

Intracranially protruded bilateral posterior and superior SCCs with multiple dehiscences in a patient with positional vertigo: CT and MR imaging findings and review of literature

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Abstract

We report a rare case of intracranially protruded posterior and superior semicircular canals beyond the margins of temporal bone with bony roof dehiscence in bilateral posterior and left superior semicircular canals in a patient with benign paroxysmal positional vertigo (BPPV).

Key words: Intracranial protrusion; magnetic resonance imaging and computed tomography; multiple dehiscences; semicircular canal

Introduction

Semicircular canal (SCC) dehiscences are uncommon abnormalities of the temporal bone and involvement of posterior semicircular canal (PSCC) and/or multiple dehiscences is even rare.^[1] It was found in current review of literature that magnetic resonance imaging (MRI) and computed tomography (CT) scan findings of intracranially protruded semicircular canals (SCCs) and multiple dehiscences have not been reported. We report a rare case of intracranially protruding PSCC and SSCC beyond the margins of temporal bone with bony roof dehiscence in bilateral posterior and left superior SCCs in a patient with benign paroxysmal positional vertigo (BPPV).

Case Report

A 22-year-old male patient with clinically diagnosed BPPV came to the radiology department for MRI evaluation of posterior fossa. Clinically positive Tullio phenomenon (sound-induced vertigo or nystagmus or both; can be attributed to both Superior and Posterior semicircular canal dehiscences) and Hennebert sign (nystagmus following external ear pressure) were noted. Typical type of nystagmus related to PSCC was also noted with Dix-Hallpike maneuver on both sides. Since 2 years, the patient had been having increase in vertigo, predominantly on the left side, and was on medication intermittently. No hearing loss was noted. No history of trauma was also noted.

Patient had undergone routine MRI brain examination earlier elsewhere which was reported as normal. Otorhinologist at our hospital suggested initial MRI examination to rule out posterior fossa neurovascular and inner ear evaluation. MRI (1.5T MRI scanner, MAGNETOM Essenza, Siemens Healthcare, Muenchen, Germany) examination [using highly T2-weighted 3D sequence like constructive interference in steady state (CISS) or fast imaging employing steady-state acquisition (FIESTA)]

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10.4103/0971-3026.143904

revealed protrusion of PSCC and SSCC on both sides and findings suggestive of bony roof defects in bilateral posterior and right superior SCCs. The findings were again confirmed on high-resolution computed tomography (128 slice CT scanner, Optima 660, GE Healthcare, Wisconsin, USA) (HRCT) scan of temporal bones.

On both MRI and CT scan axial and oblique sagittal and coronal images, protrusion of PSCCs was seen behind the oblique temporal plate of posterior fossa and protrusion of SSCCs was above the horizontal plane of temporal bone on both sides [Figures 1A-F and 2A-C, respectively]. Bony roof defect was seen in the postero-superior aspect of both posterior and left superior SCCs. Both PSCC bony roof defects were demonstrated on axial images [Figure 1A-D] and the defect of SSCC was seen in axial and oblique planes [Figure 2]. Defect in PSCC was also shown by reformatted images in the coronal plane [Figure 1E]. These defects are very difficult to appreciate on coronal planes, [Figure 3] especially the postero-superior defect of SSCC. Coronal planes can better demonstrate most superior defects of SSCCs and most lateral defects of lateral SCCs.

The plane parallel to PSCC was perpendicular to SSCC (similar to Stenver's plane), and clearly demonstrated and confirmed the defects in both SCCs than the plane parallel to SSCC [Figure 2A].

No evidence of stenosis or filling defects was seen in SCCs to account for cupulolithiasis and canalithiasis.

No evidence of bony defect was seen in relation to left superior and both lateral SCCs. Vestibule and cochlea were

normal on both sides. VII-VIII nerve complex, internal auditory canal, and posterior fossa vascular channels did not reveal any abnormality.

Patient refused surgery due to its complexity and associated complications. Thus, otorhinologists suggested him to continue medical therapy. At 6 months follow-up, the patient's vertigo is under control with medical management.

