in the noon and returning to normal (Picket fence type). He was free from any vestibular symptoms, facial asymmetry or meningitis symptoms. On examination, there was

discharge of right ear with sagging of posterior and superior canal wall. The examination of left ear was unremarkable. The patient did not have mastoid tenderness or erythematous over right mastoid region indicating a negative Griesinger's sign. Crowe beck test was performed and was negative. A baseline Pure tone Audiometry was performed. Ophthalmology opinion was taken and there was no diplopia. He was immediately admitted in the Intensive Care Unit and started with intravenous Meropenem 1g thrice a day and intravenous linezolid 600mg twice a day with Polymyxin B sulphate, Chloramphenicol and Dexamethasone ear drops four drops twice a day over the right external auditory canal.



**Fig 1: Sagging Of Posterior And Superior Canal Wall**



**Fig 2: MRI Showing Empty Delta Sign**

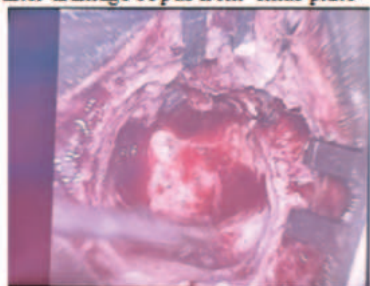
High resolution Computed Tomography of the temporal bone revealed soft tissue density in right middle ear and mastoid air cells involving erosion of ossicles and disruption of ossicular chain indicating otomastoiditis. Magnetic Resonance Venogram revealed partial sinus thrombosis with empty delta sign.

The patient underwent right canal wall down mastoidectomy by post aural incision. Intraoperatively, there was granulations over Tympanic membrane and attic cholesteatoma was seen. Also, posterior external Auditory canal was eroded. On exploring the mastoid cavity, sinus plate erosion was also found. Cholesteatoma sac was exposed from all sides and removed. Incus was eroded – removed, malleus head was engulfed with cholesteatoma flakes and was removed. Pus was drained from sinus plate and partial sinus thrombosis was observed. Wide meatoplasty, saucerization of the cavity was done and flap was placed under the handle of malleus. Soft tissue was placed over the sinus plate and incision was closed in layers.

**Fig 3: MR venogram showing Partial sinus thrombosis**



**Fig.4 showing intra operative picture of canal wall down mastoidectomy cavity after drainage of pus from sinus plate**



Postoperatively patient recovered well in the ward with no signs and symptoms of sepsis and meningitis. The intraoperative pus culture came out as beta hemolytic streptococcus and was sensitive to meropenem. He was discharged after completing 2 weeks of intravenous antibiotics. During follow up, his right ear mastoid cavity was found to be healthy. No thrombolytic agents were used in this patient.

## DISCUSSION

LST is the second deadliest intracranial complication of otitis media after meningitis.<sup>[2,3]</sup> Hence, it is crucial to understand the pathogenesis and recognize the initial clinical presentation of this condition. The mastoid emissary vein establishes interconnection between the posterior auricular vein and the lateral sinus through the mastoid foramina. Although the mastoid emissary vein is a sluggish vein that carries blood away from the brain, it can transmit infection in the retrograde direction back to the intracranial region due to its valveless tubular structure.<sup>[4]</sup> When there is a source of infection from the middle ear or mastoid region, the mastoid emissary vein can become thrombosed and act as a route of infection to the skull bone and intracranial parenchyma, leading to bony erosion and perisinus abscess.<sup>[10]</sup> The consequence of inflammation and granulation formation in the mastoid area will cause reactive thrombophlebitis and intramural thrombus formation in the sigmoid sinus and other adjacent venous sinuses. This thrombus can be either infected or noninfected and tends to propagate as a septic embolus to a distant organ area. Fortunately, our case only involves the peri sinus region which is amenable for surgical removal during mastoidectomy.

intermittent release of septic emboli due to the lytic action of hemolytic streptococcus.<sup>[9]</sup> Other common presenting symptoms can be unspecific and vague such as headache, otalgia, otorrhea, neck pain, visual change, photophobia, and nausea but cannot take it lightly.<sup>[9]</sup> Griesinger's sign is regarded as a pathognomonic sign of LST and reflex thrombosis of the mastoid emissary vein characterized by tenderness and oedema over the mastoid area. In this antibiotic era with the early administration of antibiotics, this classic presenting symptom can be absent as in our case here. Thus, clinicians should always be vigilant with a high index of suspicion even though there is no warning sign of intracranial complication.

Thus, diagnosis of LST depends on radiographic imaging techniques or direct intraoperative detection. Contrast-enhanced computed tomography of the head and neck is the most commonly performed radiographic investigation when intracranial complications of otitis media are suspected. Lateral sinus thrombosis may be diagnosed by the presence of the pathognomonic "empty delta" sign. Early use of the MRI/MRV for the diagnosis of LST has been advocated in the last decade, because they have more sensitivity in detecting this complication, and are also useful for excluding other intracranial complications like adjacent subdural empyema, cerebritis, or cerebral abscess. The gold standard for the diagnosis of LST remains MRV, which is a non-invasive method that can obviously outline the patency of the central venous sinuses, and can be performed simultaneously with a brain MRI exam.<sup>[7]</sup> Magnetic resonance venography enables the distinction between a slow venous flow and an occlusive thrombus, which may be difficult to decide based on spin echo MRI sequences.

Various studies suggest that LST should be considered in patients with persistent fever, otalgia and otorrhea despite receiving antibiotics.<sup>[11]</sup> In regard to the broad usage of antibiotics in this era, it could cause a masking effect of the initial presentation. The commencement of antibiotics has resulted in patients having minimal and even absence of clinical symptoms. The benefit of thrombolytics is still under debate since many studies suggest the use of it and at the same time there are studies showing positive outcomes without the use of thrombolytics including our case.<sup>[11,12]</sup>

## CONCLUSION

Lateral sinus thrombophlebitis though a rare complication can still occur and we need to have a high degree of suspicion especially in developing countries like India. Timely referral to the Tertiary Care Centre is essential as combining parenteral antibiotics with surgical intervention is the treatment of choice.

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The classic symptom of LST is picket fence fever as a result of