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Dehiscence of the bony roof of the superior semicircular canal in the middle cranial fossa

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Abstract

Spontaneous dehiscence of the superior semicircular canal (SSC) in the middle cranial fossa is rare and may cause clinical problems. This dehiscence was investigated in cadaveric and dried temporal bone specimens. One cadaveric specimen showed a spontaneous defect: the dehiscence was a symmetrical, elongated ellipse with smooth margins. Four of 244 dry bone specimens showed bony defects in the roof of the SSC, however, only one specimen was thought to have a spontaneous defect. Based on computer-simulation models, we hypothesized that spontaneous defects of the SSC may arise during the fetal period. Although rare, this defect may cause problems in middle cranial fossa surgery and may relate to certain vertiginous disorders.

Key words: Temporal Bone; Abnormalities; Skull Base; Microscopy, Electron, Scanning; Semicircular Canals

Introduction

The superior semicircular canal (SSC, anatomical alias: anterior semicircular canal) is situated in the most superior part of the inner ear and is in close proximity to the middle cranial fossa (MCF).[1,2] Dehiscence of its bony roof is rare, however, this may cause problems with surgery or lead to disorders, for example, inner ear damage during elevation of dura mater and pressure-related dizziness or vertigo.[1,3–6]

This report describes an investigation of this dehiscence and its possible aetiology.

Materials and methods

Sixty-nine cadaveric MCFs (41 asian: male: female = 18: 23, 54 to 94 years old, mean 79.1 and 28 Causcasian: male: female = 15:13, 61 to 95 years old, mean age 83.5) were examined. Dura mater on the MCF was carefully peeled in all specimens. If bony defects over the SSC were suspected, the bones around the defect were carefully removed from the cadaver and soaked in bleach (7% sodium hypochlorite) for a day to remove organic matter.[7] Then the specimens were coated with gold and observed under a scanning electron microscope (SEM) (Coater: Sputter Coater 108 Auto, Cressington, Watford, UK. SEM: S-530, Hitachi, Tokyo, Japan and JSM-5410-LV, JOEL, Tokyo, Japan). The overall shape and margin of the defect were carefully observed. If the margin of defect was regular and smooth and did not seem to be caused by any artefactual force, this defect was determined to be spontaneous.

One hundred and eighty-four bone specimens (244 sides; 60 skulls and 124 temporal bones, right: 132, left: 112) were examined in order to estimate the frequency of this defect. Once a bony defect was found, its surface was carefully cleaned and the varnish was removed using varnish remover and chloroform. After cleaning, silicone putty was carefully applied to the bony surface around the defect to make a mould (President fast putty soft, Coltène AG; Altästten, Switzerland). An epoxy resin cast was made from this silicone putty.[8] Resin specimens were also coated and observed under SEM.

Finally, the shape of the defect was compared with computer-generated simulation models. The SSC is assumed to be a half doughnut (torus) with a thickness of 1 mm and outer diameter of 6 mm. The shapes of the cross sections of a torus obtained by cuts with various depths of plane were assessed and compared to defects observed in cadaveric and dry bones (Software: Mathematica ver 4, Wolfram Research, Champaign, Illinois, USA, Hardware: Power Macintosh G4, Apple Computer, Cupertino, California, USA).

Results

In cadaveric specimens, two bony defects of the roof of the SSC were found in both MCF in one

