

**INTERVENTIONAL DIAGNOSIS AND TREATMENT OF VASCULOGENIC PULSATILE TINNITUS**

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**Abstract**

**Objective** To retrospectively study clinical features and diagnostic imaging of vasculogenic pulsatile tinnitus, and the feasibility and efficacy of transvascular interventional treatment for this condition. **Methods** Data from 82 cases of arterial or venous pulsatile tinnitus were reviewed. DSA characteristics and possible pathophysiological mechanisms of pulsatile tinnitus in these cases were studied. Diagnoses in this group included intracranial arterovenous fistula (AVF) (n=3), spontaneous skull base dural AVF (n=16), traumatic carotid-cavernous sinus fistula (n=5), subclavian artery stenosis (n=2), internal carotid artery stenosis (n=3), intracranial arterial stenosis (n=1), kinked and/or elongated vertebrobasilar artery (n=2), venous sinus diverticulum (n=2), venous sinus stenosis on the dominant drainage side (n=46) and occipital sinus stenosis (n=2). Treatments included embolization and stenting using coils, NBCA glue, Balt balloons, self-expansion stents and intracranial micro-stents via either the femoral artery or femoral vein. **Results** Procedures were successful in all cases with no surgery-related complications. Tinnitus disappeared within 2 days after the procedure in all cases. Follow up duration was 5-36 months. Recurrence occurred in 4 cases of arterial tinnitus within 3 months following the initial procedure, which improved after revision embolization or symptom management. There was no recurrence in venous tinnitus cases following stent plastic or stent-coiling embolization treatments. **Conclusions** Endovascular intervention provides a new approach to the diagnosis and treatment of intractable pulsatile tinnitus. It is also effective in differentiating and studying other types of tinnitus.

**Key words:** Pulsatile tinnitus; AVF; cerebral artery; Venous sinus; Stenosis; Embolization; Stent vasculoplasty

**Introduction**

Epidemiology studies indicate that prevalence of tinnitus is 14.5% among those under 40 years of age and 17.5 - 35% among those over 40<sup>[1,2]</sup>, most of it being sensorineural in nature. Although prevalence of pulsatile tinnitus is merely 4%<sup>[3]</sup>, considering the size of the population, it carries a considerable negative impact on the wellbeing and quality of life among people. Pulsatile tinnitus that is synchronized to heart beat can be further characterized as “objective” and “subjective”. Objective pulsatile tinnitus is often seen in atherosclerotic stenosis of skull base carotid artery and vertebrobasilar ar-

tery (VBA), carotid-cavernous sinus arterovenous fistula (AVF), meningeal AVF, skull base aneurysms and arteries of aberrant courses. Due to lack of specific physical signs, subjective pulsatile tinnitus is sometimes mistaken as depression or other forms of “psychological disorders”. Patients with severe pulsatile tinnitus can suffer greatly when appropriate medical care is not available. With recent advances in cerebral vascular imaging, it has been recognized that some arterial stenosis, dural venous sinus stenosis, high rising jugular bulb and venous sinus diverticulum in the skull base area can be the cause of subjective pulsatile tinnitus<sup>[4-6]</sup>.

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The current study is a review of 82 cases of pulsatile tinnitus treated by the authors between January 2003 and January 2013, reporting their diagnoses, etiologies, interventions and treatment outcomes.

## Clinical data

### Patients

The series included 53 females and 29 males, aged 13 - 61 years (mean = 37.3 years). Symptom duration ranged 1 - 24 months. Auscultation revealed bruits synchronized to heart beat over the ipsilateral subclavian artery or near the mandibular angle in some cases. In most patients with skull base meningeal AVF, bruits were audible behind the ear, above the orbit or in the temporal area. Auscultation was usually negative in patients with intracranial arterial stenosis, kinking or elongation, or in those with venous sinus stenosis or diverticulum. Otolological consultation ruled out diseases of the outer or middle ear.

### Diagnostic imaging

Magnetic resonance imaging (MRI) and digital subtractive angiography (DSA) were performed in all 82 cases. T1 and T2 weighted MRI clearly revealed intracranial or skull base AVF draining into intracranial vessels in 15 cases, and kinked and elongated VBA compressing on the VIIIth cranial ver (vestibulocochlear nerve) near the superior olivary fossa and other neural structures close to the brainstem in 1 case. Computed tomography (CT) scan showed internal lamina erosion next to a sigmoid sinus diverticulum in 2 cases. DSA was performed to visualize the morphology and course of cervical and intracranial arterial and venous systems, and to determine blood flow distribution and circulation time, as well as the location and degree of arterial stenosis. DSA also provided information on the location of AVF and its supplying and draining vessels, the extent of kinking and elongation of VBA, characteristics of venous drainage and presence of stenosis, and the location, size and morphology of venous sinus diverticula. Diagnoses based on DSA in this group included intracranial AVF (n=3), spontaneous skull base dural AVF (n=16), traumatic carotid-cavernous sinus fistula (TCCF) (n=5), subclavian artery stenosis (n=2), internal carotid artery stenosis (n=3), intracranial arterial stenosis (n=1), VBA kink and/or elongation (n=2), venous sinus diverticulum (n=2), venous sinus stenosis on the dominant drainage side (n=46) and occipital sinus stenosis (n=2).

