ORIGINAL ARTICLE

Effect of Semicircular Canal Dehiscence on Contralateral Canal Bone Thickness∗

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KEYWORDS

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Abstract

Objectives: Our objective was to determine if the existence of dehiscence in the superior or posterior semicircular canal was associated with the thinning of the bone roof in the rest of the vertical canals (superior or posterior).

Methods: The thickness of the superior and posterior semicircular canals contralateral to a dehiscence was studied using computerized tomography and compared statistically.

Results: When a superior semicircular canal had a dehiscence, the contralateral canal showed a significant mean decrease in its thickness of 0.5 mm (SD: 0.3 mm). This was not the case if the dehiscence was in the posterior semicircular canal, where the thickness of 2.1 mm remained unchanged (SD: 1.2 mm; P= .49).

When a posterior semicircular canal showed dehiscence, no significant thinning was shown in the superior semicircular (1 mm; SD: 0.4) or in the posterior contralateral (1.3 mm; SD: 0.3) canals.

Conclusion: The existence of a dehiscence in the superior semicircular canal is associated with bone thinning in the canal on the opposite side, but not with the posterior semicircular canal. In contrast, if the dehiscence is in the posterior semicircular canal, contralateral and superior canal thickness is not modified.

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Influencia de la existencia de una dehiscencia en un canal semicircular en el espesor óseo de los canales contralaterales

Resumen

Objetivos: Determinar si la existencia de un canal semicircular superior o posterior dehisciente se asocia con el adelgazamiento de la cubierta ósea en el resto de los canales verticales (superior o posterior).

Métodos: Se estudia mediante tomografía computarizada y se compara estadísticamente el espesor de los canales semicirculares superiores y posteriores contralaterales a una dehiscencia.

Resultados: Cuando un canal semicircular superior presentaba una dehiscencia, el canal contralateral mostraba un adelgazamiento significativo de su espesor con una media de 0,5 mm (DE: 0,3 mm). No sucede lo mismo con los posteriores que no modifican su grosor de 2,1 mm (DE: 1,2 mm; p = 0,49).

Cuando un canal semicircular posterior presentaba dehiscencia no se observó adelgazamiento significativo en los canales semicirculares superiores 1 mm (DE: 0,4), ni en el posterior contralateral 1,3 mm (DE: 0,3).

Conclusión: La existencia de una dehiscencia del canal semicircular superior se asocia al adelgazamiento del hueso del canal del lado contrario, pero no de los canales semicirculares posteriores. No ocurre lo mismo si la dehiscencia es del canal semicircular posterior, que no altera de manera significativa el espesor del canal del lado contralateral ni en los superiores.

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Introduction

At present, the aetiology of semicircular canal dehiscence is not yet fully known, although various theories have been postulated. Tsunoda and Terasaki suggested that dehiscence is due to a defect in embryonic development by malposition of the primitive otocyst. Crovetto et al. linked it with a defect in bone reorganisation of the channel during the prenatal period. Zhou et al. linked it to a canal defect due to abnormal development of the middle cranial fossa. Carey et al. and Teixido et al. linked it to a postnatal alteration of the top of the canal and Mikulec et al. to a congenital predisposition. Other hypotheses have suggested that dehiscence occurs by a rupture of the coverage of extremely fine canals caused by cranial trauma, a sudden increase in intracranial pressure or erosion caused by the weight and pressure of the temporal lobe. Recently, Nadir et al. investigated whether superior canal dehiscence was congenital or acquired. After observing that prevalence increased with age, they suggested that the most common origin was acquired, rather than congenital.

We performed a study of patients with radiological dehiscence in a vertical semicircular canal (superior or posterior). We analysed the mean thickness of the cover of the other vertical canals to verify whether a pathological condition of the former determined variations in the mean thickness of the latter. We took normal values as reference and established their correlation.

Material and Methods

The study was conducted at 3 healthcare centres: Hospital de Basurto, Hospital de Cruces and Hospital General de la Defensa, in Zaragoza, between September 2007 and March 2008. The study was approved by the Ethics Committees of the respective centres according to the guidelines of the Helsinki Declaration of 1964. All patients were informed about the study and signed the relevant informed consent forms.