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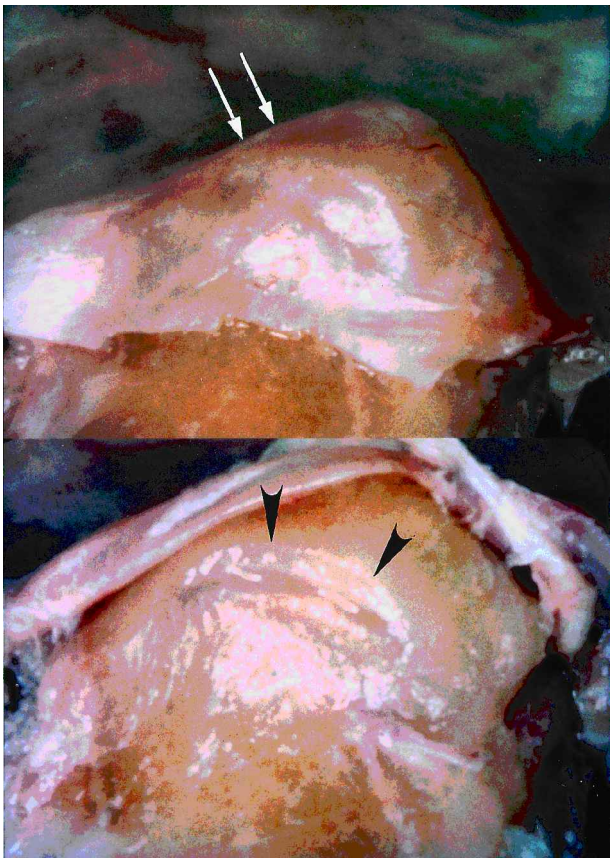

Fig. 1

Defect of the antero-superior wall of the right SSC seen in an 83-year-old female, with dura (upper) and without dura (lower). Bony defect (arrow head) was seen medial to the arcuate eminence (arrow).

Caucasian female. The area of the defect was noted to be covered with soft tissue which seemed to consist in part of endosteum (Figure 1). The defects observed in the cadaver measured 5.2 mm (right) and 3.5 mm (left) in size. Both mastoids were well developed, with thin bone over the air-cells. The bony wall of the SSC in the sulcus of the superior petrosal sinus was intact.

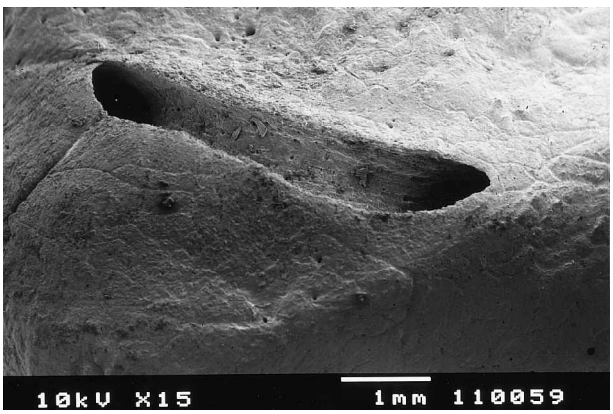

Fig. 2

The SEM image of the defect on the right MCF of an 83-year-old Caucasian female. Shape of defect is smooth and symmetrical and no abnormal change is observed around the defect (× 15)

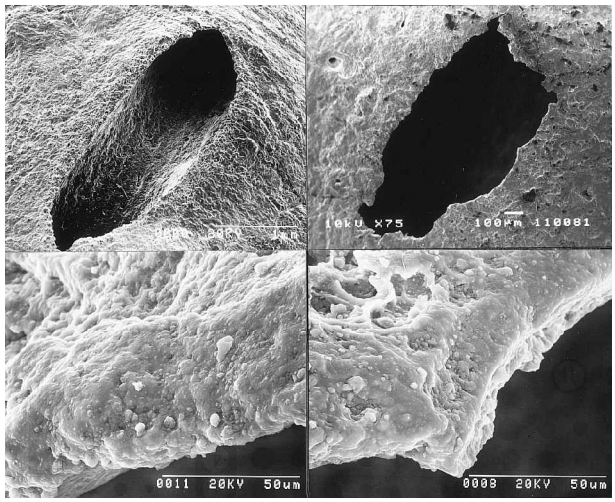

Fig. 3

Defect seen in the left MCF. Margin of the defect is also smooth and no abnormal change is observed around the defect (× 20, upper left). Even in high power field, margins are smooth (× 1000, lower left). Artificial defect have been made over the SSC after the bleaching and arcuate eminence lies between both defects (× 75, upper right). Different from margin of the original defect, sharp fractured edge is seen in artificial one (× 1000, lower right).

