Case Report :

Pulsatile tinnitus secondary to sigmoid sinus diverticulum: A case report.

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Received on Aug 01, 2022, Accepted on Sep 11, 2022 , dx.doi.org/10.5455/mjhs.2022.04.010

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Abstract

Sigmoid sinus diverticulum (SSD) has been recently recognized as a cause of pulsatile tinnitus (PT). The cause of the SSD and tinnitus caused by a sinus diverticulum is unknown. Numerous cases have reported successful treatment with surgical or radiological intervention. We report the first case of SSD in our region that was treated surgically with a successful outcome. A 40-year-old female presented to the Ear Nose Thorat (ENT) clinic with the chief complaint of right ear ringing for three years. The symptoms were relieved on compression of the ipsilateral internal jugular vein during clinical examination. Computed tomography showed a bony defect and sigmoid sinus diverticulum. Surgical excision was performed under general anesthesia using the trans-mastoid approach. The diverticulum was identified and obliterated. Temporalis fascia and conchal cartilage were used as grafts. No postoperative complications were noted. A 3-year follow-up showed no signs of recurrence. Tinnitus is a relatively common condition. SSD should be investigated as a probable cause. A successful outcome is governed by the surgeon's expertise.

Key Words:
Pulsatile tinnitus; Case report; Sigmoid sinus diverticulum; Otolaryngology .

Introduction

Pulsatile tinnitus (PT) is a progressive condition that often affects the patient's quality of life and is typically associated with a vascular cause. Clinical and radiological examination results may indicate cerebral arterial or venous compartments effected in PT. Our findings were obtained from a patient with high-pitched unilateral PT and unilateral sigmoid sinus diverticulum (SSD), likely of venous hum origin. A trans-mastoidal approach was employed to treat the anomaly. This is the first case reported in the Gulf region that utilized surgery to treat PT secondary to SSD. It highlights the need to investigate tinnitus, particularly when associated with an unusual etiology.

Case Presentation

A 40-year-old, formerly healthy female presented to our clinic with a 3-year history of right-sided synchronous PT. Tinnitus was perceived to be louder in a quiet environment and on exertion. Turning the patient's head to the ipsilateral side relieved her symptoms. The patient did not experience changes in the pitch of the sound over time and did not suffer from hearing loss, vertigo, ear pain, facial weakness, or disturbed sleep. The bruit interfered with her daily activities but did not affect her sleep. The patient gradually developed depression secondary to tinnitus and was treated with antidepressants. The patient was rated with grade 5 tinnitus based on the tinnitus handicap inventory (THI). There is no history of previous tinnitus. The family history was unremarkable. Physical examination revealed a mildly obese female with a disturbed disposition due to her symptoms. Otoscopic examination revealed no abnormalities in either ear. No bruits were detected on auscultation. Tinnitus was relieved on compression of the ipsilateral internal jugular vein. The patient had a normal neurological and ophthalmologic functions. Pure tone audiometry revealed normal hearing and word discrimination. Routine blood test results were within normal range. A bony defect in the right sigmoid plate with vascular protrusion from the sigmoid sinus was detected on computed tomography (CT) (Fig. 1A).

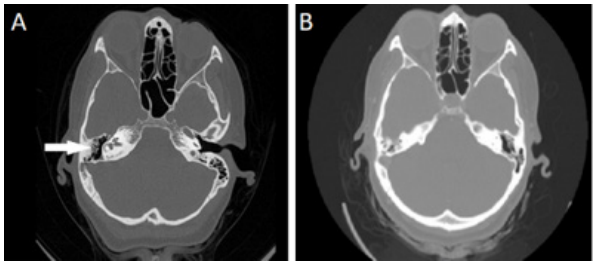


Figure. 1. Axial computed tomography scan of the brain (A): preoperative scan which shows a small right bony defect in the inner table of the right sigmoid plate (white arrow) containing vascular structure that is connected to the sigmoid sinus without obvious connection to any other vascular structure. (B): postoperative scan showing obliteration of the diverticulum with bone remodeling around the harvested cartilage.

No other vascular neoplasm, anomaly, or malformation was observed. The treatment plan included obliteration of the SSD using a trans-mastoidal approach to relieve the symptoms and improve the quality of life.

The procedure was performed under general anesthesia. A curvilinear post-auricular incision was made 1 cm posterior to the post-auricular sulcus. The skin, temporalis fascia, and periosteal flap were elevated. A bluish mass was visible under an operative microscope, and the mastoid cortex around this mass was drilled until the complete identification of the sigmoid diverticulum (Fig. 2A).

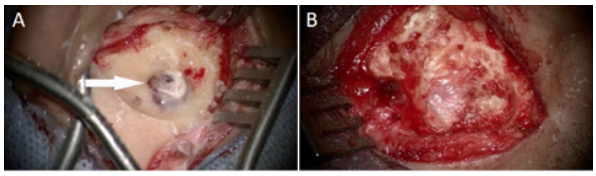


Figure. 2. Gross intraoperative view

(A): Well-identified sigmoid sinus diverticulum (white arrow). (B): Temporalis fascia graft covering the obliterated diverticulum.