The study was conducted on consecutive patients, selected from those attending the radiology departments of these hospitals to undergo a computed tomography (CT) scan of the temporal bones for various reasons (hearing loss, facial paralysis, vertigo, tinnitus, chronic otitis media). CT scans were reviewed by 3 radiologists with over 10 years of experience in neuroradiology, one for each hospital.

We excluded patients with any type of anatomical alteration of the labyrinth. We also excluded all those patients whose CT scans did not have sufficient quality to determine the parameters under study.

All studies were performed with helical, multi-slice CT devices, obtaining images in the axial plane and with the neck in hyperextension in order to avoid direct radiation on the ocular lens. Subsequently, we conducted coronal reconstructions and in the plane of the superior semicircular canal (SSC) of each ear and in axial sections for the posterior semicircular canals (PSC). The "raw data" were reconstructed using a bone algorithm.

In all temporal bones we measured the minimum thickness covering the roof of the SSC and the middle cranial fossa, as well as the minimum thickness of the PSC and the posterior cranial fossa. We defined the minimum thickness of the bone covering the SSC as the thinnest point of bone observed in the CT scan obtained in the same plane as the canal (Pöschl) between the outer surface of the SSC and the free surface of the middle cranial fossa. We defined the minimum thickness of bone covering the PSC as the thinnest point of bone observed in the axial sections of the CT scan between the PSC and the intracranial space.

We used the following radiological protocols for the acquisition and formatting of images: collimation 2 mm × 0.6 mm, section thickness 0.65 mm, section increase...
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0.32 mm, rotation time 0.75 s, pitch 0.38, kV 120, mAs 300, matrix 1024 × 1024, field of view 180 mm, reconstruction thickness 0.5 mm and reconstruction increase 0.5 mm.

For the statistical method, we described the variables considered by their absolute frequencies, percentages and 95% confidence interval. In bivariate analysis, for quantitative variables we verified normality through the Kolmogorov-Smirnov test. If they were normal, comparison was performed using Student’s t-test.

Results

The study was conducted on 318 patients, with 2 temporal bones being studied in 286 cases and only 1 in 32 cases. The age range of patients was between 2 and 88 years, with a mean value of 50.3 years (SD: 19.32 years). A total of 20 patients presented SSC dehiscence, which was bilateral in 2 cases (3.6%). Two subjects presented unilateral dehiscence of the PSC (0.3%).

Of the 20 cases with radiological dehiscence of the SSC, only 4 showed cochleovestibular signs or symptoms consistent with dehiscence (1 of them was a bilateral case). The remaining 16 patients with radiological dehiscence of the SSC and the 2 cases with radiological dehiscence of the PSC showed no suggestive symptoms or signs, which was interpreted as asymptomatic dehiscence.

The mean thickness of the SSC to the middle cranial fossa, excluding dehiscent cases (which gave a total of 602 observed cases) was 1.9 mm (SD: 0.6 mm).

Dehiscent Superior Semicircular Canal and Contralateral Canal

In the general sample, the mean thickness of the SSC was 1.1 mm (SD: 0.5 mm). The contralateral superior semicircular canals to the dehiscence (n=16) presented a mean thickness of 0.5 mm (SD: 0.3 mm). When comparing the mean thickness of the SSC without involvement with the mean SSC values on the contralateral side to the dehiscence we observed statistically significant differences (P<.05).

In a separate analysis, we also compared the SSC on the contralateral side to the dehiscent canal by age groups and noted that those patients under 45 years of age presented a mean thickness of 0.5 mm (SD: 0.3 mm), whilst those over 45 years of age presented a value of 0.6 mm (SD: 0.3 mm), without significant differences when comparing their means (P=.871 vs P=.793) (Fig. 1 and Table 1).

Dehiscent Superior Semicircular Canal and Posterior Semicircular Canal

In the overall sample, the mean thickness of the bone separating the PSC from the posterior fossa was 1.9 mm (SD: 0.6 mm). The posterior semicircular canal (n=39) of patients with SSC dehiscence showed a mean thickness of 2.1 mm (SD: 1.2 mm). These differences were not statistically significant (P=.49) (Table 1).
Table 1  Comparison of the Mean Thickness of Superior and Posterior Semicircular Canals.

