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A New Therapeutic Procedure for Treatment of Objective Venous Pulsatile Tinnitus

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Abstract: Pulsatile tinnitus usually means a turbulent flow within the vessels. We describe a 54-year-old man with a disabling objective pulsatile tinnitus due to a diverticulum of the sigmoid sinus toward the ipsilateral mastoid. We performed a surgical intervention via the endovascular route using coils to obliterate the diverticulum and a stent to avoid coil migration. The patient had a complete and immediate remission of the pulsatile tinnitus. This procedure has been described only in arterial circulation.

Key Words: arteriography; pulsatile tinnitus; sigmoid sinus; vascular malformation

Objective tinnitus is very uncommon in the daily practice of an otolaryngologist. Owing to this rarity, patients seldom are investigated in a correct manner, decreasing the possibility of having an adequate diagnosis and a compatible treatment. Objective tinnitus usually arises from muscular or vascular structures and can be pulsatile (when synchronous with the heartbeat) or nonpulsatile [1–6]. Pulsatile tinnitus may arise in arterial or venous structures, resulting from a turbulent blood flow due to an increase in blood volume or pressure or to changes in the vessel lumen [4, 7]. Its severity is directly related to the subjective annoyance provoked in patients' lives and, in severe cases, can even lead to suicide [1, 4, 8].

Very few articles have described the treatment of patients with pulsatile tinnitus besides the description of the case itself. The objective of our study is to report a case of disabling objective pulsatile tinnitus, the surgical treatment of which via an endovascular route led to its cure, reestablishing the patient's quality of life.

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CASE REPORT

Anamnesis and Physical Examination

P.R.S., a 54-year-old man, complained about a pulsatile tinnitus in his left ear for the prior 3 years, with spontaneous onset and no remission since then. He was strongly convinced that his tinnitus was responsible for his hearing difficulty because compression of the left part of his neck caused tinnitus to disappear and hearing to improve. Tinnitus thoroughly disrupted his life, prohibiting normal sleep and concentration patterns and leading to suicidal intentions. No other complaint was present. The ear, nose, and throat and neurological examination results were normal. However, during auscultation of the ear, we noticed a pulsatile rough bruit coming from the left external canal and from the mastoid, which completely disappeared with a light compression of the ipsilateral jugular vein.

Complementary Examinations

Three pure-tone audiometric examinations were performed within 2 months and showed different results, with a sensorineural hearing loss varying from mild to severe in the left ear. The results were considered inconclusive once the patient reported great difficulty in performing such examinations because he wanted to

compress his jugular vein many times to decrease tinnitus and to be able to distinguish the pure-tone signal. The audiologist also reported a significant difficulty in obtaining the pure-tone thresholds because the patient worried too much about compressing his neck to ascertain the given tone. This fact was considered mainly responsible for this fluctuation in pure-tone thresholds. However, vocal audiometry and discrimination were within normal limits. Laboratory examination results were normal and excluded the systemic causes of pulsatile tinnitus, such as anemia and hyperthyroidism. The auditory brainstem response was normal, although the morphology was poorer in the left side. Once again, the patient remained very tense during the entire examination. The computed tomography scan showed an extension of the left transverse-sigmoid sinus, forming a diverticular image protruding into the mastoid area (Fig. 1). Magnetic resonance angiography was normal. Cerebral angiography confirmed the lesion seen in the computed tomography scan, demonstrating a slow and turbulent blood flow within the diverticular image projected from the left transverse-sigmoid sinus (Fig. 2).

TREATMENT

Owing to the important decrease in the patient's quality of life, we opted for obliteration of the venous diverticulum. The procedure was performed under general anesthesia, puncturing the left internal jugular vein and introducing a self-expanding 8- × 60-mm stent covering the transverse sinus and the opening of the diverticulum. Through a No. 2.3 F. microcatheter, the mesh of the stent was crossed, and coils were left filling the diverticulum. The stent avoided the migration of the coils into the transverse sinus once the opening of the diverticulum was fairly large. The pulsatile tinnitus immediately

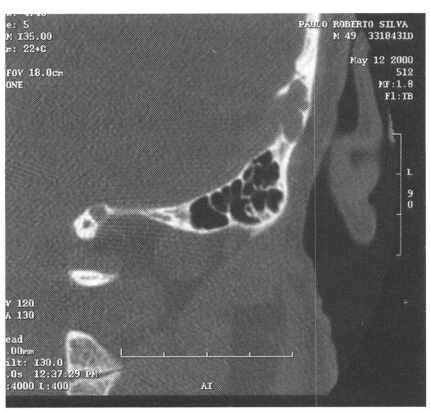

Figure 1. Preoperative computed tomography scan showing enlargement of the left transverse-sigmoid sinus with the diverticular image protruding toward the mastoid.

