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Editorial

Lateral Sinus Thrombosis

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Received: January 17, 2018; **Accepted:** January 24, 2018; **Published:** January 31, 2018

Abstract

Lateral sinus thrombosis is now a rare complication of otitis media. It was first described in 1826. Three decades later, the pathology of lateral sinus thrombosis was first described by Lebert. In 1888, Lane performed the first successful surgery for lateral sinus thrombosis. It was a very well known complication in the pre antibiotic days. Its incidence is reduced due to early antibiotic therapy during initial otitis and mastoiditis. Previous antibiotic therapies often modify the classical picture and make the diagnosis of the occasional case of lateral sinus thrombosis more difficult by altering the expected clinical course. An awareness of this condition is necessary for early diagnosis and treatment. Prognosis is good if treatment with antibiotics and surgery is instituted early.

Introduction

Lateral Sinus Thrombosis (LST) is a well-known complication of otitis media [1]. The proximity of the middle ear and mastoid air cells to the dural venous sinuses predisposes them to thrombosis and thrombophlebitis secondary to infection and inflammation in the middle ear and mastoid [2]. In the past, LST accounted for 6% of all intracranial complications in the era of antibiotic treatment of suppurative ear disease [3]. It is generally considered the third or fourth most common complication among all intracranial complications of chronic otitis media. In the present era, the dramatic drop in the incidence of LST can be attributed to the introduction of antibiotics, earlier diagnosis and prompt effective treatment. The classical picture is often modified because of previous antibiotic therapy making the diagnosis of the occasional case of lateral sinus thrombosis more difficult by altering the expected clinical course [4-6]. Prompt diagnosis of LST requires high index of suspicion and firm understanding of its varied clinical presentations [7].

Demographical Distribution

Presently, lateral sinus thrombosis is most common in adults or older children with cholesteatoma and less common from the other types of chronic mastoiditis. Many reports have documented a clear male predominance [8].

Pathophysiology

The spread of otogenic infection to the lateral sinus may be through coalescent or cholesteatomatous bone erosion or through a thrombophlebitic phenomenon. Thrombosis of the lateral sinus usually forms as an extension of a perisinus abscess that develops after mastoid bone erosion from cholesteatoma, granulation tissue, or coalescence. The perisinus abscess exerts pressure on the dural outer wall of the sinus leading to necrosis. The necrosis extends to the intima and attracts fibrin, blood cells, and platelets. A mural thrombus forms, then becomes infected, enlarges, and occludes blood flow through the sinus [9]. Fresh thrombus can propagate and extend in a retrograde direction to the transverse sinus. In the opposite direction, the clot can extend via the jugular bulb into the internal jugular vein in the neck, and can extend to the cavernous sinus via the inferior or superior petrosal sinus. The infected clot frequently showers the bloodstream with bacteria, giving rise to the signs and symptoms of septicemia and the possibility of metastatic abscesses (most commonly to the lungs). Thrombus formation helps to localize the infection and it may be considered as a protective mechanism.

Because the sinus is a continuation of the cerebellar dura mater, extension of an infection by only a few millimeters can result in meningitis, epidural abscess, subdural empyema, cerebritis, or cerebellar abscess, all equally grave complications. Deep cervical lymph nodes are closely adjacent to the internal jugular vein, throughout its course. Thrombosis of internal jugular vein can manifest as tender mass in the neck along or across sternocleidomastoid muscle [10].

Clinical features

Clinical features vary according to the stage of the disease. The most frequent presenting symptoms were headache, otalgia, fever and vomiting and pain in the neck [11]. With the occlusion of the lumen of the sinus, interruption of cortical venous circulation results in headache, papilloedema and increased intracranial pressure.

The picket-fence fever pattern with diurnal temperature spikes exceeding 103˚F has been described with this condition for decades. Although some more recent articles have pointed out that this pattern is not seen as frequently, in part because many patients present with previous or current antibiotic therapy. Nonetheless, a single high fever reading should alert the clinician to the possibility of sigmoid sinus thrombophlebitis. Tenderness and edema over mastoid (Griesinger's sign) are pathagnomonic of lateral sinus thrombosis and reflex thrombosis of mastoid emissary vein [12].

More ominous are the signs of sudden intracranial hypertension resulting from decreased venous drainage from the skull. This results in progressively worsening headache. This headache is more likely to occur with the obstruction of the dominant venous drainage system (the right side in 60% of patients). 9th, 10th, 11th cranial nerve may be paralyzed by the presence and pressure of clot in the jugular bulb.

