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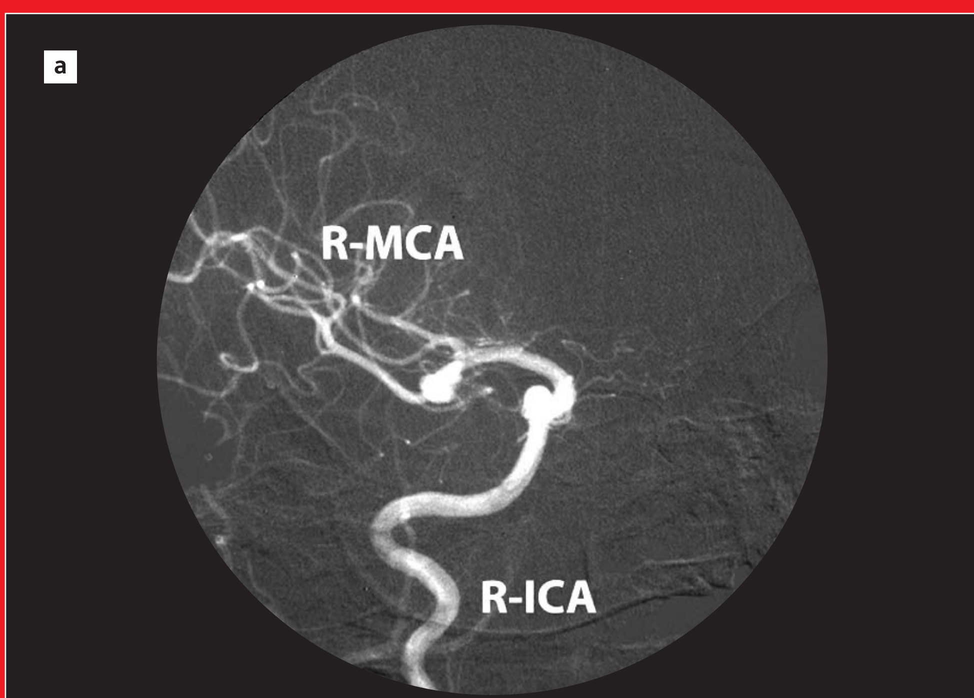

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Coracoclavicular joint: clinical significance and correlation to gender, side and age

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Abstract

Objectives: The aim of this study was to evaluate the incidence of coracoclavicular joint in the Greek population, review the clinical significance for the orthopaedic surgeon and investigate differences between genders, sides and age that may contribute to a better understanding of the accessory joint development.

Methods: The study was performed on the scapulae and claviculae of 140 dried skeletons taken from a local ossuary. After exclusion of damaged bones, the sample of the study consisted of 216 pairs of scapulae and claviculae. Each pair of bones was inspected for the existence of a definite articular facet on the conoid tubercle of the clavicle and also on the superomedial surface of the coracoid process of the scapula. A coracoclavicular joint was considered to be present only when both of these structures existed. Pearson's chi-square test was used to investigate differences between the genders, sides and age of the specimens.

Results: Coracoclavicular joint articular facets were found in 14 out of the 216 bone pairs examined (6.5%). A statistical significant difference was found only between the age groups. The coracoclavicular joint surfaces were significantly more frequently found in the elderly age group (p=0.002). No bones from the youngest age group (45–60 years old) demonstrated a coracoclavicular joint surface, whereas three bones from the median age group (61–75 years old) and 11 from the oldest age group (76+ years old) presented accessory joint surfaces.

Conclusion: The findings of the present study favor those who claim that the coracoclavicular joint could be the result of degenerative changes. From a clinical point of view, this accessory joint may be incidentally noticed in a plain radiograph in asymptomatic patients, but has also been associated with various clinical manifestations of the shoulder region.

Keywords: anatomical variation; clavicle; osteology; scapula; shoulder pain; shoulder radiology

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Introduction

The coracoclavicular syndesmosis consists of two separate ligaments, the trapezoid and the conoid ligaments. Close to the posterior border of the clavicle, at the junction of the lateral fourth with the rest of the bone, there is a prominent conoid tubercle which gives origin to the conoid part of the coracoclavicular ligament. The trapezoid ligament originates from the oblique or trapezoid ridge which starts from the conoid tubercle and runs anteriorly and laterally. Both ligaments insert on the coracoid process, with the trapezoid ligament being anteriorly and the conoid posteriorly.[1]

The conoid tubercle of the clavicle may be enlogated, having a wide ending which is usually an articular surface that articulates with a corresponding articular surface on the footprint of the conoid ligament on the coracoid process. The synovial joint that may be found between those two surfaces is an accessory joint, named the coracoclavicular joint.[2,3] Several articles have been published on the frequency of the coracoclavicular joint and the geographical distribution of this anatomical variation is interesting.[2–6] However, the literature is scarce in studies which analyze the potential effect of the characteristics of the

This study was presented at the 6th Scientific Congress of Medical School of Aristotle University of Thessaloniki, April 7–9, 2011, Thessaloniki, Greece.

sample on the coracoclavicular joint presence. Moreover, there is an ongoing debate in the literature about whether it is hereditary,[4] or an acquired characteristic.[5]

It is well-known that shoulder pain is multifactorial. Several pathologies, such as inflammation, degenerative changes, trauma and entrapment neuropathies which are located in different regions around the shoulder might be presented with shoulder pain as the main complaint. Although the presence of the coracoclavicular joint is usually asymptomatic, it has been associated with shoulder pain due to degenerative changes and alterations in shoulder biomechanics. Thus, orthopaedic surgeons should bear in mind this accessory joint since its incidence is not rare and also it can be identified in an anteroposterior shoulder radiograph.

The purpose of the present study was to evaluate the incidence of coracoclavicular joint in the Greek population, review its clinical significance for the orthopaedic surgeons and also to examine for differences between genders, sides and age that may contribute to a better understanding of the accessory joint development.

Materials and Methods

The study was performed in the scapulae and claviculae of 140 dried skeletons (280 pairs of scapulae and claviculae) taken from a local ossuary. After exclusion of bones with evidence of fracture, postmortem damage, or arthritis, the sample of the study consisted of 216 pairs of scapulae and claviculae (110 right and 106 left), which belonged to 127 individuals (66 females and 61 males). When a scapula or a clavicle was damaged, then both bones of that side were excluded. The donors' mean age was 68.5±10.72 years (range: 46–96 years).

Regarding the age of death, the study sample was divided into three age groups. The first group consisted of 55 pairs of specimens aged 46–60 years, the second group consisted of 82 pairs of specimens aged 61–75 years and the third one consisted of 79 pairs of subjects aged over 75 years.

The presence of the coracoclavicular joint was documented by inspecting the occurrence of a definite articular facet on the conoid tubercle of the clavicle and also on the superomedial surface of the coracoid process of the scapula. Those criteria were based on previous osteological studies.[5,7]

Pearson's chi-square test was used to investigate differences between the two genders, sides and age of the specimens. A p-value of <0.05 was considered statistically significant. All analyses were conducted using SPSS 19.0 software (SPSS Inc, Chicago, IL, USA).

Results

The coracoclavicular articular facets were bilaterally present in four and unilaterally in six individuals (**Figure 1**). Thus, a coracoclavicular joint was considered to be present in 14 out of the 216 bone pairs examined (6.5%). Seven coracoclavicular joint surfaces were present on the right and seven on the left side (p=0.943), while the male to female ratio was 8:6 (p=0.509).

As for the age groups, the coracoclavicular joint surfaces were found significantly more frequent in the elderly age group (p=0.002). Namely, no bones from the youngest age group (45–60 years old) demonstrated a coracoclavicular joint surface, whereas three bones from the median age group (61–75 years old) and 11 from the oldest age group (76+ years old) presented those accessory joint surfaces. These findings are summarized in **Table 1**.

Discussion

The first description of coracoclavicular joint was made by Gruber, in 1861.[8] According to Mann and Hunt[9],

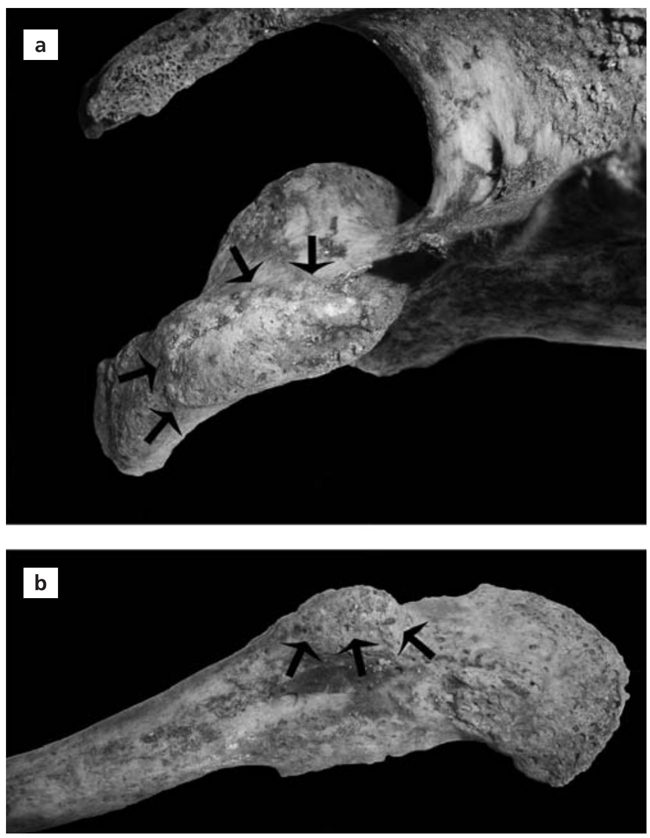

Figure 1. Articular facets (**arrows**) (**a**) on the superomedial aspect of the coracoid process, and (**b**) the conoid tubercle of the clavicle. [Color figure can be viewed in the online issue, which is available at www.anatomy.org.tr]

the frequency of this anatomical variation is 1–1.2%. However, Cockshott[4] reported that the coracoclavicular joint originated in Central Asia many years ago, where the incidence was up to 40.7% demonstrating a gradual decrease as we move away from that region. In Greece, we found the coracoclavicular joint surfaces in 6.5%, whereas in two osteological studies which were carried out in Southern Europe, the incidence of this accessory joint was reported as 0.3% and 2.8%, respectively.[7,10]

Based on the geographic distribution of the coracoclavicular joint, Cockshott argued that there is an autosomal dominant pattern of inheritance.[4] Actually, the development of the coracoclavicular joint has been a controversial issue in the literature. Kaur and Jit[11] and Pillay[12] supported that the joint may develop more by genetics than by environmental factors or congenital abnormalities. On the other hand, Lane[13] proposed that it is an acquired characteristic, resulting from occupational stress. Kaur and Jit[11] suggested that the coracoclavicular joint appears after the first decade. Nalla and Asvat[6] reported that this joint results from larger scapulae and longer claviculae coupling with a normal-sized thoracic inlet, which may restrict associated movements of the scapula. The findings of Cho and Kang,[5] study are in accordance with the present study where the coracoclavicular joint surfaces were significantly more common in the oldest age group. The existence of the coracoclavicular joint facets has been more frequently in males than females, but no study reported any significant difference.[2,14] In the present study, the accessory joint surfaces were more frequently found in males than females (8:6), without any statistical significance as well.

Nalla and Asvat found the coracoclavicular articular facets bilaterally in 47.9% and unilaterally in 30.4% of specimens.[6] The relative percentages of Olotu et al. were 11% bilateral and 89% unilateral.[3] In the osteological study of Nehme et al., the coracoclavicular joint was equally found bilaterally and unilaterally, while in the radiological study of the same authors, the joint was bilaterally present in 40% and unilaterally in 60% of specimens.[14] We observed the coracoclavicular joint facets in ten individuals, four bilateral and six unilateral. According to the majority of the literature, the coracoclavicular joint is more often located in the right side,[2,3,5,14] while Nalla and Asvat found a higher incidence in the left side.[9] In our study, both sides presented the same prevalence of coracoclavicular joint surfaces.

Table 1
Incidence of coracoclavicular joint facets, and distribution by gender, side and age.

		n	Coracoclavicular joint facets	p
Gender	Male	105	8 (7.6%)	0.509
	Female	11	6 (5.4%)	
Side	Right	110	7 (6.4%)	0.943
	Left	106	7 (6.6%)	
Age	46–60	55	-	0.002
	61–75	82	3 (3.7%)	
	76 +	79	11 (13.9%)	
Total		216	14 (6.5%)	

Although a large conoid process of the clavicle usually articulates with the coracoid process of the scapula, in our series one clavicle was found to have an elongated conoid process with a rough and slightly sharp tip, but without any joint surface. In this case, there was no articular facet on the coracoid process of the ipsilateral scapula. Furthermore, raised bony lips around the insertion of the conoid ligament were found in eight clavicles without any sign of neoarthrosis in the corresponding scapula. Raised bony lips were also found around the insertion of the coracoclavicular ligaments on the coracoid processes of four scapulae without any sign of a coracoclavicular joint on the clavicle. A bony crest or lip can also be caused by a thickened and strong ligament. Thus, we believe that the raised bony lips only on one side of those 12 bones were caused by calcifications of the coracoclavicular ligaments, and therefore they were not considered as a coracoclavicular joint.

The articular facet of the coracoclavicular joint has been recognized in plain radiographs.[14–16] CT scans may also reveal this joint.[15,16] Although the presence of the joint is usually asymptomatic, occasionally it may be related with various clinical manifestations of the region. In particular, it has been associated with a humeral head fracture,[17] shoulder pain,[18] cervicobrachial syndrome[19] and thoracic outlet syndrome.[20] Wertheimer[18] and Hall[21] supported that the presence of this accessory joint may alter the biomechanics of the shoulder since it probably decreases the shoulder mobility.[18,21] Coracoclavicular joint arthritis may be accompanied by pain in the shoulder with reflection in the neck, arm and chest.[22] Moreover, this joint might be responsible for degenerative changes on neighboring joints.[2] The coracoclavicular joint may be recognized in an anteroposterior radiograph of the shoulder. CT scans may also reveal and confirm the presence of the joint. In cases with residual symptoms, surgical resection of the accessory joint might be necessary.[20,23,24]

The limitation of the present study is that, as any osteologic study,[5,14] the presence of the coracoclavicular joint can only be assumed by findings of the joint surfaces on the clavicle and the coracoid process. The existence of a true synovial joint can only be confirmed in cadaveric studies.

Conclusion

The findings of the present study favor that the coracoclavicular joint could be the result of degenerative changes. From a clinical point of view, this accessory joint may be incidentally noticed in a plain radiograph in asymptomatic patients, but it has been also associated with various clinical manifestations of the shoulder region.

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X-ray analysis of foramen rotundum for preliminary diagnosis of fossa pterygopalatina lesions

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Abstract

Objectives: Fossa pterygopalatina is located posterior to maxillary sinus, anterior to pterygoid plates and inferior to greater sphenoid wing. Tumors that arise in fossa pterygopalatina are usually asymptomatic and often discovered at late stage. Because of its deep and relative inaccessible location, clinical examination of fossa pterygopalatina is difficult. Aplastic or nonvisible foramen rotundum may give an early indication for the regional tumors.

Methods: In the present study, foramen rotundum was visualized as a round structure with prominent sclerotic margins located near ethmoid cells, maxillary sinus or orbital cavity in X ray images. X-rays (radiographs) of 17 dry skulls and 7 half dry skulls were performed with Phillips Digital Diagnost X-Ray device. All dry skulls were positioned similar to Caldwell's view. We have designed a fixation apparatus to optimize the positioning of dry skulls and half skulls. After fixation of the skulls with the apparatus, the X-ray beam centered about 3–4 cm below the external occipital protuberance with an angle of 15 degrees to the chantomeatal line. Each radiologic image was examined for the location of foramen rotundum in relation with orbital cavity, ethmoidal cells and maxillary sinuses. We described the characteristic of foramen rotundum with count and percent values.

Results: In the present study, 41 foramina rotunda were analyzed. Out of 41 foramina rotunda, twenty were right-sided, twenty one were left-sided. Of the 20 right-sided foramina rotunda, 19 were visible on X-ray images. On the other side 18 of 21 left-sided foramina rotunda were apparent. 14 of 19 visible right-sided foramina rotunda were identified in the orbital cavity (73.68%). Three of them were identified in ethmoidal cells, and two in the maxillary sinus. 11 of 18 visible left-sided foramina rotunda were identified in the orbital cavity (61.11%). Two of these were found in ethmoidal cells, eight in the maxillary sinus. Out of 17 dry skulls, one had bilateral nonvisible foramina rotunda.

Conclusion: Foramen rotundum must be taken into consideration in evaluation of routine X-rays to prevent misdiagnosis of the patients with persistent non-specific symptoms.

Keywords: cranium; foramen rotundum; pterygopalatine fossa; X-ray

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Introduction

Fossa pterygopalatina is located posterior to maxillary sinus, anterior to pterygoid plates and inferior to greater sphenoid wing.[1] It has connections with infratemporal fossa *via* the pterygomaxillary fissure, nasal cavity *via* the sphenopalatine foramen, orbit *via* the inferior orbital fissure, oral cavity *via* the palatine foramina and middle cranial fossa *via* the foramen rotundum.[1,2] Maxillary nerve, maxillary artery and pterygopalatine ganglion are contents of fossa pterygopalatina.[3] Maxillary nerve originates at the Gasserian ganglion, passes via the foramen rotundum to exit the cranium, crosses fossa pterygopalatina and enters the infraorbital canal.[2] Fossa pterygopalatina tumors are usually asymptomatic and often discovered at late stage.[3] Clinical examination of fossa pterygopalatina is difficult due to its deep and relative inaccessible location.[4]

Orbital pseudotumors extending into fossa pterygopalatina have been reported.[5] Lesions originating in oral and maxillofacial regions and tumors from the sinonasal region may also extend to the pteryogopalatine fossa.[3] These sinonasal region tumors may be overlooked by patients and physicians because of their non-specific symptoms.[3] Some of the non-specific symptoms reported in the literature related to the pathologies of fossa pterygopalatina are nasal obstruction, headache, infraorbital pain, facial numbness, nausea and vomiting, hyposmia, rhinorrhea, hearing impairment, diplopia, exophthalmos and epiphora.[6] Foramen rotundum may be absent in a rare neurocutaneous disorder named Gómez-López-Hernández syndrome characterized by rhombencephalosynapsis, parieto-occipital alopecia, brachycephaly, facial malformations, trigeminal anesthesia.[7] Thus, aplastic or nonvisible foramen rotundum may give an early indication for aforementioned diseases. In the present study, foramen rotundum was visualized as a round structure with prominent sclerotic margins located near ethmoid cells, maxillary sinus or orbital cavity in X-ray images.

Materials and Methods

Study data were collected from Departments of Anatomy of Ankara Yıldırım Beyazıt University and Hacettepe University. 17 craniums and 7 half craniums were evaluated. X-rays (radiographs) of 17 dry skulls and 7 half dry skulls were taken at TOBB ETU University using Phillips Digital Diagnost C50 X-ray device (Philips Medical Systems, Hamburg, Germany). All dry skulls were positioned to Caldwell's viewwith the forehead and nose placed against X-ray detector. This projection is one of the most commonly used cranial X-ray projections for visualization of the paranasal sinuses, especially the frontal sinuses. We positioned the skulls and half skulls as shown in **Figure 1**. The X-ray beam was centered about 3–4 cm below the external occipital protuberance with an angle of 15° to the canthomeatal line.

Results

Each radiologic image was examined for the location of foramen rotundum in relation to the orbital cavity, ethmoidal cells and maxillary sinuses. We described the characteristics of foramen rotunda with count and percent values. In the present study, 41 foramina rotunda (20 on the right side and 21 on the left side) were examined. Of the 20 right-sided foramina rotunda, 19 were visible on X-ray images. On the other side, 18 of 21 left-sided foramina rotunda were apparent.

14 of the 19 visible right-sided foramina rotunda were identified in the orbital cavity (73.68%) (**Figure 2**), while three were identified within the ethmoidal cells, and two in the maxillary sinus (**Figure 3**). 11 of 18 visible left-sided foramina rotunda were identified in the orbital cavity (61.11%) (**Figure 2**), two were within ethmoidal cells (**Figure 4**), and 8 in the maxillary sinus (**Figure 3**). Out of 17 dry skulls, one had bilateral nonvisible foramina rotunda (**Figure 5**).

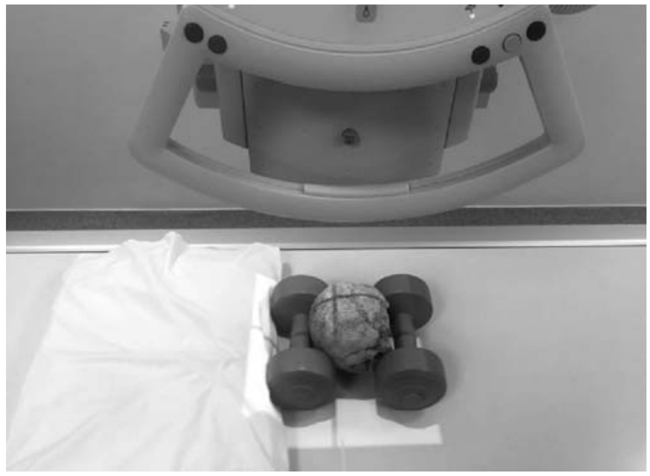

Figure 1. Cranium positioned similar to Caldwell's view against X-ray detector.

