

# Angioplasty and Stenting for Intractable Pulsatile Tinnitus Caused by Dural Venous Sinus Stenosis: A Case Series Report

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**Objective:** Pulsatile tinnitus caused by dural venous sinus (DVS) stenosis is a newly identified form of tinnitus. Its persistent nature can severely affect patients' sleep and quality of life, leading to depression in severe cases. The aim of this report is to investigate the efficacy and safety of angioplasty and stenting in treating this form of tinnitus.

**Study Design:** Retrospective review.

**Setting:** Chinese PLA General Hospital.

**Methods:** Clinical data of 46 cases of pulsatile tinnitus caused by DVS stenosis treated between December 2009 and October 2012 using angioplasty and stenting were reviewed. Diagnosis of DVS abnormality was confirmed in all cases using digital subtraction angiography (DSA). Among these cases, stenosis was located in

the transverse-sigmoid sinuses junction area ipsilateral to tinnitus in 44 cases and on both sides in the remaining 2 cases. Stenosis was treated with angioplasty and stenting in all cases.

**Results:** Pulsatile tinnitus disappeared immediately after the procedure in all 46 cases. There was no procedure-related complication. During the 2 to 36 months' follow-up, there was no recurrence.

**Conclusion:** These results indicate that DVS stenosis is the cause of pulsatile tinnitus in these cases and that angioplasty and stenting are an effective and safe treatment for intractable pulsatile tinnitus caused by DVS stenosis. **Key Words:** Angioplasty and stenting—Dural venous sinus—Stenosis, Tinnitus.

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Epidemiologic studies on tinnitus show that tinnitus prevalence increases along with age. The prevalence is 14.5% among people younger than 40 years, 17.5% among those between 40 and 60 years, 22.2% among those older than 60 years, and 35% among those older than 65 years (1,2). Although most tinnitus is related to sensorineural hearing loss and pulsatile tinnitus accounts for only 4% of tinnitus cases (3), because of the sheer large size of the tinnitus population, pulsatile tinnitus can impose a significant impact on people's well-being and quality of life.

Many cases of the pulsatile tinnitus synchronized to heartbeat are caused by cerebral vascular diseases. Common underlying conditions include atherosclerotic carotid stenosis, arterovenous fistula such as from abnormal communication between the internal carotid and cavernous sinus, posterior fossa dural arterovenous fistula, and so on.

As cerebral vascular imaging technology advances, pulsatile tinnitus caused by venous abnormalities (e.g., dural venous sinus or DVS stenosis, venous sinus diverticulum and high jugular bulb) has attracted increasing attention. Because of their often insidious symptoms and physical signs, venous system diseases are frequently undiagnosed or misdiagnosed, or sometimes patients are thought to have psychological disorders such as depression. This leaves many patients with rather severe venous disease-related ailment untreated and in a great deal of suffering.

Arterial pulsatile tinnitus is often associated with murmurs audible to auscultation. Its source can be readily determined on computed tomographic angiography (CTA), magnetic resonance angiography (MRA), and digital subtraction angiography (DSA) and effectively treated with embolization or resection procedures. In recent years, there have been reports on treating pulsatile tinnitus from DVS abnormalities using intravascular stents and coils (4–8). However, when pulsatile tinnitus is caused by DVS stenosis, despite sometimes clearly located perception, it is not always easily detected with a stethoscope. In addition, the actual location of stenosis is often hidden at the transverse-sigmoid sinus junction area and difficult to identify on

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regular CTA or MRA. DSA and measurement of pressure gradient across suspected stenosis are often needed to confirm the source of pulsatile tinnitus. Because of these reasons, DVS stenosis often goes undiagnosed as the cause of pulsatile tinnitus. These patients therefore fail to receive effective treatment and continue to experience the annoying noise. Some of these patients may be diagnosed as having depression and receive wrongly targeted treatment. In severe cases, persistent pulsatile can severely impair a patient's functionality and quality of life.