This was followed by mastoidectomy and obliteration of the diverticulum. The conchal cartilage and temporalis fascia were used as grafts to obliterate the diverticulum (Fig. 2B). An absorbable hemostatic agent was used to achieve hemostasis. The patient reported that the sounds disappeared immediately after the procedure and was discharged with no adverse reactions the following day. Postoperative venous CT was performed after three months, which showed obliteration and reduction of the diverticulum with bone remodeling around the harvested cartilage (Fig. 1B). No recurrence of tinnitus was noted during the subsequent 3-year follow-up. Repeated THI showed grade 1 of tinnitus severity.

Discussion

The perception of noise in the absence of an auditory stimulus is known as tinnitus1. When tinnitus is perceived as rhythmic and synchronizes with the heartbeat, it is defined as PT2. PT can be produced by vascular organs (arteries or veins) or non-vascular organs (neoplasms or middle ear conductive hearing loss)[3]. Sigmoid sinus wall anomalies (SSWA), such as SSD or sigmoid sinus dehiscence (SSDe), are the venous cause of PT in 35% of the cases1. Most cases of SSWA have been reported in middle-aged obese females, suggesting a gender predilection[4]. One of the theories behind the epidemiology is that SSWA can be a manifestation of idiopathic intracranial hypertension (IIH), which is common in this sub-group1. Prolonged turbulence in sigmoid sinus blood flow eventually leads to SSWA and SSD. Turbulent blood flow and bony erosion can also occur secondary to cerebrospinal fluid (CSF) pulsation and increased intracranial pressure. The above processes contribute to the pathophysiology of IIH1. In addition, most cases of PT occur on the right side, which is the dominant venous system in most populations5. This is due to the nonsynchronous development of the venous sinuses during embryonic development[6]. Unilateral dominance of the venous system leads to irregular vessel diameter, resulting in irregular blood flow, followed by PT[7]. Venous PT is eliminated or attenuated when the ipsilateral internal jugular vein is compressed, and the volume is often altered by head positioning[1,5]. Symptoms usually last for several months, and some patients have other symptoms, such as hearing loss or

vertigo[1,8]. Otoscopy usually reveals a normal tympanic membrane[5,8-10]. The reported case is in conjunction with the epidemiology and presentation of this disease.

Since SSD is a common cause of venous PT, CT is used for the diagnosis due to its low cost and better visualization of bone anatomy compared to magnetic resonance angiography (MRA)[2]. Most diverticula occur in the sigmoid-transverse junction of the anterior, anterolateral, and lateral walls[5,11]. SSWA is often an incidental finding on CT and is found in 18% of patients undergoing imaging for other symptoms1. Concurrent abnormalities include prediverticular stenosis, a high jugular bulb, or superior semicircular canal dehiscence[1,8-10]. There is debate on the role of temporal bone pneumatization in the transmission of sigmoid sinus vibration. All the patients had extensive temporal bone pneumatization in a study by Liu et al. [11]. They concluded that pneumatization amplifies PT secondary to SSD. In contrast, another study did not show a statistically significant association between the severity of PT and pneumatization of the temporal bone[12].

The most common treatment options for SSD are sigmoid sinus wall reconstruction (91.4%), endovascular procedures (7.9%), and the use of endovascular clips and cardiac U-clips (0.7%)4. The trans-mastoid approach involves reducing the diverticulum and reinforcing the sinus bone[13]. Several materials, such as temporalis fascia, autologous bone chips, bone pate, and bone cement, have been employed to achieve multilayer closure[4]. The major surgical complications are extraluminal compression and intravascular thrombosis[2]. Other minor complications include ear fullness and peri-auricular numbness[4]. Endovascular treatment is minimally invasive and can address diverticulum and stenosis but cannot address bony wall defects[10]. Complications of this procedure include stent thrombosis, which requires prolonged anticoagulation therapy and coil migration[4]. A less common treatment method is the retro-sigmoid approach and diverticulum clipping. It is less invasive than other surgical treatments, but no long-term follow-up study has been conducted to date[14,15].

Trans-mastoid sinus wall reconstruction has been shown to be successful in 90% of patients[2]. Recurrence is minimal but can occur several years later[4,13].

Conclusion

In conclusion, this is the first report of an SSD-causing PT in Saudi Arabia. Since a delayed diagnosis is associated with morbidity and poor quality of life, physicians should be aware of the vascular causes of PT and include them in the differential diagnosis.

Informed consent statement:

Informed written consent was obtained from the patient for publication of this report and any accompanying images.

Conflict-of-interest statement:

The authors declare that they have no conflict of interest.

Financial support:

There are no financial conflicts of interest to disclose

Acknowledgement:

I would like to thank the Deanship of Scientific Research, Majmaah University for providing guidance and support in writing the manuscript.

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