Discussion

Most of the patients with non-specific symptoms (*i. e.* nasal obstruction, headache, infraorbital pain, facial numbness, nausea and vomiting, hyposmia, rhinorrhea, hearing impairment, diplopia, exophthalmos and epipho-

Figure 2. Bilateral foramina rotunda in orbital cavities on X-ray.

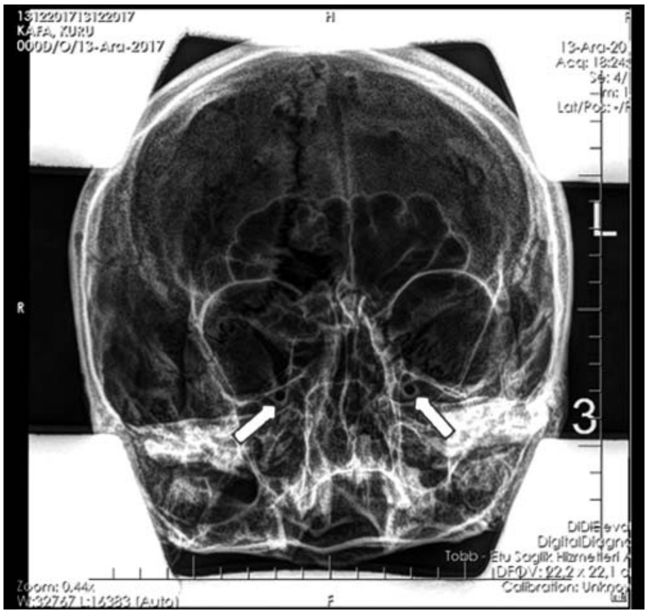

Figure 3. Bilateral foramina rotunda in maxillary sinuses on X-ray.

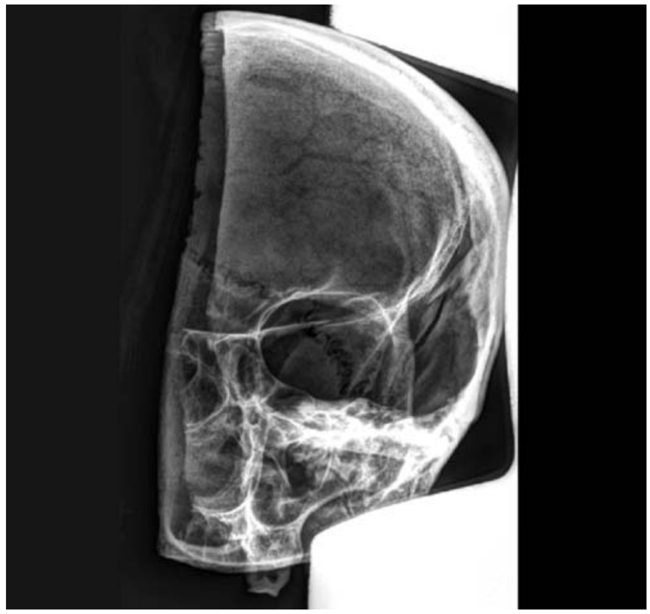

Figure 4. Unilateral foramen rotundum in ethmoidal cells on X-ray.

ra) undergo X-ray visualization as a first order examination in family medicine centers. Foramen rotundum is a structure which is not generally taken into consideration during routine X-ray image evaluation. This anatomic structure is located at a key point at skull base.[1,2] It is important to realize foramen rotundum on routine X-rays to diagnose and distinguish pathologies developed near this structure from normal findings. We suggest that tumors which arise in fossa pterygopalatina and tumors which extend to fossa pterygopalatina from oral, orbital and maxillofacial regions may obscure radiologic visualization of this key structure.

Thus, there is need for comparative studies for radiologic evaluation of foramen rotundum between patients with normal findings and with patients who have fossa pterygopalatina pathologies. X-ray evaluation of foramen rotundum in large series of patients with no evidence of disease that might alter foraminal anatomy should be base for the following studies. Besides tumors detection of aplastic foramina rotunda may give an early indication of Gómez-López-Hernández syndrome; a rare neurocutaneous disorder which is characterized by rhombencephalosynapsis, parieto-occipital alopecia, and trigeminal anesthesia.[7]

There are studies in the literature, performed on both dry skulls and CT (computed tomography) scans. Liu et al.[8] studied the size of foramen rotundum of 51 patients with computed tomography (CT) to indicate the etiology of trigeminal neuralgia. Berlis et al.[9] analyzed the size of foramen rotundum on 60 dry skulls using CT and measured the average diameters as 3.9×3.13 mm. Edwards et al.[10] reviewed the size of foramen rotundum and claimed that a narrow foramen due to achondroplasia, fibrous dysplasia, osteopetrosis, malignant schwannoma may present severe clinical consequences. Hedeman et al.[11] reported two cases with schwannoma of Gasserian ganglion protruding into foramen rotundum and causing

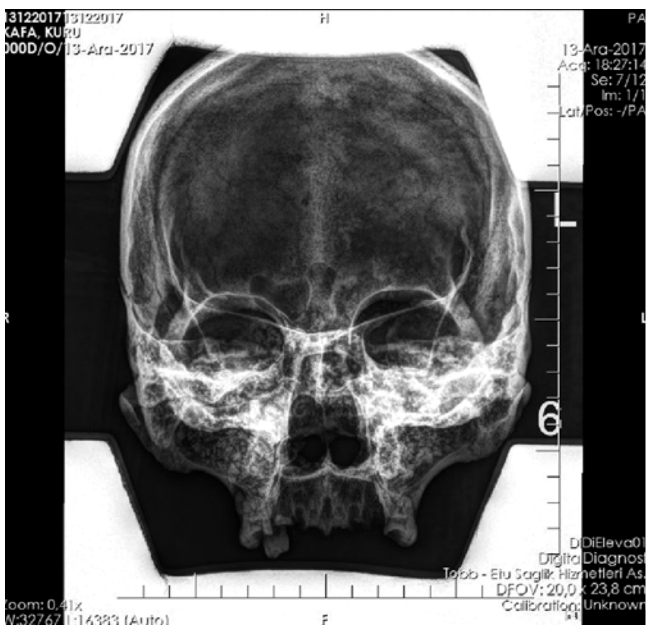

Figure 5. Nonvisible foramen rotundum.

right-sided pain and a burning sensation along the maxillary nerve.

Although CT is valuable to measure the size of the foramen rotundum, it is not a routine imaging technique. Indications of the patients undergoing CT imaging must be well-defined. Foramen rotundum can be visualized with cheap and easily performed X-ray imaging to reveal soft tissue masses obscuring foramen rotundum.

Conclusion

Foramen rotundum must be taken into consideration in evaluation of routine X-rays to prevent misdiagnosis of the patients with persistent non-specific symptoms mentioned above.Therefore, radiologists should be familiar with the X-ray view of this important structure and attaching this key structure to the reporting templates will make a difference.

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Castration induces progressive increase in the carotid intima-medial thickness of the male rat

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Abstract

Objectives: The role of androgens in the development of cardiovascular diseases remains controversial. The current study therefore aimed to determine the changes in the carotid intima-medial thickness of the male rat in surgically-induced hypogonadism.

Methods: Twenty-two *Rattus norvegicus* male rats aged two months were used. The rats were randomly assigned into baseline (4), experimental (9) and control (9) groups. Hypogonadism was surgically induced in the experimental group by bilateral orchiectomy under local anesthesia. At experiment weeks 3, 6 and 9, three rats from each group (experimental and control) were euthanized, their common carotid artery harvested, and routine processing was done for paraffin embedding, sectioning and staining. The photomicrographs were taken using a digital photomicroscope for morphometric analysis.

Results: The mean carotid intima-median thickness was 321.97 μm at baseline. There was a progressive increase in the carotid intima-median thickness by 17.6%, 37% and 67.1% of the baseline values in the castrated group at the end of the third, sixth and ninth week respectively ($p<0.001$). Although the carotid intima-median thickness increased in the non-castrated group with increasing duration of the study, these increases were not statistically significant ($p=0.110$). The increase in carotid intima-median thickness was associated with hyperplasia of the intimal layer as well as increased deposition of collagen fibers in the medial layer.

Conclusion: Androgen deprivation by surgical castration induces a progressive increase in the carotid intima-median thickness. This may constitute an anatomical basis for the higher predisposition of hypogonadal males to cardiovascular diseases.

Keywords: androgens; cardiovascular disease; carotid intima-medial thickness; castration; hypogonadism

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Introduction

Carotid intima-medial thickness (c-IMT), defined as the combined thickness of the intimal and medial layers of the common carotid artery (CCA),[1] is an important biomarker of subclinical atherosclerosis.[2,3] It is an established indicator of the presence, extent and severity of coronary artery disease (CAD).[4] It is also independent predictor of future cardiovascular events, with an absolute 0.1 mm increase in the c-IMT correlating to a 10–15% and 13–18% increase in the risk of future myocardial infarction (MI) and stroke respectively.[5] It is therefore utilized as a surrogate end point for cardiovascular events in interventional studies.[6–8]

The role of androgens in the development of cardiovascular diseases (CVDs) remains controversial.[9] Traditionally, androgenic hormones have been associated with an increased risk of CVDs. This has largely been based on the gender disparity in the incidences of CVDs, with a higher male preponderance[10] as well the abuse of anabolic steroids by athletes which has been linked to various adverse effects on the cardiovascular system including thrombosis, hypertension and heart failure.[11] Similarly, hyper-androgenic conditions such as congenital adrenal hyperplasia (CAH) and polycystic ovarian syndrome (PCOS) has been associated with an increase in C-IMT.[12–14] However, evidence from various population studies and animal studies demonstrate beneficial effects of physiological levels of androgens, reporting an inverse correlation between androgen levels and c-IMT & incidences of CVDs.[15–18] The relationship between androgens and CVDs/c-IMT therefore remains unclear.

Of note, previous studies mainly focused c-IMT measurements in the presence of physiological or supra-physiological levels of androgens. Changes in the c-IMT in hypogonadal states remains relatively underexplored. The current study therefore sought to determine the changes in the c-IMT in orchiectomy-induced hypogonadism. This may be important in predicting the risk of cardiovascular events in patients with hypogonadism secondary to age-related decline in endogenous androgen levels,[19,20] androgen deprivation therapies[21] or disorders that either damage the testis or reduce gonadotropin stimulation.[22,23]

Materials and Methods

A total of 22 male rats aged 2 months were used in this study. Rats were used as the study model because of their ease of handling, low maintenance cost and close physiological resemblance to man. Further, the structure of the rat common carotid artery (CCA) is similar to that in man. Two-month old rats were used because this is the age at which they attain sexual maturity. Rats with visible neck or scrotal pathology were excluded. The animals were housed in cages floored with wood shavings that were changed regularly. They were kept in their cages for 2 weeks prior to commencement of the study for acclimatization. The animals were placed under a normal 12 hours' light/dark diurnal cycle and provided with standard rat pellets and water ad libitum. Ethical approval to conduct the study was granted by the Biosafety, Animal Care and Use committee of Nairobi University, Nairobi, Kenya (Ethical approval number: FVMBAUEC/2016/96).

Four rats were chosen using simple random sampling technique to demonstrate the baseline (day 0) histomorphology of the CCA. The remaining rats were divided randomly into 2 groups (11 experimental, 11 controls). Hypogonadism was induced in the experimental via bilateral orchiectomy under local anesthesia. The animals were placed in the dorsal recumbent position and under physical restrain. The scrotal skin disinfected using iodine solution. Two ml of 1% lignocaine was injected in the around the scrotal sac to provide local anesthesia. A 1.5 cm incision was made at the base of the hemi-scrotal sacs. Subcutaneous tissue was bluntly dissected to reveal the vaginal processes. This was then excised to access the testis and the spermatic cord which were then gently exteriorized. The spermatic cord was then clamped, ligated and removed together with the testis and epididymis. A 4-0 suture was then used to close the processus vaginalis. This procedure was repeated on the contralateral hemi-scrotal sac. The skin was thereafter closed with a 2-0 non-absorbable interrupted sutures. The wound was then covered with a cotton wool soaked in iodine and secured with a bandage. Post procedural pain was detected through observation based on the animals' behavior and attitude changes. These include reluctance to move, abnormal posturing, decreased appetite and vocalization. This post procedural pain was managed using ibuprofen 5 mg/kg mixed with water.

On experimental week 3, 6 and 9, three animals from both groups were picked randomly, euthanized and perfused with normal and formal saline solutions. Their CCA were harvested and processed for paraffin embedding and sectioning. The rats' CCA were fixed in 10% formalin for twelve hours. This was followed by dehydration in increasing grades of alcohol (70% up to absolute alcohol) at one hour intervals, and clearing in toluene. Thereafter, the vessels were placed in the memmert oven for wax infiltration. The CCA were embedded in paraffin wax and oriented for transverse sectioning. After cooling, the embedded tissues were blocked using wooden blocks and then serially cut into 7 µm sections using a microtome. Fifteen 7 µm sections were randomly obtained from the ten ribbons, floated on a 60 °C water bath and picked on a glass slide, then dried in an oven for 12 hours. Masson's Trichrome was used to display smooth muscle cells and collagen fibers while Wiegerts stain was used to display elastic fiber profile.

Photomicrographs of the sections were taken using a digital camera (Canon Powershot A640, 12 mp, Beijing, China) mounted on a photomicroscope (Carl Zeiss, Axiostar Plus Microimaging, Jena, Germany) for morphometric analysis using the Fiji-ImageJ. This is an open source software developed by the United States National Institute of Health for processing and analyzing images. The variable obtained was the carotid intima-medial thickness (c-IMT). The c-IMT measurements were obtained from four different parts of the vessel wall and an arithmetic mean of the four values used for analysis.

The collected were entered into the using the Statistical Package for Social Sciences (SPSS for Windows, version 21.0, Chicago, IL, USA) for coding, tabulation and statistical analysis. The c-IMT was expressed in micrometers. The data were grouped into two: Control group (non-castrated) and experimental (castrated) group. After confirming that the data was not normally distributed (using box plots and histograms), non-parametric tests were used for univariate analysis. Kruskal-Wallis H test was used to compare the medians of the c-IMTs along the various harvesting periods within each group. Mann-Whitney U test was used to compare the medians in c-IMTs between control and experimental groups. A p-value <0.05 was considered significant at 95% confidence interval. Data is presented in tables and photomicrographs.

Results

The mean carotid intima-median thickness (c-IMT) was 321.97 μm at baseline. There was a progressive increase in the c-IMT by 17.6%, 37% and 67.1% of the baseline values in the castrated group at the end of the third, sixth and ninth week, respectively (p<0.001) (**Table 1**). These increases in c-IMT were more marked in rats exposed to hypogonadism over a longer period of time (**Figures 1a-f**). The increase in c-IMT was associated with hyperplasia of the intimal layer as well as deposition of collagen fibers in the medial layer (**Figures 1c** and **d**). Although the c-IMT increased in the non-castrated group with increasing duration of the study, these increases were not statistically significant (p=0.110) (**Table 1**).

Discussion

The present study demonstrates that endogenous androgen deprivation by bilateral orchiectomy results in a significant increase in the carotid intima-medial thickness (c-IMT) of the male rat. To the best of our knowledge, this is the first study to describe changes in the c-IMT in induced hypogonadism. Previous studies focused mainly on the correlation between the levels of endogenous androgens and c-IMT. Nonetheless, the findings of the current study are in concordance with studies by Muller et al.,[24] Farias et al.,[25] and Chan et al.,[26] who reported an inverse correlation between androgen levels and c-IMT. The findings of the current study are however at variance with those by Allameh et al.,[13] and Kim et al.,[14] who reported increased c-IMT measurements in hyperandrogenic conditions such as congenital adrenal hyperplasia (CAH) and polycystic ovarian disease (PCOS). Pooled together, the above findings suggest that physiological levels of androgens are beneficial, whereas extreme levels (hypogonadism and hypergonadism) are detrimental to the cardiovascular system.

The increase in c-IMT could be attributed to the hyperplasia of the tunica intima as well as increased deposition of collagen fibers in the tunica media that was observed in the current study. The development of intimal hyperplasia in induced hypogonadism, hitherto undescribed to the best of our knowledge, may be attributed to the increased production of pro-inflammatory cytokines[27] and reactive oxygen species (ROS)[28] that normally accompany androgen deprivation. These are known to cause endothelial injury, a crucial initial component in the pathogenesis of intimal hyperplasia.[29] Similarly, endogenous androgens are known to have inhibitory effects on vascular smooth muscle proliferation and migration. It is therefore plausible to postulate the loss of these inhibitory effects, hence proliferation of smooth muscle cells in the tunica intima, resulting in intimal-hyperplasia. Although the current study did not investigate the changes in lipid profiles of the study animals, previous studies have reported dysregulations in lipid metabolism in hypogonadal states, with decreased levels of high density lipoproteins (HDL) as well as increased levels of low density lipoproteins (LDL) and triglycerides (TGs).[9] This may plausibly constitute another basis for the development of intimal hyperplasia in the current study.

Increased carotid intima-medial thickness (c-IMT) is an established marker of atherosclerosis[8] and is an independent predictor of the presence and severity of coronary and peripheral artery disease.[4,30] Increased c-IMT seen in this study and those from previous studies therefore suggest that low androgen levels are associated with increased risk of atherosclerotic coronary and peripheral

Table 1

Changes in the carotid intima-medial thickness in control and experimental animals.

Group	Baseline	Week 3	Week 6	Week 9	p
Control (medians)	B1 - 322.64 B2 - 323.36 B3 - 320.39 B4 - 321.49	C1 - 327.31 C2 - 324.22 C3 - 326.44	C4 - 329.98 C5 - 331.28 C6 - 329.51	C7 - 344.43 C8 - 342.05 C9 - 345.36	0.11
Experimental groups (medians)	_	E1 - 379.31 E2 - 375.76 E3 - 378.01	E4 - 442.42 E5 - 438.04 E6 - 441.38	E7 - 538.89 E8 - 540.17 E9 - 537.36	<0.001*
p	_	0.002*	0.001*	<0.001*	

*p<0.05. **B:** baseline animals; **C:** control animals; **E:** experimental animals.

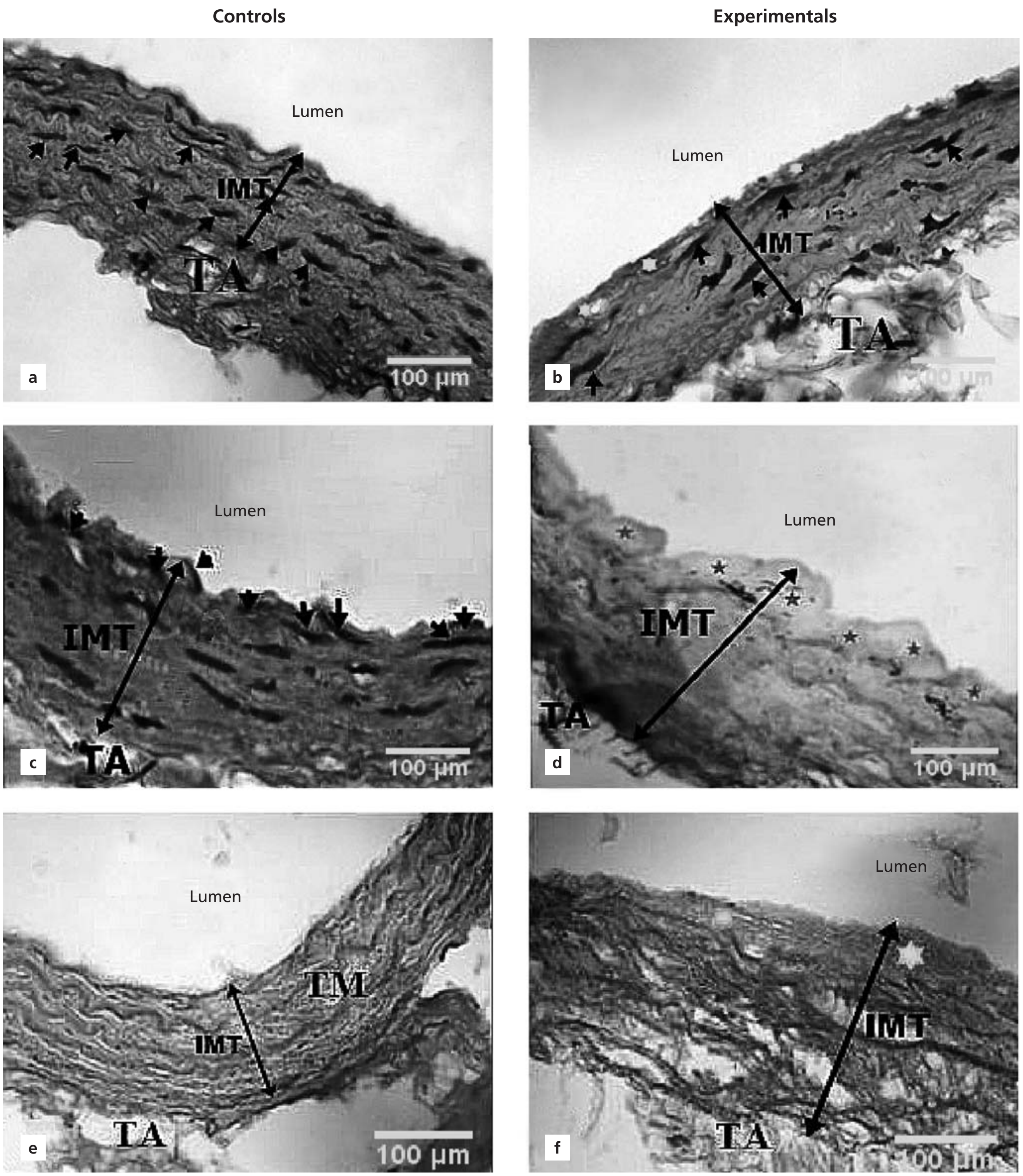

Figure 1. Photomicrograph showing the structure of the CCA of (**a**) a normogonadic rat at week 3, (**b**) three weeks after castration (note the increase in intima-medial thickness compared to the control in Figure 1a), (**c**) 9 weeks after castration (note the intima hyperplasia with smooth muscle cell nuclei), (**d**) hypogonadic rat seen 9 weeks after castration (note the intimal hyperplasia that's thicker than that in the control animal in Figure 1c) (**green stars**). Hematoxylin and Eosin stain. Photomicrograph showing the organization of tunica media of common carotid artery of (**e**) a normogonadic rat at week at week 6 of the study, (**f**) a hypogonadic rat seen 6 weeks after castration (note the increased intima-medial thickness as well as the intimal hyperplasia). IMT: intima-medial thickness; TA: tunica adventitia; TM: tunica media. Wiegerts' elastic stain. Scale bars=100 µm. [Color figure can be viewed in the online issue, which is available at www.anatomy.org.tr]

artery disease. This is in agreement with various observational studies that have reported higher incidences of these diseases in hypogonadal men compared to those with normal androgen levels.[15,16,18] Progressive hyperplasia and hypertrophy of the tunica intima is known to result in vascular stenosis. This may compromise vascular supply to important organs such as brain and heart affected by atherosclerosis making them more vulnerable to ischemic insults.[31] This may explain the higher incidences of stroke,[32] myocardial infarction[33] as well as ischemic lower limb amputations[34] among hypogonadal men.