The current article reports a series of 46 cases of bothersome pulsatile tinnitus caused by DVS stenosis treated by the primary author between December 2009 and October 2012. After failing medical treatment, these patients were successfully treated with angioplasty and stenting with satisfying outcomes, which also served to confirm the causality of DVS stenosis in pulsatile tinnitus in these cases.

## MATERIALS AND METHODS

### General Data

Pulsatile tinnitus caused by DVS stenosis was diagnosed and confirmed in all 46 cases (44 female and 2 male subjects; aged from 22 to 58 years; mean, 42.1 yr). Duration of pulsatile tinnitus ranged from 2 months to 20 years (mean, 3.5 yr) with sudden onset in 29 cases and gradual onset in 17 cases. Tinnitus was rated as severe and seriously affecting daily functioning and quality of life in all cases. Medical treatment and masking therapies were not beneficial in these cases.

### Diagnosis

The primary complaint in all 46 cases was "whooshing" pulsatile tinnitus synchronized to heartbeat with a definite perception location. Its loudness could be reduced by head rotation or ipsilateral neck compression. Tinnitus was unilateral in 44 cases and bilateral in 2 cases, involving the right side in 34 cases and the left side in 12 cases. Three cases had intracranial hypertension.

### Otologic Evaluation

A pure tone audiometer was used to approximate tinnitus pitch and loudness via sound frequency and level matching (9,10). The pulsatile tinnitus in this series was matched to frequencies between 125 and 1,000 Hz (except for 1 case in which it was matched to 8,000 Hz) with loudness between 30 and 85 dB HL. Otologic diseases, such as otitis media and benign paroxysmal positional vertigo, were all ruled out. No audible noise could be detected in these cases, except in 1 case in which imaging studies showed a venous sinus diverticulum proximal to the stenosis that had eroded the petrous bone.

### Imaging Studies

Magnetic resonance venography (MRV) was completed in 20 cases with clear indication of stenosis in the transverse-sigmoid sinuses junction area ipsilateral to tinnitus perception. In 3 cases with intracranial hypertension, stenosis was seen in distal transverse sinus area. DSA was performed in all 46 cases and showed drainage dominancy on the tinnitus side, stenosis in the transverse-sigmoid sinuses junction area, and diverticulum-like dilation proximal to stenosis (n = 29). In the 2 cases of

bilateral pulsatile tinnitus, DSA showed greater than 70% stenosis in the transverse-sigmoid sinuses junction area on both sides, with greater than 13-second arteriovenous transit time and retention of contrast distal to stenosis (longer than 4 s). The arteriovenous transit time was greater than 15 seconds in the 3 cases with intracranial hypertension (>260 mm H<sub>2</sub>O).

## Treatment

Angioplasty and stenting were elected as treatment after these cases had failed medical therapies.

### Procedure

Under general anesthesia, a 4F catheter (Renegade; Boston Scientific, MA, USA) was passed through the femoral artery to the internal carotid artery or vertebral artery. Angiography was performed to determine the location of venous sinus stenosis and venous drainage to stenotic sinus from cerebral and cerebellar hemispheres and was used to provide guidance for angioplasty and stenting. An 8F guidance catheter was then passed through the contralateral femoral vein to the jugular foramen level on the tinnitus side, followed by passing a 2.4F catheter (Boston Scientific) through stenosis into the posterior sagittal sinus for intrasinus angiography to confirm the location and length of stenosis and to measure pressure across the stenosis. A 3 m X-celerator exchange guidewire (ev3 Neurovascular, USA) was passed to and anchored in the posterior sagittal sinus. After removing the catheter, a 6 × 20 mm Aviator dilation balloon (Cordis, USA) was used to dilate the stenotic section of the venous sinus, followed by placement of 7 × 40-mm Precise self-expanding stents (Cordis) to complete angioplasty. The 2.4F catheter (Boston Scientific) was placed again and the guidewire removed. Venoangiography was repeated and pressure measured again. This was followed by repeat artery angiography via the carotid or vertebral artery to assess the effects of the angioplasty and stenting and venous drainage into the venous sinus system from the Labby's vein, petrous vein, and superior and middle cerebellar veins and to ensure that there was no risk of compression (11–13).

