Prevalence of Carotid Canal Dehiscence Facing with Middle Ear and its Relationship with Tinnitus

Mehmet Emre Sivrice1, Hasan Yasan2, İsmail Çoban3, Mustafa Kayan4

1Department of Otorhinolaryngology, Tavşanlı Docent Doktor Mustafa Kalenli State Hospital, Kutahya,
2Department of Oto-Rhino-Laryngology and Head&Neck Surgery, Faculty of Medicine, S.Demirel University, Isparta,
3Department of Oto-Rhino-Laryngology and Head&Neck Surgery, Antalya Atatürk State Hospital, Antalya,
4Department of Radiology, Faculty of Medicine, S.Demirel University, Isparta, Turkey

Canal Dehiscence and its Relationship with Tinnitus

Özét


Karotis Arter; Pulsatil Tinnitus; Temporal Kemik

Abstract

Aim: To assess the frequency of carotid canal dehiscence facing with middle ear cleft and its relationship with pulsatile tinnitus. Material and Method: High resolution computed tomography scans (reformatted from the axial plane images taken for paranasal sinus investigations) of 1026 temporal bone images of 513 patients were retrospectively examined for the presence of dehiscent internal carotid artery. The presence of pulsatile tinnitus complaint was noted. The relationship between carotid canal dehiscence in the middle ear and pulsatile tinnitus was investigated. Results: One thousand and twenty-six (1,026) temporal bone images of 513 patients were investigated for the presence of carotid canal dehiscence in the middle ear (%30.9). Three hundred and eighteen (318) out of 1026 temporal bone images proved to present carotid canal dehiscence in the middle ear. There were 16 ears with pulsatile tinnitus at the site of dehiscent carotid canal. There were 19 ears with pulsatile tinnitus at the site of the temporal bone without carotid canal dehiscence. There was no statistically significant correlation between the carotid canal dehiscence and pulsatile tinnitus. Discussion: Neither age nor gender is a determining factor for the presence of carotid canal dehiscence. Pulsatile tinnitus seems not to be related with carotid canal dehiscence. Keywords

Carotid Artery; Pulsatile Tinnitus; Temporal Bones

DOI: 10.4328/JCAM.4805  Received: 09.09.2016  Accepted: 29.09.2016  Printed: 01.05.2017  J Clin Anal Med 2017;8(3): 195-7

Corresponding Author: Mehmet Emre Sivrice, Department of Otorhinolaryngology, Tavşanlı Docent Doktor Mustafa Kalenli State Hospital, 43300, Kutahya, Turkey.
GSM: +905447724519 F.: +90 2746151425 E-Mail: emresivrice@gmail.com
**Introduction**

Internal carotid artery and the jugular bulb are two major vascular structures that pass through the temporal bone, and if dehiscent, face with the middle ear cavity. It is well known that the dehiscence of the jugular bulb may cause pulsatile tinnitus. Although carotid canal dehiscence (CCD) has been documented in previous studies, its relationship with pulsatile tinnitus has not been established [1,2,3]. Here we have evaluated the frequency of carotid canal dehiscence in the middle ear and its relationship with pulsatile tinnitus.

**Material And Method**

The outpatient and inpatient charts of all patients who underwent computed tomographic (CT) evaluation of the paranasal sinuses from March 2008 through September 2010 were reviewed. CT evaluation of the paranasal sinuses was performed in the axial projection without the administration of intravenous contrast material. We performed the CT examinations with the patient supine on the scanner bed with the head in extension. Multi-slice 128 CT scanning (Definition AS, Siemens Medical Solutions, Forchheim Germany) was obtained in the axial plane with the following parameters: 120 kV, gantry rotation of 1 second and 110 mA second, 0.8 pitch factor, and mean total acquisition time 5 seconds. Examinations were evaluated using both bone and soft tissue windows. The axial CT scans of patients with paranasal sinus evaluation were reformatted to obtain coronal plane images. Temporal bones were normally visualized in the paranasal sinus being investigated. Reformatted images of temporal bone (axial and coronal plane images) were investigated for carotid canal dehiscence in the middle ear (Figure 1).

This evaluation was carried out by one radiology specialist and one ENT specialist. Clinical findings of all patients were taken from charts. In cases of discrepancy or insufficient information in the files, then the patients were asked by phone about the nature of complaints and clinical history. Exclusion criteria were as follows: patients who had a history of previous ear operation, known cardiovascular disease, ototoxic drug usage, head trauma, aberrant internal carotid artery (ICA), intratemporal or intratympanic mass and/or fluids, and age older than 50 years. During the evaluation, CCD is accepted as positive if any part of the carotid canal is facing the middle ear cleft, and the petrous and eustachian part has dehiscence. Dehiscence of the carotid canal on the base of cranium was not evaluated as CCD.

**Results**

The number of patients included in this study was 513 and the number of temporal bone images was 1206. The mean age and range of age of patients were 32.80 ± 12.39 and 10-50 years, respectively. The numbers of male and female patients were 263 (51.3%) and 250 (48.7%) respectively. Three hundred and eighteen (318) out of 1026 temporal bone images (30.99%) showed the presence of CCD in the middle ear. There were 16 ears with pulsatile tinnitus at the side of CCD. There were 19 ears with pulsatile tinnitus at the side of temporal bone without CCD. One hundred and thirty-nine patients have had bilateral CCD (278 temporal bones), 28 patients have had only right-sided CCD, and 12 patients have had only left-sided CCD. That is, 167 out of 513 right-sided temporal bones have had CCD in the middle ear (32.6%), and 151 out of 513 left-sided temporal bones have had CCD in the middle ear (29.4%). There were 30 patients in the pulsatile tinnitus group. Five of these 30 patients had bilateral pulsatile tinnitus. In the pulsatile tinnitus group, 21 right-sided ears and 14 left-sided ears were involved. Fifteen of the right-sided pulsatile tinnitus and eight of the left-sided pulsatile tinnitus patients had high jugular bulb with dehiscence. The etiology of pulsatile tinnitus in the remaining 12 ears was not diagnosed by the present findings. Right-sided pulsatile tinnitus was seen in nine ears with CCD, and 12 ears without CCD. Left-sided pulsatile tinnitus was seen in seven ears with CCD, and seven ears without CCD (Table 1).