The current study was limited by the fact that castration is a surgical procedure that causes tissue injury. Therefore, some of the changes observed in this study may have been due to reactive processes to tissue injury. This was delimited by the fact that the surgical procedure was carried out in the scrotum and did not tamper with the common carotid arteries located in the neck. Also, based on the fact that the changes observed in this study were proportional to the duration of androgen deficiency, they are most likely to be due to gonadal hormone deficiency rather than surgical trauma.

Conclusion

Androgen deprivation by surgical castration induces a progressive increase in the c-IMT. This may constitute an anatomical basis for the higher predisposition of hypogonadal males to cardiovascular diseases.

Acknowledgements

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Incidence of agger nasi and frontal cells and their relation to frontal sinusitis in a Turkish population: a CT study

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Abstract

Objectives: The aim of this study was to determine the incidence of agger nasi and frontal cells in a Turkish population and their relation to frontal sinusitis.

Methods: A total of 412 non-contrast paranasal sinus computed tomography (CT) images taken between March 2017 and June 2018 were examined retrospectively. Frontal cells were classified into four types according to Kuhn's classification. The relation of agger nasi and frontal cells to frontal sinusitis was evaluated.

Results: Of the 412 patients, 202 were males (mean age 34.8±14.9) and 210 were females (mean age 35.1±13.9). agger nasi cell was detected in 214 (51.9%), and frontal cell in 198 patients (48%). Frontal sinusitis was detected in 68 patients (16.5%). According to Kuhn's classification, Type 1 frontal cell was detected most frequently. A significant relationship was observed between the presence of agger nasi and frontal cells and frontal sinusitis (p<0.001). When the right and left frontal sinusitis were evaluated separately, the relationship of frontal cell types of Kuhn's classification with frontal sinusitis was found to be significant on the right side, but not on the left side.

Conclusion: Agger nasi and frontal cells are common paranasal sinus variations that play a role in the development of frontal sinusitis. Although most of the paranasal sinus variationsare considered as predisposing in the development of sinusitis, there are obvious differences in studies. For this reason, a higher number of comprehensive studies are necessary to reveal the relation between the presence of agger nasi and frontal cells and sinusitis.

Keywords: agger nasi cell; frontal cell; frontal sinusitis; Kuhn's classification

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Introduction

There are a number of anatomical variations of paranasal sinuses. These anatomical variations may cause narrowing or obstruction in sinus drainage canals, leading to a reduction in the ventilation of sinuses and a deterioration mucociliary activity.[1] Particularly the inflammation of the cells around the sinus ostium leads to a narrowing in the sinus ostium, which disrupts the ventilation of sinuses and in turn leads to development of sinusitis.[2]

In the surgical treatment of paranasal sinus diseases, knowledge of the anatomy of paranasal sinuses and their variations yields better results in surgery and reduces complications.[3] Computed tomography (CT) is an imaging technique used as golden standard in the evaluation of the patients preoperatively. Detailed anatomical information can be obtained preoperatively with CT and imaging findings are used as a surgical maps.[4]

In this study, we aimed to determine the incidence of agger nasi (AN) and frontal cells in a Turkish population and to reveal whether there is a relation with these and frontal sinusitis.

Materials and Methods

This study was conducted in the Department of Radiology of Kırıkkale University, School of Medicine between January 2018 and June 2018. Ethics Committee approval was obtained from Kırıkkale University Ethics Committee. Paranasal sinus CT images obtained with

64-section multislice CT (Brilliance CT system; Philips Healthcare, Cleveland, USA) were evaluated retrospectively. The informed consent was waived due to the retrospective design of the study. The conformed with the principles outlined in the appropriate version of 1964 Declaration of Helsinki. The patients under the age of 18 and over the age of 75, and those with nasal polyposis, a history of operation on the sinonasal area, trauma history or a benign or malignant tumor were discluded from the study. CT images were obtained on coronal plane, with the patients lying prone (face down). Sagittal and axial images were reconstructed when necessary.

Radiologically, frontal sinusitis was defined as the thickening of sinus ostium or sinus walls at least for 3 mm in CT images. AN cell was accepted as the foremost anterior cell which was pneumatized in the frontal recess as described by Messerklinger.[5] Right and left recesses were evaluated separately. The frontal cells superior to the AN cell were divided into four groups in CT according to Kuhn's classification.[4] Based on this classification, Type 1 is a single cell located on AN cell; Type 2 is in the form of two or more cells on AN cell; Type 3 is a single large cell pneumatized into the frontal sinus; and Type 4 is in the form of a cell within the frontal sinus. The presence of frontal cells was evaluated separately on the left and right sides. All measurements were performed by a single researcher with seven years of experience in head and neck radiology.

Statistical analysis was performed using the Statistical Package for Social Sciences (SPSS for Windows, version 20.0, Chicago, IL, USA). Categorical variables were presented as frequencies and percentages, and data were expressed as mean±standard deviation. Groups were compared with Pearson's chi-squared test. To compare non-categorical changes, independent samples t-test was used. A p-value <0.05 was considered statistically significant.

Results

Of the 412 patients, 202 were males (mean age 34.8±14.9) and 210 were females (mean age 35.1±13.9). AN cell was detected in 214 patients (51.9%); 74 on the right side (17.9%), 36 on the left side (8.7%), and 104 bilateral (25.2%) (**Figure 1**).

Frontal cells were detected in 198 patients (48%); 54 (13.1%) of these were on the right side, 46 (11.1%) on the left side, and 98 (23.7%) bilateral. The most common frontal cell was Khun Type 1 and the least common Khun Type 4 (**Figure 2**). The incidence of frontal cells according to Kuhn's classification is shown in **Table 1**.

Table 1
The incidence of frontal cells on the right and left sides according to Kuhn's classification.

Kuhn's classification	Right (%)	Left (%)
Type 1	78 (18.9%)	58 (14%)
Type 2	56 (13.5%)	50 (12.1%)
Type 3	12 (2.9%)	24 (5.8%)
Type 4	6 (1.4%)	14 (3.3%)

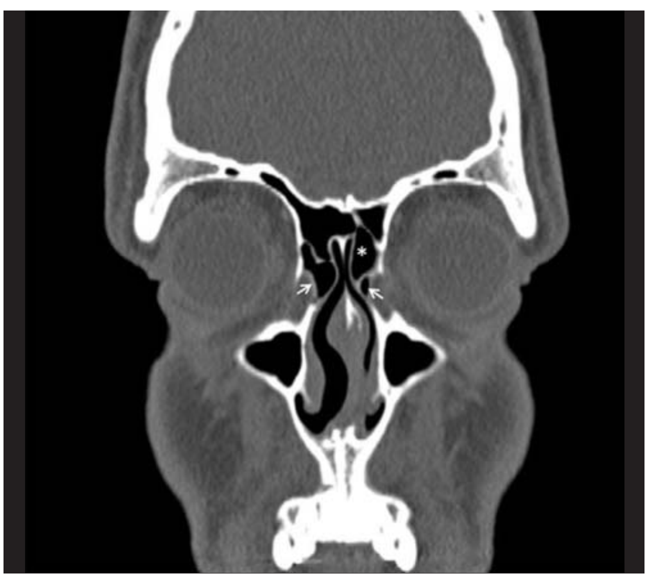

Figure 1. Coronal non-contrast paranasal sinus CT image shows bilateral agger nasi cells (**white arrows**) and frontal cell Type 1 on the left side (*).

Figure 2. Coronal non-contrast paranasal sinus CT image shows a frontal cell Type 4 in the left frontal sinus (*).

Table 2
The incidence of agger nasi cell and frontal cells and their relationship between frontal sinusitis.

		None (%)	Right (%)	Left (%)	Bilateral (%)	Total (%)	p
Agger nasi cell	None	96 (23.3%)	0 (0%)	0 (0%)	0 (0%)	96 (23.3%)	<0.001
	Right	58 (14%)	8 (1.9%)	2 (0.4%)	6 (1.4%)	74 (17.9%)	
	Left	30 (7.2%)	2 (0.4%)	4 (0.9%)	0 (0%)	36 (8.7%)	
	Bilateral	160 (38.8%)	10 (2.4%)	16 (3.8%)	20 (4.8%)	206 (50%)	
	Total	344 (83.4%)	20 (4.8%)	22 (5.3%)	26 (6.3%)	412 (100	
Frontal cell	None	198 (48%)	4 (0.9%)	6 (1.4%)	6 (1.4%)	214 (53.1%)	<0.001
	Right	40 (9.7%)	8 (1.9%)	2 (0.4%)	4 (0.9%)	54 (13.1%)	
	Left	36 (8.7%)	2 (0.4%)	4 (0.9%)	4 (0.9%)	46 (11.1%)	
	Bilateral	70 (16.9%)	6 (1.4%)	10 (2.4%)	12 (2.9%)	98 (23.7%)	
	Total	214 (51.9%)	54 (13.1%)	46 (11.1%)	98 (23.7%)	412 (100%)	

Frontal sinusitis was detected in 68 patients; 20 (4.8%) of these were on the right side, 22 (5.3%) on the left side, and 26 (6.3%) were bilateral (**Figure 3**). A statistically significant realtion was found between the presence of both AN and frontal cells and frontal sinusitis (p<0.001) (**Table 2**).

When right and left paranasal sinuses were evaluated separately, frontal cell types according to Kuhn's classification had a significant relationship with frontal sinusitis on the right side (p=0.002), whereas there was no significant relationship on the left side (p=0.942) (**Table 3**).

Figure 3. Axial non-contrast paranasal sinus CT image shows a frontal cell Type 3 in the right side (*) and right frontal sinusitis (white arrow).

Discussion

Non-contrast paranasal sinus CT taken on a coronal plane is the most commonly preferred imaging technique in revealing the anatomy of paranasal sinuses in detail.[4] The variations of paranasal sinuses are often detect in routine paranasal CT examinations, and have been reported in different studies with wide ranges.[6,7] In this study, we

Table 3
The relation between frontal cell types and frontal sinusitis.

Frontal cell types			Absent (%)	Present (%)	Total	P
Right frontal sinusitis	Right	Type 1	30 (7.2%)	9 (2.1%)	39 (9.4%)	0.002
		Type 2	26 (6.3%)	2 (0.4%)	28 (6.7%)	
		Type3	5 (1.2%)	1 (0.2%)	6 (1.4%)	
		Type 4	0 (0%)	3 (0.7%)	3 (0.7%)	
		Total	61 (14.8%)	15 (3.6%)	76 (18.4%)	
Left frontal sinusitis	Left	Type 1	23 (5.5%)	6 (1.4%)	29 (7%)	0.942
		Type 2	20 (4.8%)	5 (1.2%)	25 (6%)	
		Type 3	10 (2.4%)	2 (0.4%)	12 (2.9%)	
		Type 4	5 (1.2%)	2 (0.4%)	7 (1.6%)	
		Total	58 (14%)	15 (3.6%)	73 (17.7%)	

aimed to identify whether there was a relations between the presence of AN and frontal cells and frontal sinusitis. AN incidence was reported at different rates in different studies. Kayalioglu at al.[6] reported AN incidence as 7.77%. In contrast, in another study, Bradley and Kountakis[7] reported this incidence very high, 93%. The possible reason for this wide range of results in these studies is the difference in the definition of AN cell. In this study, the incidence of AN cell was 51.9%, and bilateral AN cell 25.2%.

Similarly, differences exist in earlier studies regarding the incidence of frontal cells; Gümüş et al.[8] reported as 15.2%, Küçükgünay et al.[9] 20.9%, and Eweiss et al.[10] 78.5%. In this study, the incidence of frontal cells was 48%, and bilateral frontal cells 23.7%. The most common frontal cell was Khun Type 1 and the least common Khun type 4.

Sinusitis is defined as the infection or inflammation of the paranasal sinuses. Its pathology can be explained with deterioration in the ventilation of sinus through the sinus ostium. The factors that affect the size of the sinus ostium play a role in the development of sinusitis.[11] This clinical situation can be seen in all sinuses. Among these factors, frontal sinusitis comes to the fore due to its anatomical position and intracranial complication risks.[11]

There are also differences in literature regarding the relationship between AN cell and frontal sinusitis. Eweiss et al.,[10] Kubota et al.,[12] and Del Gaudio et al.[13] found no relation between AN cell and frontal sinusitis, wheras Yegin et al.[14] reported the presence of sinusitis as significantly higher on the side of the AN cell. We also found a statistically significant relationship between frontal sinusitis and AN cell ($p<0.001$).

Del Gaudio et al.[13] reported no relationship between frontal cells and frontal sinusitis. In contrast, Meyer et al.[4] reported a significant relationship between Type 3 and Type 4 frontal cells and frontal sinusitis. Küçükgünay et al.[9] noted that Type 3 frontal cells had a relationship with sinusitis, but other types were not related. Gümüş et al.[8] detected a significant relationship between the presence of frontal cells and ipsilateral frontal sinusitis. In this study, when the right and left sides were evaluated separately, the relationship with frontal sinusitis was found to be significant on the right, but not on the left side.

The improvements in endoscopic sinus surgery have rendered the detailed anatomy of paranasal sinuses and their variations more important. Knowledge of variations is essential in order to avoid the complications which could arise during the operation and to apply surgical procedures in a safe way.[15] Therefore, the evaluation of paranasal sinus anatomy and its variations preoperatively is crucial in eliminating complications.[15] CT is the best imaging method for the evaluation of inflammatory paranasal sinus pathologies and the osteomeatal complex. Contrary to the axial plane, coronal CT evaluation is considered to be a more efficient imaging method for endoscopic approaches.[3,4]

Conclusion

The presence of AN and frontal cells plays a role in the development of frontal sinusitis. Although it is accepted that most of the paranasal sinus variations may be predisposing in the development of sinusitis, there are obvious differences between studies. For this reason, a higher number of comprehensive studies are required to reveal the relation between the presence of AN and frontal cells and sinusitis.

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Demonstration of craniocervical junction abnormalities for diagnosis of atlanto-occipital assimilation using MRI

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Abstract

Objectives: Atlanto-occipital assimilation (AOA) is one of the most common skeletal anomalies of the craniovertebral junction (CVJ). Because its clinical symptomatology is non-specific and it has several variations, many cases go unnoticed which may lead to additional and unnecessary radiological examinations. In this study, we aimed to present CVJ abnormalities with MRI to improve diagnostic accuracy of AOA.

Methods: Cervical MRIs of the patients registered in PACS between January 2008 and October 2011 were scanned and AOA was detected in 40 cases. Sagittal FSE T1 and T2-weighted cervical MRIs and axial T2*-GRE sequence images were re-evaluated for AOA typing, anterior atlantodental interval (AADI), posterior atlantodental interval (PADI) measurements, spine fusion anomalies, basilar invagination, tonsillar herniation, myelomalacia, suboccipital muscles and vertebral arteries (VAs).

Results: CVJ abnormalities were present in all cases and the most frequent association was observed in suboccipital muscles (100%) and VAs (95%). 60% of the cases had decreased PADI, 32% C2–3 vertebrae fusion, 25% increased AADI, 22.5% basilar invagination, 15% myelomalacia and 5% tonsillar herniation.

Conclusion: Suboccipital muscle abnormality was found in all AOA cases whatever the severity and type of the bony fusion. VA anomaly was observed as the second most common abnormality and accompanied preferably the cases with lateral body involvement. Being aware of additional CVJ abnormalities in MRI examinations may reduce unnecessary radiological examinations by increasing the AOA diagnosis rate.

Keywords: assimilation; craniocervical; magnetic resonance imaging

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Introduction

Atlanto-occipital assimilation (AOA) refers to the fusion of the atlas with the base of the occipital bone and is one of the most common bone abnormalities of the craniovertebral junction (CVJ).[1,2] AOA can be either congenital or acquired. The prevalence of AOA in the general population varies from 0.08% to 2.76%.[3] AOA may be in the form of partial or complete fusion. Although complete type is more common, several variations of the partial form were also identified. The classification of atlas includes 4 patterns according to the anatomic site of the fusion of the occiput. Type 1 involves the anterior arch of the atlas fusion (20%), Type 2 lateral body fusion (17%), Type 3 fusion of the posterior arch (13%) and Type 4 fusion is with the zonal combinations (50%).[2] Because they are probably asymptomatic, most of the patients with assimilation are not diagnosed. It is mostly accompanied by basilar invagination (BI), and atlantoaxial dislocation frequently results in cervicomodullary compression of odontoid process.[4]

Neurological disorders in patients with AOA often occur in the 3rd and 4th decades of life and require surgical treatment.[4,5] Occipitalization also results in spinal cord compression caused by soft tissue and bone abnormalities which lead to weakness and ataxia in the lower extremities and numbness and pain in the upper limbs.[4]

Vertebral artery (VA) compression causing dizziness, seizures, mental disorders and syncope or occlusion of the bone channel were also reported.[6] Preoperative angiogram is strongly recommended for the demonstration of the course of the VA.[7]

Considering its symptomatology and prevalence, AOA may be overlooked and this may lead to unnecessary radiological examinations of different anatomical regions. As well as unnecessary costs caused by the delays in diagnosis, surgical procedures scheduled in patients who did not get a diagnosis may also pose risks for this patient group. AOA diagnosis is of vital importance for the radiologists, neurologists, orthopedists and neurosurgeons. To our knowledge, there was no performed MRI study on AOA cases in the literature. For this reason, especially by considering the partial-type AOA, we aimed to present additional CVJ abnormalities with MRI which might provide evidence to improve diagnostic accuracy of AOA.

Materials and Methods

A total of 11.813 cervical MRIs taken between January 2008 and October 2011, and registered in picture archiving and communication system (PACS) were scanned. AOA was detected in 40 cases. The images were acquired using 1.5 T MRI scanner (Signa Excite, GE, Milwakuee, WI, USA).

Of the 40 cases, 9 were males and 31 were females. The mean age was 40.6 years (range: 25–69 years). Ethics committee approval was obtained from Local Ethics Committee in April 2017. Written informed consent was obtained from all patients. The patients had no history of trauma or surgery, but as head-neck, back and shoulder pain, unsteadiness and dizziness.

AOA typing, anterior atlantodental interval (AADI) measurements, sagittal diameter between posterior edge of foramen magnum and atlas posterior arch (or posterior atlantodental interval, PADI) measurements were performed with sagittal FSE T1(TR/TE: 442–460/10–16 ms, slice thickness: 2.5 mm, slice spacing: 3 mm, matrix: 512×512), FSE T2-weighted images (TR/TE: 3310–3420/82–99 ms, slice thickness: 2.5 mm, slice spacing: 3 mm, matrix: 512×512) and axial T2*-weighted gradient echo (GRE) sequence images (TR/TE: 400/14 ms, slice thickness: 2.5 mm, slice spacing: 3 mm, matrix: 512×512, flip angle: 20).

The presence of accompanying possible spine fusion anomalies, BI, tonsillar herniation and myelomalacia were noted. Suboccipital muscles [rectus capitis posterior minor (RCPM), rectus capitis posterior major (RCPMA) and obliquus capitis posterior inferior (OCPI)] were evaluated. The obliquus capitis posterior superior (OCPS) muscle which is not involved in the study area was excluded from the assessment. Skull base portion and intracranial component of the VA were also evaluated. The sagittal diameter of foramen magnum above 18 mm according to the PADI measurements was considered normal.[8,9] AADI values greater than 3 mm were considered abnormal.[10] Extension of the tip of dens more than 5 mm over the Chamberlain's line was considered as basilar invagination.[11] Extension of cerebellar tonsils under the opistion-basion line more than 5 mm was regarded as the tonsillar herniation.[12] Statistical analysis was performed using SPSS 15.0 for Windows (SPSS, Inc., Chicago, IL, USA). Data were expressed as percentages with using frequency tests.