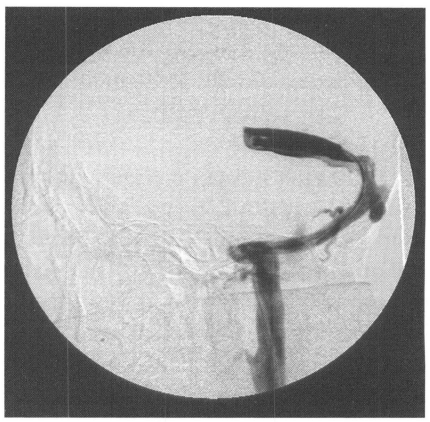

Figure 2. Preoperative arteriography demonstrating the diverticular image projected from the left transverse-sigmoid sinus and a slow and turbulent blood flow.

disappeared after the patient recovered from anesthesia. Although no recurrence was seen during the procedure, the patient developed a small cerebellar area of ischemia, resulting in an ataxic walk, which progressively disappeared during the next 2 months. Postoperative angiography showed absence of the diverticulum with preservation of the blood flow within the transverse-sigmoid sinus (Fig. 3). After 12 months of follow-up, there was no recurrence of the pulsatile tinnitus.

DISCUSSION

The pulsatile tinnitus runs the gamut of differential diagnosis, including vascular tumors (paragangliomas, hemangiomas), vascular malformations (dural arteriovenous fistula), and other congenital or acquired vascular abnormalities [4, 9–11]. Owing to the proxim-

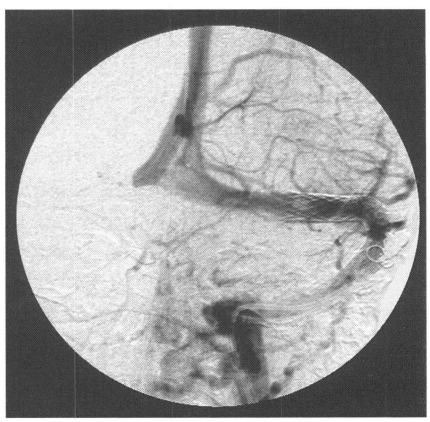

Figure 3. Postoperative arteriography showing the venous diverticulum obliterated by the coils and the mesh of the stent prohibiting them from migrating back to the transverse sinus.

ity between the ear and large vessels, such as the carotid artery, the sigmoid sinus, and the jugular bulb, pulsatile tinnitus was thought to be more common as a complaint of disease in such vessels [12].

The transverse-sigmoid sinus can be asymmetrical in 50–80% of the normal population and absent in up to 5% of people, according to retrograde jugular venographic scans [13]. Occasionally, it may be aberrant, in a more lateral or anterior position [14]. Then, it usually receives drainage of inconstant veins from the scalp and deep cervical region, called *emissary* and *diploic* veins.

An aberrant sinus or thrombosis of venous anastomosis can lead to a turbulent blood flow responsible for an objective pulsatile tinnitus. Because such anomalies do not cause evident neurological symptoms, they traditionally were treated in a conservative manner. Owing to its low incidence [15], venous tinnitus is an exclusion diagnosis. Benign intracranial hypertension, compression of the internal jugular vein by the atlas lateral process, a high-dehiscent jugular bulb, and the presence of an abnormal mastoid emissary vein are known causes of venous tinnitus [5, 16, 17]. The disorder may be related to hemodynamic changes that occur in systemic diseases (anemia, thyrotoxicosis, valvular cardiac disease) and in the presence of predisposing factors (pregnancy, oral contraceptives, collagenous diseases, hypercoagulability states) [18, 19].

The pulsatile tinnitus in our patient was due to an anatomical variation of the transverse-sigmoid sinus, which insinuated itself on mastoid cells by probable thrombosis of an emissary vein (inside the diploë), modifying the regular laminar blood flow and causing its turbulence. As this condition does not implicate a neurological risk, it is advisable to employ a less aggressive treatment, aimed at normalization of blood flow and venous drainage and at anatomical preservation of the involved sinus. However, our patient's quality of life was so disrupted because of the pulsatile tinnitus that we were obliged to discover an alternative solution.

Therapeutic embolization is a viable alternative in cases of pulsatile tinnitus caused by flow alteration, vessel abnormality and, even in some cases, paraganglioma [20]. In our patient, a technical alternative able simultaneously to preserve the flow within the transverse sinus and to allow the occlusion of the diverticular structure was necessary. One alternative would be the placement of coils into the diverticular structure [21], but its large opening would increase the possibility of coil herniation, with consequent occlusion of the transverse sinus. Therefore, we chose instead first to introduce a stent, which originated as a tool for keeping the arteries open after angioplasties, followed by introduction of the coils through its mesh, using the stent as protection. Thus, the coils were kept safe inside the diverticular structure, separating the diverticulum from the normal blood circulation and maintaining a low risk of herniation.

The use of the stent to avoid coil migration had been described previously but only in arterial circulation and for the treatment of pseudoaneurysms [22, 23]. We did not find any report associated with coils in pulsatile tinnitus, but this technique significantly increased the safety of the procedure, avoiding coil migration and retaining sinus permeability. The possibility of the diverticular structure emerging as the cause of the pulsatile tinnitus was clearly proved by the disorder's immediate disappearance after such endovascular treatment.

CONCLUSION

The adequate identification of the possible etiology of pulsatile tinnitus is of utmost importance to determine the appropriate treatment for each case. Surgical intervention via an endovascular route using coils and stents was an effective procedure for our patient. Thus, it may be performed in patients with a disabling pulsatile tinnitus with an etiological diagnosis that calls for such treatment.

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