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PARAMETERS OF THE SECOND GENU OF THE FACIAL NERVE IN PEOPLE OF MESO- AND BRACHIOCEPHALIC TYPE OF THE CEREBRAL SKULL

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ABSTRACT

Otitis media, mastoiditis or the pressure effect of tumorous lesions such as cholesteatoma can be the cause of facial canal dehiscence and facial nerve paralysis. The most common segment involved in dehiscence is the tympanic segment and the second most common is the lateral aspect of the facial canal in the oval window area. To determine the prevalence of the facial canal dehiscence and the relationship between the angle at the second genu of the facial nerve and facial canal dehiscence. We evaluated the surgical findings in 113 patients who underwent surgery for cholesteatoma. Facial canal dehiscence was detected in 62 of the 113 patients. Patients were divided into two groups: Group 1, with dehiscence of the facial canal and Group 2, without dehiscence of the facial canal. The mean angles at the second genu of the facial nerve in Groups 1 and 2 were 117.8◦ ± 9.63◦ and 114◦ ± 9.9◦, respectively. There was a statistically significant difference between the mean angles at the second genu for the two groups ($p = 0.04$). In patients with dehiscence of the facial canal, the angle at the second genu was found to be wider than those without dehiscence.

1. Introduction

Otitis media, mastoiditis or the pressure effect of tumorous lesions such as cholesteatoma can be the cause of facial canal dehiscence and facial nerve paralysis [1], [2]. Facial canal dehiscence can be either congenital or acquired. Congenital facial canal dehiscence is a developmental defect in the bony covering of the facial nerve [3]. Acquired dehiscence is often associated with atticoantral chronic suppurative otitis media with cholesteatoma [4]. It may also develop due to long-standing inflammation, prior ear surgery and trauma [2]. The incidence of facial canal dehiscence has been reported to be between 0.5% [5] and 74% [6] based on intraoperative findings. The most common segment involved in dehiscence is the tympanic segment (84.6%), and the second most common is the lateral aspect of the facial canal in the oval window area (69.2%) [7]. The roof of the tympanic segment is very thin, so tumoral lesions such as cholesteatoma

or otitis media can easily be the cause of facial canal dehiscence due to the pressure effect. Normally, the angle at the second genu of the facial nerve is 95◦---25◦. When the angle at the second genu increases, the mastoid part of the facial nerve is displaced posteriorly in the mastoid, away from the chorda tympani and round window [8]. Because of the increased angle at the second genu, the tympanic segment of the facial nerve has a wider surface area which may lead to dehiscence in a wider surface area in patients with cholesteatoma [9], [10]. A limited number of studies related to the angle at the second genu of the facial nerve are available [8], [11]. However, there has been no investigation into the relationship between the angle at the second genu and facial canal dehiscence in patients with cholesteatoma. Here, the aim of the study is to determine the prevalence of facial canal dehiscence and the relationship between the angle at the second genu and facial canal dehiscence in patients with cholesteatoma.

2. Patients and Methods

Institutional Review Board approval was obtained to review the records of all patients who underwent temporal bone Multidetector Computed Tomography (MDCT) between 2011 to 2016 years. The approval protocol number from the Ethics Committee of our institution is B.30.2.ODM.0.20.08/495. A total of 113 patients (37 female, 76 male) with pathologically proven middle ear cholesteatoma who underwent primary surgery from 2011 to 2016 were enrolled in this study. The medical records of the intraoperative assessment of the facial canal were reviewed using the hospital's database. The facial canal was divided into five segments: geniculate ganglion, tympanic segment, second genu, oval window niche and mastoid (Table 1). Facial canal dehiscence was defined as any discontinuity in the bony structure of the facial canal which resulted in a connection between the facial nerve and any middle ear space or mastoid air cell system. According to surgical findings, facial canal dehiscence was detected in 62 of the 113 patients. A total of 51 patients with cholesteatoma had no dehiscent facial canal. Patients were divided into two groups: Group 1, with dehiscence of the facial canal and Group 2, without dehiscence of the facial canal. Demographic data of the study population is shown in Table 2.

3. Results

All 9 patients had advanced Hodgkin lymphoma. FGD-PET was done at start of treatment and after 4 cycles of ABVD. All children except one received 6 cycles of ABVD with radiotherapy for those with bulky disease. One child died during course of treatment due to septic shock. Out of 9 children 3 had complete remission, 1 after 4 cycles of ABVD and 2 after 6 cycles of ABVD. The disease stage, organ dysfunction and ferritin level is shown in Table 1. Five of 9 had partial remission 3 of whom had bulky disease and received radiotherapy. All of the children received ABVD cycles barring one who had initial raised Bilirubin level and was given 1 cycle of CHOP and then ABVD was introduced. The children were grouped into mild, moderate and sever ferritin values [Mild elevation-Serum Ferritin less than 500 ng/ml, Moderate elevation-Serum ferritin 500-1000 ng/ml, severe ferritin elevation-Serum ferritin >1000 ng/ml]. The patients were grouped into 3 categories based on outcome values. The results are discussed in Table 2. The outcome in general was good for stage III disease, indicating clinical stage is an independent prognostic factor. However patients with elevated serum ferritin either moderate or severe lead to more partial remission states versus low serum ferritin levels. The results are however limited by the small sample size. The study needs more validation with larger samples. After 4/6 cycles of ABVD the reduction in ferritin was correlated with the remission status. It was high in non-responders and low in responders.

4. Discussion

The facial nerve canal begins to develop as a narrow groove or sulcus within the cartilage of the otic capsule. Ossification then starts from the apical otic ossification center at 21 gestational weeks and from the canalicular ossification center at 26 gestational weeks near the stapedius muscle. The two centers fuse near

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the region of the oval window until one year after birth [2], [12]. From an anatomical and radiological standpoint, the facial canal is completely developed by four years of age [13]. However, middle ear inflammations can affect the development of the facial canal in children [12]. Also, facial canal dehiscence may develop due to prior ear surgery, trauma and the pressure effect from tumorous lesions [2]. The incidence of facial canal dehiscence was reported in a relatively wide range from 0.5% [5] to 74% [6] based on histologic and surgical studies. Dehiscence of the facial canal must be at least 1 mm in size to be detected during surgery [12]. However, the incidence of facial canal dehiscence is higher in histological studies, since it can be detected in microdehiscences of less than 1 mm in cadaveric studies [2]. Takashi and Sando found that 40% of all dehiscences were detected on the inferior to inferomedial aspect of the facial canal in the posterior half of the oval window area [6]. Baxter revealed that 85% of all dehiscences occurred through the inferior surface of the tympanic segment toward the oval window niche [14]. In fact, it is not possible to see these dehiscent areas with routine otologic surgery.

5. Conclusions

The prevalence of facial canal dehiscence was 54.8% in patients with cholesteatoma and was most commonly seen in the tympanic segment of the facial canal. Additionally, the angle at the second genu of the facial nerve in patients with dehiscent facial canal was greater than in those without dehiscence. An increased angle at the second genu can be the cause of the wider surface area of the tympanic segment. Thus, patients with an increased angle at the second genu may be more prone to dehiscence of the facial canal.

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