Results

On MRI, there was a bony fusion of any part (lateral body, posterior or anterior arch) of the atlas with occipital condyle in all cases. Of the cases (n=40), bilateral fusion of the lateral bodies of atlas with occipital condyle was seen in 14 (35%) (**Figure 1**), complete fusion of atlas and condyle in 12 (30%) (**Figure 2**), symmetric fusion of lateral bodies and anterior arch with condyle in 7 (17.5%) (**Figure 3**), symmetric fusion of lateral bodies and posterior arch with condyle in 3 (7.5%), unilateral lateral body fusion with condyle in 3 (7.5%) and fusion of unilateral partial lateral body fusion in 1 (2.5%) cases. Atlas had hypoplastic posterior arch in 16 cases, posterior arch defect in 3 and unilateral agenesis of posterior arch in 3.

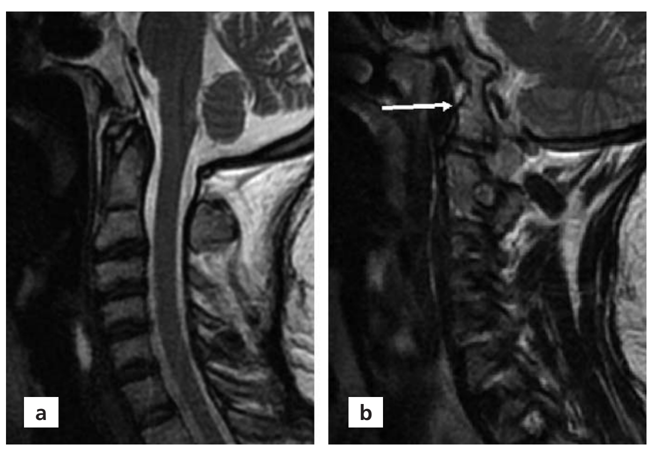

Figure 1. (**a**) A 48-year-old man with AOA. No bony fusion of anterior and posterior archs of the atlas with occipital condyle seen at midsagittal MR image, but there is severe atrophy of suboccipital muscles. (**b**) Parasagittal T2-weighted image shows a bony fusion of lateral body with occipital condyle (**arrow**).

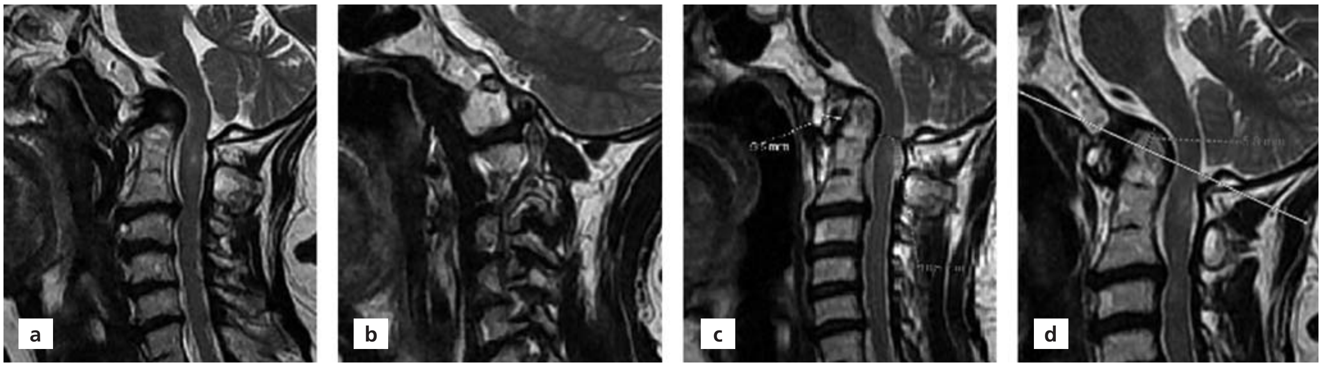

Figure 2. (**a**, **b**) A 58-year-old female presented with chronic head and neck pain. At sagittal T2-weighted images, complete form AOA is seen as fusion of anterior arch, posterior arch and lateral masses of C2 vertebra. Spinal cord myelomalacia at the C2 level and fusion of C2 and C3 vertebra also accompanied. (**c**) AADI increased to 5.5 mm whereas PADI decreased to 10.5 mm, revealing craniocervial instability. (**d**) The tip of odontoid process was projecting more than 3 mm (5.8 mm) above the Chamberline's line, compatible with basilar invagination.

The anomaly was accompanied with BI and C2–3 vertebrae fusion in 9 and 13 cases, respectively. PADI was < 14 mm (mean 10.46 mm) in 14 cases (35%). This value ranged from 14 to 18 mm in 20 patients and was greater than18 mm in 6 cases. AADI was >3 mm in 10 cases (25%).

Spinal cord myelomalacia at CVJ was present in 6 patients (**Figure 2**). With the evaluation of suboccipital muscles, RCPM muscle was found abnormal in all cases. In 25 cases, no muscle structure was observed on MR examination and the remaining 15 cases had further findings of atrophy related to the muscle. While RCPMA muscle was symmetrically atrophic in 13 patients, 8 patients had unilateral atrophy. OCPI muscle was symmetrically atrophic in 5 patients, whereas 1 patient had unilateral atrophy (**Figure 4**). Tonsillar herniation was observed in 2 (5%) patients.VA abnormality was detected in 38 (95%) cases. In 30 cases, VA was in bone tunnel formed by the fusion of the atlas lateral body and occiput. VAs in 8 cases in this group showed entrapment with fused lateral bodies, odontoid

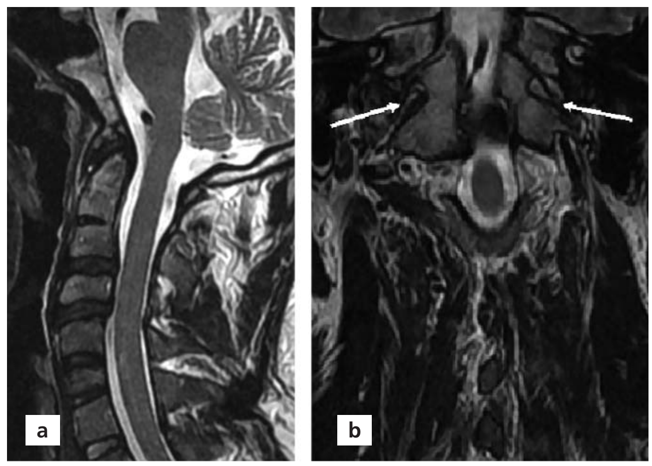

Figure 3. (**a**) 44-year-old woman with AOA. Fusion of the anterior arch of atlas with occipital condyle, C2–3 vertebrae fusion and suboccipital muscle atrophy were observed on sagittal T2-weighted MR image. (**b**) Coronal T2-weighted image shows bilateral fusion of lateral bodies (arrows) with occipital condyle. Vertebral arteries passing through a bony channel were also observed.

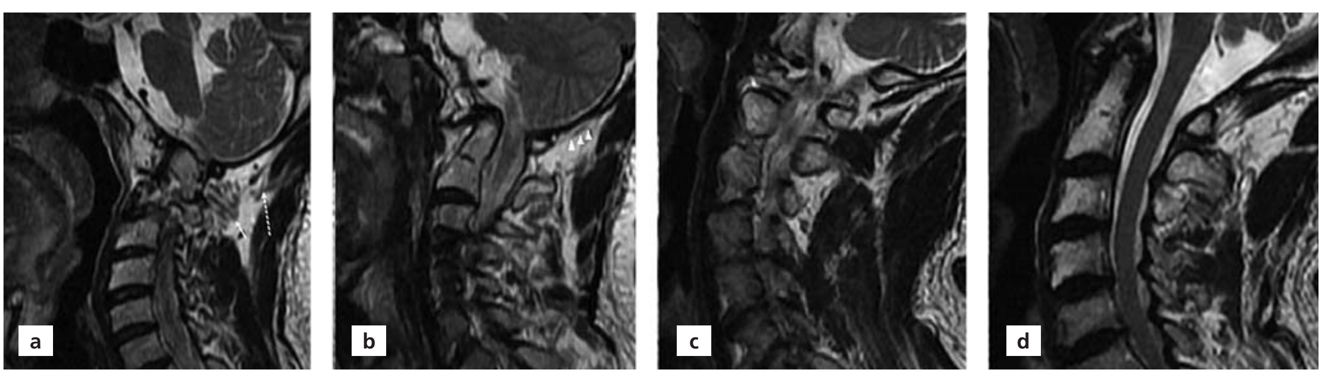

Figure 4. (**a**) A 52-year-old female with AOA. Sagittal T2-weighted MR images representing severe atrophic changes of rectus capitis posterior major (short arrow) and obliquus capitis inferior muscles (long arrow). (**b**) Nearly absence of rectus capitis posterior minor muscle (arrowheads). (**c**) Normal rectus capitis posterior major-minor (**d**) Obliquus capitis inferior muscles in a 52-year-old female on sagittal T2-weighted MR images.

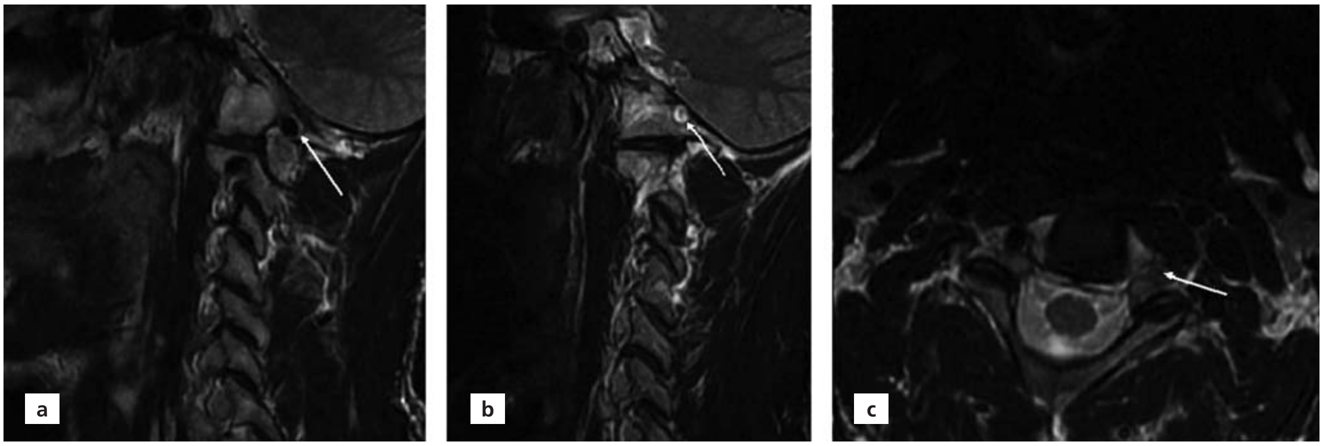

Figure 5. A 38-year-old male with AOA. (**a**) Right vertebral artery (**arrow**) coursing in the bony tunnel is seen on sagittal T2-weighted MR image. (**b**) Sagittal T2-weighted MR image shows empty bony tunnel (**arrow**). (**c**) T2-weighted MR image shows empty transverse foramen on the left suggesting agenetic left vertebral artery.

process or brainstem formations. In 7 cases, VA was in the tunnel close to the upper surface of fused lateral body and entrapped between the occiput and cerebellum. In 20 cases, VA was extending from transverse foramen of the axis to the intracranial space by passing through the bottom of the fused lateral body. VA agenesis and advanced hypoplasia were present in 2 and 8 cases, respectively (**Figure 5**). While 4 cases had unilateral normal VA, bilateral normal VAs were present in only 2 cases. Additional CVJ abnormalities according to the AOA types are shown in **Table 1**.

Discussion

CVJ is much more mobile than any other joint of the cervical spinal column. It is necessary to know the important biomechanical properties when spinal stabilization is planned in order to ensure the proper instrumentation in the case of trauma, tumor or degenerative disease. Occiput-C1 and C1–2 joints have unique biomechanics. While bone structures are important for stabilization in occiput-C1 joint, ligamentous structures provides much more stabilization compared to the bone elements in C1–C2 joint.[13] Most of the pathological conditions affecting CVJ become symptomatic with the involvement of neural structures. Signs and symptoms are variable; typically late-onset and slowly progress, remain stable at some time and recur rarely.[14]

Although AOA is often congenital, most of the cases do not develop symptoms up to the 2nd decade of their lives. This condition is thought to be due to the gradually increase in ligamentous laxity and the increase in the degree of instability with age.[15] The incidence of AOA in our study was 0.33%; female-male ratio was 3.4. With respect to age, patients were mostly in the 4th and 5th decades.

Table 1
CVJ abnormalities according to the AOA types

Type of AOA	Suboccipital muscle abnormality	VA abnormality	Increased AADI	Decreased PADI	C2–3 fusion	BI	Myelomalacia	Tonsillar herniation
Symmetric lateral body (n=14)	14	14	3	12	4	2	1	1
Complete (n=12)	12	12	6	12	7	6	4	-
Symmetric, anterior arch and lateral body (n=7)	7	5	1	5	2	1	1	1
Symmetric, posterior arch and lateral body (n=3)	3	3	-	2	-	-	-	-
Unilateral, lateral body (n=3)	3	3	-	2	-	-	-	-
Unilateral, partial lateral body (n=1)	1	1	-	1	-	-	-	-
Total (n=40)	40 (100%)	38 (95%)	10 (25%)	34 (85%)	13 (33%)	9 (23%)	6 (15%)	2 (5%)

AADI: anterior atlantodental interval; AOA: atlanto-occipital assimilation; BI: basilar invagination; CVJ: craniovertebral junction; PADI: posterior atlantodental interval; VA: vertebral artery.

Menezes[5] reported that anterior arch assimilation with BI is accompanied with instability explained by the restricted movement caused by weakening of supportive myoligamentous structure resulting from transfer of craniospinal first mobile segment into the C1–2 vertebrae, atlantoaxial joint weakness and progressive atlantoaxial subluxation. According to Wackenheim, cranial translocation of odontoid process is the main criterion for AOA, and other skeletal anomalies are accompanied, especially C2–3 vertebrae fusion.[16]

Gholve et al.[17] identified 2 types of BI directly causing brainstem compression and showing association with Chiari malformation and reduction in posterior cranial fossa and showed that the risk of atlantoaxial instability is high in cases with fused C2–3 vertebrae. In our study group, AOA was accompanied by posterior arch hypoplasia (n=16), midline defect of posterior arch (n=3) and unilateral agenesis of the posterior arch (n=3). BI was present in 9 (22.5%) cases and C2–3 vertebrae fusion in 13 (32.5%) cases. Both anomalies were more common in complete form AOA cases. Isolated anterior arch fusion was not observed in the study group. Of the cases in which BI was detected, 6 had complete form fusion, 2 had symmetric lateral body fusion, and one showed symmetric fusion of both anterior arch and lateral body. In other words, 7 of 9 cases with BI had anterior arch involvement. 4 of the cases accompanied by BI had C2–3 vertebrae fusion. Tonsillar herniation was detected in 2 cases which had isolated symmetric lateral body and lateral body with anterior arch type fusion accompanied with hypoplasia of the posterior arch.

AOA may narrow foramen magnum diameter and lead to neurological complications resulting from spinal cord compression.[1,18] BI and dorsal displacement of odontoid process typically results in reduced anteroposterior diameter of foramen magnum and significant compression on craniomedullar junction (CMJ). Sudden death cases associated with AOA have been reported in the literature.[19] Although increased AADI indirectly supports the possibility of instability, this increase may remain constant or there may not be neural compression. Therefore, increased AADI does not indicate neurological abnormality. On the other hand, decrease in PADI does not mean a neurological abnormality, either. However, the decrement of PADI can cause anterior spinal, VA and basilar artery failure without leading to direct spinal cord compression.[20] It has been reported that PADI value is correlated with the presence of paralysis and its severity. It is the most important potential indicator of neurological recovery after surgery in patients with rheumatoid arthritis.[21]

Greenbery[9] reported that PADI value of 14 mm or less means spinal cord compression; 14–18 mm means possible spinal cord compression, and 18 mm or above means no compression. In our study group, PADI was below 14 mm in 14 cases and mostly associated with complete form AOA. In 20 cases, PADI was measured between 14 to 18 mm. AADI was greater than 3 mm in 10 cases and mostly (6 cases) were associated with complete form AOA. 7 cases with decreased PADI (7/14) also had increased AADI. In 5 of 6 cases with myelomalacia at the CMJ, PADI was lower than14 mm, and in 3 cases AADI was above 3 mm. In 7 cases (7/14) with C2–3 vertebrae fusion, PADI was below 14 mm, in 5 cases between 14–18 mm and in 1 case PADI was above 18 mm. In all cases, in which BI was accompanied with myelomalacia, PADI was lower than 14 mm and, in this group C2–3 vertebrae fusion was present in 2 cases.

Wackenheim[16] stated that assimilation was always accompanied with VA abnormalities. Wang et al.[22] reported 4 different types of VA at the CVJ level in AOA. In Type 1, VA extends into the spinal canal and foramen magnum at the bottom of the C1 posterior arch immediately after leaving transverse foramen of the axis. VA course is below the assimilated C1 lateral mass. In Type 2, VA enters into the spinal canal and foramen magnum below the assimilated C1 posterior arch. In Type 3, VA passes through transverse foramen of the atlas, enters into osseous foramen generated by fused atlas and occiput. In Type 4, VA is unilaterally agenetic. Tubbs et al.[23] examined VAs in the skulls of 5 adult cadavers with AOA and reported the presence of abnormal osseous pathway into the cranium that mostly had posterior arch or hemiarch fusion. In this study, VA was abnormal in 38 cases (95%). VAs passing through the tunnel by crossing the bone bar formed by the fused occiput and lateral mass of atlas after leaving the transverse foramen of the axis was present in 30 cases and they showed Type 3 feature according to Wang's classification. In 20 cases, VAs were featured as Type 1 and 2. Agenetic VA (Type 4) and advanced hypoplastic VA was detected in 2 and 8 cases respectively. In 8 cases showing Type 3 VA property (in 6 cases VA was accompanied with BI), VAs showed entrapment between odontoid process, fused lateral mass and the brain stem. Again, VAs of 7 cases in this group were in the tunnel located near the upper surface of fused lateral mass and entrapped between the occiput and cerebellum. VA was completely normal in 2 cases and both cases had fusion of anterior arch and lateral mass accompanied by posterior arch hypoplasia. While right and left VA exhibited the same type of abnormality in most of the cases, in some cases, different types of VA abnormality were present in the same patient.

As well as the reduction of the luminal diameter caused by lateral mass protrusion to the foramen magnum, compression of the first cervical nerve affect suboccipital muscles and lead to abnormal head position and unstable walking.[24] Bodon et al.[8] examined the CVJ and suboccipital muscle changes in cadaver skull with occipitalization. In some studies, in cases with chronic head and neck pain, dizziness and imbalance complaints, atrophic changes were identified with MRI in suboccipital muscles especially in RCPM and RCPMA muscles.[25] In our study, RCPM, 1 of the suboccipital muscles, was abnormal in all patients. There was no identifiable muscular tissue in 20 cases. In 15 cases, RCP was severely atrophic. This condition may arise as a result of compression of the first cervical nerve and is also attributed to the atrophy of the muscle that is effective in flexion and extension of the head resulting from the movement restriction caused by assimilation.

With review of the study results, we realized that as the severity and distribution of bony fusion in AOA increased, the frequency of accompanying CVJ abnormalities also increased. In complete and symmetric lateral body/arch fusions, various CVJ abnormalities were accompanied, while partial and/or unilateral forms had less abnormalities. We also noticed that the type and severity of the fused bony segment can predict the nature of CVJ abnormality. In this context, cases with VA abnormality mostly had lateral body involvement. CVJ abnormality for suboccipital muscles was remarkably seen in all cases whatever the type and severity of assimilation.

Our study has several limitations. First, because the cervical MRIs were obtained by standard protocols, OCPS muscle, not included in the study area, could not be evaluated. Second, since axial plan images did not include the CVJ level in all cases, vertebral artery was only evaluated on sagittal images in some patients. To exclude these limitations as possible, we preferred sagittal T2-weighted imaging with thin slices (2 mm) and low slice spacing parameters (2 mm), especially in the selected cases. Sagittal scanning can be done more laterally for imaging of the suboccipital muscles.

Conclusion

Considering AOA has various types, ranging from complete to a minute bony fusion and according to the fused bony part of the atlas, cases with AOA can be simply overlooked. In order to improve the diagnostic rate of AOA and thus minimize the unnecessary radiologic examinations and reduce the complications especially in the patients scheduled for surgery, it is useful and important for the radiologists, neurologists, orthopedic surgeons and neurosurgeons to display severe atrophy of suboccipital muscles, VA abnormality, BI, C2–3 vertebrae fusion, reduction of foramen magnum sagittal diameter, and myelomalacia.

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Ultrasonographic assessment of spleen, kidney and liver size in licensed football players

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Abstract

Objectives: The aim of this study was to find out the effect of training on dimensions of spleen, kidney and liver in licensed football players.

Methods: The study group consisted of 53 licensed male football players and 52 male non-athlete controls. The visceral organ measurements were evaluated by ultrasonography. Independent samples t test and linear regression analysis were used for statistical analysis.