On the right, the bony defect was an elongated ellipse, whose shape was distorted but symmetrical under observation of SEM. Its margins were even and smooth, with a maximum width of the defect equal to the diameter of the canal lumen and a minimum depth equal to the lumen radius (Figure 2). Similar findings were observed on the left side; however, its shape wsa similar to an elongated ellipse without distortion (Figure 3). No bony pathology was observed around either defect or lumen of the SSC. These defects had smooth margins and did not show bony fracture (Figure 3).

No other lesions were found in these temporal bones. No tumours were detected intracranially and the corresponding temporal lobe seemed to be normal for the age of the patient. Her cause of death was old age (83 years old) and no obvious systematic skeletal abnormalities were observed.

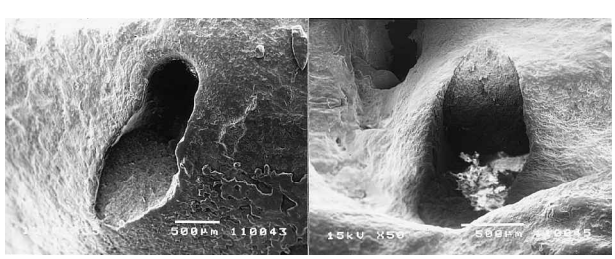

Fig. 4

Defect seen in dry bone specimen. The margin is partially damaged, however, the preserved margin seems to be smooth (× 35, left). The same findings are seen in the defect in the sulcus of the superior petrosal sinus (× 50, right). The defects in this specimen are not well preserved and putty remains in the lumen of the SSC due to technical difficulties. However, even with their conditions and technical difficulties, they seem spontaneous defects, not artefacts.

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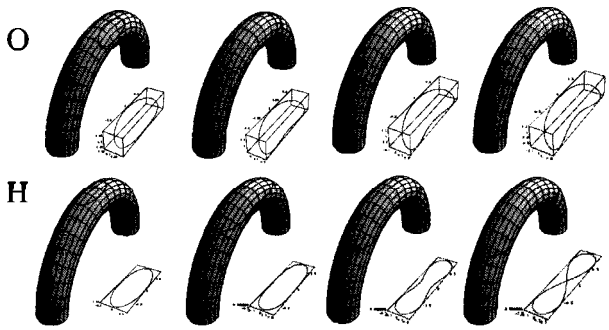

Fig. 5

Computer-generated model of those defects. Various shapes of the cross section were obtained by cut with various depth of plane. O: oblique plane. H: horizontal plane. This ellipse* is then pinched and divided into two as depth of plane increases, however, a dumbbell shape will not appear. To obtain a dumbbell, another factor might take place (Figure 6,7).

*Above mentioned ellipse is not an exact mathematically defined ellipse, but an approximate one. The shape of the cross section (x,y,z_0) of a horizontal plane ($z = z_0$) and a doughnut (torus) is described as:

$$x^2 + [\surd(y^2 + z_0^2) \pm R]^2 = r^2$$

where r and R denote the canal's inner radius and the rotation radius around X-axis, respectively.

In case of an oblique plane ($z = ax + z_0$), the cross section (x,y,z) is described by the solution of the simultaneous equations

$$z = ax + z_0$$
$$x^2 + [\surd(y^2 + z_0^2) \pm R]^2 = r^2$$

However, because no medical record was obtained, it was not known if the patient had a history of vertigo.

Four temporal bones from 244 dry bone samples showed bony defects over the SSC. However, in three specimens, the shape of the defects was irregular and the margins of defect were not smooth by observation under a stereoscopic microscope and/or SEM. The possibility of artefact caused by postmortem change or artefactual force could not be denied in these three defects.

A fourth specimen showed defects not only in the middle cranial fossa but also in the sulcus of the superior petrosal sinus. The shapes of the two defects were similar to the elongated ellipse with a maximum width of each defect equal to the diameter of the canal lumen and a minimum depth equal to the lumen radius. Some regions of the defects showed smooth margins. No pathological changes were found on the surrounding bone and shapes of defects resembled those observed in the cadaver (Figure 4). These defects were observed in the left temporal bone specimens. No information was obtained about the opposite side.

On computer simulation, the shape of the cross sections had changed according to cutting depth. The elongated elliptical shape observed in specimens' defects was reproduced by computer modelling. A horizontal plane, which was perpendicular to the semicircular canal, produced an elongated ellipse with no distortion, mimicking the defect of the cadaver's left side and those of the dry bone specimen (Figure 5). On the other hand, an oblique plane produced a distorted ellipse and this shape was similar to that of the cadaver's right side (Figure 5). These computer-generated elongated ellipses could only be reproduced by cutting through the SSC such that its depth was equal to the lumen radius. Since the specimens showed defects with a depth equal to the lumen radius, these computer models seemed to be consistent with the actual defects.

Discussion

Dehiscence of the bony roof of the SSC is known among otologists, however, Minor *et al.* first reported the relationship between dehiscence or thinning of the bone overlying the SSC and pressure-induced vertigo.[3] The detection of this dehiscence is easily made by a coronal section of high resolution computed tomography.[3–5] However, compared to defects in other MCF areas, frequency of this dehiscence is rare.[9–11] From a histological study of 1000 temporal bone specimens, five defects were found in the bony covering of the SSC and only one defect was found in the MCF.[6] In the present study, a defect over the SSC was found in four out of 244 bone specimens, however, a spontaneous defect was observed in only one specimen. Defects were found in both sides of one Caucasian cadaver and no defect in Asian cadavers. This finding in the Caucasian specimen might be incidental, however, a discussion of the incidence or population differences is beyond the scope of this study.

Bony dehiscence often occurs as a developmental failure. Concerning the aetiology of this dehiscence, it has been ascribed to a postnatal failure in the development of the outer and/or middle layer of bone coupled with minor head trauma or barotrauma.[5,6] However, the shapes of the defects in our specimens were regular and quite symmetrical, with a depth equal to the lumen radius. We propose the following hypothesis for the production of this defect.

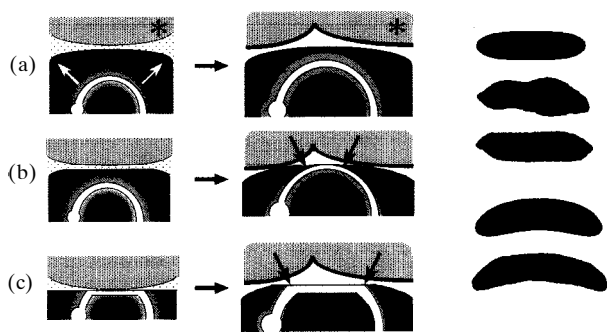

Fig. 6

The models explaining the aetiology of the defect over the SSC. The precartilage bordering the developing membranous labyrinth dedifferentiates into loose reticular mesenchyme, permitting growth of labyrinth (white arrow, (a)). During this stage, if the otocyst was situated close to the developing brain (*) and a space for growth of the SSC was not enough, the otic capsule might directly contact with dura mater (black arrow, (b)). The shapes of defects are shown in the right row. From above, simulation (horizontal cut), temporal bone specimen, cadaver's left side, simulation (oblique cut), cadaver's right side. If cartilage space is too little, the canal between the ampulla and common crus will be stretched, but the space for the endolymphatic duct will be preserved. (c) This may result in a dumbbell-like defect.

