




Evaluation of facial canal and mastoid pneumatization characteristics in Bell's palsy

Bell paralizisinde fasiyal kanal ve mastoid havalanma özelliklerinin değerlendirilmesi

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ABSTRACT

Objectives: This study aims to evaluate the facial canal diameter and length and mastoid bone pneumatization on the affected side, compared to the contralateral side, in patients with unilateral Bell's palsy (BP).

Patients and Methods: Between January 2014 and November 2016, a total of 30 patients (16 males, 14 females; mean age 42.3±12.7 years; range, 18 to 65 years) who were admitted to our outpatient clinic with unilateral BP were retrospectively analyzed. The temporal bone computed tomography images of the patients were examined by multislice and three-dimensional (3D) reconstruction method and the facial canal diameters were measured in the mastoid segment, labyrinthine segment, and foramen stylomastoideum level in both temporal bones. The length of the facial canal was also measured along the mastoid segment. Using the 3D images, each mastoid cell volume was measured separately to calculate the total cell surface area and to identify the level of mastoid pneumatization.

Results: The mean facial canal diameter was statistically significantly narrower in the mastoid segment, labyrinthine segment, and foramen stylomastoideum level on the affected side (p=0.001). The mean facial canal length was also statistically significantly longer along the mastoid segment on the affected side (p=0.043). There was no statistically significant difference in the level of mastoid bone pneumatization between the two sides (p=0.359).

Conclusion: Our study results suggest that the diameter of the facial canal is particularly relevant in BP etiology as a risk factor. The length of the facial canal course along the mastoid segment may be also considered a risk factor. However, there is no significant relationship between the mastoid pneumatization level and BP.

Keywords: Bell's palsy, facial nerve, mastoid, three-dimensional computed tomography.

ÖZ

Amaç: Bu çalışmada tek taraflı Bell paralizisi (BP) olan hastalarda karşı tarafa kıyasla etkilenmiş tarafta fasiyal kanalın çapı ve uzunluğu ve mastoid kemiğin havalanması değerlendirildi.

Hastalar ve Yöntemler: Ocak 2014 - Kasım 2016 tarihleri arasında tek taraflı BP nedeniyle polikliniğimize başvuran toplam 30 hasta (16 erkek, 14 kadın; ort. yaş 42.3±12.7 yıl; dağılım, 18-65 yıl) retrospektif olarak incelendi. Hastaların temporal kemik bilgisayarlı tomografi görüntüleri çok kesitli ve üç boyutlu (3D) rekonstrüksiyon yöntemi ile incelendi ve her iki temporal kemiğin mastoid segment, labirint segment ve foramen stilomastoideum düzeyinde fasiyal kanal çapları ölçüldü. Mastoid segmentte fasiyal kanal uzunluğu da ölçüldü. Üç boyutlu kesitlerde her bir mastoid hücre tek tek ölçülerek toplam hücre yüzey alanı hesaplandı ve mastoid havalanma düzeyi belirlendi.

Bulgular: Etkilenen tarafta fasiyal kanalın ortalama çapı mastoid segment, labirint segment ve foramen stilomastoideum düzeyinde istatistiksel olarak anlamlı düzeyde daha dardı (p=0.001). Mastoid segment boyunca ortalama fasiyal kanal uzunluğu da, etkilenen tarafta istatistiksel olarak anlamlı düzeyde uzundu (p=0.043). Mastoid kemik havalanma düzeyi açısından her iki taraf arasında istatistiksel olarak anlamlı bir farklılık yoktu (p=0.359).

Sonuç: Çalışma sonuçlarımızın fasiyal kanal çapının BP etiolojisinde risk faktörü olarak özellikle ilintili olduğunu göstermektedir. Mastoid segment boyunca fasiyal kanal uzunluğu da bir diğer risk faktörü olarak kabul edilebilir. Bununla birlikte, mastoid kemik havalanma düzeyi ile BP arasında anlamlı bir ilişki izlenmemiştir.

Anahtar sözcükler: Bell paralizisi, fasiyal sinir, mastoid, üç boyutlu bilgisayarlı tomografi.

