Computational Fluid Dynamics Simulation of Hemodynamic Alterations in Sigmoid Sinus Diverticulum and Ipsilateral Upstream Sinus Stenosis After Stent Implantation in Patients with Pulsatile Tinnitus

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■ OBJECTIVE: **To investigate the relationships between upstream venous sinus stenosis and pulsatile tinnitus (PT), and to assess the correlation with diverticulum growth and the effectiveness of stent implantation.**

■ METHODS: **Patient-specific geometric models were constructed using computed tomography venography images from a patient with PT, with sigmoid sinus diverticulum, and with upstream transverse sinus stenosis, in whom stenting of the upstream sinus stenosis alone achieved complete remission of PT. Computational fluid dynamics simulation based on this patient-specific geometry was performed using commercially available finite element software (ANSYS-14) to qualitatively and quantitatively compare the flow velocity, flow rate, velocity vector, pressure, vorticity, and wall shear stress on the affected side transverse and sigmoid sinuses, before and after stent implantation.**

■ RESULTS: **Stenting improved the flow direction and magnitude. After stenting, the flow pattern became smoother and more regular. High-speed blood flow at the level of the diverticulum neck was confined to a smaller area, and its direction changed from approximately perpendicular to the diverticular dome to the distal side of the diverticular neck. The diverticulum showed obvious flow reduction, with decreases of 80.7%, 68.7%, 96.1%, and 91.3% in peak velocity, inflow rate, pressure gradient, and peak vorticity, respectively. The abnormally low wall shear stress at the dome of diverticulum was eliminated.**

■ CONCLUSIONS: **Our findings strongly support a major role of diverticulum stenosis before in PT development and suggest that such stenosis is a causative factor of diverticulum growth. They also confirm the effectiveness of stent implantation for the treatment of PT.**

INTRODUCTION

Pulsatile tinnitus (PT) is characterized by the perception of a rhythmic sound, which is synchronous with the cardiac rhythm, with no acoustic stimulus. Although PT is a benign condition, it reduces patients' quality of life and can even lead to depression or suicide. It is often of vascular origin, and it is classified as objective or subjective, depending on whether it can be detected by an examiner. PT is relatively rare, accounting for only 4% of tinnitus cases.[1] Venous-origin pulsatile tinnitus (VPT), first described by Ott in 1977,[2] is more common than PT of arterial origin[3] and is often objective. The volume of VPT decreases with increasing venous pressure, such as that caused by compression of the ipsilateral jugular vein or turning of the head. VPT is often the result of a venous anomaly or variant, such as stenosis or diverticulum of the dural venous sinus (DVS) adjacent to the mastoid air cells, sigmoid plate dehiscence (SPD), a high jugular bulb, or aberrant mastoid emissary veins. An increasing number of cases with sigmoid sinus diverticulum (SSD) has been noted

Key words
- Computational fluid dynamics
- Pulsatile tinnitus
- Sigmoid sinus diverticulum
- Sinus stenosis
- Stent implantation

Abbreviations and Acronyms
CFD: Computational fluid dynamics
DVS: Dural venous sinus
PT: Pulsatile tinnitus
SPD: Sigmoid plate dehiscence
SSD: Sigmoid sinus diverticulum
VPT: Venous-origin pulsatile tinnitus
WSS: Wall shear stress

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recently, making this anomaly the most common recognizable cause of VPT[3,4]; it was previously considered to be an infrequent cause of PT.

Although upstream (before diverticulum) venous sinus stenosis and SSD frequently co-occur in patients with PT, the relationship between venous sinus stenosis and PT was not reported until 1997.[5] Thereafter, this condition has been studied intensively as a potential cause of PT. Endovascular treatment of VPT has emerged recently as a safe and effective option.[6-10] Stent implantation to remodel upstream stenosis has been demonstrated to effectively eliminate tinnitus,[8,11] suggesting a causal relationship between such stenosis and PT. Kefayati et al[12] hypothesized that PT was caused by localized turbulent downstream flow from the site of stenosis to the opening of the diverticulum. A retrospective review showed that about 70% of patients with PT have multiple vascular anomalies and variants on the affected side,[13] which may explain the persistent faint VPT in some patients who have undergone sigmoid sinus wall reconstruction alone, without the treatment of upstream venous sinus stenosis.