### Treatments

Three techniques were used to restore anatomical normalcy of cervical and cerebral vascular and neural structures via transvascular interventional approach under general anesthesia with endotracheal intubation. 1) In the 56 cases of arterial or venous stenosis, balloon dilation and stent were used to restore vascular structural normalcy, changing turbulent into laminar flows and thus eliminating tinnitus. 2) For the 24 patients with various AVF and venous diverticula, balloons, coils, NBCA glue and stents were used in various combinations for embolization and elimination of abnormal arterovenous communications to restore normal vascular anatomy. 3) To treat VBA kinking and abnormal elongation, micro-stents were used to reshape kinked vessels, creating a separation from the brainstem or nerve roots for a decompressing effect<sup>[7,8]</sup> (see example cases).

### Treatment outcomes and follow up

Procedures were successful in all 82 cases. There were no surgery-related complications. Pulsatile tinnitus was reported to have disappeared within the first 2 days following the procedure by all patients in this series. During the 5-36 months follow up, pulsatile tinnitus recurred in 3 cases of skull base spontaneous AVF, which disappeared again after revision embolization. In a case of intracranial arterial stenosis, mild tinnitus of different pitches from the original tinnitus appeared 3 months after stent treatment, which improved following treatment with vasodilating agents. There was no recurrence in cases of pulsatile tinnitus from venous system abnormalities following treatments with stents or coils.

## Discussion

### Clinical features and classification of tinnitus

Various types of tinnitus exist, with dozens of etiologies and variable loudness and pitch features. However, tinnitus is probably originated from within the auditory system including the tympanic membrane, cochlea, auditory nerve, auditory nuclei in the brainstem, thalamus and auditory cortex. Pathological changes involving any of these structures have the potential to generate abnormal auditory perceptions, or tinnitus. Due to the difficulties in determining the mechanisms and locations of tinnitus generation, clinical research on and treatment of tinnitus have been challenging for a long time, leading to dissatisfaction in both clinicians and patients. From our experiences in treating vascular disorders and reports in the literature, the authors classify tinnitus into the following four categories based upon its anatomical location and clinical features.

Otogenic tinnitus, which originates from acoustic/vibratory energy involving the outer, middle or inner ear or nearby arteries or veins in the neck region, sometimes as a result of bony structure defects such as those involving the jugular foramen. Such tinnitus can be constant or pulsatile and can be diagnosed via otological examination, electrophysiological tests and CT scans.

Vasculogenic tinnitus, which comes from vascular structures in the head, neck or chest, transmitted to the cochlea via the bone, blood vessel or blood flow. Anatomically, the internal carotid canal is located close to the cochlea. Elongated/kinked petrous carotid artery, especially when its canal is defected, can enter the middle ear or directly stimulate the cochlea to generate pulsatile tinnitus. The VBA can take an abnormal course and compress the auditory nerve and/or cochlear and superior olivary nuclei at a location near the superior olivary fossa or pontine. The rhythmic pressure on these structures may lead to abnormal auditory perception, i.e. pulsatile tinnitus. In pulsatile tinnitus from the venous system, most pathologies are located at the junction of the transverse and sigmoid sinuses on the dominant draining side (more than 80% in this series). Arachnoid granulations are present at this location, which may demonstrate inflammatory reactions to infection, autoimmune process or hormonal changes, leading to constrictive changes and stenosis. Blood flow from a stenotic section into a distal dilation or diverticulum tends to generate turbulent and noise that can be transmitted to the cochlea via the skull or mastoid, leading to perception of pulsatile tinnitus. Because the loudness of venous pulsatile tinnitus is related to the velocity of local blood flow, a clinical characteristic of such tinnitus is that it decreases or disappears when ipsilateral jugular vein is compressed which slows down blood flow inside the culprit venous sinus<sup>[10]</sup>.

