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Original Article

Can carotid canal diameter be an indicator of anterior cerebral vascular variations and abnormalities?

Karotid kanal çapı anterior serebral vasküler varyasyonların ve anomalilerin gösterilmesinde yol gösterici olabilir mi?

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Abstract

Aim: To measure the bony carotid canal diameters and to determine whether the variations of bony carotid canal width could be an indicator of cerebral vascular abnormalities and variations.

Material and Methods: Seven-hundred neck-brain CT angiographies were assessed retrospectively. Of the patients, 283 (40.4%) were women and 417 (59.6%) were men. Bilateral bony carotid canal diameter was measured. Cerebral vascular variations and aneurysms were recorded.

Results: Normal canal diameter on the right, in all patients was 5.631±0.502mm, in males 5.797±0.475mm and 5.388±0.441mm in females; on the left side, 5.666±0.512mm overall, 5.825±0.492mm in males and 5.432±04.49mm in females (p=0.039, <0.001 and <0.001 consequently).

In vascular hypoplasias, in all other vascular agenesias other than posterior communicating (Pcom) artery, in Moyamoya disease, in anterior and middle cerebral artery aneurysms the canal is narrow.

In the presence of fetal originated vessels, in dolichoectasias of vessels, except PCom, in all anterior communicating artery (ACom) variations and aneurysms, in internal cerebral artery aneurysm the canal is wide.

PCom agenesia, anterior cerebral artery A2 trifurcation and ACom fenestration is accompanied by narrow canal on the right and wide canal on the left.

Conclusion: Abnormal canal diameter may indicate to vascular variation or abnormaliti. The increased incidence of aneurysm in carotid canal anomalies implicates of the necessity of further studies with larger groups.

Keywords: cerebral; vascular; carotid; canal; CTA

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ÖZ

Amaç: Karotid kanal çapının ölçülmesi ve kanal çapındaki değişikliklerin serebral vasküler anomaliler ve varyasyonların göstergesi olup olamayacağının belirlenmesi amaçlandı.

Gereç ve Yöntemler: Yedi yüz beyin-boyun bilgisayarlı tomografi incelemesi retrospektif olarak değerlendirildi. Hastaların 283'ü (40.4%) kadın, 417'si (59.6%) erkekti. İki taraflı karotid kanal çapı ölçüldü. Serebral vasküler varyasyonlar ve anomaliler kaydedildi.

Bulgular: Sağ karotid kanal çapı tüm hastalarda 5.631±0.502mm, erkeklerde 5.797±0.475mm ve kadınlarda 5.388±0.441mm; sol karotid kanal çapı tüm hastalarda 5.666±0.512mm, erkeklerde 5.825±0.492mm ve kadınlarda 5.432±04.49mm bulundu (p=0.039, <0.001 ve<0.001).

Vasküler hipoplazilerde ve posterior komünikan arter (PCom) dışındaki diğer vasküler agenezilerde, Moyamoya hastalığında, anterior ve orta serebral arter anevrizmalarında karotid kanal dardır.

Fetal orjinli posterior serebral arter varlığında, PCom dışındaki dolikoektazilerde, anterior komünikan arter (ACom) varyasyon ve anevrizmalarında, internal serebral arter anevrizmasında karotid kanal geniştir.

PCom agenezisi, anterior serebral arter A2 trifukasyonu ve ACom fenestrasyonu sağda dar kanal solda geniş kanal ile birliktelik göstermektedir.

Sonuç: Karotid kanal çapındaki anormallikler, serebral vasküler varyasyon ve anevrizmaların göstergesi olabilir. Karotid kanal anormalliklerinde artan anevrizma insidansı daha geniş hasta gruplarında daha geniş çalışmaların gerekliliğini göstermektedir.