<table>
<thead>
<tr>
<th></th>
<th>Contralateral SCC</th>
<th>PSC</th>
<th>Dehiscent SCC</th>
<th>Contralateral PSC</th>
<th>SSC</th>
<th>PSC: posternal semicircular canal; SD: standard deviation; SSC: superior semicircular canal.</th>
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<td></td>
<td>0.5 mm (SD: 0.3)</td>
<td>.05</td>
<td>2.1 mm (SD: 1.2)</td>
<td>.49</td>
<td>1 mm (SD: 0.4)</td>
<td>.062</td>
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</table>

Dehiscent Posterior Canal and Contralateral Canal

The posterior semicircular canals contralateral to a dehiscent homonym (n=3), presented a mean thickness of 1.3 mm (SD: 0.3 mm), with no statistically significant differences being observed when comparing this mean value with that found among control subjects without dehiscence (P=.062) (Table 1).

Dehiscent Posterior Canal and Superior Semicircular Canal

The superior semicircular canals of patients with posterior semicircular canal dehiscence (n=4) presented a mean thickness of 1 mm (SD: 0.4 mm), with no statistically significant differences with respect to the SSC without dehiscence associated with any of the posterior (P=.815) (Table 1).

Discussion

Among the factors which produce dehiscence are those caused by the rupture of the bone coverage by a second episode (cranial trauma, excessive pressure in the intracranial space) due to thinned canals.

The thickness of the bone layer which separated the SSC from the middle cranial fossa in our sample varied between 0.1 and 2.9 mm, with a mean value of 1.1 mm (SD: 0.5 mm). These figures were very similar to those described by Tsunoda,11 whose mean value was 1.2 mm (±0.6 mm), whilst the thickness separating the PSC from the posterior cranial fossa varied from 0.2 to 6.7 mm, with a mean value of 1.9 mm (SD: 0.6 mm). In turn, these figures were very similar to those described by Nomiyama et al.,11 whose mean value was 1.96 mm. With these data we established that the thickness of the bones covering the SSC in our study presented values that could be considered as a “thin pattern”, below 0.6 mm in the SSC and 0.8 mm in the PSC.

Our data suggest that the SSC contralateral to the dehiscent SCC presented a thin bone cover, since their mean values were of 0.5 mm, a fact that proved significant when compared with the mean thickness of the control subjects (P<.005), unlike those observed by Nadigier et al.,10 who did not link this canal dehiscence to a reduced bone thickness on the opposite side (P=.73).

Age was not a factor which influenced the thickness of the SSC on the opposite side to the dehiscence. Thus, we observed that this showed a value of 0.5 mm (SD: 0.3 mm) among those patients aged over 45 years, and 0.6 mm (SD: 0.3 mm) among those aged over 45 years, without significant differences being observed upon comparing their means (P=.871 vs P=.793). We observed no significant variations when comparing these data taking into account the gender of patients. The selection of 2 age groups separated by the barrier of 45 years was not arbitrary, since this is the age of onset of a hormonal dysfunction which leads to bone thinning, which could cause differences.13

Our results regarding the mean thickness of the contralateral SSC to the dehiscence showed very similar values to the studies of Hirvonen et al.,14 who found a decrease of this bone thickness relative to control subjects who were not affected by unilateral dehiscence.

As reported by Chien et al.,15 dehiscence of the PSC is a rare clinical entity with a very low incidence (0.3% as reported by Crovetto et al.16 and 0.2% as reported by Nomiyama et al.13), so although our results are difficult to assess, they do not lack validity.

We did not find any works in the literature linking the thickness of the PSC contralateral to a dehiscent PSC with dehiscence of the superior canal.

Our study showed that presenting SSC dehiscence did not alter the thickness of the PSC, since the mean thickness thereof under these conditions was 2.1 mm, whereas the mean thickness of controls without dehiscence was 1.9 mm (P=.49).

When comparing the thicknesses of dehiscent PSC with those on the opposite side, we found no statistically significant differences enabling an association of these observations. Nevertheless, given a value of P=.062 and a sample size equal to 3, we believe that these data could vary with a larger number of dehiscent patients under study, thus enabling a more conclusive result.

The thickness of the SSC of patients with PSC dehiscence presented a mean value of 1.3 mm, with no statistically significant differences (P=.815).

Conclusion

The presence of dehiscence in the SSC is associated to bone thinning in the contralateral canal, but not in the PSC. However, this is not true for dehiscence in the PSC, which is not linked to the thickness of the contralateral or superior canals in a significant manner.

Conflict of Interests

The authors have no conflict of interests to declare.

References