Neurogenic tinnitus is probably the most common form of tinnitus with ill-understood etiologies, although ischemic or degenerative changes in the brainstem or auditory nerve are suspected causes. In recent years, it has been found that development of systemic or cerebral atherosclerotic changes in middle aged or older people, acute or chronic blood supply deficiencies, anoxic or demyelination changes in the auditory nucleus or nerves, involvement of the auditory system in posterior fossa neoplasms, and inflammation or adhesion of the arachnoid membrane can all result in electrophysiological disorders within the auditory system, leading to abnormal auditory perception or tinnitus<sup>[11]</sup>.

Systemic etiology related tinnitus may result from hyperthyroidism, renal deficiency, aminoglycoside toxicity, anemia and cardiac dysfunction that affect the auditory system, often leading to bilateral tinnitus, sometimes with hearing loss in severe cases.

### *Characteristics and mechanisms of vasculogenic tinnitus*

Vasculogenic tinnitus can be of arterial or venous origin and is characterized by pulsation synchronized to heart beat. Anatomically abnormal arteriovenous communication, stenosis and dilation cause alteration of streamline blood flow into turbulent flow, which turn the velocity energy with systolic/diastolic changes into sound energy with the same rhythmic variations. Such rhythmic sound energy is transmitted to the cochlea or auditory structures in the brainstem via the skull base bony structures, resulting in sound perception consistent with heart beat, or pulsatile tinnitus. Vasculogenic tinnitus can be caused by abnormalities of an artery or vein. Venous pulsatile tinnitus not only can be caused by structural abnormalities of a vein, but also by increased intracranial pressure. Tinnitus originating from non-arterial or venous structures is called non-vasculogenic tinnitus and can be subjective or objective. While subjective tinnitus is perceived only by the patient, objective tinnitus can be detectable by the examiner<sup>[13,14]</sup>.

From pathogenesis, skull base spontaneous AVF is thought to be secondary to skull base thrombotic phlebitis. Traumatic carotid-cavernous sinus fistula (TCCF) is often caused by breakthrough of carotid artery wall near the cavernous sinus by sharp skull base fracture end. Aggravated cervical or intracranial atherosclerotic changes can lead to local arterial stenosis. Venous diverticula are a result of bulging from defects in the venous wall. Venous sinus stenosis tend to be seen around the junction of transverse and sigmoid sinuses, probably related to the existences of abundant arachnoid granulations in this area which are prone to inflammation and subsequent constrictive changes as a result of infection or other insults. Despite the numerous possible causes, various kinds of pulsatile tinnitus share a common feature, i.e. the presence of a pressure differential across the stenosis or dilation or at the opening of a AVF, which leads to acceleration of blood flow and turbulent, thus the noise consistent with heart beat that can be transmitted to the cochlea to generate pulsatile tinnitus. Perhaps the only exception is a kinked/elongated VBA, which causes rhythmic irritation to the auditory system and subsequently abnormal auditory signals by direct mechanic compression on the auditory nerve root or brainstem, leading to a perception of pulsatile tinnitus<sup>[15-17]</sup>.

**Case 1** A 53 years old male presented with left side pulsatile tinnitus of 3 months. A bruit was detected near left mandibular angle. Doppler sonography showed stenosis of left common carotid artery, which was confirmed by DSA (>80% occlusion) (Figure 1a). The stenosis was dilated using a balloon under local anesthesia, followed by placement of a Protégé 9×40 mm stent. Follow up angiography indicated satisfactory correction of the stenosis

(Figures 1 b and c). Tinnitus immediately resolved following the procedure with disappearance of bruit. There was no recurrence at 10 months follow up.



Figure 1

**Case 2.** A 61 years old female reported bilateral pulsatile tinnitus of 18 months synchronized to heart beat. Failing treatments at multiple medical centers, the tinnitus gradually increased and caused the patient feeling anxious. Physical examination showed no focal neurological signs or bruits. Tinnitus was not reduced by carotid or jugular compression. Tinnitus was matched to a 125 Hz tone at 40 dB HL. Transcranial Doppler ultrasound indicated accelerated blood flow in left internal carotid artery, anterior cerebral artery, and mid cerebral artery (223 cm/s) with turbulent and bruits, while blood flow in right mid cerebral artery was normal. MRA showed severe atherosclerotic changes inside intracranial arteries and severe stenosis of intracranial left carotid artery with diminished distal filling (Figure 2a). DSA showed a 2 mm section of 70% occlusion in the posterior communicating branch of the left internal carotid artery with sharp “Z” shaped kinking and slight distal dilation (Figure 2 b). Under general anesthesia and guided by a wire, the stenosis was dilated using a 3×10 mm balloon followed by placement of a 4×20 mm Wingspan stent (Figure 2 c). Repeat DSA showed good correction of the stenosis and adequate distal filling (Figure 2d). Pulsatile tinnitus disappeared on the day following the procedure with no recurrence at 6 months. The tinnitus was believed to be from the turbulent caused by the stenosis and transmitted via the skull base bone to both cochleae.