Discussion

Vertigo is caused by many conditions, either due to the failure of end organs or the failure of vestibular nerves or vestibular connection to the brain stem and cerebellum. BPPV is the most common disorder of the peripheral vestibular system. It causes short episodes of vertigo when the patient moves his/her head in certain directions. BPPV rarely demonstrates abnormal imaging findings.^[2] It may account for 20-40% of all vertigo patients. PSCC is involved most commonly than lateral and superior SCCs. Bilateral or multicanal involvement can be noted in some patients. BPPV is most commonly idiopathic and trauma is the most common cause in the secondary form. Incidence of BPPV recurrence varies from 7 to 50%.^[3] Even though SCC defect can present with BPPV,^[4] however, BPPV due to specific SCC defect has not been described in literature.

Positional vertigo in our patient may be attributed to the defect in PSCCs on both sides with typical nystagmus on Dix-Hallpike maneuver.

Recurrent and persistent BPPV (for both primary and secondary) is most commonly idiopathic; however, trauma

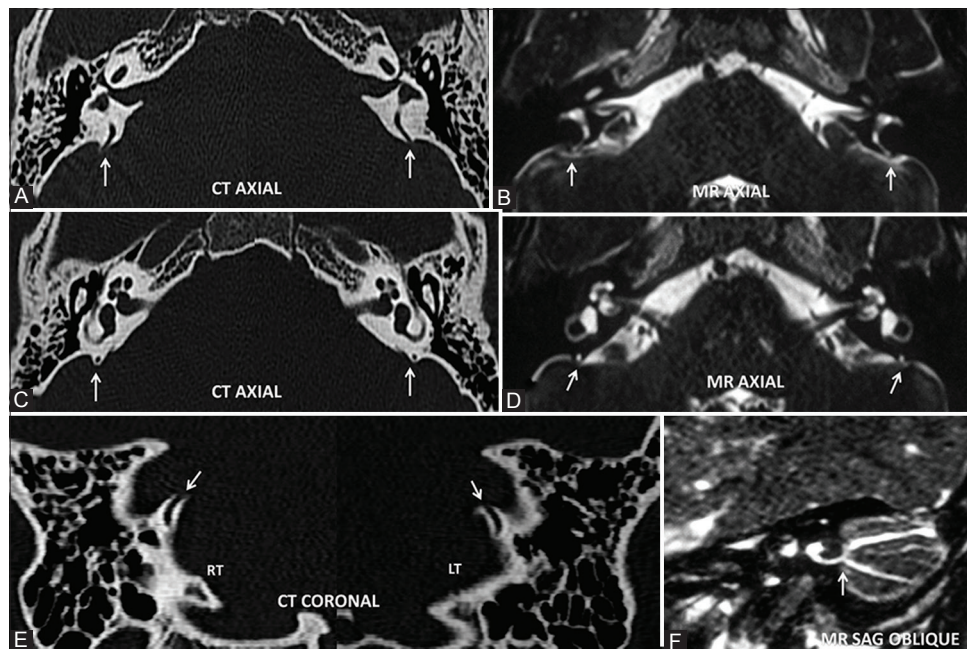


Figure 1 (A-F): HRCT and MR (CISS sequence) correlation, showing postero-superior bony roof defect in PSCC on both sides (arrows). Both protruded PSCCs were seen beyond the oblique temporal plate of posterior fossa

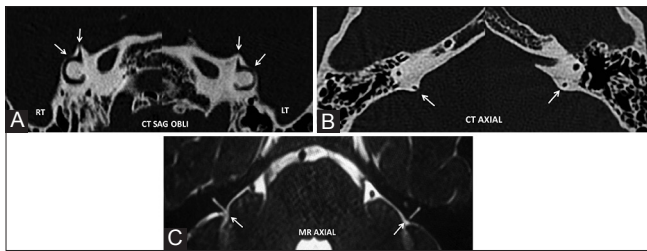


Figure 2 (A-C): HRCT reformatted images showing bony roof defect in SSCC on the right side and PSCC on both sides, with both protruded SSCCs above the horizontal plate of tegmen temporal bone (arrows in A). HRCT (B) and MR CISS sequence (C) show intracranially protruded both SSCCs with bony roof defect on the right side (arrows)

is more common in secondary causes. PSCC involvement is most common in recurrent BPPV cases.^[5,6]

Even though recurrent and persistent BPPV can be successfully treated with frequently repeated canalith repositioning procedures (CRPs), it was unsuccessful in our patient.

On MRI, intractable BPPV cases may show stenosis and filling defects in SCCs, which may indicate cupulolithiasis and canalithiasis.^[7] There was no filling defect or stenosis seen in our patient [Figures 1B, D, F, and 2C].

Coronal reformatted images are usually sufficient for the radiologic diagnosis of SSCC defects. Reformatted images of the temporal bone in the 45° oblique plane, perpendicular and parallel to SCC, i.e. Stenvers and Pöschl planes, respectively, may be reserved for difficult or equivocal cases.^[8,6] In our case, it was very difficult to demonstrate postero-superior defect in right SSCC on the coronal plane, for which we used Pöschl and Stenvers plane reformatted images. Axial and sagittal reformatted images may be adequate for evaluation of PSCC defects; however, reformatted images in the plane parallel to PSCC will increase the diagnostic confidence. The plane parallel to PSCC can also demonstrate SSCC defects [Figure 2A] compared to the plane parallel to SSCC revealing PSCC defects.

Many times, patients with vertigo will undergo either routine CT or MRI examination alone. Thus, radiologist and/or otorhinologist should suspect bony roof defect if intracranially protruded SCCs are seen on routine MRI and CT examination and should request for detailed 3D MR/HRCT examination with reformatted images to confirm the same.

El Hadi *et al.*^[9] reported higher incidence of bony dehiscence in intracranially protruded SCCs than in non-dehiscent cases (92.3% vs. 30%, respectively). According to the table in their report, superior and posterior (i.e. multiple) SCC defect was noted in 15.3% of all cases of dehiscence and in

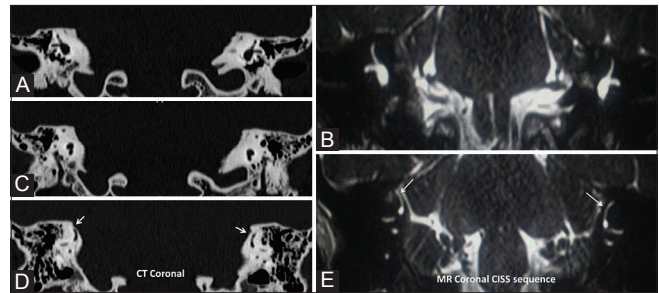


Figure 3 (A-E): CT coronal and MR CISS coronal images showing thinned bone overlying the postero-superior aspect of right SSCC compared to the left side. Small defects in SCCs may be difficult to detect on true coronal plane; thus, we need special planes like Stenvers plane for PSCC and Pöschl for SSCC

50% of those with bilateral intracranially protruded PSCCs and SSCCs. No isolated cases of PSCC defect and protrusion of PSCC were mentioned.

Embryologically, otic capsule, including all SCCs, develops from cartilage model and ossifies completely by 22-24 weeks of gestation. Subsequent remodeling after birth occurs to a lesser extent, but internal auditory canal, vestibular aqueduct, mastoid, and external auditory canal continue to grow after birth.^[10] Thus, presence of intracranially protruded SCCs may represent either abnormal ossification or development of temporal bone, and exposes the SCCs to pressure effects from intracranial structures.^[11]

Even though Manzari *et al.* have reported a case of multiple dehiscence of SCC, they have not mentioned whether the SCCs were intracranially protruded or not. Also, their CT scan images represent reformatted sections in Pöschl plane and the plane parallel to PSCC for demonstration of dehiscence in superior and posterior SCCs, respectively, but not sagittal section. Thus, our case, along with Manzari *et al.*'s case, will give further scientific information in relation to intracranially protruded SCCs, multiple dehiscences, and the radiological features.

Conclusion

The MRI and CT scan findings of the present case will increase the diagnostic confidence of both radiologists and otorhinologists in evaluating SCC dehiscence.

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Cite this article as: Kundaragi NG, Mudali S, Karpagam B, Priya R. Intracranially protruded bilateral posterior and superior SCCs with multiple dehiscences in a patient with positional vertigo: CT and MR imaging findings and review of literature. *Indian J Radiol Imaging* 2014;24:406-9.

Source of Support: Nil, **Conflict of Interest:** None declared.