Results: The mean age was 22.11±3.36 years in footballers and 22.71±3.92 years in controls. The spleen's transverse axis length (4.89±0.52 cm) and liver's anteroposterior length (11.9±2.35 cm) were significantly higher in the footballers compared to the controls (4.42±0.65 cm and 10.39±1.9 cm; p<0.001 and p<0.001; respectively). However, the lengths of the long axis of the right kidney (10.3±0.86 cm), long axis of the left kidney (10.4±0.77 cm) and the transverse axis length of the right kidney (4.05±0.57 cm) were significantly lower in the footballers compared to the controls (10.69±0.68 cm, 10.97±0.68 cm, 4.35±0.74 cm; p=0.012, p<0.001, p=0.02; respectively). Furthermore, weekly training time was negatively correlated with transverse axis lengths of the right kidney (Beta=0.656; p<0.001) and of the left kidney (Beta=-0.275; p=0.042).

Conclusion: We consider that knowing the normal sizes of the visceral organs of footballers will be useful in determining the appropriate diagnosis and treatment and in accelerating the footballers to come back to competitions.

Keywords: football players, kidney, liver, spleen, ultrasonography

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Introduction

Football game is one of the most popular sports in the world. Modern football game requires strength, power, and speed from players.[1] Intensive training programs cause significant alterations in various tissues from microscopic to macroscopic scales. These alterations have been attributed to increased metabolic rate of striated muscle.[2] Internal organs such as liver and kidney respond to increased demand by functional alterations.[2,3] For instance, the responses of liver to increased demand due to sportive performance are increased glucose synthesis and glycogen degradation, caused by fall in insulin and rise in glucagon blood levels.[3] Exercise induces significant decrease in renal plasma flow, glomerular filtration rate and consequently diminished urine output as well.[4,5]

In many diseases, the size and morphology of these visceral organs also undergo significant changes. Perceived findings with different viral illness are splenomegaly, hepatomegaly and renomegaly.[6] The knowledge of the normal range of values of the various viscera is of importance in identification of early pathological changes, treatment and may prove very useful in making clinical management decisions regarding safe return to competitions.[6] Physical examination, such as palpation or percussion might be lacking to identify these changes.[7] Ultrasonography (USG) is a straightforward and solid approach to evaluate visceral organs with non-ionizing radiation. Another advantage is that it is available even in prime healthcare units.[8]

The aim of this study was to find out the effect of training on dimensions of spleen, kidney and liver in football players. There are few studies in the literature on the visceral organ dimensions of athletes in various branches.[6,9–14] Other studies mainly aimed to create reference values in healthy adults.[8,15–26] To our knowledge, this is the first study that has evaluated spleen, kidney and liver dimensions in the football players.

Materials and Methods

Our study group consisted of 53 licensed football players, who were 18 years old or above, and playing in a football club in Kahramanmaraş or its nearby cities for at least five years. These players trained regularly for at least 5 h a week. 52 healthy men who did not perform any physical activity were included to the study as controls. The liver, spleen and both kidney measurements of the football players and the control group were evaluated using US. Body mass index (BMI) of every subject was calculated to determine obesity based on World Health Organization (WHO) obesity classification. BMI ranges were as follows; underweight: under 18.5 kg/m2; normal weight: 18.5 kg/m^2 to 24.99 kg/m^2; overweight: 25 kg/m^2 to 30 kg/m^2; and obese: over 30 kg/m^2. The study was approved by the Clinical Research Ethics Committee of Kahramanmaraş Sütçü İmam University. The written informed constent was obtained from all subjects and the study was carried out in accordance with the Declaration of Helsinki. No sedation or preparation was applied before the examination. Patients who had no disease associated with the organs measured, and admitted to our radiology clinic with the request of USG for various reasons were examined. Patients with congenital anomalies or systemic diseases were not included in the examination. All organs examined were ultrasonographically in normal size, shape, and echostructure. Sonographic examinations were performed using an Aplio-400 (Toshiba, Tokyo, Japan). The Aplio-400 PVT-375BT Transducer/ Probe had been used with a 3.5 MHz center frequency.

Liver measurements were made for the long axis and anteroposterior length. The long axis measurement of the liver was done on right mid-clavicular line while the patient was in the left lateral decubitus position. During this measurement, the longest distance between the right and left lobe extreme corners was measured by imaging the inferior vena cava and gallbladder on the same plane. The lateral segment of the left lobe was considered to be the extreme boundary in the medial, and the posterior inferior segment of the right lobe was considered to be the extreme boundary in the lateral margin (**Figure 1a**). The measurement for the anteroposterior length was performed on the midsagittal plane (horizontal plane passing through the xiphoid process) while the person was in the supine position. The measurement was performed at the origin points of the three hepatic veins by imaging on the same plane. In this position, the top edge of the liver under the diaphragm was considered the uppermost border (**Figure 1b**).[27]

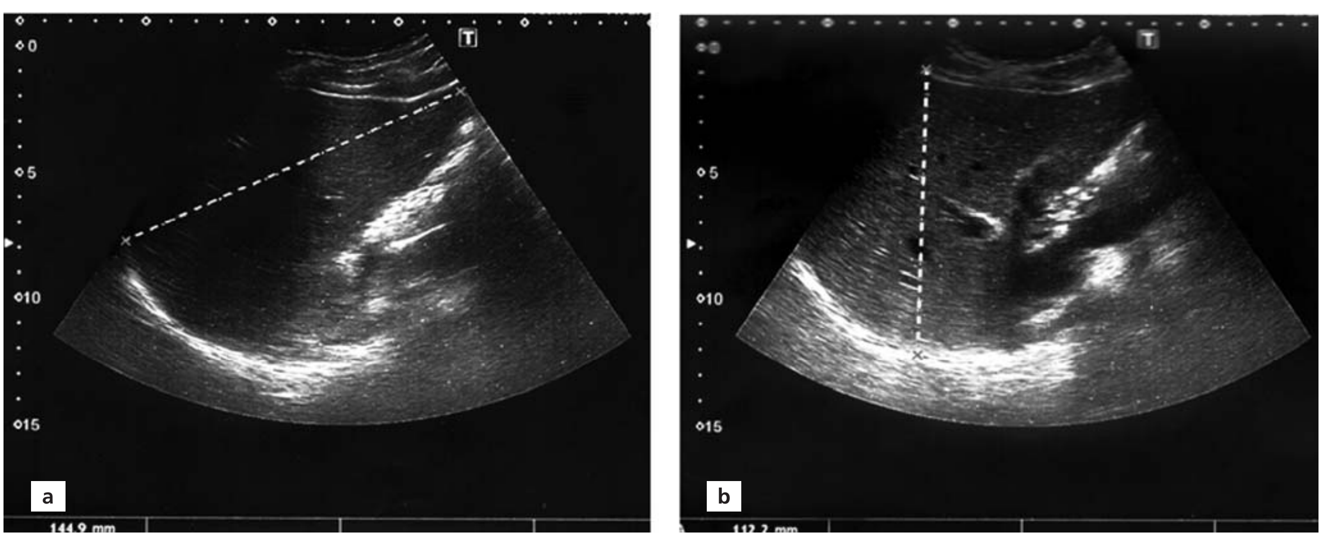

Figure 1. USG images showing measurements of (**a**) long axis length, and (**b**) anteroposterior length the liver.

Spleen long axis measurements, on the other hand, were made by taking to the splenic hilum examination plan while the subjects were in supine position or slightly right lateral decubitus position. In this measurement, imaging of splenic vein invading splenic hilum was taken as the landmark. Long axis measurement was performed by measuring the distance between the most superomedial and inferolateral extremities of the spleen (**Figure 2a**). Measurement of the transverse axis of the spleen was performed by measuring the distance between the most superolateral border of the spleen and the hilum (**Figure 2a**).[27]

Both kidney measurements were obtained by imaging the coronal plane passing through the renal hilum while the subject was in supine position or in cubital position slightly in the right or left lateral direction. The extreme distance from the pole to the pole was considered as the long axis of the kidney (**Figure 2b**). Transverse axis length was obtained by measuring in such a way that it will be perpendicular to the long axis of the kidney in the hilar region (**Figure 2b**).[27] All visceral organs measurements were carried out when the patient was in deep inspirium by same experienced radiologist.

Three sequential measurements were taken and the mean was calculated to ensure minimum intraobserver variation and greater accuracy and reliability of measurements.

For statistical analysis, Kolmogorov-Smirnov test was applied for the suitability of normal distribution of variables. The case and control group comparisons in normal distributed variables were performed with independent samples t-test. Multivariate regression analysis was performed for the effect of training, age and BMI on visceral organ size. Autocorrelation was examined by the Durbin-Watson test. Descriptive statistics were expressed with mean±SD. Statistical significance level was accepted as $p<0.05$. Data were evaluated using the Statistical Package for Social Sciences (SPSS for Windows, version 22.0, IBM Corporation, Armonk, NY, USA).

Results

The study group consisted of 53 (50.5%) licensed football players who had been keeping up their active football careers in Kahramanmaraş or its nearby cities. Controls were 52 (49.5%) healthy men who did not perform any physical activity. The mean age was 22.11±3.36 years, BMI was 23.60±1.74 kg/m^2, weekly training time was 7.89±1.27 hours and the length of their football careers was 9.19±3.45 years in the footballers. The mean age of the control group was 22.71±3.92 years and BMI was 24.09±2.82 kg/m^2. There were no significant differences for age and BMI between footballers and controls (p=0.402, p=0.282, respectively) (**Table 1**).

The spleen's transverse axis length (4.89±0.52 cm) and the liver's anteroposterior length (11.9 ±2.35 cm) were significantly higher in the footballers compared to controls (4.42±0.65 cm, 10.39±1.9 cm; p<0.001, p<0.001, respectively). However, the lengths of the long axis of the right kidney (10.3±0.86 cm), the long axis of the left kidney (10.4±0.77 cm) and the transverse axis of the right kidney (4.05±0.57 cm) were significantly lower in the footballers compared to controls (10.69±0.68 cm, 10.97±0.68 cm,

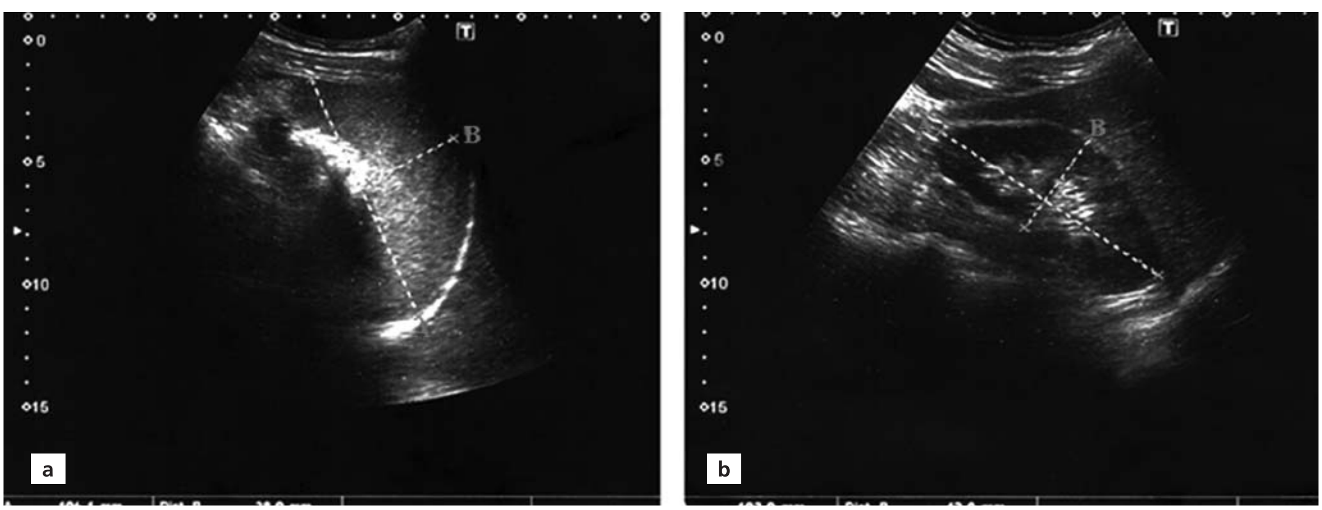

Figure 2. USG images showing measurements of (**a**) long axis length (dashed lines A) and transvers axis length (dashed lines B) of the spleen, and (**b**) long axis length (dashed lines A) and transvers axis length (dashed lines B) of the right kidney

4.35±0.74 cm; p=0.012, p<0.001, p=0.020; respectively) (**Table 2**).

Weekly training time and age were positively correlated with the long axis of the spleen (Beta=0.280; Beta=0.043, r=0.283; p=0.04, respectively). Furthermore, weekly training time, age, and BMI were positively correlated with the anteroposterior length of the liver (Beta=0.558; p=0.001; Beta=0.388; p=0.001; Beta=0.258, p=0.006, respectively). BMI was positively correlated also with the long axis of the liver (Beta=0.292; p=0.04). Moreover, age was positively correlated with the long axis of the right kidney (Beta=0.415; p=0.003). On the other hand, weekly training time was negatively correlated with transverse axis lengths of the right (Beta=-0.656; p<0.001) and the left kidney (Beta=-0.275; p=0.042) (**Table 3**).

Discussion

To our knowledge, this is the first study to investigate the effect of training on dimensions of spleen, kidney, and liver in football players. We found that the spleen's transverse axis length and the liver's anteroposterior length in football players were higher compared to the controls. However, the lengths of the long axis of the right kidney, the long axis of the left kidney and the transverse axis of the right kidney were lower in the footballers compared to controls. Furthermore, weekly training time was negatively correlated with transverse axis lengths of the right and left kidneys.

Kidneys are located in the retroperitoneal space at the level of T12–L3 vertebrae. Changes in the size of the kidneys are important in diagnosis and follow up of many diseases. Size of the kidneys has a particular importance in the evaluation of clinical cases such as hypertension due to renal artery stenosis, renal failure, frequent urinary tract infection complaints, and tracking renal transplant recipients.[15,28] Buchholz et al.[16] in their study on 194 healthy subjects (98 males, 96 females) aged between 13 and 80 years reported a left kidney length of 10.4±0.7 cm and a width of 4.7±0.5 cm for subjects aged 21–30 years. The researchers measured the right kidney length as 10.3±0.6

Table 1

Descriptive data of the study population using independent samples t test.

	Controls (n=52) Mean±SD	Footballers (n=53) Mean±SD	p
Age (year)	22.71±3.92	22.11±3.36	0.402
Weight (kg)	72.36±10.82	70.02±8.02	0.211
Height (cm)	176.87±6.74	175.81±6.86	0.429
BMI (kg/m^2)	24.09±2.82	23.60±1.74	0.282
Weekly training time (hours per week)	-	7.89±1.27	-
Active football career (year)	-	9.19±3.45	-

Table 2

Correlation of visceral organ dimensions (cm) among the study population using independent samples t test.

	Controls (n=52) Mean±SD	Footballers (n=53) Mean±SD	p
Long axis length of spleen	10.66±1.05	10.91±1.28	0.273
Transverse axis length of spleen	4.42±0.65	4.89±0.52	<0.001*
Long axis length of liver	14.73±1.25	14.41±1.03	0.152
Anteroposterior length of liver	10.39±1.9	11.9±2.35	<0.001*
Long axis length of right kidney	10.69±0.68	10.30±0.86	0.012*
Transverse axis length of right kidney	4.35±0.74	4.05±0.57	0.02*
Long axis length of left kidney	10.97±0.68	10.4±0.77	<0.001*
Transverse axis length of left kidney	4.81±0.68	5.03±0.56	0.07

*p<0.05

Table 3

Multivariate regression analysis of the effects of age, BMI and training time to visceral organ dimensions in football players.

	Regression Model	Beta X_1	Beta X_2	Beta X_3	R^2	P (X_1)	P (X_2)	P (X_3)
Long axis length of spleen	$Y=5.788+0.108X_1+0.028X_2+0.282X_3$	0.283	0.038	0.28	0.193	0.04*	0.775	0.043*
Transverse axis length of spleen	$Y=5.409-0.016X_1-0.002X_2-0.016X_3$	-0.104	-0.008	-0.039	0.014	0.488	0.958	0.789
Long axis length of liver	$Y=11.626-0.011X_1+0.172X_2-0.112X_3$	-0.035	0.292	-0.138	0.111	0.802	0.040*	0.326
Anteroposterior length of liver	$Y=9.799+0.271X_1+0.346X_2+1.033X_3$	0.388	0.258	0.558	0.624	<0.001*	0.006*	<0.001*
Long axis length of right kidney	$Y=3.962+0.020X_1+0.015X_2+0.003X_3$	0.415	0.108	0.121	0.233	0.003*	0.407	0.353
Transverse axis length of right kidney	$Y=5.361-0.007X_1+0.051X_2-0.295X_3$	-0.042	0.157	-0.656	0.485	0.699	0.142	<0.001*
Long axis length of left kidney	$Y=5.978+0.057X_1+0.103X_2+0.113X_3$	0.249	0.232	0.185	0.182	0.071	0.087	0.172
Transverse axis length of left kidney	$Y=5.139-0.039X_1+0.074X_2-0.121X_3$	-0.236	0.232	-0.275	0.202	0.083	0.083	0.042*

Multivariate regression analysis and Durbin-Watson test. **R2:** explanatory coefficient; **X1:** age; **X2:** BMI; **X3:** training time. *p<0.05.

cm and the width as 4.3±0.8 cm. Udoaka et al.[17] measured the left kidney length as 10.4±1.08 cm, and width as 5.21± 0.76 cm, the right kidney length as 10.0±0.99 cm and width as 5.68±0.7 cm. In other study performed on 140 healthy subjects (69 males, 71 females), the left kidney length of the 18–30 year-old group was reported as 9.78±0.75 cm and the width as 4.5±0.48 cm; the right kidney length was reported as 9.66±0.84 cm and width as 4.4±0.68 cm.[18] Yadav et al.[19] studied 110 healthy subjects between 15—80 years of age and measured the left kidney length has 9.97±0.77 cm, the width as 4.01±0.95 cm, the right kidney length as 9.88±1.01 cm and width as 4.06±0.63 cm. El-Reshaid and Abdul-Fattah,[20] in a study on 252 healthy subjects between 18–30 years of age, measured the left kidney length as 10.6±1.2 cm and the right kidney length as 10.75±1.3 cm. They measured the left kidney length as 10.8±1.1 cm and the right kidney length as 10.5±0.9 cm in healthy subjects between the ages of 60 and 81. They also reported that increasing of age does not affect the kidney length, but decreases the glomerular filtration rate. Additionally, in a study conducted in Europe, it has been reported that the normal kidney length for men should be between 9.2 cm and 13.3 cm and the normal kidney width for men should be between 3 and 7.1 cm.[29] Consistent with the studies in the literature, in our study, the control group's long axis length of the right kidney was measured as 10.69±0.68 cm, transverse axis length was measured as 4.35±0.74 cm; long axis length of the left kidney was measured as 10.97±0.68 cm and the transverse axis length was measured as 4.81±0.68 cm. We think minor differences between the measurements in the studies depend on the race, gender, age, BMI and measurement technique used. We found that long axis and transverse axis lengths of the right kidney, and long axis length of the left kidney in football players were shorter than those of the non-athletes. It has been reported that moderate and high-intensity exercises included in the football training programs cause a marked decrease in the urine volume, the flow of the kidney plasma, the circulating blood flow in the kidneys and the renal filtration rate.[4,5] Also, the finding of the decreased renal cortical thickness of subjects with decreased glomerular filtration rate obtained from previous studies explains why the kidney lengths of footballers are smaller than those of the control group.[3,30,31] Furthermore, weekly training time was negatively correlated with transverse axis lengths of the right kidney and of the left kidney.

The liver is the largest organ in the human body that lies at the right hypochondriac region and it is divided into large right and small left lobes.[32] Changes in the normal sizes of the liver are an important indicator of disease development.[33] Knowing the normal sizes is important in surgical planning, in the tracking and treatment of the disease.[34,35] Hepatomegaly is a term used to describe an enlarged liver beyond its normal sizes (longitudinal length ≥16 cm).[21,22] Hepatomegaly alone is not a disease, but rather a potential indication of the process that causes it.[36] Many researchers have reported the long axis length of the liver as 145.15±16.22 mm,[18] 14.0±1.7 cm,[22] 13.7±1.42 cm,[37] 12.68±2.57 cm[32] and the anteroposterior length as 11.4±1.94 cm.[37] In our study, the long axis length of the liver was measured as 14.73±1.25 cm and the anteroposterior length as 10.39±1.90 cm in the control group in accordance with these studies. We found that the anteroposterior length of the liver in football players is longer than that of the controls. The liver is an important organ that contains enzymes that meet the increased energy needs of the body during high-density aerobic exercises.[10] Growth in the liver size is to meet the increased energy needs of the organ.[12] High protein diets are also given to the athletes' diet, taking into account this high energy requirement.[11,38] It was reported that those who have been active in the American football team professionally for 2–3 years had heavier liver (0.29 kg), heart (0.08 kg) and kidney (0.09 kg) mass compared to the newcomers.[12] Furthermore, weekly training time, age, and BMI were positively correlated with the anteroposterior length of the liver. Also, BMI was positively correlated with the long axis of the liver. Not taking the blood sample from football players and the control groups for various tests including the visceral organs, not measuring the portal vein diameter, and not knowing the football players' eating habits, training program, and their medical history were limitations of this study.