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Bell's palsy (BP) is a sudden onset, idiopathic weakness of the peripheral part of the facial nerve. It is the most common cause of facial paralysis with an annual frequency of 20 to 30 per 100,000 individuals.^[1] Its etiology constituting 60 to 70% of all facial paralysis cases dwells on genetic, vascular, autoimmune, anatomic and infectious agents, although the most common culprit is herpes simplex virus.^[1] Several studies have shown that the virus settles in the geniculate ganglion and, then, upon activation it initiates the paralytic process. It has been suggested that nerve edema and disruption of the circulation in the part of the Fallopian canal due to the emerging infection causes ischemic damage and loss of function.^[2] The presence of edematous nerve in the canal as an intraoperative finding in patients undergoing facial decompression due to BP is supported by this view.^[3,4] However, in the etiology of idiopathic facial paralysis, anatomic variations in various points of the facial nerve anatomy may also play a role. Therefore, the anatomical structure of the Fallopian canal in which the facial nerve is located within the temporal bone gains importance in facial paralysis. It has been reported that, in patients with BP based on anatomical and radiological examinations of the temporal bone, the diameter of the facial canal is narrower than normal.^[5,6]

The anatomical development process of the temporal bone begins in the second and third months of the prenatal period. The pneumatization of the mastoid bone continues from prenatal period to the end of childhood and puberty.^[7] Therefore, it is possible that mastoid bone pneumatization may alter the temporal bone anatomy. In this respect, one of the most important structures is facial nerve and Fallopian canal. Although there are studies evaluating this subject, no definite relationship has been demonstrated, yet.^[8,9] Three-dimensional (3D) multiplanar reconstruction of computed tomography (CT) imaging of the temporal bone allows the evaluation of the anatomic structure of both the mastoid pneumatization and nerve. Therefore, in the present study, we aimed to investigate the facial canal diameter and length and mastoid bone pneumatization on the affected side, compared to the contralateral side, in patients with unilateral idiopathic BP through the 3D high-resolution temporal bone CT.

PATIENTS AND METHODS

Medical data and CT findings of patients older than 18 years diagnosed with unilateral idiopathic peripheral facial palsy who were admitted to our

otorhinolaryngology clinic between January 2014 and November 2016 were retrospectively reviewed. Inclusion criteria were as follows: having unilateral sudden idiopathic peripheral facial paralysis findings without traumatic, infectious, vascular, oncologic etiologies; and having no infection. Those with a known chronic disease history and previous history of ear operation were excluded from the study. In all patients, no feature other than facial paralysis was found on otorhinolaryngological examination. Also, there was no any abnormality on neurological examination. The level of facial paralysis of all patients was graded and recorded based on the House-Brackmann facial paralysis grading system. The patients were started with an appropriate medical treatment protocol and followed.

A written informed consent was obtained from each patient. The study protocol was approved by the Sakarya University Medical Faculty Ethics Committee. The study was conducted in accordance with the principles of the Declaration of Helsinki.

High-resolution temporal bone CT scans which were performed to identify the etiologic cause were examined. The images obtained with the Aquilion 64-Slice CT device (Toshiba Medical Systems, Otawara, Japan) in the Picture Archiving and Communication System (PACS) (TeraRecon Inc., San Mateo, CA, USA) in our hospital were evaluated. The CT scans were obtained with the patients in supine position tilted at 0-degree with the parameters of 120 Kv, 150 mA, rotation time of 1 sec, matrix size 512×512, and 1 mm of section thickness. The images were installed to the PACS to assess the presence of a temporal bone pathology. In addition, 3D multiplanar reconstruction images in sagittal and axial planes with 1 mm in thickness and 0.2 mm reconstruction intervals were created with the TeraRecon Aquarius iNtuition version 4.4; workstation computer (TeraRecon San Mateo, CA, USA). All axial and sagittal reconstruction images were evaluated on a standard bone tissue window. Both temporal bones were marked as affected and unaffected sides in each image. All measurements were calculated by a single radiologist who was blinded to patients' data and side of paralysis.

The mean diameter of the canal in the middle part of the mastoid segment and the stylomastoid foramen level were measured and recorded at sagittal plane. The length of the facial canal along the mastoid segment was also measured at sagittal plane (Figure 1). The mean diameter of the labyrinthine segment was measured at axial plane (Figure 2). Mastoid air cells with a gray-scale level similar to air in the temporal bone were determined on CT for volumetric measurement. To calculate the total mastoid cell area, individual polygonal area measurements were used for each of the

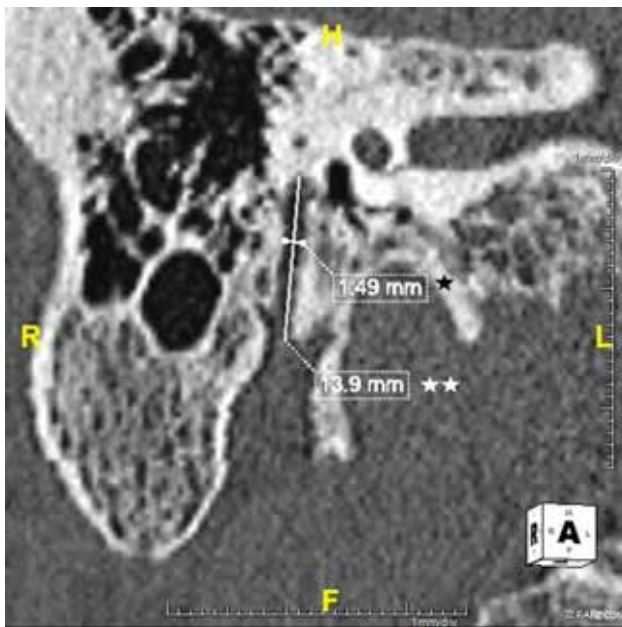


Figure 1. * Measurement of facial canal diameter at mean part of mastoid segment. ** Measurement of facial canal length of mastoid segment at sagittal plane.

cells of independent air density in the workstation. The total mastoid cell aeration surface area was calculated by summing up all the obtained measurements (Figure 3). All measurements were recorded separately for each side in all patients.

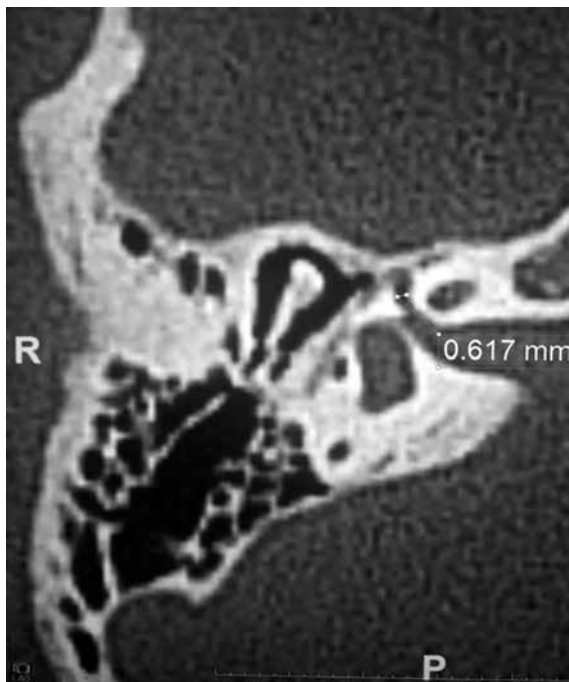


Figure 2. Measurement of labyrinthine segment diameter at axial plane.

Statistical analysis

Statistical analysis was performed using the IBM SPSS version 24.0 software (IBM Corp., Armonk, NY, USA). Descriptive data were expressed in mean±standard deviation (SD), median (min-max), or number and frequency. The Shapiro-Wilk test was used to evaluate the normality distribution of variables. The Mann-Whitney U test was used to compare the surface area of the mastoid cells and the facial canal length. The independent samples t-test was used to compare the diameters of the canal at the mastoid segment and foramen level. A *p* value of <0.05 was considered statistically significant.

RESULTS

Of all patients, six had complete facial paralysis and 17 had incomplete facial paralysis. Thirteen of the patients had right-side involvement and 17 had left-side involvement.

The mean diameter of the canal along the mastoid segment on the affected side was 1.509±0.1349 (range, 1.21 to 1.83) mm while the diameter of the canal on the unaffected side was 1.6239±0.1302 (range, 1.31 to 1.87) mm. The mean diameter of the labyrinthine segment was 1.12±0.48 (range, 0.61 to 2.23) mm on the affected side and 1.27±0.53 (range, 0.95 to 1.49) mm on the unaffected side. The mean diameter of the mastoid segment was 1.509±0.134 (range, 1.33 to 1.84) mm on the affected

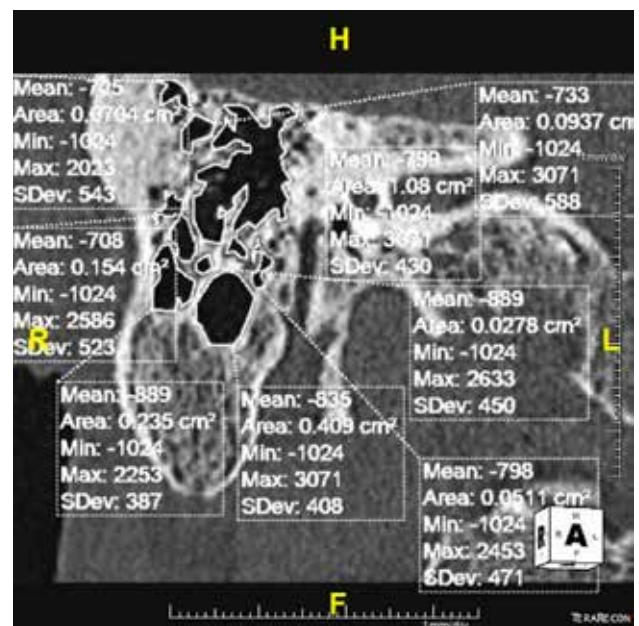


Figure 3. Total mastoid cell pneumatization was calculated by measuring each cell surface area at sagittal plane.

Table 1
Facial canal diameter at three different segments

	Affected side	Unaffected side	<i>p</i> *
	Mean±SD	Mean±SD	
Mastoid segment (mm)	1.509±0.134	1.623±0.13	0.001
Labyrinthine segment (mm)	1.12±0.48	1.27±0.53	0.001
Foramen stylomastoideum (mm)	1.561±0.124	1.681±0.119	0.001

SD: Standard deviation; * Independent sample t test.

side and 1.623±0.13 (range, 1.45 to 1.86) mm on the unaffected side. At the foramen level, the mean diameter of the canal was 1.5615±0.124 (range, 1.23 to 1.81) mm on the affected side and 1.6815±0.119 (range, 1.4 to 1.87) mm on the unaffected side (Table 1). In addition, the mean length of the canal was 15.335±1.588 (range, 11.9 to 17.7) mm on the affected side and 14.485±1.263 (range, 11.2 to 18.1) mm on the unaffected side (Table 2). The mean mastoid cell surface area was 4.137±1.298 (range, 2.482 to 8.361) cm² on the affected side and 4.559±1.393 (range, 2.84 to 7.121) cm² on the unaffected side (Table 3).

The mean facial canal diameters measured along the mastoid segment, labyrinthine segment, and at the level of stylomastoid foramen were significantly lower than the unaffected side (*p*=0.001 for all) (Table 1). There was also a statistically significant difference between the two groups in terms of the mean canal length (*p*=0.043) (Table 2). The mean canal length on the affected side was significantly longer than the unaffected side. However, there was no significant difference between the mean total cell surface area on both sides (*p*=0.359) (Table 3).

Table 2
Facial canal length in mastoid segment at both sides

	Mean±SD	<i>p</i> *
Mastoid segment		
Affected side (mm)	15.335±1.588	0.043
Unaffected side (mm)	14.485±1.263	

SD: Standard deviation; * Mann-Whitney U test.

Table 3
Total mastoid cell pneumatization level at both sides

	Mean±SD	<i>p</i> *
Affected side (cm ²)	4.137±1.298	0.359*
Unaffected side (cm ²)	4.559±1.393	

SD: Standard deviation; * Mann-Whitney U test.

DISCUSSION

Bell's palsy is the most common cause of peripheral facial paralysis and arises from acute dysfunction of the facial nerve in the temporal bone. Several factors are blamed for the etiology in BP including such as viral infections, peripheral vascular diseases, thromboembolism, and immunological factors until now.^[10] Today, the most common view in BP etiology is inflammation due to the reactivity of the herpes simplex type 1 virus located in the geniculate ganglion.^[2] Thus, depending on the developing inflammation, the nerve is trapped in the canal, its nutrition is impaired, and nerve functions are lost due to developing ischemia. The anatomical structure of the facial canal also plays a role in the development of ischemia. It has been suggested that nerve damage occurs most commonly at the level of the labyrinthine segment.^[4] This is attributed to the fact that the narrowest part of the facial canal throughout the infratemporal course is the labyrinthine segment. However, May and Schaitkin^[10] reported that the narrowest region of the facial canal was the point at which the nerve enters the canal and that the mean diameter there was about 0.68 mm. In addition, previous studies have shown that the facial nerve has different thicknesses in each segment of the canal, and the percentage of free space varies. At the meatal segment level, the facial nerve fills the 83% of the canal, whereas this percentage is about 23% in the tympanic segment, and 64% in the mastoid segment.^[11] Therefore, the other segments of the facial nerve being narrower than normal may play a predisposing role in etiology. The narrowest part of the facial nerve canal is at the internal auditory canal entrance with a mean diameter of 0.68 mm.^[10]