Although the possible existence of a link between VPT and turbulent blood flow in irregular or stenotic vascular structures around the petrous bone is widely accepted, the role of upstream stenosis in sound generation remains controversial[8,10-12,14,15] and evidence based on clinical treatment and computational fluid dynamics (CFD) is lacking. Upstream sinus stenosis has been proposed as a key causative factor of diverticularization.[11,15-18] However, to our knowledge, the relationship between these 2 factors has not been investigated. Motivated by such considerations, we hypothesized that turbulent blood flow from upstream venous sinus stenosis was a major cause of PT and was associated with SSD growth and that stent implantation could eliminate it effectively. We performed patient-specific CFD simulation to compare hemodynamic characteristics before and after stent deployment in upstream sinus stenosis under realistic conditions, to provide a theoretical basis for interventional therapy for this condition.

METHODS

Patient Information

This CFD simulation study was based on a patient with PT, with right SSD and SPD, and ipsilateral upstream transverse sinus stenosis, who was treated with a carotid stent in real-life. The 54-year-old patient presented with a 15-year history of severe and gradually worsening right-sided PT. Tinnitus was eliminated by compressing the ipsilateral internal jugular vein. Otherwise, the results of physical examination and otoscopic and audiometric evaluation were unremarkable. Computed tomography venography revealed a wide-necked diverticulum of the right sigmoid sinus, absence of the bony plate around the sigmoid sinus, and ipsilateral upstream transverse sinus stenosis. Digital subtraction angiography confirmed the diverticulum and upstream stenosis, and revealed a pressure gradient of 8.99 mm Hg across the stenosis. Thus, the decision of endovascular treatment of the venous anomaly was made, with the patient's consent. The implantation of an 8- × 40-mm carotid stent (Cordis Co., Miami Lakes, Florida, USA) in the stenotic segment, without addressing the SSD or SPD, achieved immediate and complete remission of PT and significant reduction of the pressure gradient across the upstream sinus stenosis from 8.99 to 1.92 mm Hg. Tinnitus did not recur during 8 months of outpatient follow-up. All medical data used in this study were obtained from our institution, and applied with the patient's consent.

Vascular Model Construction

Patient-specific raw computed tomography venography data consisted of 150 slices acquired before stent implantation and 173 slices acquired after implantation (512 × 512 pixels, 6.25-μm slice thickness), in Digital Imaging and Communications in Medicine format. Image files were imported into MIMICS software (version 10.0; Materialise, Belgium) for the construction of 3-dimensional models of the right DVS. Through image segmentation and 3-dimensional model creation, geometry models were obtained (Figure 1). After processing, surface geometries were saved in standard tessellation language format.

Computational Models and Simulations

The standard tessellation language format files were imported into the ANSYS workbench 14.0 (ANSYS, Inc., Cecil Township, Pennsylvania, USA) for processing. Before and after stent implantation analyses were conducted using typical tetrahedral meshes with 0.9 and 1.0 million elements, respectively. At this mesh density, the deviation in velocity and wall shear stress (WSS; the frictional force exerted by blood flow tangential to the vessel lumen) values relative to those generated with a finer mesh is fairly minor.[19,20] After meshing, ANSYS CFX 14.0 software (ANSYS, Inc.) was used for the simulation of hemodynamics. The venous wall was assumed to be rigid and a no-slip boundary condition was applied. The ignoring of wall motion when studying intracranial arterial blood flow was considered to be acceptable.[21] As the DVS is constrained between the brain and the skull and the vein is less elastic than the artery, we considered that the DVS would undergo little deformation, and thus that the neglect of wall elasticity was acceptable. The blood was assumed to be a laminar and incompressible Newtonian fluid with a density of 1050 kg/m^3 and viscosity of 0.0035 Pa/s. A steady flow condition was applied at the inlet boundary (due to microcirculation exit and far from the effects of breathing and cardiac pumping), and a pressure condition was used at the outlet boundary. Because patient-specific blood flow information was not available, a velocity of 13.0 cm/s, derived from phase-contrast magnetic resonance measurements from the same vessels in healthy subjects, was prescribed at the inlet[22] and 1333 Pa pressure was prescribed at the outlet boundary. The Navier-Stokes formula, solved with ANSYS CFX 14.0, was used as the governing equation for calculations. Based on the simulation results, several flow parameters were calculated to evaluate the effects of stent implantation on the affected-side transverse and sigmoid sinus hemodynamics qualitatively and quantitatively. These parameters included flow velocity, flow rate, velocity vector, pressure, vorticity, and WSS.

RESULTS

Figures 2–5 show the results of CFD simulation before and after stenting conditions. Before stenting, blood flow was faster in the stenosis and downstream region of the stenosis showed increased twisting and curling (Figure 2). The main site of recirculation was