Spleen size is an integral part of abdominal ultrasonography because both enlarged and small spleens can be indicative of a variety of physical conditions.[9,23] In addition, splenomegaly (longitudinal length ≥13 cm) may be a risk factor for splenic rupture.[13] False-positive labeling of a patient as having splenomegaly can lead to conducting unnecessary medical tests and anxiety for the athletes as well as delaying for participation in contact sports activity.[6,9] In various studies, the long axis length of the spleen was reported as 10.76±1.84 cm,[24] 11.1±0.9 cm[25] and the average as 10.9 cm[23] for healthy adults. There are also studies reporting transverse axis length as 5.27±0.93 cm,[28] 4.4 cm±0.5 cm[25] and the average as 4.5 cm.[23] In accordance with these studies, we found that the control group's long axis length of the spleen was 10.66±1.05 cm and the transverse axis length was 4.42±0.65 cm. In previous studies on athletes, the long axis length of the spleen was reported as 10.82±1.55 cm,[14] 12.79±6.46 cm,[6] 11.4±1.7 cm,[9] and transverse axis length was 5.20±1.21 cm,[14] 5.77±6.70 cm[6] and 5±0.8 cm.[9] Consistent with these studies, the long axis length of the spleen was measured as 10.91±1.28 cm and the transverse axis length was measured as 4.89±0.52 cm in our study. In addition, the transverse axis length of the spleen in footballers was found higher than the control group, and weekly training time and age were positively correlated with the long axis of the spleen.

Conclusion

We conclude that knowing the normal sizes of the visceral organs of footballers will be useful in determining the appropriate diagnosis and treatment and accelerating the footballers come back to competitions.

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Views of medical students on anatomy education supported by plastinated cadavers

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Abstract

Objectives: The purpose of this study was to assess the awareness of medical students who receive education with anatomic plastinated cadavers and determine their views on the education model that is provided to them.

Methods: This study was performed on İstanbul Medeniyet University first (n=150) and second (n=190) semester volunteer medical students (n= 340). Data were collected using questionnaire that consisted of 27 closed-ended questions.

Results: 68.8% of the students did not have any knowledge about fixation methods, while 47.4% did know about plastination. 60.2% of the students believed that the anatomy education provided with plastinated cadavers had a positive effect on their anatomical knowledge. 39.4% of the students believed the anatomy education provided with plastinated cadavers affected their theoretical anatomy exam success positively. 60.8% of the students believed that anatomical structures were sufficiently represented in three dimension in the plastinated cadaver. 76.2% of the students described the system where the anatomical structures could be seen most easily as the musculoskeletal system, and 50% described most difficulty as neuroanatomy.

Conclusion: We believe that the data of this study will benefit the studies that will assess the effectiveness of plastinated cadavers in anatomy education.

Keywords: cadaver; education; medical students; plastination

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Introduction

Cadavers are indispensable tools that have been used by students and educators for teaching anatomy since the Renaissance.[1] Students are able to see the relationships among anatomic structures macroscopically and in three dimensions in anatomy education with cadavers. Cadavers are usually preserved by the method formaldehyde fixation. However, there are some disadvantages of the method of fixation by formaldehyde. The negative effects of formaldehyde on different systems on body have been reported previously.[2,3]

With the fixation method that was first developed by Gunther von Hagens in 1977[4] and named plastination, the exposure of students and lecturers to formaldehyde was prevented. The histological characteristics of the tissues that are fixated with this method are also preserved.[5] Tissues may be stored for a long time in a nontoxic, dry, durable and odorless way.[6] Therefore, plastinated cadavers have been a significant tool of education in anatomy training in time and their usage has become prevalent.[7,8]

Due to the lack of cadaver donation in Turkey and the increasing number of medical schools, problems are experienced in obtaining cadavers for undergraduate and postgraduate anatomy education.[9] Being able to observe the anatomic structures clearly three dimensionally and being helpful to conceptualizing and understanding diagnostic tests increased the interest of medical schools on plastinated cadavers.[6,10] Therefore, the aim of this study was to assess the awareness of medical students educated using plastinated cadavers, and determine their views on this education model.

Materials and Methods

Educational Methodology

The anatomy classes that are provided in the School of Medicine of İstanbul Medeniyet University (IMU) where this study was carried out are given in semesters I and II in all committees. In semester I, locomotor system subjects including bones, joints and muscles are studied. In semester II, the cardiovascular system, respiration system, gastrointestinal system, neuroanatomy and urogenital system are studied in this order. In anatomy education where subjects are systematically given, practical classes are held in anatomy laboratories following theoretical classes. Practical classes are held with study groups of 10–15 people. The students selected in the workgroups prepare for the content of the subject to be taught by consulting with the educators before the practical class and take an active role in the classes. Firstly, they examine the structures related to the subject on anatomy models under the coordination of the educators. The students' working time with the anatomy models changes between 45 and 60 mins based on the subject. Then, the structures in question are presented to the workgroups on plastinated cadavers and plastinated pieces by expert educators for about 15–20 min. In this study, imported plastinated cadavers and pieces prepared for anatomy education were used. The students were able to ask questions comfortably to the educators at all stages of the education. In the practical applications, one whole-body plastinated cadaver, plastinated pieces for one upper extremity and one lower extremity were used. The whole-body plastinated cadaver was in two halves as left and right in the mid-sagittal plane. While surface structures were visible on one half of the body, surface structures were partly removed from the other half so that the deeper structures could be observed. Anatomy practical examination was held at the end of each committee. In these examinations, questions were asked on anatomy models and plastinated specimens. The numerical weight of the questions that were asked over plastinated cadavers constituted about 10% of all questions based on the anatomy subjects that were learned.

Procedure

This study was carried out in the period of March–May 2018 with a total of 340 students that were enrolled in IMU faculty of medicine in semester I (n=150) and in semester II (n=190). Ethics approval was received before this cross-sectional descriptive study (Approval no: 2018/00071; date: 06.03.2018). The data were collected by a questionnaire that consisted of 27 closed-ended questions based on the principle of volunteerism. Thirteen questions employed a 5-point Likert-type scale where the options were "Always", "Mostly", "Frequently", "Sometimes" and "Never". For the reliability of the responses, the students were given the freedom to include or not include their names. The questionnaire contained questions on the demographic characteristics and success levels of the students, questions on their levels of knowledge on fixation and plastination, questions on the contribution of the education that is provided with plastinated cadavers on their levels of knowledge in anatomy, and questions on the subjects that they utilized the least and the most in the education provided with plastinated cadavers.

Statistical Analysis

After obtaining percentages and frequencies, data were analyzed using the Statistical Package for Social Sciences (SPSS for Windows, version 22.0, Armonk, NY, USA). Means and standard deviations were provided for the nominal data, while frequencies and percentages were provided for the categorical data. Pearson's chi-squared test and Fisher's exact test were used to determine the relationships among the categorical variables. $p<0.05$ was accepted as the level of statistical significance.

Results

The mean age of the 340 students who participated in the study was 20.04±1.37 (min:18–max:26); 178 (52.4%) were females and 162 (47.6%) were males. The descriptive characteristics, success statuses and weekly durations of personal study for anatomy of students are shown in **Table 1**.

68.8% of the students did not have any knowledge on fixation methods, while 47.4% did know about plastination. The distribution of the knowledge levels of the students on the concepts of fixation, plastination and cost of plastinated cadavers are presented in **Table 2**.

The views of the students on the methodology of the education provided to them with plastinated cadavers, its contribution to their levels of theoretical and practical knowledge, effect on their theoretical and practical examination success and contribution of the clinical practices they will conduct in the future were assessed on a 5-point Likert-type scale. The responses that were provided to these questions by the students are given in **Table 3**.

The anatomical systems where the structures can be seen the least and the most on plastinated cadavers were asked. 76.2% of the students described the system where the anatomical structures could be seen the most as the musculoskeletal system, and 50% described the system where structures could be seen the least as neuroanatomy.

While 53.8% of the students responded to the question "Did you have a perception of meeting a real human body when you met a plastinated cadaver for the first time?" as "No", the rates of those who provided answers

Table 1
The descriptive characteristics, success statuses and weekly durations of personal study for anatomy of students in semester I and II.

General information of students		Academic semester Semester I (n)	Semester II (n)	Total number of students n (%)
Gender	Female	77	101	178 (52.4)
	Male	73	89	162 (47.6)
Repeated courses	No	129	166	295 (86.8)
	Yes / 1 time	19	18	37 (10.9)
	Yes / 2 times	1	6	7 (2.1)
	Yes / 3 times	-	-	-
	Yes / 4 times	1	-	1 (0.3)
Grade point average	0–49	9	4	13 (3.8)
	50–59	50	27	77 (22.6)
	60–69	48	78	126 (37.1)
	70–79	33	55	88 (25.9)
	80–89	10	23	33 (9.7)
	90–100	-	3	3 (0.9)
The weekly anatomy study time	0–1 hours	60	96	156 (45.9)
	1–3 hours	53	62	115 (33.8)
	3–5 hours	25	24	49 (14.4)
	More than 5 hours	12	8	20 (5.9)

of "Yes" and "Undecided" were 26.6% and 20%, respectively.

The students responded to the question "Would you consider joining a course about methods for preparing plastinated cadavers in the following years?" as "Yes" by 30%, "No" by 37.6% and "Undecided" by 32.4%.

82.6% of the students responded to the question "Would you like to receive anatomy education by making a dissection on cadavers by yourselves?" as "Yes", while the rates of those who responded as "No" and "Undecided" were 6.8% and 10.6%, respectively.

The relationship between the responses of the students to questions that asked the adequacy of the duration of the education provided with plastinated cadavers, its contribution on the levels of knowledge on anatomy and its contribution on theoretical and practical examination success and their class levels they were enrolled in is shown in **Table 4**.

Table 2
The distribution of the knowledge levels of the students on the concepts of fixation, plastination and costs of plastinated cadavers and the statistical significance levels.

		Academic semester Semester I n (%)	Semester II n (%)	Total number of students n (%)	p
Do you have any knowledge of fixation methods?	Yes	4^a (2.7)	9^a (4.7)	13 (3.8)	
	No	106^a (70.7)	128^a (67.4)	234 (68.8)	0.572
	I have less information	40^a (26.7)	53^a (27.9)	93 (27.4)	
Do you have any knowledge of plastination?	Yes	38^a (25.3)	23^b (12.1)	61 (17.9)	
	No	52^a (34.7)	109^b (57.4)	161 (47.4)	0.0001*
	I have less information	60^a (40)	58^a (30.5)	118 (34.7)	
Do you have any knowledge of the costs for plastinated cadavers?	Yes	63^a (42)	28^b (14.7)	91 (26.8)	
	No	48^a (32)	114^b (60)	162 (47.6)	0.0001*
	I have less information	39^a (26)	48^a (25.3)	87 (25.6)	

Each subscript letter (a, b) denotes a subset of class categories whose column proportions do not differ significantly from each other at the 0.05 level. For each pair of columns, the column proportions (for each row) are compared using a z test. If a pair of values is significantly different, the values have different subscript letters assigned to them. *p<0.05.

Table 3
Views of students on the education provided with plastinated cadavers.

	Always n (%)	Mostly n (%)	Frequently n (%)	Sometimes n (%)	Never n (%)
Do you think the duration of education that is provided with plastinated cadavers is sufficient within the total education time for anatomy?	6 (1.8)	25 (7.4)	31 (9.1)	141 (41.5)	137 (40.3)
Do you think the anatomy education provided with plastinated cadavers provided significant contributions on your theoretical anatomy knowledge levels?	46 (13.5)	96 (28.2)	63 (18.5)	94 (27.6)	41 (12.1)
Do you think the anatomy education provided with plastinated cadavers provided significant contributions on your practical anatomy knowledge levels?	56 (16.5)	104 (30.6)	63 (18.5)	90 (26.5)	27 (7.9)
Do you think the anatomy education provided with plastinated cadavers affected your theoretical anatomy examination success positively?	27 (7.9)	55 (16.2)	52 (15.3)	122 (35.9)	84 (24.7)
Do you think the anatomy education provided with plastinated cadavers affected your practical anatomy examination success positively?	35 (10.3)	80 (23.5)	40 (11.8)	111 (32.6)	74 (21.8)
Do you think the anatomy education provided with plastinated cadavers will provide benefits in the clinical applications that you will perform in the future?	58 (17.1)	75 (22.1)	68 (20.0)	98 (28.8)	41 (12.1)
Do you think anatomical structures were sufficiently represented in three dimensions in the plastinated cadaver?	29 (8.5)	98 (28.8)	80 (23.5)	109 (32.1)	24 (7.1)
Do you think the relationships of the anatomical structures that you worked on in the plastinated cadaver with surrounding structures were represented clearly?	16 (4.7)	84 (24.7)	86 (25.3)	129 (37.9)	25 (7.4)
Are you satisfied that anatomic structures are presented on a plastinated cadavers in groups of 10-15 people?	22 (6.5)	52 (15.3)	54 (15.9)	106 (31.2)	106 (31.2)
Do you think you have sufficiently contacted the plastinated cadaver?	16 (4.7)	28 (8.2)	29 (8.5)	62 (18.2)	205 (60.3)
Do you think the numbers of plastinated cadavers and pieces at the laboratory where you receive education are sufficient?	10 (2.9)	37 (10.9)	43 (12.6)	108 (31.8)	142 (41.8)
Are you pleased that a part of the examination questions in the anatomy practical examination are asked through a plastinated cadaver?	20 (5.9)	39 (11.5)	48 (14.1)	92 (27.1)	141 (41.5)
Would you like for the numerical values of the questions that are asked through a plastinated cadaver in the anatomy practical examination to be increased?	23 (6.8)	20 (5.9)	25 (7.4)	60 (17.6)	212 (62.4)

Discussion

Although cadaver dissection has been seen as the main component of anatomy education for centuries,[11] educational instruments such as computer-assisted educational tools (simulation and animation), radiographical images, three-dimensional plastic anatomical models and virtual reality applications are used prevalently due to advances in technology today.[3,12–14] In recent years, plastinated cadavers also became alternative educational tools to wet cadavers, and they have been started to be included in the educational process especially in countries where there are problems in cadaver donation.[15] This is why feedback that is received from students regarding the methodology of education provided with plastinated cadavers will provide opportunities for developing more effective education strategies.

Plastination is known as a fixation method where tissue fluids and partly tissue lipids are slowly replaced by polymers. In this method, students can comfortably touch the cadaver and the exposure to toxic substances that are used in classical fixation is prevented.[15] Despite the advantages of plastinated cadavers, the knowledge of students in different regions of the world regarding plastination is insufficient. 70% of the students in Nigeria and 98.23% of the students in India had no knowledge on plastination.[6,7] In this study, the rate of students who had no knowledge on

Table 4

Distribution of the views of students on the education provided with plastinated cadavers based on class levels and their statistical significance levels.

		Academic semester		
		Semester I n (%)	Semester II n (%)	p
Do you think the duration of education that is provided with plastinated cadavers is sufficient within the total education time for anatomy?	Always	4[a] (2.7)	2[a] (1.1)	0.001*
	Mostly	19[a] (12.7)	6[b] (3.2)	
	Frequently	22[a] (14.7)	9[b] (4.7)	
	Sometimes	74[a] (49.3)	67[b] (35.3)	
	Never	31[a] (20.7)	106[b] (55.8)	
Do you think the anatomy education provided with plastinated cadavers provided significant contributions on your theoretical anatomy knowledge levels?	Always	24[a] (16)	22[a] (11.6)	0.005*
	Mostly	53[a] (35.3)	43[b] (22.6)	
	Frequently	28[a] (18.7)	35[a] (18.4)	
	Sometimes	35[a] (23.3)	59[a] (31.1)	
	Never	10[a] (6.7)	31[b] (16.3)	
Do you think the anatomy education provided with plastinated cadavers affected your theoretical anatomy examination success positively?	Always	15[a] (10)	12[a] (6.3)	0.007*
	Mostly	26[a] (17.3)	29[a] (15.3)	
	Frequently	30[a] (20)	22[b] (11.6)	
	Sometimes	55[a] (36.7)	67[a] (35.3)	
	Never	24[a] (16)	60[b] (31.6)	
Do you think the anatomy education provided with plastinated cadavers affected your practical anatomy examination success positively?	Always	22[a] (14.7)	13[b] (6.8)	0.006*
	Mostly	40[a] (26.7)	40[a] (21.1)	
	Frequently	22[a] (14.7)	18[a] (9.5)	
	Sometimes	42[a] (28)	69[a] (36.3)	
	Never	24[a] (16)	50[b] (26.3)	
Do you think anatomical structures were sufficiently represented in three dimensions in the plastinated cadaver?	Always	21[a] (14.0)	8[b] (4.2)	0.001*
	Mostly	55[a] (36.7)	43[b] (22.6)	
	Frequently	36[a] (24)	44[a] (23.2)	
	Sometimes	31[a] (20.7)	78[b] (41.1)	
	Never	7[a] (4.7)	17[a] (8.9)	

Each subscript letter (a, b) denotes a subset of class categories whose column proportions do not differ significantly from each other at the 0.05 level. For each pair of columns, the column proportions (for each row) are compared using a z test. If a pair of values is significantly different, the values have different subscript letters assigned to them. *p<0.05.

fixation was 68.8%, while the rate of those who had no knowledge on plastination was 47.4%. Moreover, the plastination knowledge levels of semester I students were higher in comparison to those of semester II students. Although the plastination knowledge levels of the students in this study were higher than those in previous studies, it was observed that almost half of them did not have any knowledge on the concept of plastination. In this study, semester I and II students were not informed about the plastination techniques before the lectures. In this context, providing basic information about plastination techniques in anatomy classes may increase students' awareness on plastination. Higher utilization of plastinated cadavers by semester I students based on the subjects they learned may have caused their higher levels of knowledge in comparison to semester II students.

Debates about the effectiveness of educational tools that are used in anatomy education have been going on for years. As in the past, it is stated today that cadaver dissection is the main and most valuable element of anatomy education.[16,17] Additionally, the value and interest paid by students to dissection in anatomy education are still ongoing.[18,19] While the students in this study have used models and plastinated cadavers, 82.6% stated that they would like to receive anatomy education by making dissections by themselves.

On the other hand, the prevalence of using plastinated cadavers has made it necessary to assess the effectiveness of these educational materials on students. In study of Fruhstorfer et al.,[20] 93.6% of the students (the sum of "good" and "very good" answers) stated that plastinated cadavers were valuable in learning anatomy. Similarly, in the study of Azu et al.,[7] 75% of the students believed that plastinated cadavers were useful in learning anatomy. While Lattore et al.[15] found that plastinated cadavers were useful for both anatomy students (mean 2.34/3) and anatomy educators (mean 2.43/3). In this study, the rate of the students who stated that plastinated cadavers had positive contribution on their theoretical anatomy knowledge was found as 60.2% (the sum of frequently, mostly and always). We also found that the views of semester I students on the positive contribution of plastinated cadavers to their theoretical knowledge levels were more positive than those of semester II students. The reason why semester I students found plastinated cadavers to be more useful may be that the structures of the locomotor system, especially muscles, that are studied in semester I are more clearly seen in plastinated specimens. These results show the positive contribution of plastinated cadavers on anatomy education.

In a well-dissected plastinated cadaver, anatomical structures can be more easily distinguished from each other in comparison to a wet cadaver. In a previous study, students reported that structures are more clearly seen in plastinated cadavers and their relationship with surrounding structures are represented better.[6] The non-toxic nature of plastinated cadavers and the fact that students can comfortably touch them are anticipated positively by students. However, the fact that the structures are hard and breakable when forced is a disadvantage for students in reaching deeper structures.[20] Making the dissection before plastination, in compliance with the education program, and conducting especially the dissection of muscles from the surface towards the deep in a topographical discipline may increase the effectiveness of plastinated cadavers in anatomy education. In this study, the students thought that anatomical structures were sufficiently represented in three-dimension in plastinated cadavers. Removal of surface structures (especially muscles) on the plastinated cadaver that was used in this study in a topographical discipline led to the better observation of deeper structures such as nerves and vessels. This situation may have been reflected in the satisfaction levels of the students in their feedback.

The students educated using plastinated cadavers in this study were in groups of 10–15 people. As the number of plastinated materials was not sufficient, the time spent with plastinated cadavers in each group was limited to 15–20 minutes. This situation reflected negatively on the feedback of the students. 60.3% of the students stated that they could not touch the plastinated cadaver and 40.3% said the duration of the education provided with the plastinated cadaver was not sufficient. Making the number of plastinated cadavers sufficient for the number of students will increase the effectiveness of plastinated cadavers in anatomy education and reflect in student feedback more positively.

Conclusion

Anatomy education provided to the students using plastinated cadavers contributed positively to the anatomy knowledge levels of the students. However, the knowledge levels of students on plastination were not sufficient. We believe that the data of this study will be useful to studies that will assess the effectiveness of plastinated cadaver usage in anatomy education.

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Case Report

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Unusual enlargement of genial tubercle on cone beam computed tomography (CBCT): case report

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Abstract

Enlargement of the genial tubercles, a group of four tubercles located at the inner part of symphysis menti, is very rare especially in dentulous patients. These may lead to problems in speaking, chewing, swallowing and further rehabilitation of the total prosthesis. A 39-year-old female patient was referred to the clinic for the assessment of general dental examination. The patient had difficulty in speaking and swallowing. When the floor of the oral cavity was examined, a bony expansion was detected on the lingual surface of the mandibular symphysis region. Panoramic radiography and cone beam computed tomography (CBCT) were performed. When images were evaluated, enlarged genial tubercle was observed only in CBCT but not in panoramic radiography. The patient was operated under general anesthesia and the enlarged genial tubercle was completely removed. In the post-operative period, speech and swallowing difficulties ceased to exist. To our knowledge, there is no other case reported with such long genial tubercles (22 mm) especially in a dentulous patient. We conclude CBCT is very useful in the diagnosis of enlarged genial tubercle exostosis and planning of the surgical procedure.