## RESULTS

The angioplasty and stenting procedure was successful in all 46 cases with no neural or vascular complications. Compared with before the procedure, contrast retention from venous sinus stenosis was completely corrected by the procedure. All patients reported complete resolution of tinnitus upon waking up from anesthesia. In the 2 cases with bilateral stenosis, stenting on the dominant draining side eliminated tinnitus on both sides. Average hospital stay was 5 days. Patients were maintained on clopidogrel (75 mg/d) and warfarin (2.5 to 3.75 mg/d) for 6 months, with a prothrombin time between 16 and 25 seconds. Patients were followed on the phone for 3 to 24 months. There were never signs of recurrence in 44 cases. In 2 cases, mild dizziness and mild high pitched nonpulsatile tinnitus were reported around 3 months postoperative. DSA follow-up showed well-maintained stenting and no signs of local restenosis or disturbed flow. The symptoms in these 2 cases disappeared 6 months later.

## DISCUSSION

### Classification of Pulsatile Tinnitus

Pulsatile tinnitus is an auditory perception that is synchronized to the heartbeat or pulse. It is often described as a rhythmic “roaring” or “whooshing” sound and usually caused by vascular structural abnormalities including stenosis that result in accelerated or turbulent blood flows. Pulsatile tinnitus can be from arterial or venous abnormalities (14–16) and can be labeled as objective or subjective depending upon if it can be detected by an examiner. Arterial pulsatile tinnitus commonly originates from skull base dural arteriovenous fistula, large aneurysm, and carotid stenosis, as well as from high heart output situations as in severe anemia, hyperthyroidism, hypertension, and atherosclerosis (14,16–18). Venous pulsatile tinnitus can be caused by DVS stenosis or diverticulum, high riding jugular bulb or deformed mastoid emissary veins, as well as intracranial hypertension or venous sinus thrombosis (19–21).

### Tinnitus Caused by DVS Stenosis

The concept of “venous pulsatile tinnitus” was first proposed by Ott (22) in 1977. It is often caused by stenosis or diverticulum in the transverse or sigmoid sinus in the mastoid area, high jugular bulb, or aberrant mastoid emissary veins. As pressure in the venous system is lower than that in the arterial system, the loudness of venous pulsatile tinnitus is noticeably lower than that of arterial pulsatile tinnitus and hard to detect by auscultation. Venous pulsatile tinnitus is therefore often thought to be subjective tinnitus, leading to misdiagnosis. In 1980, Stern and Goldenberg (23) found that DVS diverticulum was associated with pulsatile tinnitus. In 1985, Kennedy et al. (24) indicated that high riding jugular bulb could be the cause of venous pulsatile tinnitus. In 1986, Lampert and Cantrell (25) found that large size mastoid emissary vein could cause severe pulsatile tinnitus. However, tinnitus associated with DVS stenosis was not mentioned until Russell’s first report in 1995 (26). Little has been added since Russell’s first report on pulsatile tinnitus caused by DVS stenosis, probably because of the complexity and difficulties in its diagnosis and treatment. DVS stenosis was confirmed on DSA in the transverse-sigmoid sinus junction area in all the 46 cases in our series, with abnormal distal dilation in 29 cases that could lead to turbulent flow. The noise from turbulent flow in the venous sinuses can be transmitted to the cochlear via bone conduction to produce tinnitus. Because this noise is correlated to changes in blood flow dynamics, it is synchronized to heart beat. The consistent resolution of pulsatile tinnitus in response to angioplasty and stenting treatment in our series also clearly indicates the causality of DVS stenosis in these patients’ pulsatile tinnitus.