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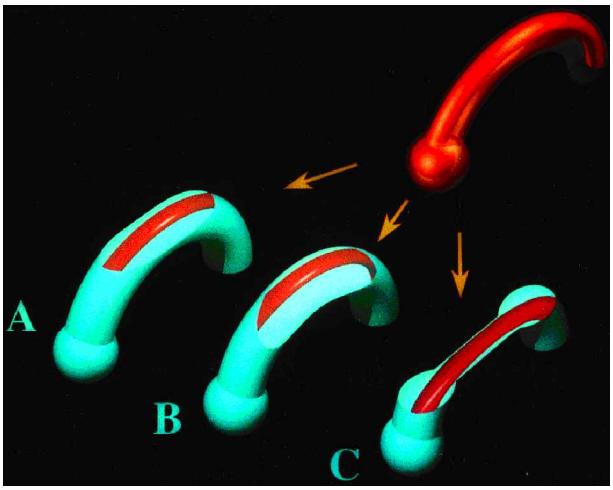

Fig. 7

Three-dimensional simulation model (Software: Strata Vision 3d ver 5.5, Strata, St George, Utah, USA. Hardware: Powerbook, Apple Computer). Endolymphatic duct is housed in the semicircular perilymphatic space (upper-right). Figures (a) and (b) correspond to model of Figure 6(b). (A): horizontal defect, (B): oblique defect. (C) corresponds to model of Figure 6(c).

The shape of the otocyst becomes mature around the 10th week and grows to adult size keeping its shape by the 23–25th weeks.[12–14] However, the condensation of mesenchyme around the otocyst occurs in the fifth week and mesenchyme envelopes the membranous labyrinth in the sixth week. The mesenchyme enclosing the otocyst becomes chondrified to form the otic capsule at the eighth week. At the ninth week, the pre-cartilage ordering the developing membranous labyrinth de-differentiates into loose reticular mesenchyme, permitting growth of the labyrinth (Figure 6A). As the membranous labyrinth expands, the otic capsule remodels and in places undergoes de-differentiation to form fluid-filled spaces that eventually become the perilymphatic spaces.[12–14]

During this stage, if the otocyst was close to the developing brain and the cartilage superior to the otocyst did not have enough size and space for growth of the SSC, the otic capsule, i.e. the future endolymphatic space of the SSC, might make contact directly with the dura mater which arises mainly from paraxial mesoderm (Figure 6B). In such a situation, both inner and outer periosteal ossification might not occur. These cartilaginous or bony defects might approximate the simulated defect presented in Figure 5 since the shape of the SSC itself keeps its mature shape after the 10th week of fetal development. Therefore, any defect caused by this mechanism should be smooth and symmetrical although this shape would be slightly changed according to postnatal development (Figure 7). The shape of the defect is also influenced by the shape and position of the arcuate eminence which corresponds to the occipito-temporal sulcus.[2,15,16] According to the position of the gyrus or sulcus of temporal lobe, defects may occur horizontally or obliquely (Figure 5, 7). All of the dehiscence in the bone covering the SSC may not have been caused by this mechanism alone. However, based on this hypothesis, even and symmetrical bony dehiscence may plausibly occur.

Interestingly, Minor reported a case with a dumbbell-shaped defect over the SSC.[5] From our computer simulations, however, a 'dumbbell' could not be generated even with extreme erosion (Figure 5). Based on our hypothesis, if cartilaginous space for growth is too little, then the canal between the crus would be prolonged and bone would only develop around the endolymphatic duct. This would result in a dumbbell-shaped defect and can explain its aetiology (Figure 6(c), 7(c)).

Since this defect appears to be congenital, it should be observed in the neonate, or even in the fetus.[6] We also found one defect in a part of the superior petrosal sinus from one infantile temporal bone. This specimen also seemed to be a spontaneous defect under SEM observation. However, sample numbers were not large enough, and investigations of infant or the fetus were excluded from the present study.

Conclusion

A spontaneous defect of the roof of the superior semicircular canal is rare, occurring in 0.4 per cent of the temporal bones according to our study. Such a defect might occur in the early stages of the formation of the fetus and may cause clinical problems.

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