Anahtar kelimeler: serebral; vasküler; karotid; kanal; BTA

Introduction

In the absence of internal carotid artery (ICA), the bony carotid canal does not develop as well. In ICA hypoplasia, on the other hand, the canal is narrower than normal.[1] Other rare variations such as persistent stapedial artery and aberrant internal carotid artery also cause abnormalities in the carotid canal [2]. Moyamoya disease is characterized by ICA stenosis, carotid canal narrowing and increase in leptomeningeal vascularization secondary to ICA stenosis [3].

Starting from the effect of these variations and abnormalities on carotid canal, it does not seem extraordinary to think that changes in carotid canal diameter can provide clues about cerebral vascularization.

The objective of this study is to measure the bony carotid canal diameters and to determine whether the variations of bony carotid canal width could be an indicator of cerebral vascular abnormalities and variations.

Material and Methods

The research was reviewed and approved by the local ethics committee (Ankara Numune Educational and Research Hospital Ethics Committee) with the file number B.10.4.ISM.4.06.00.13/40045.

Images containing motion artifacts impairing the evaluation of the vascular tree or carotid canal, or metalic artefacts were excluded from the study. Cranial trauma and surgical history, malignities, acromegaly, fibrous displasia and all tumors and inflammatory disorders affecting the skull base were of the exclusion criterias.

Otherwise, patients older than 18 year of age with computerized tomography angiography (CTA) examinations done with different indications such as transient ischemic attack and stroke, vertigo, headache, or balance, movement, hearing and visual disorders between January 2013 and January 2014 were evaluated retrospectively. The study included 700 patients, of which 283 women (%40,4) and 417 men (%59,6).

CT Angiography Imaging Protocol:

Examinations were performed with 64-detector computerized tomography device (Aquilion 64, Toshiba Medical Systems, 2011, Japan). The parameters used in the examinations are as follows: collimation 64x0.5, gantry rotation time 0.5 sec, slice thickness 0.5 mm, pitch value 0.64 and X-ray tube 120kV & 450mA.

The patients were placed in supine position, their heads placed into a cranium headpiece in order to immobilize the head. Scanning in caudocranial direction was done after

determining localization on lateral topography and telling the patients to breathe shallow and not to swallow. Contrast material containing intravenous 100 ml non-ionic, high iodine concentration (350-400 mg/ml iodine concentration) was injected from an antecubital vein via 18-20 G catheter with 4 ml/sec speed using an automatic pump (Ulrich medicine technical version, 2004, Germany). After the contrast material, 40 cc of normal saline was administered. Bolus tracking technique was used during scannings. A cursor was placed on the aortic arch and the scan was started when density reached 130 Hounsfield Units (HU). Total scanning lasted approximately 9.6 seconds.

The obtained CTA images were examined using Aquarius® (iNtuition edition version, California, USA) software. After axial sections were examined, multi-planar reconstruction, volume rendering and maximum intensity projection reconstructions were used for assessement. HP ZR2440W (24", 1920x1200@60Hz) and ASUS PB278Q (27", 2560x1440@60Hz) monitors were used to assess the obtained images.

After the patient is layed on the table the head positioning may not always be symetrical. Furthermore, after being positioned the patient may disrupt his/her position. Therefore, prior to the measurements, sagittal plane adjustments of the hard palate, coronal plane adjustments parallel to the large sphenoid wings have been made and the measurements were obtained considering the internal acoustic canal symmetry (Fig. 1a, b.). All the measurements were made by the same radiologist experienced in head and neck radiology. Before the measurements were made, intraobserver reliability was calculated by obtaining double measurements of 30 carotid canals on images with optimized positions as described above (Intraclass correlation coefficient 0.999, p=<0.001), thus the measurements were found to be compatible.

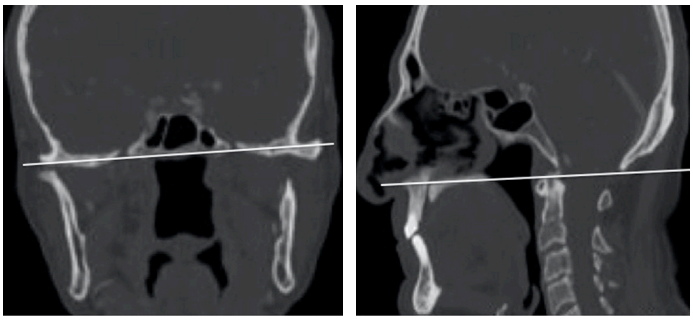

Figure 1a, b. Prior to measurement, symmetry was obtained to the large sphenoid wing on coronal plane, and parallel to the hard palate on sagittal plane.

Bone-to-bone measurements were done on axial sections, parallel to the skull base in CT bone window. Measurement was taken from the middle region of the transverse part of the petrous carotid canal (Fig. 2). Variations and abnormalities in anterior cerebral circulation were recorded. Since variations and anomalies of anterior communicating artery and anterior cerebral artery of A2 segment are in midline, they were accepted as bilateral because of affecting both ICAs.

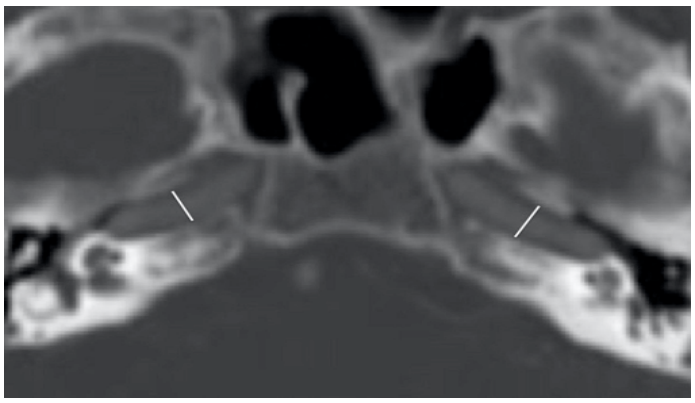

Figure 2. Measurement method of the carotid canal. Bone-to-bone measurement from the middle of the canal perpendicular to the canal wall was made.

Statistical Analysis

The data obtained at the end of the study were analyzed using SPSS 15.0 (Statistical Package for Social Sciences, 15.0, SPSS Inc., Chicago, USA) package software.

Intraclass correlation coefficient of 30 patients was calculated in order to evaluate the intraobserver reliability.

Continuous data distribution was heterogeneous, therefore non-parametric tests have been used. When comparing independent groups, Mann-Whitney U test was used for continuous variables, whereas Wilcoxon test was used when comparing dependent groups. Comparison of discrete variables between groups was assessed with Chi-square test.

Right-left canal diameters were compared with paired samples test, and canal diameters with respect to gender were compared with independent samples test.

P<0.05 value was accepted as significant. Descriptive statistic values were given as number and average percentage.

Results

The mean age of 700 cases assessed was 60.04 (19-89), 58.95 (24-89) for women and 60.79 (19-88) for men. In 345 (49.2%) (205 male (59.4%), 140 female (40.6 %) of these patients no variations or abnormalities have been detected. Normal canal diameters and gender distribution are presented on the Table. The canal is bilaterally more narrow in females (p<0.001). The canal diameter is more narrow on the right side of all patients (p=0.039).

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Variations or abnormalities were grouped as narrow compared to normal canal diameter or with wide canal diameter and presented on the Table. As a result of this classification, in ACA A2 trifurcation, ACom fenestration and PCom agenesia the canals were not bilaterally wide or narrow; narrow on the right and wide on the left. Others were found to be narrow or together with a wide canal.

Agenesia, hypoplasia and dolichoectasia were detected in the internal cerebral artery (ICA). Carotid canal diameter difference exists in hypoplasia (Figs. 3a and b) and dolichoectasia (Figs. 4a and b), but only the difference in hypoplasia was found significant ($p<0.05$). Both two carotid canals are also prominently narrow in Moyamoya case, which mainly affects the ICA (Figs. 5a and b). Agenesia, hypoplasia, dolichoectasia and fenestration were detected in anterior cerebral artery (ACA) A1 segment and anterior communicating artery, while hypoplasia, dolichoectasia, trifurcation and azygos were detected in A2 segment.

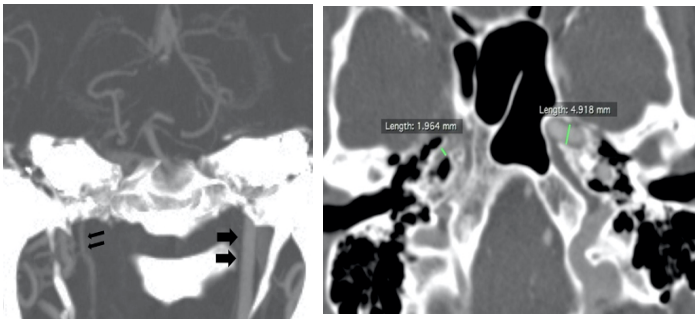

Figure 3a, b. 50-year-old man with internal cerebral artery (ICA) hypoplasia. Coronal maximum intensity projection CT image **(a)** shows right internal cerebral artery thinning (narrow arrows), the left is with normal calibration (wide arrows). On axial CT image **(b)**, right carotid canal is small, supporting ICA hypoplasia.

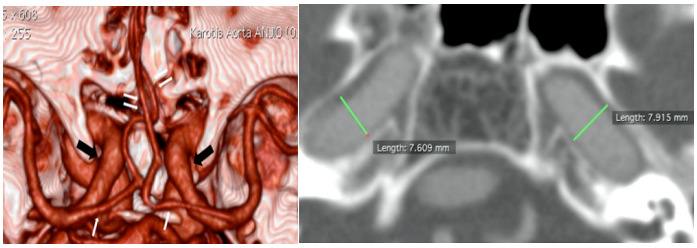

Figure 4a, b. 70-year-old woman with internal cerebral artery (ICA) and anterior cerebral artery (ACA) dolichoectasia. Volume rendering CT image **(a)** shows dolichoectasia both in ICA (wide black arrows) and ACA (narrow white arrows). On axial CT image **(b)** carotid canals are widened together with dolichoectasia.

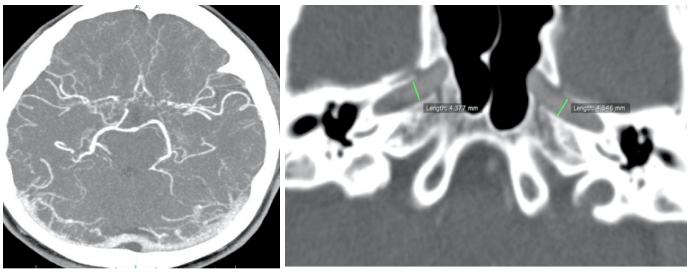

Figure 5a, b. 48-year-old woman with Moyamoya disease. Axial maximum intensity projection CT image of Moyamoya case **(a)** showing increased leptomeningeal vascularization secondary to anterior cerebral artery occlusion seen bilaterally. Axial CT image **(b)** shows bilaterally smaller carotid canals.

Ipsilateral carotid canal is narrow in cases of agenesia and hypoplasia at ACA A1 segment, while in fenestration and dolichoectasia the carotid canal is wider. Canal narrowness is significant in ACA A1 agenesia and right sided hypoplasias ($p<0.05$).

In ACA A2, hypoplasia and trifurcation are present with canal narrowness, while dolichoectasia and azygos A2 cases are present with expansion in canal (Figs. 6a, b and c).

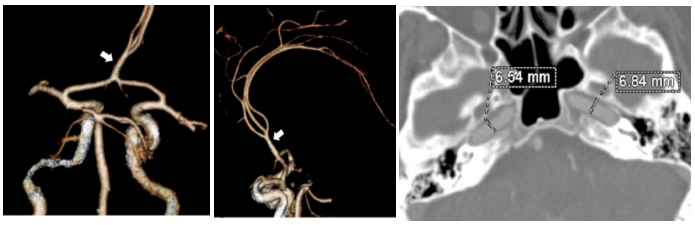

Figure 6a, b, c. 62-year-old man with azygos anterior cerebral artery. Volume rendering oblique axial **(a)** and sagittal **(b)** CT images show azygos anterior cerebral artery A2 segment. Axial CT image **(c)** shows widening of the carotid canal.

Anterior communicating artery agenesia, hypoplasia and dolichoectasia are present with expansion in canal, while fenestration does not fit any certain rule.

Carotid canal was observed narrower in middle cerebral artery (MCA) hypoplasia cases. MCA duplication did not affect carotid canal diameter.

Carotid canal is significantly wider in fetal origin of posterior cerebral artery cases ($p<0.05$). Carotid canal was detected minimally narrower in posterior communicating artery hypoplasia, and in agenesia cases wide at one side and narrow at the other side. All these differences were not found statistically significant.

The carotid canal was wider in the presence of persistent trigeminal artery and hypoglossal artery (Figs. 7a, b and c). However, statistical difference was not detected.

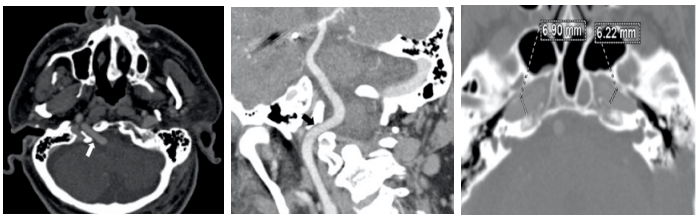

Figure 7a, b, c. 60-year-old man with persistent hypoglossal artery. On the axial **(a)** and oblique **(b)** reformatted CT images, persistent hypoglossal artery passes through the widened right hypoglossal canal. Axial CT image **(c)** shows widening of the carotid canal.

The carotid canal was observed wide in one of the cases of ICA clinoid segment aneurysm, while it was observed narrow in 4 cases. On the other hand, on left supraclinoid segment aneurysms, the carotid canal is narrow and the difference is significant (p=0.018).

The carotid canal was observed wide in anterior communicating artery aneurysms (Figs. 8a and b). In right-sided A1 and A2 aneurysms, the canal was observed narrow whereas it was observed wide in left-sided A2 aneurysms. But the difference is insignificant (p>0.05).

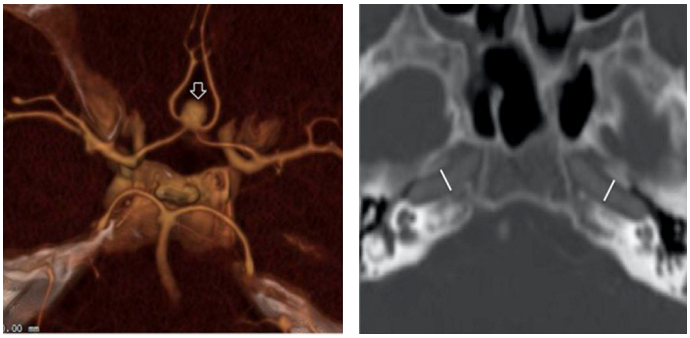

Figure 8a, b. 36-year-old man with anterior communicating artery aneurysm. Volume rendering image shows anterior communicating artery aneurysm **(a).** Axial CT image **(b)** shows widening of the carotid canal.

The carotid canal is generally narrower than normal in MCA aneurysms as well. The narrowing at right side in MCA bifurcation/trifurcation aneurysms is near to the significance limit (p=0.065), and significant at left side (p=0.019). Moreover, the difference in other segment aneurysms is not significant.

In posterior communicating artery aneurysms, one smaller and one wider carotid canal in comparison to normal were detected in two patients, which is insignificant.

When aneurysms are generally considered, carotid canal was found smaller in aneurysm cases.

Discussion

In medicine, particularly in radiology, there is an effort of clarifying of more complex diseases/issues using simple findings. An example for this effort is whether the abnormalities in bony carotid canal diameter would be a clue about cerebral circulation. Although partially, this subject is known. Indirect findings such as absence of bony carotid canal in ICA agenesia and bilateral small canal in Moyamoya disease are known [3-6]. However, variations in carotid canal diameter have not been verified in all cerebral arterial variations and abnormalities.

In this study, carried out for the reasons described above, the diagnosis was made with the absence of carotid canal in the case with ICA agenesia, thus it was distinguished from full occlusion [4,6]. Similarly, ICA hypoplasia was accompanied by small carotid canal and as a result a diagnosis was made [1]. This is because bony canal develops after ICA. Tanaka et al. [7] have found that the flow velocity in carotid and basilar artery changes significantly in the existence of Willis variations in 125 cases using cine phase-contrast magnetic resonance imaging. Besides, in an empirical study performed with three-dimensional phantom models, agenetic arteries have been found to alter the cerebral hemodynamics [8]. In the presented study, ICA and ACA A1 agenesia, ICA, ACA, MCA and PCom hypoplasias were accompanied by a narrow carotid canal; ACA A1 fenestration, azygos ACA A2, ACom agenesia and hypoplasia and MCA duplication were accompanied by a wide canal. ACA A2 trifurcation, ACom fenestration and PCom agenesia were accompanied by a narrow canal on the right and wide canal on the left. In a study of intracranial MR angiograms by Kane et al. [9] the correlation between ipsilateral absence or hypoplasia of the A1 segment and a reduced caliber of the ICA has been also reported. Similarly, the canal was also observed slightly smaller in posterior communicating artery hypoplasia.

Two of the very few studies done on carotid canal width are about Moyamoya disease. Bilateral bony carotid canal was revealed significantly small in Moyamoya cases according to the researches done in Japan, where the disease is frequently seen [3, 5]. In the study done by Watanabe et al. [3], carotid canal diameter was found as 5.27±0.62mm in the control group. The bony carotid canal diameter was measured as 3.31±0.44mm in 11 cases having Moyamoya disease. In another study, carotid canal diameter in the control group was 5.62±0.61mm, whereas it was measured as 4.70±0.61mm for 25 person group having Moyamoya disease.[5] Moyamoya disease, which is seen fairly seldom, is characterized by progressive stenosis of ICA's terminal segment and main branches. Its incidence is higher in East Asia[3]. In the presented study, it was seen that bilateral carotid canals in one case are smaller in comparison to the control group.

One of the abnormalities presented with the expansion of the canal is dolichoectasia. Dolichoectasia is characterized with arterial elongation and expansion. It is an arteriopathy apart from atherosclerosis and even if association is seen, there

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Table. Classification of normal carotid canal widths, variationts or abnormalities based on wide or narrow canal

	Right			Left	
Name of Variable	n	mean±SD (min-max)	p	n	mean±SD (min-max)
Normal canal diameters and gender distribution					
Normal diameter	345	5.631±0.502 (4.1-7.0)	0.039a	345	5.666±0.512 (4.4-7.2)
Men	205	5.797±0.475 (4.5-7)	<0.001	205	5.825±0.492(4.6-7.2)
Women	140	5.388±0.441 (4.1-6.9)	<0.001	140	5.432±04.49(4.4-6.9)
With narrow canal diameter					
ICA agenesia	1	∞	NA	1	∞
ICA hypoplasia	4	4.414±0.121 (2.4-5.6)	0.002*	4	5.275±0.340 (5.0-5.7)
Moyamoya disease	1	4.377	NA	1	4.546
ACA A1 agenesia	12	5.175±0.543 (4.2-6.5)	0.003*	3	5.500±0.435 (5.0-5.8)
ACA A1 hypoplasia	79	5.441±0.506 (3.6-6.6)	0.005*	34	5.603±0.532 (4.4-6.6)
ACA A2 hypoplasia	11	5.609±0.550 (5.0-6.5)	0.74	6	5.450±0.197 (5.3-5.7)
ACA aneurysm	9	5.400± 0.400 (5.0-6.0)	0.23	19	5325±0.472 (4.5-6.2)
ACA A2 trifurcationb	6	5.483±0.828 (4.4-6.7)	0.67	-	-
ACom Fenestrationb	2	5.550±0.156 (5.4-5.6)	NA	-	-
MCA aneurysm	24	5.344±0.439 (4.7-6.3)	0.004*	24	5.514±0.554 (4.3-6.7)
MCA hypoplasia	5	5.217±0.519 (4.5-6.1)	0.05	5	5.380±0.295 (5.0-5.8)
PCom agenesiab	14	5.414±0.704 (4.3-6.5)	0.24	-	-
PCom hypoplasia	33	5.564±0.476 (4.6-6.6)	0.40	29	5.628±0.390 (5.0-6.5)
PCom ectasia	3	5.433±0.493 (5.1-6.0)	0.47	1	5.200
TOTAL	204			127	
With wide canal diameter					
ICA ectasia	3	6.833±0.125 (5.4-7.7)	0.09	3	6.867±0.110 (5.8-8.0)
ICA aneurysm	7	5.783±0.560 (5.1-6.5)	0.52	0	-
ACA A1 ectasia	4	6.050±0.118 (4.9-7.7)	0.55	5	6.383±0.890 (5.7-8.0)
ACA A2 ectasia	2	6.300±0.198 (4.9-7.7)	NA	3	6.500±1.300 (5.7-8.0)
ACA A1 Fenestration	0	-	NA	1	5.700
Azygos ACA A2	7	6.043±0.591 (5.1-6.7)	0.06	7	6.090±0.414 (5.5-6.8)
ACA A2 trifurcationb	-	-	NA	6	5.650±0.852 (4.6-6.8)
ACom Fenestrationb	-	-	NA	2	5.750±0.354 (5.5-6.0)
ACom agenesia	5	5.820±0.572 (5.1-6.5)	0.44	5	6.160±0.487 (5.3-6.5)
ACom hypoplasia	8	5.800±0.462 (5.2-6.4)	0.36	8	5.938±0.489 (5.2-6.8)
ACom ectasia	1	6.000	NA	1	6.200
ACom aneurysm	16	5.733±0.613 (4.7-6.7)	0.51	16	5.794±0.446 (5.1-6.7)
MCA duplication	0	-	NA	2	5.700±0.707 (5.2-6.2)
PCom agenesiab	-	-	-	18	5.717±0.543 (5.0-6.9)
Fetal PCA	53	5.750±0.395 (5.0-6.5)	0.06	51	5.875±0.450 (4.6-7.0)
P. trigeminal arteryc	1	6.124	NA	1	5.700
P. hypoglossal arteryc	2	6.300±0.282 (6.1-6.5)	NA	0	-
TOTAL	109			129	

aComparison of left-right normal canal diameter. NA: Not available (No sufficient data for comparison).
bVariations with narrow carotid canal on one side and wide carotid canal on the other side.
c P=Persistent
Ectasia: is used to mean dolichoectasia.
ICA: Internal cerebral artery, ACA: Anterior cerebral artery, PCA: Posterior cerebral artery, MCA: Middle cerebral artery, AcomA: Anterior communicating artery, PcomA: Posterior communicating artery.
*p<0.05.

are proofs indicating them to be different entities [10,11]. In the present study, it was seen that the carotid canal was enlarged in dolichoectasia of ICA, ACA and ACom. It is claimed that dolichoectasia is a systemic pathology [10,11] and the reason for canal extension in ACA dolichoectasia is probably because of segmental dolichoectasia in concomitant ICA. The enlargement of left ICA and ACA A1 dolichoectasias were found to be statistically significant.

If an enlarged canal is accompanied by a variation, then it is fetal originated PCA, which is frequently encountered. It can be asumed that carotid canal diameter would also enlarge in persistent carotid-vertebrobasilar anastomosis. Because generally the posterior system is hypoplastic and it is supplied with a persistent artery extending from the anterior to the posterior. Enlargement in fetal originated PCA is near significance level (p=0.06) on one side, and significantly enlarged (p=0.004) on the other side. The canal is also enlarged in persistent trigeminal and hypoglossal artery cases, however the number of participants was insufficient for statistical evaluation.

Aneurysms affect carotid canal diameter as well. A high correlation was detected between the unilateral or bilateral internal carotid artery absence and Willis polygon aneurysm [12]. It is thought that Willis polygon variations affect hemodynamics and can cause aneurysm development. There are only few studies researching the correlation between Willis polygon variations and aneurysm [13-16]. In the study done by Krasny et al. [15] using digital subtraction angiography, ACA A1 segment variations were observed more frequently in aneurysm group compared to the control group. Besides, there are studies indicating the correlation between PcomA variations and aneurysm[13,17,18]. Lazzaro et al. [19] have detected that Willis polygon variations are more frequent in ruptured AComA and PComA aneurysms in comparison to the group without rupture. However, on contrary to all those studies no correlation was detected between variation existence and aneurysm in the presented study.

In the presented study, ACA and MCA aneurysms were accompanied by bilateral narrow carotid canals, the narrowness level was statistically significant on the right in ACA aneurysms and on the left in MCA aneurysm. The carotid canal is wide in ICA and ACom aneurysms. However, this findings was statistically insignificant probably because of the low number of participants. Similarly, in the study of Kim et al. [20] supraclinoid ICA was found wide in AcomA aneurysm cases. In two studies, the ACA A1 segment was found wider at the side of AComA aneurysm [20,21]. In one of those studies, it was reported that A1:A2 ratio increased, in other words A2 is thinner in comparison to A1[21]. In our study, parent artery diameter was not measured. In future studies, researching the correlation between parent artery diameter and same side carotid canal diameter could be instructive on aneurysm detection.

What is of importance in aneurysms is the time of occurrence. Congenital aneurysms may lead to changes in the carotid canal. However, aneurysms seen in most of the adults may not lead to any changes. Therefore, unless aneurysms are differentiated as congenital/acquired, a precise rule regarding the canal width might not be made. If we consider that the canal diameter affects the aneurysm, on contrary of the aneurysm affecting the canal, the increase of flow in a narrow canal might trigger an aneurysm distally to the narrowing. This should also be proved with larger series.

The width of carotid canal may be affected from reasons other than abnormalities such as aneurysm and Moyamoya disease, and variations of the circle of Willis. Therefore, disorders such as acromegaly, fibrous dysplasia, tumors and inflammatory diseases of the skull and the skull base, and due to the risk of metastasis all malignities are accepted as exclusion criteria. It should be kept in mind that extravascular factors may inhibit the fisibility of canal diameter measurement.

Normal carotid canal diameter values obtained from the presented study can be used in routine health screening. However, significant diameter difference was detected between right-left sides. Left carotid artery originates from the aortic arch. Therefore, left carotid artery might have wider diameter since it is exposed to higher pressure. The difference between men-women was found significant as well. Therefore, it should be known that the measurement alters with respect to gender and side.

This study has two important limitations. Firstly, there are relatively few cases per each abnormality when the cases are distributed, and secondly only CTA imaging was performed. Due to varying incidence of the mentioned abnormalities, homogeneous distribution was not observed in patient groups admitted to the study. Therefore, statistically significant result cannot be obtained. Multicentered studies including larger numbers of patients would be of benefit. CTA sensitivity decreases in very small aneurysms [22]. Thus, there is a probability of missing some aneurysms.

As a result, if we exclude extravascular reasons that may affect the canal, carotid canal diameter different than normal ranges

may indicate to abnormalities such as vascular variations or aneurysm. Variations are not very important alone, however some previous studies have shown their cooccurrence with increased aneurysm incidence. Although the relatively low number of patients in this study was an obstacle to create a rule, further multicentered larger studies may clarify this topic, and provide the premise of CTA use.

Declaration of conflict of interest

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