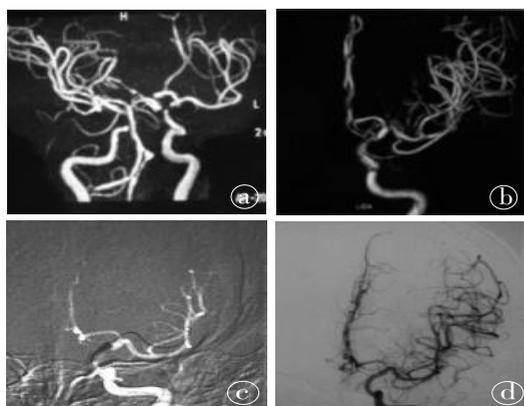


Figure 2

Most studies on vascular compression on the brainstem and cranial nerves are about trigeminal neuralgia and hemi-facial spasm. Micro-vascular decompression provides consistent benefits in such cases and has been widely accepted as an effect treatment. The etiologies in these patients are aberrant arterial branches from the anteroinferior cerebellar artery or superior cerebellar artery mechanically compressing on the Vth or VIIth cranial ne-

ve exit root zone. As the nerve sheath at this location is composed of oligodendric glial cells instead of Schwann's cells, the nerve is susceptible to demyelination under mechanical pressure. When the culprit vessel and the nerve are surgically separated, nerve function often improves, probably as a result of correction of causes of demyelination injury<sup>[18]</sup>. In the 2 cases of kinked/elongated VBA in this series, pulsatile tinnitus was likely a result of similar compression on the VIIIth cranial nerve as it enters the brainstem. The plastic effects of a stent on the kinked artery reduces its curvature and causes a slight anterior displacement, which leads to diminished pressure on the brainstem and auditory nerve and resolution of pulsatile tinnitus. Kinking and elongation of VBA can be from abnormal vascular development or atherosclerosis. There have few reports on similar cases, but the authors believe that the kinking and elongation in our cases are probably related to age-related increase of atherosclerotic changes on the arterial wall.

**Case 3** A 64 years old male complained of left ear tinnitus of 5 years for no particular causes. The tinnitus was composed of a pulsatile component synchronized to his heart and a low grade “electric buzzing”. Multiple treatments had failed and he was experiencing anxiety symptoms. Hearing was normal in both ears and auscultation was negative. MRA showed a kinked and elongated course of the left vertebral artery at the superior olivary fossa level, shifting anteromedially and compressing the side of the pontine and medulla and the auditory nerve (Figure 3 a). DSA showed the left vertebral artery taking a sharp anteromedial turn at the pontomedullary groove level, joining the basilar artery (Figure 3 b and c). He was diagnosed with vasculogenic tinnitus. Under general anesthesia, a 4×30 mm Solitare micro-stent was placed in the kinked intracranial vertebral artery for angioplasty (Figure 3 d). Postoperative DSA showed reduced left vertebral artery kinking (Figure 3 e and f). Patient reported resolution of tinnitus the next day following the procedure and no recurrence at 2 years.



Figure 5.

#### Diagnosis and differential diagnosis of vasculogenic tinnitus

From anatomy and pathophysiology, vasculogenic tin-

nitus can originate from intra- or extra-vascular sources, i.e. turbulent in a vessel or compression on the nerve or nucleus by an aberrant vessel. It is characterized by pulsation synchronized to heart beat, which can be used to differentiate from neurologic or other forms of tinnitus. Other features that can be used for differential diagnosis include relative low pitch of tinnitus (usually lower than 600 Hz) and DSA or CT angiography (CTA) data showing abnormal vascular structures.

Bruits are often audible in arterial pulsatile tinnitus, which can be diagnosed with CTA, magnetic resonance angiography (MRA) and DSA, and successfully treated using embolization or resection. In venous pulsatile tinnitus, however, despite the presumably consistent location and nature of the noise source, it is usually difficult to detect using a stethoscope. The usually ambiguous location of venous stenosis near the junction of the transverse and sigmoid sinuses makes diagnosis using CTA and MRA less than reliable. DSA is often needed which also allows measuring pressure differentials across the stenosis as an evidence to identify the source of pulsatile tinnitus. There have been some reports on treating venous pulsatile tinnitus associated with venous sinus diverticula using surgical reduction and transvascular stenting/coiling<sup>[19-23]</sup>, but many cases of pulsatile tinnitus related to venous sinus stenosis and their etiologies often go undiagnosed, leaving the patient suffering unnecessarily. Some of these patients are misdiagnosed and treated as having depression, leading to lost quality of life and productivity.