### Diagnosis and Differentials in Venous Pulsatile Tinnitus

In diagnosing pulsatile tinnitus, otologic and audiometric evaluation is critical to rule out external and middle ear issues, including Schwartze sign under otomicroscopy

that may represent a high jugular bulb, aberrant carotid artery, or glomus tumor. Auscultation in and around the ear, at the orbit, and in the neck and chest areas is also important (27). Arterial pulsatile tinnitus is often audible in the postauricle, supra-orbit, and occipital areas, which may decrease when the carotid is compressed in the neck because of reduced blood flow and intravascular pressure. In comparison, tinnitus caused by DVS stenosis may decrease from simple head/neck rotation (28), probably from the pressure on the jugular vein by the sternocleidomastoid muscle and transverse process of atlas, as well as from gentle compression of the jugular vein in the neck (29). When pulsatile tinnitus is associated with benign intracranial hypertension caused by thrombosis, optic papilloedema, and vision deterioration are often present (30).

Imaging studies are an important part of diagnosis in pulsatile tinnitus. CTA and MRA are capable of demonstrating the location and degree of most cerebral artery stenosis, as well as the supplying artery and draining vein in skull base dural arteriovenous fistula. For venous sinus diseases, MRV can show the dominance of the DVS system, location and degree of existing stenosis, as well as presence of diverticulum or high riding jugular bulb. MRV may help determine relations between these abnormalities and pulsatile tinnitus (14,31). DSA is an invasive test and associated with certain levels of risk of complications. However, in cases in which the patient’s tinnitus is severe and seriously affects functioning and quality of life or causes significant psychological complications, DSA can be considered which not only can demonstrate DVS stenosis and stasis of venous flow but also help determine the potential benefits of surgical intervention. DSA was selected in the 46 cases in this series after routine noninvasive imaging studies to confirm diagnosis and to facilitate planning of angioplasty. It provided reliable evidence basis regarding cerebral venous drainage structures, which was critical in guiding the angioplasty procedure.

### Treatment of Venous Pulsatile Tinnitus

Treatment of pulsatile tinnitus depends upon the nature of the underlying condition. Intravascular interventional procedures have been reported to be successful for cerebral arterial pulsatile tinnitus, including ultraslective arterial embolization for arterial malformation and arteriovenous fistula, angioplasty and stenting for arterial stenosis, and surgical ligation or arterial bypass procedures when appropriate.

Because compression of jugular vein in the neck often results in decreased venous pulsatile tinnitus, some have suggested ligation of the jugular vein as the treatment for venous pulsatile tinnitus. In 1978, Ott (22) reported using jugular vein ligation under local anesthesia for suspected venous pulsatile tinnitus. This treatment, however, remained controversial over the next 30 years. Duvillard et al. (32) and George et al. (33) believe that jugular vein ligation is effective and safe in clearly diagnosed pulsatile tinnitus associated with DVS abnormalities. Zhang et al. (34) have reported 12 cases of pulsatile tinnitus, in which tinnitus decreased or disappeared after

jugular vein ligation in 7 cases but remained unchanged in the other 5 cases. On the other hand, Jackler et al. (35) believe that ligation of jugular vein can result in serious neurologic complications including DVS thrombosis, headaches and vision deterioration. Mahasin et al have reported a case of unilateral jugular vein ligation that was followed by thrombosis in the transverse sinus (36). Because up to 50% of normal population can have unilateral aplastic transverse sinus and rely on the contralateral dominant transverse and sigmoid sinuses for venous draining, ligation of dominant jugular vein without timely establishment of collateral circulation will likely lead to intracranial hypertension and secondary thrombosis. In 1997, Mathis et al. (37) first reported a case of intracranial hypertension, in which tinnitus went away after stenting for DVS stenosis. There have been no additional similar reports since then.