Keywords: cone beam computed tomography, enlargement, genial tubercle, mandible

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Introduction

The genial tubercles consist of a group of four small bony elevations located located at the inner part of symphysis menti, midway between the superior and inferior borders of the mandible, and surrounding the lingual foramen bilaterally.[1] The geniohyoid and genioglossus muscles are attached to the genial tubercles. The disfunction of these muscles is correlated with difficulty in chewing, speech and swallowing.[2] Hence, the presence of an enlarged genial tubercle may lead to problems in normal speech, chewing and swallowing. In addition, it may lead to some problems in further rehabilitation of total prosthesis. In some cases, the enlarged genial tubercles may also cause recurrent ulcerations of mucosa or inhibit the use of prosthesis.[3]

While two-dimensional (2D) in intraoral and panoramic radiographs are used essentially for oral hard tissues in dentomaxillofacial radiology, these can be visualized more accurately using cone beam volumetric tomography (CBCT) in three-dimension (3D).[4] 3D imaging of normal and abnormal anatomical structures has important potential in diagnosis and planning of treatment.[5]

Enlargement of genial tubercles is very rare, especially in dentulous patients. This case report presents a genial tubercle enlargement in a dentate patient identified incidentally during a general dental examination, and highlights the importance of the benefit of CBCT in the diagnosis of enlarged genial tubercles and planning of the surgical procedure.

This case report was a poster presentation at the 2nd International Congress and 7th Scientific Meeting of Oral Diagnosis and Maxillofacial Radiology Society, April 13–15, 2017, Eskişehir, Turkey.

Case Report

A 39-year-old dentulous female patient was referred to the Necmettin Erbakan University School of Dentistry Dental Clinic for general dental examination. The patient had difficulty with speech and swallowing. When the floor of the oral cavity was examined, a bony expansion was detected on the lingual surface of the mandibular symphysis region. In the panoramic radiograph, we did not observe any pathology in the anterior region (**Figure 1**). A CBCT scan was done for further investigation. When the images were evaluated in axial, sagittal and coronal planes, an overgrowth the genial tubercle was observed (**Figures 2a**-**c**).

The CBCT scan revealed an enlarged genial tubercle 12.8 mm wide and 14.59 mm high, extending 22.82 mm beyond the mandibular ridge. The morphometry of enlarged genial tubercles before and after the surgery is shown in **Figures 3a**-**c**. 3D reconstruction allowed volumetric visualization of the genial tubercle region before and after surgery (**Figures 4a** and **b**).

The patient was well informed about the condition, its implications and treatment. The informed consent was obtained and surgical removal of the genial tubercle was planned under general anaesthesia and aseptic conditions. The enlarged genial tubercle was completely removed (**Figures 5a**-**c**). Histopathological examination revealed only bony particles. The patient was recalled for follow-up at regular intervals. In the post-operative period, the patient's speech and swallowing difficulties ceased to exist.

Written informed consent for the case to be published (including images, case history and data) was obtained from the patient for publication of this case report.

Figure 1. Digital panoramic radiography of the enlarged genial tubercles before surgery.

Discussion

The present case, using CBCT, demonstrated morphometric analysis of the enlarged genial tubercles in a dentulous patient. Genial tubercles are normally quite small with an average height 5.82 mm and average width 6.98 mm.[6] In addition to problems in normal speech, mastication, swallowing and inhibition of the use of the prosthesis,[7] there are cases with spontaneous fracture and displacement of the enlarged genial tubercles in the literature.[1,2,8] Surgical removal of enlarged genial tubercles is important in the repositioning of the genioglossus and geniohyoid muscles, so that the mandibula can adequately perform functions such as mastication, protrusion and normal speech.

The etiology of the enlarged genial tubercles is usually associated with edentulous mandibular bone loss, as a remodeling process following compressive forces and heavy loading on the alveolar bone due to imbalance of the denture. Mandibular alveolar bone generally resorbs four times higher than the maxillary alveolar bone. The masticatory force may lead to spontaneous fracture of the enlarged genial tubercles.[9] Our patient was dentu-

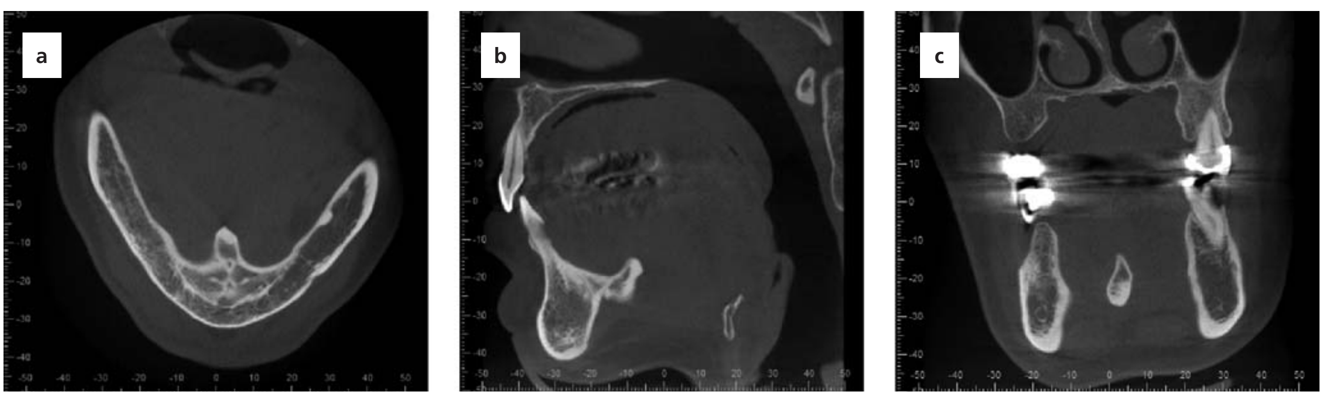

Figure 2. (**a**-**c**) Axial, sagittal and coronal CBCT images of the enlarged genial tubercles.

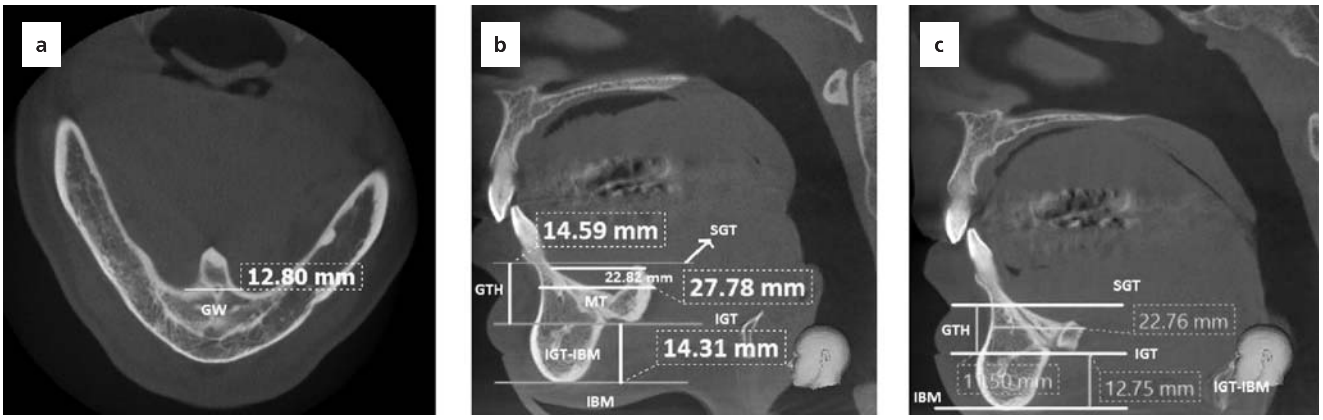

Figure 3. (**a**) Width of the enlarged of genial tubercles before the surgery. (**b**) Morphometric analysis of enlarged genial tubercles before the surgery. (**c**) Morphometric analysis of enlarged genial tubercles after the surgery. **IBM:** inferior border of the mandible; **IGT:** inferior border of genial tubercle; **IGT-IBM:** distance from IGT to IBM; **GW:** genial tubercle width; **MT:** anterior mandibular thickness; **SGT:** superior border of genial tubercle; **SGT-IGT:** distance from SGT to IGT.

lous with no trauma history and no previous radiographic documentation.

In the panoramic radiograph, the enlarged genial tubercles were not noticed due to superposition of other anatomical structures. CBCT images were used to observe and perform morphometric and volumetric analysis of the enlarged genial tubercles using 3D reconstruction. Therefore, we were able to exactly determine and measure the enlarged genial tubercles in this case.

An abnormal enlargement of the genial tubercles was first reported in 1955 in a mandible specimen of a Maori man, with a depth of 7 mm, width of 13 mm and height of 9 mm.[10] Yaed'u et al.[7] and Gallego et al.[11] reported cases with spontaneous fracture of the genial tubercles, diagnoses made using occlusal radiography and CT images, respectively. Yin et al.[6] concluded that spiral CT measurements of the genial tubercles reflect their real anatomy which is important for the planning of a surgical procedure in the region.

The panoramic radiograph could not resolve the diagnosis itself due to the superimposition of structures. The authors highlighted the importance of occlusal radiography and CT scan for the evaluation of the genial tubercles. Rubira-Bullen et al.[8] reported a genial tubercle 18 mm in length in an edentulous patient. Jindal et al.[3] in 2015 reported that genial tubercles 11 mm wide and 21 mm long in an edentulous patient. They stated that the surgical removal of the genial tubercles is important in repositioning of the genioglossus and geniohyoid muscles.

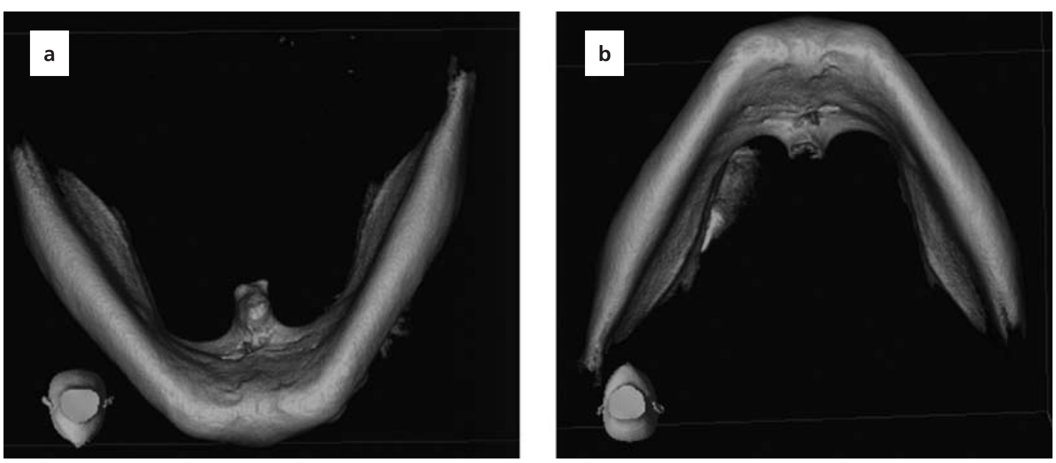

Figure 4. (**a**, **b**). 3D reconstruction images of the enlarged genial tubercles before and after surgery. [Color figure can be viewed in the online issue, which is available at www.anatomy.org.tr]

Figure 5. (**a**) Intraoral photograph during surgical operation of enlarged genial tubercles. (**b**) Post-operative photograph. (**c**) Extracted bone material. [Color figure can be viewed in the online issue, which is available at www.anatomy.org.tr]

In this case, we were able to perform an accurate measurement of the genial tubercles using CBCT images, as well as determining the exact position of the enlarged anatomic structure. 3D reconstructions in different views were also useful for the volumetric evaluation and spatial visualization of the mandible, as in the case reported by Rubira-Bullen et al.[8] To our knowledge, there is no case reported in the literature with such long genial tubercles (22 mm) especially in a dentulous patient.

Conclusion

Enlarged genial tubercles are very rare in dentulous patients. These may lead to various problems in speech and swallowing, and also in prosthetic rehabilitation leading to ulceration and bleeding. 3D imaging using CBCT is useful in diagnosis and planning the surgical procedure in cases with enlarged genial tubercles.

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Persistent carotid-vertebrobasilar anastomoses: cases of proatlantal artery Type I and Type II

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Abstract

Persistence of fetal communications between the carotid and vertebrobasilar systems is uncommon. Persistent proatlantal artery is an anastomosis between the carotid and vertebrobasilar systems, typically classified as Type I and II. In this case report, 600 angiographies are examined retrospectively and two persistent proatlantal arteries were observed - one with Type I (0.16%) and the other with Type II (0.16%) proatlantal artery. Existence of these arteries are associated with intracranial vascular anomalies, especially aneurysms. In both of our cases, an aneurysm was detected in the middle cerebral artery. Precise knowledge of these anastomoses is essential for intracranial operations and catheterizations performed in this region.

Keywords: angiography; carotid-vertebrobasilar anastomosis; persistent proatlantal artery

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Introduction

Persistent carotid-vertebrobasilar anastomoses occur as a result of discontinuation in the development of the vertebrobasilar system.[1] These are embryonic presegmental arteries supplying the primitive vertebrobasilar system from the primitive internal carotid artery (ICA).[1-4] The primitive vertebrobasilar system comprises two parallel longitudinal neural arteries supplied by the carotid system with four major anastomoses.[5] In the early stage of embryonic life, these longitudinal neural arteries supply the hindbrain. Blood flows here by the embryonic cerebral arteries that directly connect the carotid and basilar arteries. These arteries form anastomoses named presegmental arteries and are named by the adjacent cranial nerves:[1] trigeminal, otic, hypoglossal, and proatlantal. Presegmental arteries provide connection between the longitudinal neural arteries and the internal carotid artery, and disappear after the development of the posterior communicating and vertebral arteries. The one that persists most frequently is the primitive trigeminal artery with an incidence of 0.2% in cerebral angiograms. The incidence of other presegmental arteries were found as 0.1% after birth.[6] Persistant proatlantal artery (PPA) is classified Type I and Type II. Both types are rare developmental anomalies; both originate from the carotid artery and enter the cranium through the foramen magnum.

Type I originates from the ICA, takes a dorsal course cephalad to the transverse process of C1, and then travels rostral to enter the foramen magnum. Type II proatlantal artery arises from the external carotid artery (ECA) laterally, remains more lateral in position than the Type I artery, and joins the course of the horizontal portion of the vertebral artery (VA) before entering the foramen magnum[1,7] (**Figure 1**). Type I does not pass through the transverse foraminae of the cervical vertebrae; in contrast, Type II passes through the transverse foramen of C1 vertebra and then joins with the V3 of the VA.[1] We studied cerebral angiographies performed between 2011-2015 at The Department of Radiology, School of Medicine, Akdeniz University, Antalya, Turkey to reveal subarachnoidal haemorrhagies retrospectively and investigate carotid-vertebrobasilar anastomoses. Out of 600 angiographies examined retrospectively, only two cases with persistent proatlantal artery were observed.

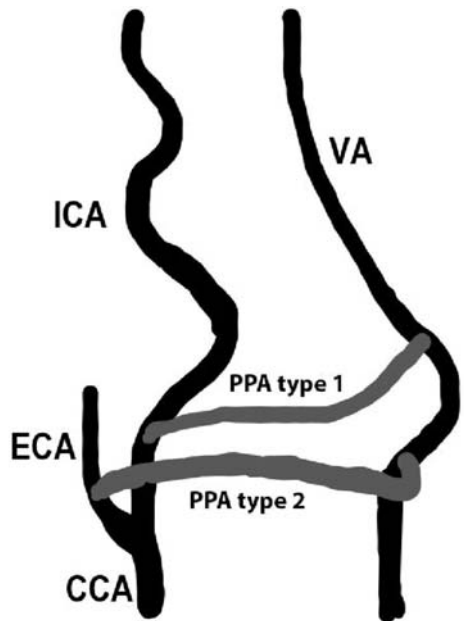


Figure 1. Illustration of proatlantal arteries; Type I and Type II. **CCA:** common carotid artery; **ECA:** external carotid artery; **ICA:** internal carotid artery; **PPA:** persistant proatlantal artery; **VA:** vertebral artery.

Case Report

Case 1

In the first case, angiography of the right common carotid artery (CCA) indicated that the CCA continued with the middle cerebral artery (MCA). At the bifurcation level of the MCA a saccular aneurysm was detected (**Figure 2a**). Angiography of the left CCA showed that Type I proatlantal artery originated from the cervical part of the ICA. The left VA arose through the Type I proatlantal artery and ICA continued with the left anterior cerebral artery (ACA) and MCA, and the right ACA was visualized via the anterior communicating artery (**Figure 2b**). The left VA was filled by the proatlantal artery and continued with the basilar artery. Two posterior cerebral arteries were also visualized (**Figures 2c** and **d**). In the angiography of the left subclavian artery, proximal part of VA was not visualized (**Figure 2e**).

Case 2

In this case, angiography of the right CCA showed that the right MCA and ACA were arising from ICA and left

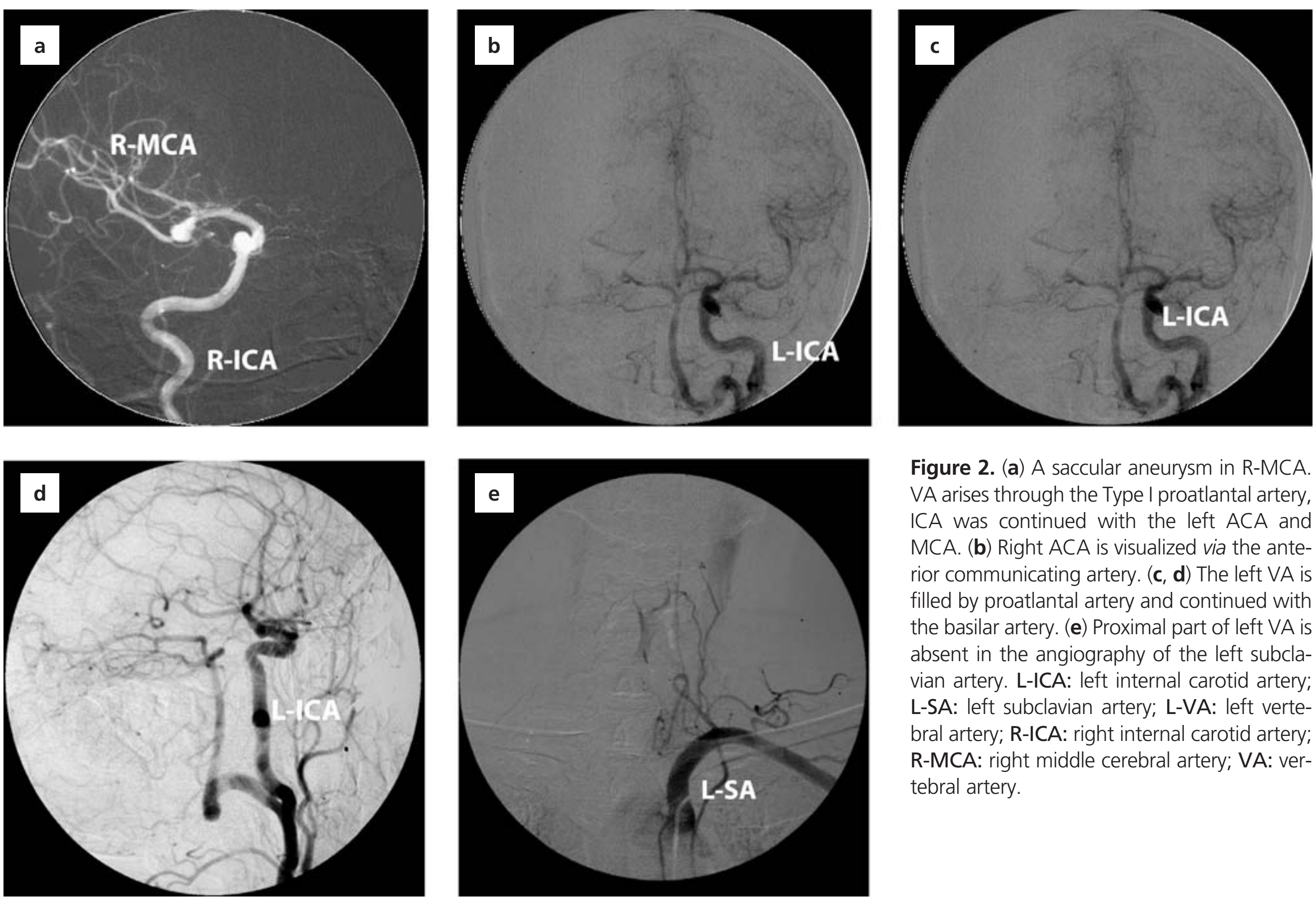


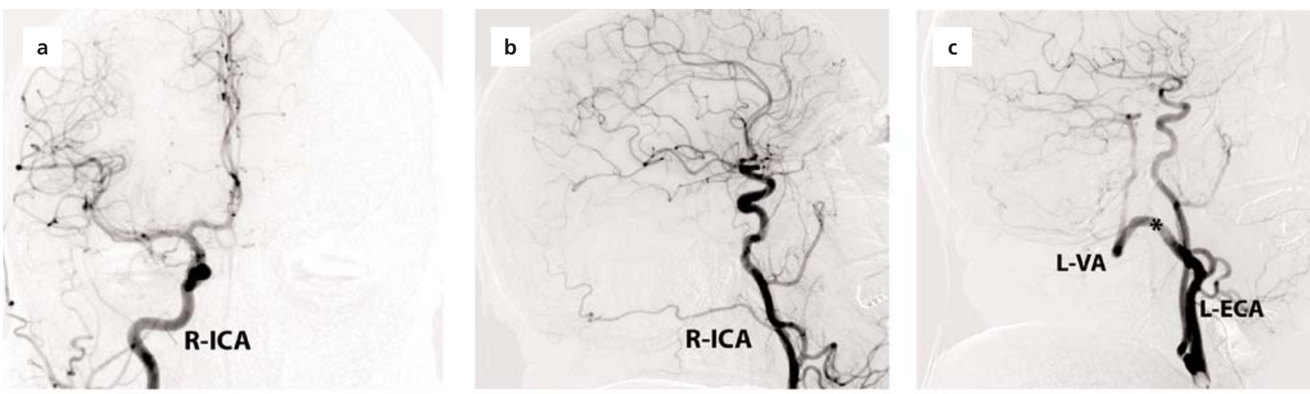
Figure 2. (**a**) A saccular aneurysm in R-MCA. VA arises through the Type I proatlantal artery, ICA was continued with the left ACA and MCA. (**b**) Right ACA is visualized *via* the anterior communicating artery. (**c**, **d**) The left VA is filled by proatlantal artery and continued with the basilar artery. (**e**) Proximal part of left VA is absent in the angiography of the left subclavian artery. **L-ICA:** left internal carotid artery; **L-SA:** left subclavian artery; **L-VA:** left vertebral artery; **R-ICA:** right internal carotid artery; **R-MCA:** right middle cerebral artery; **VA:** vertebral artery.

