LETTER TO THE EDITOR

To The Editor

Objective Audible Bruit in Idiopathic Intracranial Hypertension Resolved After Stenting

Keywords: Idiopathic intracranial hypertension, Venous sinus stent, Pulsatile tinnitus, Transverse venous sinus

Pulse-synchronous tinnitus, also referred as pulsatile tinnitus, can be disabling in some patients with idiopathic intracranial hypertension (IIH). It carries a wide differential diagnosis including atherosclerotic stenosis of skull base carotid arteries and vertebrobasilar system, carotid-cavernous sinus arteriovenous fistula (AVF), meningeal AVF, skull base aneurysms, and arteries of aberrant courses. More than half of patients with IIH report pulsatile or non-pulsatile tinnitus,[1] and this symptom can persist even after the resolution of the papilledema. Venous sinus stenting has been reported to improve pulsatile tinnitus in IIH.[2] A recent meta-analysis concluded that venous sinus stenting in medically refractory IIH has an excellent safety profile and resulted in improvement of IIH symptoms including pulsatile tinnitus.[3] We report a patient with IIH who presented with pulsatile tinnitus as the isolated symptom of IIH, who did not respond to medical therapy, but the symptom resolved after successful stenting of the right transverse venous sinus.

A 30-year-old female with high body mass index of 37 presented to an Otolaryngologist with a 4-month history of intense right-sided pulsatile tinnitus. She also noted that the tinnitus disappeared with compression of her ipsilateral anterior neck. She did not complain of headache, transient visual obscurations, diplopia, or any other visual impairment. Her past medical history was unremarkable, and she was not on any medications at the time of presentation. There was no prior history of trauma. Otological exam was normal. A CT angiogram of the head and neck arranged by the Otolaryngologist was unremarkable without any evidence of AVF or arterial abnormalities. She had a MR venogram of brain which demonstrated radiological signs of intracranial hypertension including right worse than left transverse sinus stenosis, tortuous and dilated optic nerve sheaths, flattening of posterior globes, and an empty sella (Figure 1). She was started on acetazolamide 500 mg twice daily based on the clinico-radiographic context.

She was assessed in our Neuro-Ophthalmology clinic 7 months from onset of the pulsatile tinnitus. By this time, she had been on the acetazolamide for 3 months and she had not reported any improvement in the disabling pulsatile tinnitus. This symptom was worse when she was laying down or while bending forwards. On examination, visual acuities were 20/20 in both eyes. Pupillary exam was normal without a relative afferent pupillary defect. Funduscopic exam revealed Frisen grade 2 papilledema bilaterally. Auscultation with a stethoscope revealed a loud, low-pitched, rumbling bruit in the right temporal region and over the right ear. Humphrey 24-2 Swedish interactive threshold algorithm visual field was normal. Lumbar puncture was performed after holding the acetazolamide for 5 days. Cerebrospinal fluid (CSF) opening pressure was elevated