**Case 4** A 43 years old female presented with left side pulsatile tinnitus of 6 months, gradually increasing and causing anxiety. Although there was no audible bruit, tinnitus reduced when compressing left jugular vein. DSA indicated left side dominant drainage and showed diminished filling at the junction of the left transverse and sigmoid sinuses (Figure 4 a and b). Under general anesthesia, an 8F guiding catheter was placed in the left upper jugular vein, followed by venography using a Regigate catheter which confirmed the stenosis at the junction of transverse and sigmoid sinuses (Figure 4 c and d). Following placement of a guiding wire, the stenosis (Figure 4 f and g). Patient reported complete disappearance of pulsatile tinnitus upon recovery from anesthesia and no recurrence at 14 months follow up.

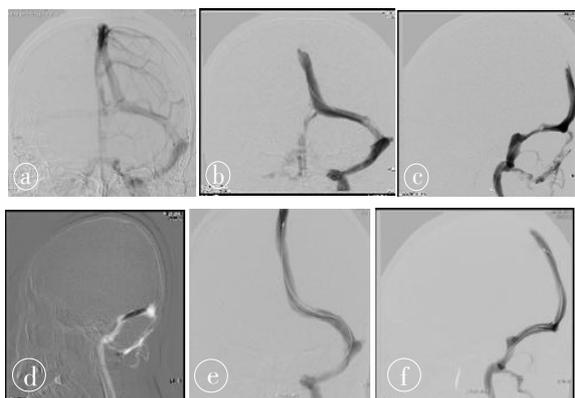


Figure 4

Cases in this series indicate a broad range of etiologies for vasculogenic tinnitus, clinically featured by pulsatile, low pitched tinnitus perception. Arterial etiologies can range from stenosis of the subclavian artery, carotid artery and artery branches around the Willi's circle, to AVF at various skull base locations. In most of these cases, a common features is the audible bruit synchronized to the heart at a surface location nearest to the culprit lesion. A bruit is often audible over the ipsilateral or contralateral eye or above the orbit in AVF in the cavernous sinus region, depending upon the direction of flow. Bruits from an AVF in the anterior or posterior fossa are usually heard above the orbit or in the temporal or retro-occipital area<sup>[24]</sup>. Compression of ipsilateral carotid artery often results in reduced tinnitus, unless it is from AVF in the VBA system. Next to abnormal development of sigmoid sinus and jugular thrombosis, stenosis of venous sinus secondary to inflammation and scarring of arachnoid granulations is also a common cause of venous pulsatile tinnitus (>80% of venous pulsatile tinnitus in this series). Other causes of venous pulsatile tinnitus include venous sinus diverticulum and enlargement of jugular bulb. The relatively quiet noise from venous sinuses makes it difficult to detect via auscultation and it tends to disappear upon compression of the jugular vein. Audible bruits consistent with heart beat and disappearance of tinnitus upon compression of cervical large vessels help differentiation from neurogenic and otogenic tinnitus and tinnitus of systemic etiologies.

**Case 5** A 27 years old male complained of pulsatile tinnitus in his head 2 months after a motor vehicle accident. A bruit was audible above left orbit and in the temporal area. DSA showed abnormal communication between the petrous carotid artery and the cavernous sinus, draining via bilateral jugular veins (Figures 5 a, b and c). A diagnosis of TCCF was made. Under general anesthesia, a 4.5 × 15 mm Jones stent was sent to the left petrous carotid artery and released. Post-operative imaging confirmed complete coverage of the fistula and normal flow through the carotid (Figure 5 d and e), with complete resolution of tinnitus. No recurrence was reported at 12 months follow up.