In pulsatile tinnitus caused by DVS stenosis, increased pressure in the sigmoid sinus from compressing the jugular vein leads to decreased pressure gradient across the existing stenosis and hence reduced tinnitus loudness, indicating that all measures that reduce pressure gradient will likely reduce tinnitus related to DVS stenosis. The primary author has been engaged in treating DVS stenosis and thrombosis using angioplasty and stenting for many years with success in most cases and experiences in technique improvement (12,38). The success in these cases has prompted the authors to extend the treatment to treating pulsatile tinnitus caused by DVS stenosis. The primary author's expertise in this field may have served as the basis for the so far satisfying outcomes.

For cases that are complicated with thrombosis and intracranial hypertension, such as in 3 cases in this series, routine thrombolysis and anticoagulation treatment should be given for 2 weeks before surgical intervention (39), which is helpful for resolution of intracranial hypertension while pulsatile tinnitus is eliminated. In some cases with mild venous pulsatile tinnitus, anticoagulation and thrombolysis treatment may reduce mural thrombus and blood viscosity while increasing relative diameter of the local vessel, which in turn can lead to reduced local pressure gradient and therefore reduced tinnitus loudness.

#### The Role of Stenting and Selection of Stents

Although anticoagulation and thrombolysis treatment may reduce venous pulsatile tinnitus in some patients, such treatment, however, seems to be less effective in severe cases. Stenting provides permanent dilation and support in stenotic venous sinuses and restores normal venous sinus anatomy, which can eliminate or reduce local pressure gradient and subsequently pulsatile tinnitus. This supported by the satisfying treatment results in the series reported in this article.

In our cases, stenosis was primarily present in the transverse-sigmoid sinus junction area. Because of the anatomic curves and size (>7 mm) of the DVS in this region, the Precise self-expanding stents of appropriate sizes are probably the most appropriate choice for best coupling to venous wall and complete coverage of the stenotic section, as well as sufficient compliance to avoid local high tension

that may cause headaches. The mesh hole size after stent expansion in this type of stent is greater than 3 mm and ensures compression of drainage from the supra-petrous, petrous, and Labbe's veins. There was no parenchymal venous infarction related to venous drainage blockage in our series, proofing the safety of these stents. Although postoperative mild dizziness and high pitched tinnitus were seen in 2 cases, the symptoms resolved after treatment with nimodipine and flunarizine, indicating no permanent mechanical blockage from stenting.

#### Perioperative Anticoagulation Indices and Duration

The inner lining in the stenotic section of venous sinus is usually less than smooth and can easily cause thrombosis that blocks blood flow. Thrombus in a venous sinus is usually red thrombus and responds to anticoagulation treatment. To minimize the risk of thrombosis in the stent, aspirin or similar agents should be used in addition to warfarin, which may also help reduce the chance of recurrence of stenosis. Based on our experiences in angioplasty and stenting for carotid artery stenosis and DVS thrombosis (40), patients in this series were routinely given heparin (30 µg/kg) before stenting procedures and maintained on warfarin and clopidogrel or aspirin from 1 week before to 6 months after the procedure. Results indicate that these measures were effective and reliable, with no acute or chronic thrombosis or restenosis within the stent.

#### CONCLUSION

Venous pulsatile tinnitus often results from stenosis or diverticulum in the DVS system, as well as abnormal drainage through mastoid emissary veins. Because of its insidious physical signs, it is sometimes thought to be subjective and misdiagnosed or undiagnosed, as in 2 cases in this series that were misdiagnosed with "depression." When routine anticoagulation treatment fails, these patients often are left to experience persistent (sometimes rather severe) tinnitus, which can lead to suicidal ideation in some patients. The results of angioplasty and stenting procedure for severe intractable pulsatile tinnitus caused by DSA confirmed DVS stenosis in this series of 46 cases provide evidence that DVS stenosis can be the cause of venous pulsatile tinnitus. The diagnosis and treatment protocol used in this series uses well-established techniques, is safe in experienced hands, and provides a new important and useful means in the management of venous pulsatile tinnitus.

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