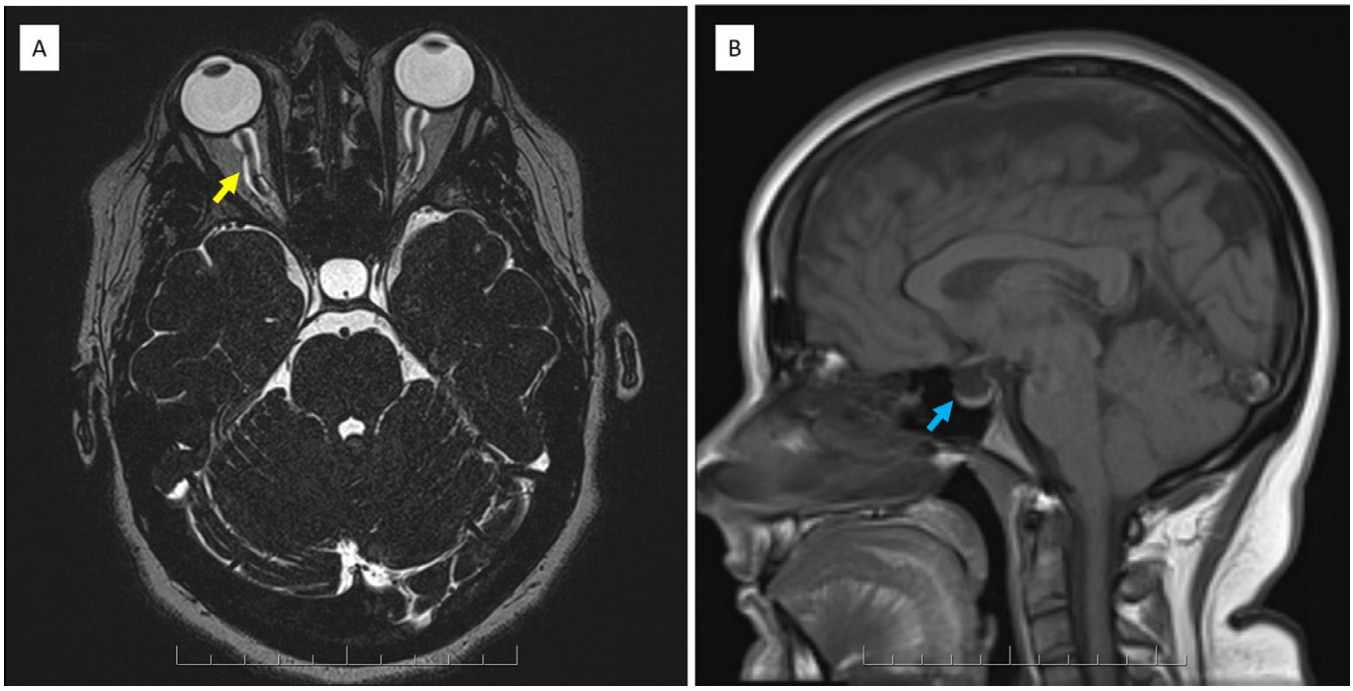

Figure 1: *Image A is an axial cut through the orbits of an MRI T2 Fast Imaging Employing Steady-state Acquisition (FIESTA) sequence showing bilateral optic nerve sheath tortuosity and dilatation (yellow arrow). Image B is a sagittal cut through the corpus callosum of a non-enhanced MRI T1 sequence demonstrating an empty sella (blue arrow).*

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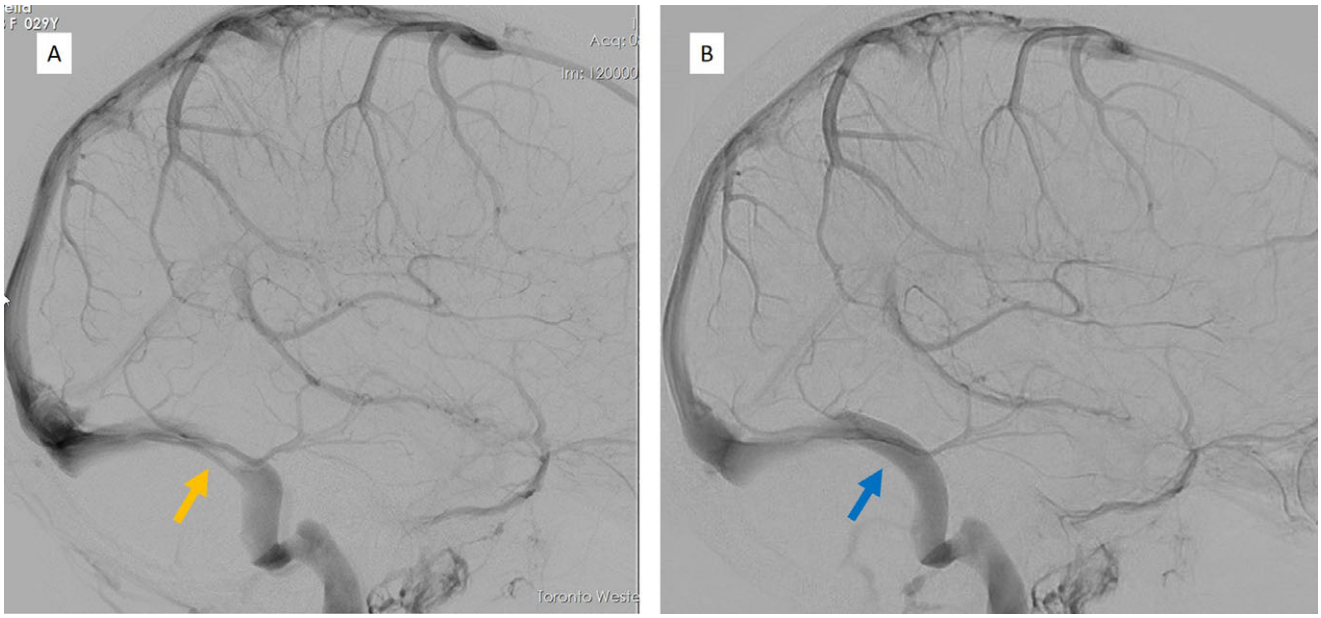

Figure 2: *Image A demonstrates cerebral angiography of the venous system in the sagittal plane showing the pre-stent stenosis of the right transverse sinus (yellow arrow). Image B displays post-stent sagittal image (blue arrow).*

at 30 cm water, and CSF constituents were normal. Paresthesia and fatigue precluded further escalation of the acetazolamide dose. As she was distressed by the ongoing tinnitus that did not improve with the acetazolamide, venous sinus stenting was proposed. Ten months after the onset of the pulsatile tinnitus, she had a cerebral angiogram which showed a pressure gradient of >15 mmHg. Following deployment of a stent in the right transverse-sigmoid sinus, the pressure gradient decreased to 3 mmHg (Figure 2). She reported improvement of the tinnitus immediately after the procedure. Lumbar puncture was repeated a month later which showed borderline CSF opening pressure of 23 cm water. Papilledema resolved and the bruit was not audible any more. Acetazolamide was weaned off. She subsequently lost 11 lbs (from a baseline weight of 230 lbs). Her most recent follow-up visit was 4 years after the venous sinus stenting. There was no recurrence of audible bruit or papilledema, and she is symptom free without the acetazolamide.