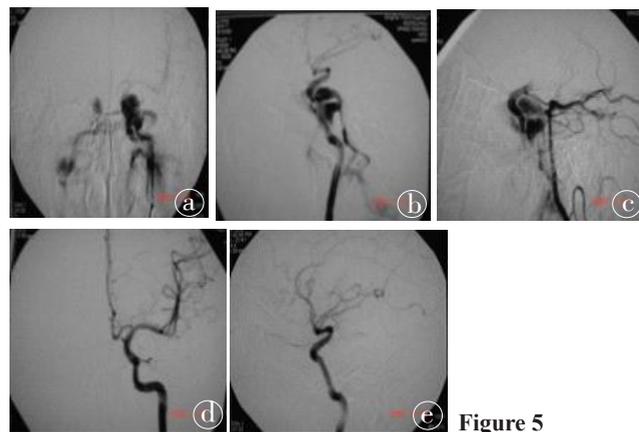
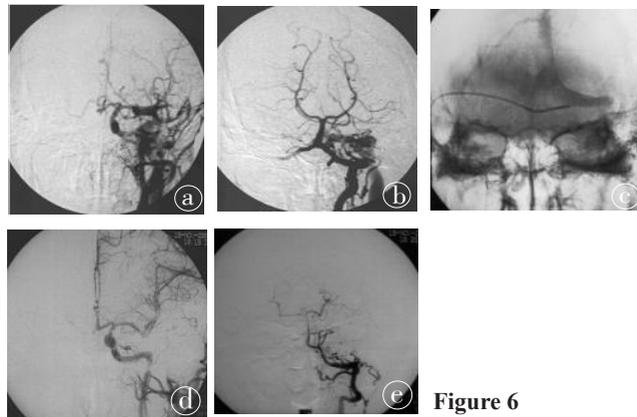


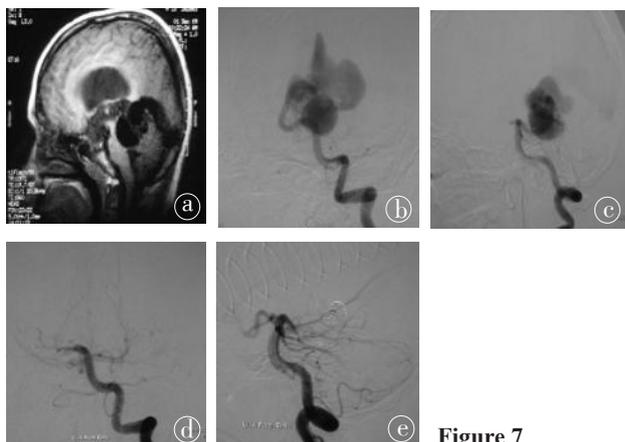
Figure 5

**Case 6** A 40 years old man presented with spontaneous pulsatile tinnitus of 1 year. Auscultation detected bruit behind the mastoid. DSA showed large left posterior fossa AVF supplied by left vertebral and external carotid branch arteries and draining into the left transverse sinus (Figures 6 a and b). The ipsilateral jugular vein was occluded with reverse flow from the sigmoid to the sagittal sinus with elevated pressure. Right side transverse and sigmoid sinuses provided main cerebral drainage. Under general anesthesia, a 4F catheter was placed in the left transverse sinus via right jugular vein (Figure 6 c). The left transverse sinus was obliterated using Cook coils and NBCA glue. Repeat angiography indicated complete closure of the fistula (Figure 6 d and e). Pulsatile tinnitus completely resolved after the procedure and remained recurrence free at 8 months.



**Figure 6**

**Case 7** A 41 years old male complained of pulsatile tinnitus with dizziness for 3 years. Symptoms had been worse for the past 3 months. Examination showed bruits over the occipital area. MRI revealed large flow void area in the brain and enlargement of the straight sinus and confluence of sinuses (Figure 7a). DSA demonstrated enlarged right posterior choroidal artery with direct drainage into the great cerebral vein, which also carried a few bulbar enlargements (Figure 7 b and c). Under general anesthesia, a  $3.5 \times 15$  mm Neuroform stent and a micro-catheter were positioned proximal to the fistula. With gradual expansion of the stent, three  $3 \times 10$  cm Microvention coils were placed, which, with support by the stent, blocked the supplying artery. Repeat DSA indicated complete fistula closure (Figure 7 d and e) with a patent ipsilateral posterior cerebral artery and no abnormal vein enlargement. Patient reported complete disappearance of pulsatile tinnitus on the day of the procedure, with no recurrence at 36 months.