There was no statistically significant difference between involvement of right-sided versus left-sided temporal bones. There were no statistically significant correlations between CCD and pulsatile tinnitus, CCD and age, or CCD and gender (Table 2).

<table>
<thead>
<tr>
<th>Parameters compared</th>
<th>P values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age – CCD (patient number)</td>
<td>0.102</td>
</tr>
<tr>
<td>Right CCD – Right pulsatile tinnitus</td>
<td>0.055</td>
</tr>
<tr>
<td>Left CCD – Left pulsatile tinnitus</td>
<td>0.076</td>
</tr>
<tr>
<td>Gender – CCD</td>
<td>0.253</td>
</tr>
<tr>
<td>Right CCD – Right pulsatile tinnitus</td>
<td>0.304</td>
</tr>
<tr>
<td>Left CCD – Left pulsatile tinnitus</td>
<td>0.087</td>
</tr>
</tbody>
</table>

1Pearson correlation test, 2Independent sample T-test.
Discussion

The carotid canal starts to develop as two laminae of the petrous bone in the 18th fetal week. In cases of incomplete closure of these laminae the carotid canal occupies dehiscence[4]. There is a wide range reported for the frequency of CCD. This discrepancy may be attributable to differences in the criteria used to define case selection and in the evaluation technique (radiologic or histologic) for identifying the dehiscence[1]. Our study results may reveal some differences from the literature due to case selection. We have included the cases only with radiologic CCD in the middle ear space. Normally, the ICA enters the petrous bone medial to styloid process via the carotid canal. The initial vertical segment is anterior to the cochlea and separated from the tympanic cavity by a thin plate of bone. Disappearance of the bony plate between the ICA and the middle ear results in dehiscence of the carotid artery in the middle ear. Sometimes this dehiscence may result in aberrant ICA in the middle ear[3]. CT can be used to investigate the CC. The best plane is the coronal, through the horizontal portion of the canal, with which one can clearly see the abnormalities of its walls[5]. We have evaluated the presence of CCD in the middle ear both in coronal and axial CT planes of temporal bones. Although there are some studies investigating the frequency of carotid canal dehiscence, most of them are related to both the ventral and dorsal carotid canals. The origin of CCD in the middle ear may be explained by failures in ossification, congenital anomaly, persistence of embryonic vessels, bone absorption throughout the years, middle ear inflammatory processes, and traumatic injuries of the temporal bone[1,2,3,4,5,6]. We have excluded the cases with a history of temporal bone trauma or middle ear inflammatory processes.

Penido et al. [2] investigated CCD in the middle ear by microscopic anatomy study. They have found 35.2% CCD in the middle ear. We have found the frequency of radiologic CCD in the middle ear as 30.9%. This finding is consistent with the literature. Previous studies are mostly related only to the frequency of CCD. Because jugular bulb dehiscence is a known cause of pulsatile tinnitus, we researched the relationship between CCD and pulsatile tinnitus. Patients who have carotid canal dehiscence also have high jugular bulb at a prevalence of more than 60%[7]. In this case, the reasons for pulsatile tinnitus may not be clear. However, our study indicates that CCD does not cause pulsatile tinnitus by itself. The prevalence of carotid canal dehiscence decreases with increasing temporal bone age[6]. In our study there is no statistically significant correlation between patient age and CCD frequency. This could be due to case selection criteria.

The differential diagnosis of pulsatile tinnitus includes: middle ear (effusion, chronic otitis media), neoplasm (glomus jugulare or glomus tympanicum, geniculate ganglion hemangiomata), arterial (arterio-venous fistulas, internal auditory vascular loops), venous (benign intracranial hypertension, sigmoid or jugular diverticulum, high jugular bulb) pathologies, and aneurysm of the internal carotid artery[8,9]. Dehiscent jugular bulb is one cause of pulsatile tinnitus. It is usually asymptomatic, but when symptoms are present, tinnitus is the most common complaint[10]. ICA-related pulsatile tinnitus can be caused by carotid artery aneurysm, carotico-jugular or carotico-cavernous fistula, or aberrant ICA. The aberrant ICA is another cause for pulsatile tinnitus. Aberrant ICA can be identified on CT scan by the following features: intratympanic mass, enlarged inferior tympanic canaliculus, absence of the vertical segment of the ICA canal, and the absence of bone covering the tympanic portion of the ICA[3].

This is the first study that investigates the radiologic frequency of carotid canal dehiscence in the middle ear cleft and its relationship with pulsatile tinnitus.

Conclusion

The frequency of carotid canal dehiscence in the middle ear cleft was 30.99% (318 dehiscences in 1026 temporal bone images). Neither age nor gender is a determining factor for the presence of CCD. Dehiscence of the carotid canal facing with middle ear cleft seems not to be one of the causes of pulsatile tinnitus.

Competing interests

The authors declare that they have no competing interests.

References


How to cite this article: