Diagnostic role of MDCT in the identification of Minor's Syndrome

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Purpose

The "superior semicircular canal dehiscence syndrome" (SCDS) has been recently considered as a possible morphologic cause of vertigo\(^1\). Increasingly, this disorder is becoming known, both inside and outside the medical profession, as Minor's syndrome.

The surgeon Lloyd B. Minor was the first one who talked about it. In 1998 he described a new clinical entity, characterized by an alteration of the superior semicircular canal (SSC), that could explain "Tullio's phenomenon" (dizziness induced by threshold sounds) and/or Hennert's phenomenon (vestibular symptoms induced by the raising of nonacoustic pressures, reaching the external acoustic duct)\(^2\).

This structural alteration of the inner ear consists of a more or less extended defect of the external bony wall of the SSC. This condition can be demonstrated by a dedicated high resolution radiological study with thin-slice collimations. SCDS is able to cause the "third mobile window" effect\(^2\): mechanical (acoustic or pressure) stimulations, activating the cochlea, may induce endolymphatic fluxes also in the SSC, causing vertiginous symptoms.

The aim of this study was to assess imaging findings of superior semicircular dehiscence on computed tomography and to evaluate incidence of superior semicircular canal dehiscence in patients presenting with vertigo and sensorineuronal hearing loss.

Computed tomography was performed in 121 patients presenting either with vertigo (\(n=28\); 4 of these patients suffered also from sensorineuronal hearing loss), other symptoms related to the inner ear, such as hearing loss or tinnitus (\(n=33\)) or symptoms unrelated to the labyrinth (\(n=46\)). All images were reviewed for presence of dehiscence of the bone, overlying the semicircular canals. 82 patients had superior semicircular canal dehiscence.

Of these patients, 83% presented with vertigo, 10% with hearing loss or tinnitus and the remaining 7% with symptoms unrelated to the inner ear.

Significant prevalence of vertigo in these patients suggests that superior semicircular canal dehiscence can cause vertigo.
Methods and Materials

121 patients with a normal drum suffering from isolated unilateral or bilateral conductive or mixed hearing loss evaluated by an audiometry were included in a prospective radiological study conducted from November 2007 to January 2010. 8 patients were found to have a unilateral and four a bilateral SCD, and underwent further etiological, clinical, and audiological evaluation.

A 10-mm collimated computerized tomography with high resolution and multidetector (16 x 0.625 mm) with axial acquisition was carried out in all the patients. Chosen pitch (table advance/collimation = moving forward/thickness of X-ray beam) was 0.5, meaning an overlapping type of 50%. Chosen constants were 140 KV at 400 mA/s. With these criteria, spatial resolution can be estimated at 0.5 mm.

In our series, SCDS was found at all ages (39-74 years) confirming the data in the literature.

We found no history of trauma, an etiology which is mentioned by some authors.

We consider a congenital origin to be highly probable.

The fact that SCD is frequently bilateral (4 patients out of 8), and associated with either a dehiscence of the tegmen tympani, supports the theory of the congenital nature of the disease.

According to some authors the tegmen tympani is formed at the fetal stage from the lateral expansion of the labyrinthine capsule, and is of ectoblastic origin which could explain the associated dehiscence of the two structures.

The SCD may arise from failure of postnatal bone development, and later by disruption or erosion of the abnormally thin layer of bone over the SCD caused by the weight and pressure of the temporal bone and/or by intratympanic depression. No patient in our group, however, presented with any clinical or radiological signs of an increase in intracranial pressure.

The third labyrinthine window theory of Minor is the most common theory advanced to explain the conductive or mixed hearing loss encountered in patients with a SCDS. SCD may induce decreases in the sensitivity to low-frequency air-conduction of sound and induce increases in the sensitivity to low-frequency (<2 kHz) bone conduction of sound. The combination of these two alterations in sensitivity can result in a significant air bone gap. With air-conducted sound, the dehiscence may act as a shunt, reducing the fluid-displacement wave that reaches the cochlea and reducing the stimulus that activates the hearing mechanism.
The effect of bone-conducted sound on the inner ear is complicated, and includes the summation of multiple stimulus pathways. Through clinical, experimental and theoretical investigations of the effect of SCD on hearing mechanisms, the validity of the third window theory has been proved and it has been shown that, in general, windows on the vestibule side of the inner ear are associated with bone conduction hypersensitivity and air-conduction losses and that windows on the cochlear side (e.g., round window fistulae) or an enlarged cochlear aqueduct have little effect on hearing function. For others authors, an improvement in bone conduction could be due to the fact that the SCD creates a communication channel between the cranial cavity and the labyrinth, thereby allowing better transmission of the acoustic waves through the fluid.

Most patients with SCDS have vestibular symptoms including eye movements evoked by stimuli such as loud tones, Valsalva maneuvers, or pressure in the external auditory canal. Some patients also have a brisk motion of the head in the plane of the SC in response to loud tones. Chronic disequilibrium, is also a common complaint. In our series, however, only two patients had associated non-disabling vestibular symptoms, as they were all referred to the radiologist for investigation of isolated conductive or mixed hearing loss with a normal eardrum.
Fig. 0: CT oblique coronal section of the left inner ear shows a dehiscence of superior semicircular canal in a 52 years old man presenting with vertigo.

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**Fig. 0:** Oblique coronal section image at different level of the left superior semicircular canal demonstrates a defect of the bone overlying the posterior branch of the superior semicircular canal.

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**Fig. 0:** In this CT oblique coronal image we can observe a pathological communication between the middle cranial fossa and the posterior branch of the superior semicircular canal.

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Results

There were six women and four men with a normal eardrum associated with a unilateral or bilateral SCDS and of age ranging from 39-74 years (mean age 60 years ± 9.47 years).

The eight patients who were found to have a SCD explaining their conductive or mixed hearing loss underwent a retrospective analysis of their medical records: five patients were found to have non-disabling vestibular symptoms, two patient had the sensation of going backwards while driving a car, and one suffered from dizziness (imbalance).

SCDS has various patterns of clinical and semeiological expressions. It is possible to distinguish three different clinical conditions by: presence of only vestibular symptoms, presence of only cochlear symptoms, presence of both vestibular and cochlear symptoms.

The new clinical-nosological panorama of the vestibular pathology, opened by Minor's observations, has been possible thanks to recent technological developments of imaging machines. Multidetector computed tomography (MDCT), thanks to its high spatial resolution, allowed us to obtain an anatomical demonstration "in vivo" of the labyrinthic capsule, evidencing in detail the existence of a bone dehiscence of SSC or its reduced thickness.

Thanks to different reconstructions on the different spatial planes and to MIP reconstructions, we are able to demonstrate the anatomy of the bony labyrinth.

CT scan may be useful in the acquisition of anatomical elements used for the neuronavigation and in the evaluation or planning of a surgical reconstruction of the SSC.
Conclusion

SCDS is a clinical radiological entity which can induce both vestibular loss and hearing loss and this latter may be the only symptom. An underlying developmental or congenital abnormality probably leads to the SCD. We did not find any clear correlation between symptoms and the size of SCD. The mechanisms causing both the vestibular and auditory manifestations are still under discussion, but the creation of a “third mobile window” in the inner ear is the most commonly accepted theory. Isolated conductive or mixed hearing loss due to SCD is relatively frequent justifying in our opinion to perform a HRCT in every patient presenting a conductive or mixed isolated hearing loss with a normal drum before operation. Surgical repair of SCD performed through a middle fossa approach is not recommended in our opinion in the case of an isolated conductive or mixed hearing loss.
References