Bacteriology

The most common organisms isolated in the post antibiotic era include a mixed flora including bacteroids, staphylococcus, enterobacteriaceae, proteus, pseudomonas and others species. Since antibiotics are commonly used during the prodromal ear infection,

Citation: Viswanatha B and Deepthi P. Lateral Sinus Thrombosis. Austin ENT Open Access. 2018; 2(1): 1005.

blood culture is often negative [13].

Radiology

CT and MRI are the investigations of choice in making a diagnosis. If the patient's condition permits, contrast-enhanced CT scan and contrast-enhanced MRI should be performed. MRI is more sensitive than CT in detecting the thrombus. CT scan is useful in demonstrating the classic 'delta sign' of perisinus dural enhancement and filling defect of the lateral sinus and also can help by ruling out other intracranial complications [14,15]. On gadolinium-enhanced MRI, thrombus appears as soft tissue signal associated with vascular bright appearance of the dural wall. Additionally, MR venography, which can demonstrate the loss of signal and the absence of flow in the sinus, has proven to be more sensitive diagnostic tool in identifying LST.

Management

Once the diagnosis is made, combination of antibiotics and surgical treatment is necessary to reduce the mortality rate16. Almost all patients with sigmoid sinus thrombosis require mastoidectomy to treat the underlying mastoid disease adequately. In selected cases of LST, medical therapy alone with intravenous antibiotics may be successful. However, medical management requires prolonged administration of antibiotics.

Lane performed the first successful surgery for lateral sinus thrombosis in 1888. Until then the mortality for this complication has been 100% [16]. Management of the clot can involve anticoagulation, ligation of the jugular vein in the neck, and opening the sinus and evacuating the infected clot. Use of anticoagulants is rarely indicated, but anticoagulation should be strongly considered when extension of the clot to the transverse sinus or cavernous sinus is suspected or documented. Removal of all perisinus infection is mandatory for effective treatment [17]. Recent reports have shown that if the surrounding granulation tissue and inflammation are removed through a mastoidectomy, the sinus will recannalize without clot evacuation. Jun et al [18] are of the opinion that the organized thrombus is an initial step for spontaneous resolution, finally inducing recanalizaiton of a sinus.

Although it is obvious that LST requires prompt mastoid surgeries with antibiotics for definitive management, the ligation of internal jugular vein is still a controversial issue in the management of LST. Internal jugular vein ligation was performed almost routinely in the pre antibiotic era, to avoid dissemination of thrombophlebitic process and septic emboli. In the modern antibiotic era, internal jugular vein ligation is reserved for those cases in which septicemia and embolization do not respond to initial surgery and antibiotic treatment [16,19].

Most authors agree that there is no place for anticoagulants in the management of LST. The use of anticoagulants is not a part of standard care of patients with LST and was more common prior to the advent of antibiotics. Unless thrombus propagates after surgery, anticoagulants are not recommended. Anticoagulants arrests the spread of thrombosis but may increase the risk of venous infarctions and therefore no longer recommended [9,11,20-24].

In the preantibiotic era, mortality after otogenic LST was nearly 100%. In the antibiotic era, this has dropped to 0% to 25%. This is due to availability of broad-spectrum antibiotics and improved diagnostic tools [25].

Children with otologic disease, presenting with headache, otalgia, and fever need careful evaluation to rule out LST, with further laboratory and radiographic testing. Contrast CT and MRI studies are essential for accurate diagnosis and treatment planning. Aggressive medical therapy with broad-spectrum intravenous antibiotics and surgical removal of the source of infection are essential for a good outcome [26].

Levine et al suggested that otitic hydrocephalus is commonly associated with lateral sinus thrombosis, but not all patients with lateral sinus thrombosis develop otitic hydrocephalus [21]. However in a recent prospective study, on complications of otitis media, association between LST and otitic hydrocephalus was not found to be statistically significant [25].

Lateral sinus thrombosis is now a rare intracranial complication of otitis media. The presence of lateral thrombosis mandates further investigation for additional complication [27].

Conclusion

Lateral sinus thrombosis has become a rare complication of otitis media, but it remains potentially fatal. An awareness of this condition is necessary for early diagnosis and treatment. It frequently occurs in association with other intracranial complications.

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Citation: Viswanatha B and Deepthi P. Lateral Sinus Thrombosis. Austin ENT Open Access. 2018; 2(1): 1005.