To date, several studies have been carried out on the role of temporal bone anatomy and facial canal structure in etiology of the development of BP. Previously, studies were performed to compare the structure of facial canal of the paralyzed side and the contralateral side. In a controlled, histopathological study of the temporal bones

of patients with facial paralysis, Vianna et al.^[12] reported that the facial canal diameter was significantly narrower at the tympanic and mastoid segment levels. However, it is not always possible to perform a histopathological study of the facial canal anatomy. Recent advances in CT imaging technology can show detailed anatomic structures. The usefulness of 3D CT in the evaluation of the facial nerve canal has been reported.^[13,14] The multiplanar reconstruction technique is one of the new imaging techniques for CT. For this purpose, Murai et al.^[13] examined the CT scans of temporal bone in BP patients with multiplanar reconstruction method and measured the mean diameter of the facial nerve at the level of the labyrinthine, horizontal, and mastoid segments on both sides. It was found that the mean facial canal diameter was significantly narrower at the level of labyrinthine segment on the affected side. Kefalidis et al.^[6] also scanned the temporal bone CT of 25 patients who experienced BP and found that the mean facial canal diameter at the meatal foramen and labyrinthine segment levels was narrower on the affected side. In our study, we found that the facial canal diameter was narrower at all three measurement points on the affected side ($p=0.001$), similar to the previous studies.

Although there are studies to evaluate the facial canal diameter, there is a very limited number of studies which evaluate the relationship between the canal length, mastoid pneumatization, and BP. Mastoid segment forming the vertical segment is the part of the nerve between the second genu and stylomastoid foramen. This is the longest part of the facial canal and is approximately 10 to 14 mm in length.^[13] It may be considered that the length of the mastoid segment, particularly the longest course of the canal, may be a predisposing factor for the BP. Kim et al.^[14] measured the length of the greater petrosal facial nerve and found the nerve length to be shorter on the side affected by paralysis. In our study, the canal length on the affected side was significantly longer than on the opposite side ($p=0.043$). A longer course of facial nerve canal may increase the risk of nerve to be affected in the canal.

Furthermore, we also evaluated the relationship of mastoid cell pneumatization level with BP in our study. Some otologic diseases are thought to be associated with mastoid cell pneumatization.^[15,16] In this context, it is prevalent that the low level of mastoid pneumatization, particularly in the development of chronic otitis media, plays a role in the etiology of the disease.^[15] There are also studies which show an association between the level of mastoid pneumatization and the etiology of otosclerosis.^[16] In the present study, the level of pneumatization might have yielded an effect on the facial canal anatomy. In addition, another theory that

pneumatization may have an impact on the development of BP was the heat exchange conduction mechanism. Some authors have suggested that dry air and cold are independent factors in the development of facial paralysis, while some others have found no relationship between the seasons and climates and facial nerve paralysis.^[17-19] Therefore, we believe that the level of mastoid bone pneumatization may be effective in terms of reflecting the sudden temperature changes in the external environment into the bone. However, based on our study results, we found no significant difference between the two sides ($p=0.359$). Similarly, no significant difference was found in a recent study evaluating the relationship of mastoid pneumatization with BP.^[20]

Nonetheless, there are some limitations to this study. First, the retrospective design with a small sample size is the main limitation. Second, we were unable to compare the results of BP patients with healthy controls. Therefore, further large-scale, prospective, controlled studies are needed to confirm these findings.

In conclusion, the anatomical features of the temporal bone and facial canal are the factors which contribute to the development of idiopathic peripheral palsy. In particular, it is possible to make a further evaluation about the 3D anatomic structure of the facial nerve and temporal bone by the multiplanar reconstruction method. In the present study, we measured the canal size at the level of mastoid segment and stylomastoid foramen and found a significant narrowness in the canal size. However, we found no significant difference in the canal length and mastoid pneumatization. Based on these results, we suggest that the diameter of the facial canal is particularly relevant in BP etiology for the anatomy of the canal. With the development of technology in the future, detailed examinations to be carried out in larger series would produce much more definitive data on this subject.

Declaration of conflicting interests

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