**Figure 7**

### Choice of treatment and indications for interventional therapies

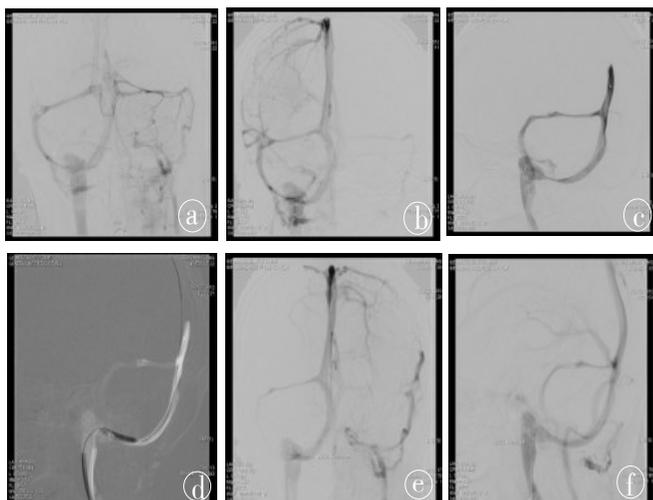
Most tinnitus cases improve with symptomatic treatments and psychological intervention, but a significant number of patients suffer from severe tinnitus with deteriorated quality of life. Since vascular structural abnormalities are present in most patients with pulsatile tinnitus, intracranial hemorrhage, stroke, demyelination changes and intracranial hypertension are some of the possible serious consequences if the condition is left untreated. Pulsatile tinnitus therefore must be taken seriously.

Because routine otology and pharmacological treatments show limited effects on pulsatile tinnitus and the underlying etiologies are often located in the skull base, when clearly diagnosed, craniotomy and lesion resection are treatment of choice in AVF of the anterior or posterior fossa. To separate and decompress the trigeminal or facial nerve, microscopic surgery via a retromastoid approach is usually chosen. Over the past 20 years, there have been some reports on efficacy of micro-vascular decompression of the auditory nerve for neurogenic tinnitus, although its long term effects have recently been found to be inconsistent and therefore the treatment has not been as widely accepted as for trigeminal neuralgia<sup>[25,26]</sup>. There has been so far no definite report on treating pulsatile tinnitus caused by aberrant VBA with micro-vascular decompression. The plastic stenting used in this series may represent an effective trial for this type of intractable tinnitus.

Because compression of jugular vein can reduce pulsatile tinnitus from venous sinus abnormalities, there have been reports in the literature on selectively ligating ipsilateral jugular vein as a treatment with some positive results<sup>[27-29]</sup>, although also with relatively high recurrence rate, probably from restoration of drainage through skull base veins and paravertebral veins following jugular closure. Without appropriate vessels, restricted venous drainage will lead to increased intracranial pressure and subsequent neurological damage. Recently, some have reported good results with retromastoid craniotomy and venous sinus diverticulum reduction for pulsatile tinnitus, although also with some risks of recurrence, probably related to uncorrected concurrent venous sinus stenosis.

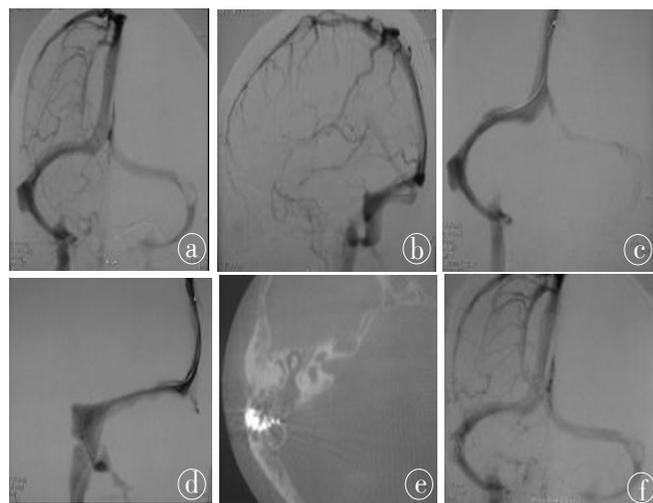
**Case 8** A 22 years old female complained of right side pulsatile tinnitus of 1 year that made her feeling anxious but could be reduced by compressing the right side of neck. Auscultation showed no bruits. DSA showed under-developed lateral sinus on both sides and direct drainage of sagittal sinus into the occipital sinus and right jugular vein through the glossopharyngeal nerve foramen. The occipital sinus was noticeably narrow through the glossopharyngeal nerve foramen (Figure 8a, b and c). Catheterization was performed under general anesthesia. Guided by a wire, dilation was completed using a 5

×20 mm balloon (Figure 8 d) followed by placement of a 6 ×30 mm Solitare stent. On follow up angiography, the occipital sinus stenosis was significantly corrected (Figure 8 e and f). Right side pulsatile tinnitus completely disappeared following the procedure, with no recurrence at 6 months.



