Treatment of Pulsatile Tinnitus Associated with Multiple Factors

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Numerous factors may lead to pulsatile tinnitus (PT), including atherosclerosis, benign intracranial hypertension, glomus tympanicum tumor, dural arteriovenous fistulas (dAVFs), abnormalities of the sigmoid sinus (dehiscence and diverticulum), and jugular bulb anomalies (glomus jugular tumor, diverticulum, high-riding or dehiscent jugular bulb). However, exact causes often cannot be found in many patients even after a detailed physical examination and extensive auxiliary examinations.[1] Moreover, no effective treatment is available for these patients. Patients with PT associated with multiple factors are seldom reported, and the condition is essentially intractable. Here, we reported three cases with PT involving multiple factors.

A 38-year-old woman presented with a chief complaint of PT on her right side for 7 years. The results of pure tone audiometry and acoustic immittance were normal. Her PT could be alleviated after compressing the ipsilateral jugular vein. Digital subtraction angiography (DSA) and computed tomography angiography (CTA) revealed a small diverticulum on the right-side of her sigmoid sinus, and a vascular malformation was found in the right frontal-parietal lobe. With a strong desire to relieve the PT and her consent, the patient was hospitalized to remove the diverticulum by surgery at the Department of Otolaryngology, Beijing Tongren Hospital, in April 2012. The diverticulum was skeletonized and reduced with extraluminal placement of the temporalis fascia and autologous bone pate. The patient experienced no change in the PT after this surgery, although the diverticulum was eliminated completely as shown in the postoperative CTA. Then, the CTA findings before and after the surgery were studied again. Extensive bone defects (dehiscence) were found in the right-side sigmoid sinus wall. She also had a high-riding and dehiscent jugular bulb, a possible underlying factor for PT.[2] With the patient's consent, a second surgery was performed under local anesthesia for reconstruction of the dehiscent wall of the sigmoid sinus and jugular bulb in December 2012. The patient experienced immediate resolution of the PT when the partial sigmoid sinus dehiscent wall was reconstructed with autologous bone pate. However, she was so nervous and anxious under local anesthesia that the surgery was discontinued. She had milder PT, approximately 30% of the loudness of the preoperative PT, immediately after the surgery. The patient's mild PT persisted stably at 15 months after the second surgery, when the last follow-up occurred. A postoperative CTA after the secondary surgery demonstrated that the dehiscent sigmoid sinus wall had been reconstructed partially, and the high-riding and dehiscent jugular bulb remained.

A 24-year-old man presented with a chief complaint of PT on his left-side for 5 years after a head injury by car accident. The results of pure tone audiometry and acoustic immittance were normal. CTA revealed a huge diverticulum on the left-side of his sigmoid sinus. To remove the diverticulum, an operation under general anesthesia was done at the Department of Otolaryngology, Beijing Tongren Hospital in March 2013, the surgical procedures were described in Case 1. The patient also experienced no change in the PT after the surgery, although the postoperative CTA shown that the diverticulum was eliminated completely. Then a DSA was done, through the DSA we found a cerebral dAVF on his left temporal lobe. After an interventional therapy in May 2013, his symptoms disappeared. This situation lasted for 12 months when the last follow-up occurred.

A 54-year-old woman presented with a chief complaint of PT on her right side for more than 10 years. CTA revealed two dehiscence of sigmoid sinus wall and a mastoid emissary vein on the right side. She accepted the operation therapy under general anesthesia at the Department of Otolaryngology, Beijing Tongren Hospital in September 2011. Because the mastoid emissary vein pulsated obviously, we judged that the vein was the main factor to cause PT. Therefore, we closed the mastoid emissary vein by bipolar coagulator. After operation, the patient feel PT improved

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slightly. A month later, the intensity of the PT returned to the preoperative status. She refused our proposal for surgical exploration once again.

Pulsatile tinnitus may arise in arterial or venous abnormalities around the ear. The sigmoid sinus diverticulum (SSD), defined as a well-circumscribed sac where the sigmoid sinus focally protrudes into the adjacent mastoid area, is an increasingly recognized factor for venous PT.[2-4] It has been reported that most cases with SSD achieve complete resolution of PT by either transmastoid surgery[2,3] or endovascular coiling/stenting.[5] However, in this paper, the first and second patients experienced no change in their PT after the first surgery, although the diverticulum was eliminated. We speculated that the diverticulum may not have been the only factor causing PT in them. The first patient did experience immediate resolution of her PT when the dehiscent wall was partially reconstructed in the second surgery. The third patients with an untreated operation sigmoid sinus wall dehiscence (SSWD), resulted in postoperative tinnitus symptoms nonimprovement. These illustrate that SSWD was apparently a significant factor in the patient's PT.

In addition to part of the SSWD being unrepaired, two additional possible factors may have caused the residual PT of the first patient. She had a high-riding and dehiscent jugular bulb, and a vascular malformation had been identified in the right frontal-parietal lobe. In second patients, dAVF was proved to be an important factor leading to the PT. In third patient, although there are reports that mastoid emissary vein can lead to PT, but the operation result show that this patient's mastoid emissary vein is not the cause of the disease. Because of her refusal to reoperation, we cannot confirm whether the SSWD is the factor which caused the PT or are there other reasons?

For the diagnosis for PT patients, we recommend CTA as a preliminary work-up modality. CTA is noninvasive, and it demonstrates relationships between temporal bone and adjacent vessels, so it is of great value in detecting anomalies of the sigmoid sinus, jugular bulb, and internal carotid artery, such as SSD, SSWD, high-riding jugular bulb, dehiscent jugular bulb, mastoid emissary vein, and aberrant internal carotid artery. However, CTA cannot replace DSA if dAVF or another vasculopathy is suspected.

We gained some insights from these cases. First, patients with PT should be given a comprehensive evaluation preoperatively because the PT may be associated with multiple factors. Second, for patients with both SSD and SSWD, it is important to eliminate the diverticulum and reconstruct the dehiscent wall simultaneously. Finally, if massive bleeding and nervousness of the patient can be avoided during surgery, local anesthesia is preferable because the surgeon may be able to gain valuable feedback from the patient in this condition.

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