The diagnostic criteria for IIH were initially defined using the Dandy Criteria and further refined by the revised criteria in 2013.[4] Narrowing of the transverse venous sinus is an established radiographic finding in IIH.[5] The association between objective pulsatile tinnitus, ipsilateral narrowing of the transverse sinus, and resolution after stenting has been well documented.[2,6] Our case is interesting as she presented with pulsatile tinnitus as the only symptom of IIH (despite the presence of bilateral grade 2 papilledema), with a striking audible bruit that signified the severity of her symptom. Her neuroimaging revealed remarkable stenosis of the right transverse venous sinus on the symptomatic side to cause the bruit. Pulsatile tinnitus audible to others is known in IIH, but a bruit heard with a stethoscope could be an objective measure in patients who present with such incapacitating tinnitus. Her symptom as well as the bruit resolved after the stenting. There remains a great deal of uncertainty regarding the pathophysiology of IIH and whether the trans-venous flow has a role in inducing IIH or is it a consequence of a more global intracranial pressure imbalance.

We wanted to report this case to emphasize the importance of being mindful of the spectrum of presenting symptomatology in IIH. Psychiatric symptoms including anxiety, depression, and chronic fatigue are common in IIH.[7] Pulse-synchronous tinnitus can result in insomnia which could add further emotional distress in patients with IIH. The presence of an audible bruit in our case signifies the severity of the cerebral venous sinus narrowing. Audible bruit has been reported previously in patients with transverse venous sinus narrowing from other vascular causes.[6] The presence of pulse-synchronous tinnitus should always prompt the clinician to investigate for a cerebrovascular anomaly. Clues to help with identifying the type of pulsatile tinnitus (i.e. venous versus arterial) could be elucidated by determining if the tinnitus abates with ipsilateral compression of the jugular system as evidenced by our patient and others. Venous sinus stenting could be an alternate intervention for medically refractory IIH.[3,8]

Disclosures

The authors have no conflict of interest to disclose.

Statement of Authorship

DM: wrote and revised the manuscript. VP: critical review of the manuscript and provided analysis of imaging. AS: conceptualization and critical review of the manuscript.

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References

1. Wall M, Kupersmith MJ, Kieburtz KD, et al. The idiopathic intracranial hypertension treatment trial: clinical profile at baseline. JAMA Neurol. 2014;71:693–701. doi: 10.1001/jamaneurol.2014.133.
2. Quintas-Neves M, Freitas E, Amorim JM, Rocha J, Pinho J. Venous sinus stenosis causing isolated pulsatile tinnitus. Can J Neurol Sci. 2019;46:591–92. doi: 10.1017/cjn.2019.73.
3. Nicholson P, Brinjikji W, Radovanovic I, et al. Venous sinus stenting for idiopathic intracranial hypertension: a systematic review and meta-analysis. J Neurointerv Surg. 2019;11:380–85. doi: 10.1136/neurintsurg-2018-014172.
4. Friedman DI, Liu GT, Digre KB. Revised diagnostic criteria for the pseudotumor cerebri syndrome in adults and children. Neurology. 2013;81:1159–65. doi: 10.1212/WNL.0b013e3182a55f17.
5. Farb RI, Vanek I, Scott JN, Mikulis DJ, Willinsky RA, Tomlinson G, terBrugge KG. Idiopathic intracranial hypertension: the prevalence and morphology of sinovenous stenosis. Neurology. 2003;60:1418–24. doi: 10.1212/01.wnl.0000066683.34093.e2.
6. Russell EJ, De Michaelis BJ, Wiet R, Meyer J. Objective pulse-synchronous "Essential" tinnitus due to narrowing of the transverse dural venous sinus. Int Tinnitus J. 1995;1:127–37.
7. Puustinen T, Tervonen J, Avellan C, et al. Psychiatric disorders are a common prognostic marker for worse outcome in patients with idiopathic intracranial hypertension. Clin Neurol Neurosurg. 2019;186:105527. doi: 10.1016/j.clineuro.2019.105527.
8. Satti SR, Leishangthem L, Chaudry MI. Meta-analysis of CSF diversion procedures and dural venous sinus stenting in the setting of medically refractory idiopathic intracranial hypertension. Am J Neuroradiol. 2015;36:1899–904. doi: 10.3174/ajnr.A4377.

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