**Figure 8**

**Case 9** A woman presented with right side pulsatile tinnitus of 1 year, increasing progressively and interfering with her sleep. A bruit synchronized to her heart beat was heard over right mastoid, which reduced when compressing on right jugular vein. DSA indicated dominant drainage on right and showed an 18 mm diverticulum in distal sigmoid sinus (Figure 9 a and b). Under general anesthesia, an 8F guiding catheter was placed in the upper jugular vein. Using a Regigate catheter, venography was repeated to confirm the diverticulum in the right sigmoid sinus (Figure 9 c and d). A 7×30 mm Precise stent was used to cover the affected sigmoid sinus section, and an Echlon 10 micro-catheter was passed into the diverticulum followed by placement of 6 Microvention coils to fill the diverticulum (Figure 9 e). Postoperative DSA showed no contrast filling in the diverticulum, indicating complete obliteration (Figure 9 f). CT showed coils in the bony defect areas in right mastoid. Patient reported resolution of right side pulsatile tinnitus upon waking up from anesthesia, together with disappearance of local bruit. There was no recurrence at 6 months follow up.



**Figure 9**

In comparison to surgical treatment, transvascular embolization and stenting provide advantages of being minimally invasive with lower complexity. This is the treatment of choice for the 82 cases in this series, which provides precise closure of AVF communication, elimination of abnormal pressure differentials, direct dilation of venous sinus stenosis, restoration of normal venous sinus anatomy and blood flow, and elimination of blood flow turbulent, ensuring a low rate of recurrence.

Transvascular interventional treatment for pulsatile tinnitus carries the same risks of complications as for other intracranial vascular diseases, including local hemorrhage and occlusion as major complications. The absence of iatrogenic complications in this series does not exclude the possibility of procedural damage to intracranial vascular structures or postoperative adverse hemodynamic changes causing blood vessel rupture, bleeding or occlusion of large vessels as potentially fatal complications associated with this type of intervention. Strict indications were adopted when determining treatment approaches in this series, including 1) the malformed vascular structure related to tinnitus was a risk of spontaneous bleeding; 2) the culprit lesion posed a threat to adjacent structures such as the eye, brainstem and cranial nerves, as indicted by imaging; 3) signs of cerebral ischemia or thrombosis related to the lesion were identified; 4) intractable pulsatile tinnitus failed to respond to routine therapies, showed signs of progression and caused anxiety/depression and severely affected patient's quality of life; and 5) there was no signs of intractable diabetes, hypertension or abnormal cardiac or renal functions or other difficult systemic diseases [30,31].

#### *Applicability and efficacy*

From this series, since the etiology of vasculogenic pulsatile tinnitus is usually located in the skull base or near brainstem or other important nerve structures, and because there lacks effective pharmacological treatments to correct the structural abnormality or tinnitus itself, surgical or interventional treatments are necessary treatment of choice in selected cases. Compared to craniotomy approaches, transvascular intervention has the advantages of being relatively simple and minimally invasive, while allowing reparative manipulation of skull base vessels and minimized risks of collateral damage to important nerve structures.

Because a local lesion causing blood flow turbulent is the key in generation of vasculogenic tinnitus, cerebral and cervical DSA is the most reliable means of diagnosis in such cases, providing opportunities to study the location, origin, morphology of the vascular lesion and if it is hemorrhagic or occlusive in nature, thus facilitating choi-

ce of treatment. In the current series, the responsible lesion originated from the arterial system in 32 cases and from the venous system in 50 cases. As for the nature of the lesion, it was occlusive in 56 cases, hemorrhagic in 24 cases, and compressive in 2 cases. The stenting and embolization techniques used in this series are consistent with those routinely used for cerebral aneurysms, artero-venous malformation and vascular stenosis over the past 20 years. The coils, NBCA glue, Balt balloons, dilating balloons, self-expansion and micro-stents used in these cases have been in clinical use for years with consistent results. These all probably provided important basis for the success in this series.

Our results indicate that transvascular intervention can effectively control and eliminate vascular lesions, provided that vigorous indications are followed, individual patient presentations carefully considered, and appropriate devices chosen. Iatrogenic complications can be minimized through individualized treatment planning, careful analysis of the lesion and hemodynamics of its arterial supplies and venous drainage, as well as diligent execution of transvascular therapy protocols.

In this series, follow ups via office visits, telephone and MRI or DSA when indicated revealed a recurrence rate of 15% (3/20) in AVE, 1.6% (1/60) in occlusive lesions and no recurrence in the 2 cases of vascular compression, indicating satisfactory short and mid-term efficacies. This study supports using transvascular interventional technique as an option for the diagnosis and treatment of intractable pulsatile tinnitus, as well as a tool in differentiating and studying other types of tinnitus.

Transvascular interventional approach is relatively simple and minimally invasive, and provides improved diagnosis and treatment outcomes. Along with its advances in treating cardiologic and neurologic disorders, it is expected that its acceptance and application in otology will increase in the near future, as an important supplement to existing diagnostic and therapeutic protocols for tinnitus and other conditions.

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