ACA was visualized via the anterior communicating artery (**Figures 3a** and **b**). Angiography of the left CCA showed that the ICA is continued isolated MCA. From the ECA, a thick Type II persistent proatlantal artery arose and VA was filled through this artery (**Figure 3c** and **d**). Right VA originated from the right subclavian artery as usual, but on the left side, proximal part of VA wasn't visualized (**Figure 3e**).

Discussion

Persistence of fetal communications between the carotid and vertebrobasilar systems is uncommon. We found the PPA artery incidence as 0.33% in our study. Bilateral or unilateral presence of PPA Type I was described in some earlier studies.[7–11] We found the incidence of Type I PPA as 0.16% in this study. Bilateral or unilateral presence of PPA Type II was also described previously.[4,12,13] We found Type II PPA in 0.16% of the angiographies. Woodcock et al.[10] found proatlantal artery Type I in 57%, Type II in 38%, and arising from the common carotid artery in 5% . As in our case, most of the proatlantal arteries were found incidentally. Purkayastha et al.[9] suggested that the actual incidence of PPA is probably higher than reported, because in most cases, the discovery is purely coincidental. Existence of these arteries are associated with intracranial vascular anomalies, especially aneurysms.[10,14,15] Yılmaz et al.[11] reported clinical and pathological findings in combination with these primitive persistent anastomoses in seven cases. Tubbs et al.[14] mentioned that, for the co-existence of PPA and aneurysms is reported in the literature, no consensus has been reached as to whether this is an association or simply incidental. In both of our cases, an aneurysm was detected in the MCA. This finding promotes the idea that PPA might be associated with vascular anomalies; however, more extensive studies are needed to clarify this.

Persistence of the proatlantal artery into adult life can be explained on an embryological basis as of a primary error in the development of the VA.[12] Therefore, PPA can be accompanied by ipsilateral, contralateral or bilateral aberrant VAs.[7] Coincidence of PPA and hypoplasia of the ipsilateral, contralateral or both VA was reported as 46%.[16] In both of our cases, proximal parts of the ipsilateral VA was absent. Bahşi et al.[17] conferred that when the VA is absent, posterior cerebral circulation is supplied by persistent arteries, and occlusion of these arter-



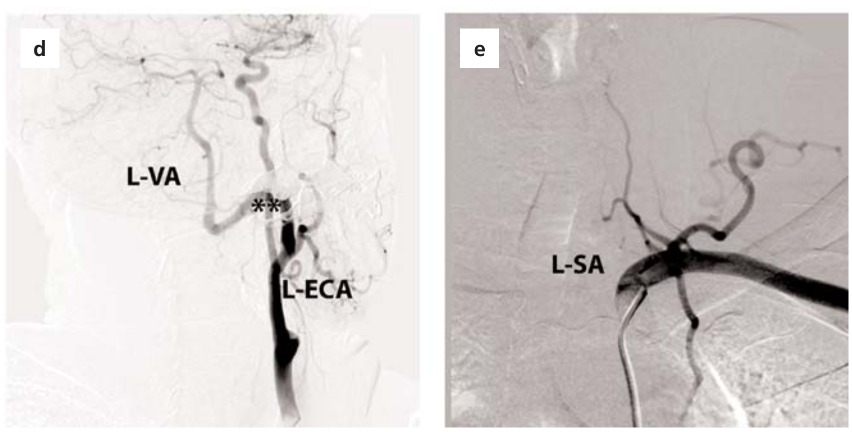


Figure 3. (**a**-**b**) Right MCA and ACA arising from the ICA and left ACA is visualized via the anterior communicating artery; (**c**) Type II proatlantal artery; (**d**) Type II proatlantal artery; (**e**) Proximal part of left VA is absent in the angiography of the left subclavian artery. *Type II proatlantal artery; **Type II proatlantal artery; **L-ECA:** left external artery; **R-ICA:** right internal carotid artery; **L-SA:** left subclavian artery; **L-VA:** left vertebral artery; **VA:** vertebral artery.

ies results in ischemia of the area.[17] Therefore, the knowledge of these persistent arteries before surgery can be crucial while designing the surgical procedure.[9,16,17]

The coincidence of PPA and aneurysms is mentioned in many studies including our study.[7,10,14,15] Besides this, some authors suspected that PPA might be associated with other symptoms or clinical findings such as tinnitus, ischemic cerebrovascular diseases, arteriovenous malformations.[12,13,18] Kolbinger et al.[18] described a case with right cerebellar infarction and ischemic lesions in the left dorsal thalamus and the right upper parietal lobe. In this case, the angiography showed occlusion of the right ICA proximal to an ipsilateral proatlantal artery Type I. They suggested their case demonstrated the clinical significance of a persistent proatlantal artery in the evolution of an atypical ischemic cerebrovascular disease.

Conclusion

The literature on the PPA and other persistent primitive arteries are mostly case reports and/or literature reviews. Thus, incidence of this artery is undecided. Although coincidence of PPA and vascular anomalies is mentioned in numerous articles, there is no consensus about this issue. Also, association with PPA and other diseases or symptoms is still not clear. When the clinical importance of this artery is considered, extended and retrospective studies like this are necessary.

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Case Report

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Effect of decompressive surgery on spine balance in congenital lumbar kyphoscoliosis: anatomical aspect with a case report

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Abstract

The lumbar vertebral column is a complex structure formed by the fusion of intervertebral joints. It protects the spinal cord by wrapping around it, carries most of the body's weight and creates a flexible structure for the body. As individuals with congenital deformities of the lumbar vertebral column age, they develop some changes in the anatomy of the spine and with some consequences. In this case, a 35-year-old male patient had a deteriorated posture together with back pain since childhood. He had walking difficulty and numbness in two legs during walking for the last three years. He fell due to a sudden brake while he was travelling afoot by bus in 2015. He was admitted to the emergency service with loss of strength in two legs. Lumbar CT and MRI showed a severe narrowing of the spinal canal due to lumbar kyphoscoliosis. The patient had neurological deficits after hyperflexion trauma. The patient underwent emergency decompressive surgery due to marked paraparesis and narrow spinal canal. At the end of the first post-operative month, his clinical complaints were almost completely resolved, and his biomechanical balance did not show radiological deterioration. While the anatomical change that occurred during the natural course of congenital kyphoscoliosis increased the stability of the lumbar spine, it severely narrowed the spinal canal and affected the spinal nerves and thereby caused severe neurological deficits.

Keywords: balance, congenital deformity, kyphoscoliosis, lumbar, narrow spinal canal

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Introduction

The lumbar vertebral column is a complex structure formed by the fusion of five vertebrae that create a robust and flexible anatomical structure for the body.[1] The lumbar functional unit is mainly divided into two parts. The anterior part consists of two vertebral corpuses and one intervertebral disc between them that bears weight and absorbs compressive loading. The posterior part consists of the facet joints that do not bear weight and provide ease of movement. The movement of the spine is greatly reduced in parallel to the increase of top-down static loading.[1,2] The lower lumbar levels need to be more stable. Therefore, connective tissue support plays an important role in the control of spinal movements. Naturally, the lower part of the vertebral column is firmly supported by abundant connective tissues to create a robust base.[3] In cases with congenital deformities, this anatomy is redesigned to the extent allowed by nature.[2–5]

The development of the spine in the embryo occurs early during the first six weeks of intrauterine life, as the complete anatomical pattern of the vertebrae is formed in mesenchyme.[5,6] The basic organization of the vertebral column is well established at the beginning of the fetal period in day 57. Vertebral body hypoplasia and aplasia represent a wide spectrum of centrum growth deficits that occur during the last seven months of pregnancy when the rapid increase in vertebral size occurs.[5,6] During this period, centrum development is independent of neural arch growth. The complete absence of the vertebral body, centrum aplasia, is the most severe form of anterior deficits.

Posterior hemicentrum and centrum aplasia produces a purely kyphotic deformity. Furthermore, lateral hemicentrum aplasia leads to scoliotic deformities.[5,6]

Congenital lumbar kyphoscoliotic deformity is a very rare spinal problem that biomechanically changes particularly the sagittal balance of the spine by causing severe deterioration of the spinal alignment. During development, the spinal column undergoes changes to protect and maintain its biomechanical balance. The deformed spinal column tries to maintain its balance at two plans. The spinal canal gradually narrows due to excessive kyphotic angulation of the spine and hypertrophy in supportive connective tissue.[4,5] The aim of this case report to present the anatomical and biomechanical changes following surgical treatment in a case of congenital kyphoscoliosis, a pathology we rarely encounter.

Case Report

A 35-year-old male patient had a deteriorated posture together with back pain since childhood. He had walking difficulty and numbness in two legs during walking for the last three years. He fell down due to a sudden brake while travelling afoot by bus in Rize province in 2015. He was admitted to the emergency service with loss of strength in two legs. Lumbar computed tomography (CT) and magnetic resonance imaging (MRI) showed severe narrowing of the spinal canal due to lumbar kyphoscoliosis (**Figure 1**). It was found that the neural tissue was crushed after

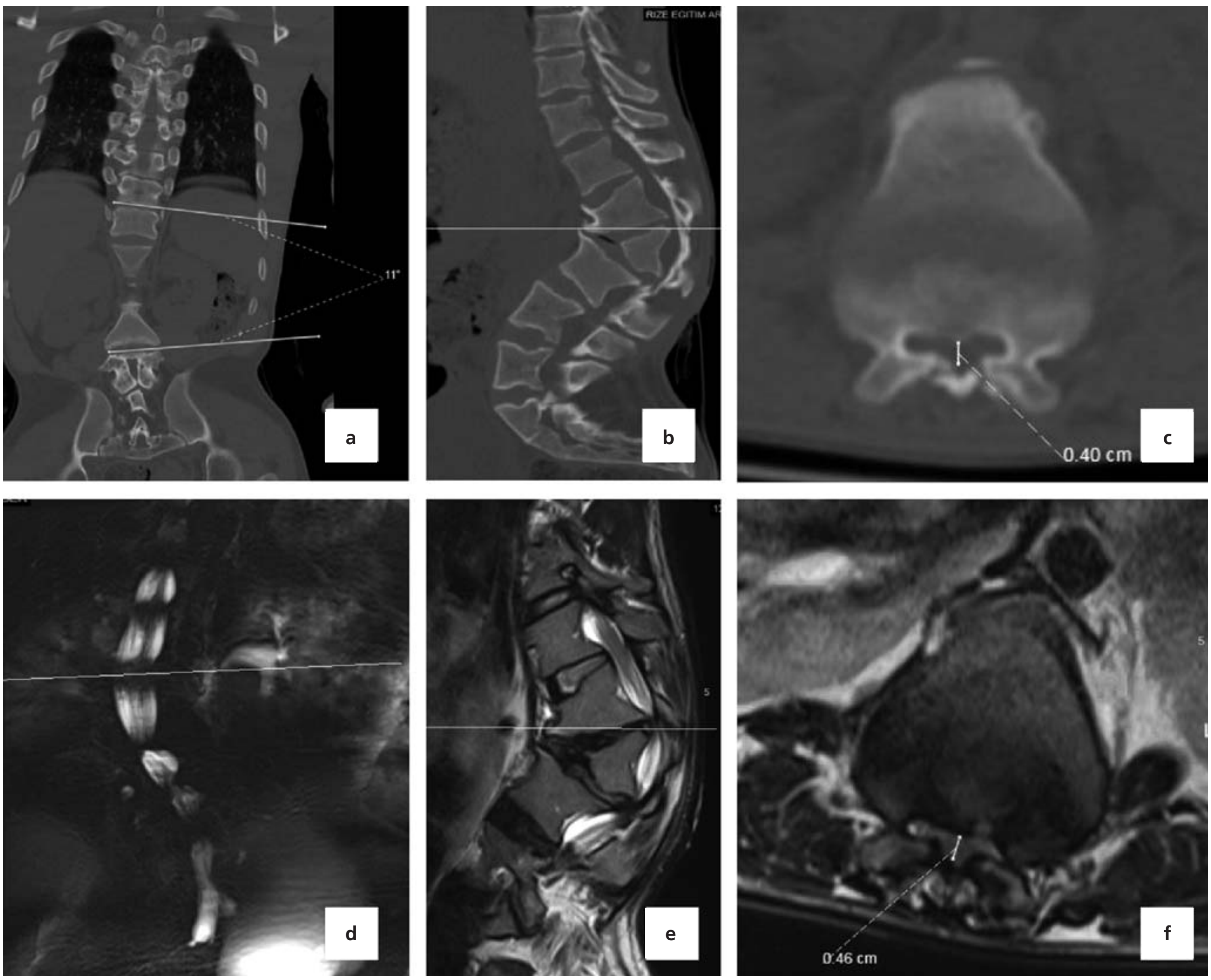

Figure 1. (**a**-**d**) Scoliosis is observed in myelo MR and CT section. (**b**-**e**) Anterior wedged vertebra on level L2 of the lumbar vertebrae is observed in sagittal CT and MR sections in the 35-year-old male patient (Type 1 deformity). (**f**) The diameter of the spinal canal decreased to 4 mm at the kyphosis segment in axial CT and MR sections.

hyperflexion trauma. The patient underwent emergency decompressive surgery due to marked paraparesis and narrow spinal canal. The posterior wall of the vertebral canal was opened with total laminectomy in level of lumbar kyphotic deformity, preserving the posterior facet joints (**Figure 2**). Neurological deficits of the patient gradually improved after surgery. Angular changes in coronal and sagittal planes were calculated using pre-operative and post-operative (1st month and 1st year) radiological images (**Table 1**). The posture of the patient became more erect when he was standing. At the end of the one-year follow-up, he had no back pain and posture deterioration. His anterior column was very strong and sagittal and coronal planes were stabilized.

Table 1
The changes before and after surgery in angles calculated by the Dicom Software related to lumbar spinal stability.

	Pre-operative	Post-operative 1st month	Post-operative 1st year
Local kyphosis angle	56°	56°	56°
Sagittal Cobb angle	65°	65°	64°
Coronal Cobb angle	11°	5°	6°
Lumbar lordosis angle	30°	26°	24°

Discussion

Congenital kyphosis and kyphoscoliosis are uncommon deformities in which there is an abnormal posterior convex angulation of a segment of the spine due to developmental vertebral anomalies.[2,4,5] These congenital anomalies are present at birth, but may not be clinically apparent except for slight changes in general posture until childhood. The development of kyphosis due to vertebral anomalies is fully established at birth and thought to develop during the later stages of chondrification and ossification.[6] Congenital kyphosis and kyphoscoliosis are less common than pure congenital scoliosis. However, these problems can lead to more serious clinical consequences such as paraplegia and spinal cord compression.[5,7]

Congenital kyphosis was first described by von Rokitansky in 1844.[5] Van Schrick (1932) differentiated a failure of vertebral-body formation from a failure of vertebral-body segmentation as a cause of congenital kyphosis.[5,8] We have our current knowledge of congenital kyphosis mainly from Winter et al. in 1973 who followed 130 patients for more than one year without treatment, and were able to make further assessments.[5] They reported that spinal deformities usually progress rapidly during

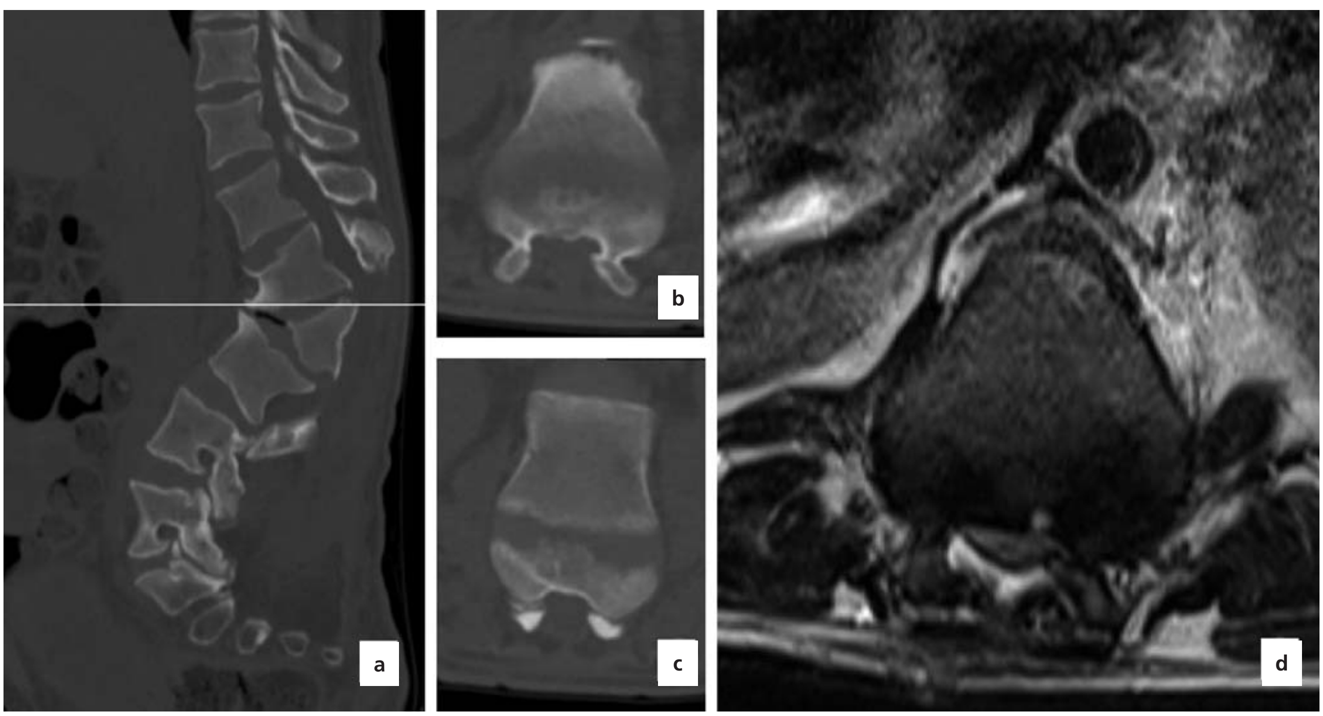

Figure 2. There are post operative changes in the spine. (**a-c**) Changes in sagittal and axial CT sections were observed after decompressive surgery. (**d**) Spinal canal enlargement at kyphosis level on axial MR section.

periods of rapid growth seen in adolescence and thereby clinical manifestations occur after childhood.[5]

Congenital kyphoscoliotic spinal deformities are classified into four types according to the shape of the vertebral column anatomy. In Type 1 deformities, there is deterioration in the anterior segment of the vertebral body. These patients are also divided into four subgroups within themselves: posterolateral quadrant vertebra, posterior hemivertebra, butterfly (sagittal cleft) vertebra, and anterior or anterolateral wedged vertebra. Posterolateral quadrant vertebra and butterfly (sagittal cleft) vertebrae affect the posture of the vertebral column in the coronal plane and cause more severe scoliotic deformities.[5] The other two problems cause more severe kyphotic deformities, because they affect the posture of the vertebral column mostly in the sagittal plane. In Type 2 deformities, vertebral segmentation is impaired, and intervertebral discs are absent or unclear. There is a long, unsegmented bony bar in the anterior or anterolateral side. The bony bar can cause a kyphotic deformity if it is in the anterior side.[5,6] Type 3 deformities are mixed-type anomalies. They consist of a combination of segmentation and vertebral body anomalies. Very severe deformities that do not fit all these classifications are typified as Type 4 (unclassified deformities). Our case had anterior wedging of the L2 vertebral body, Type 1 deformity causing kyphotic angle of 56 degrees, and mild scoliosis with a Cobb's angle of 11 degrees (**Figure 3**).

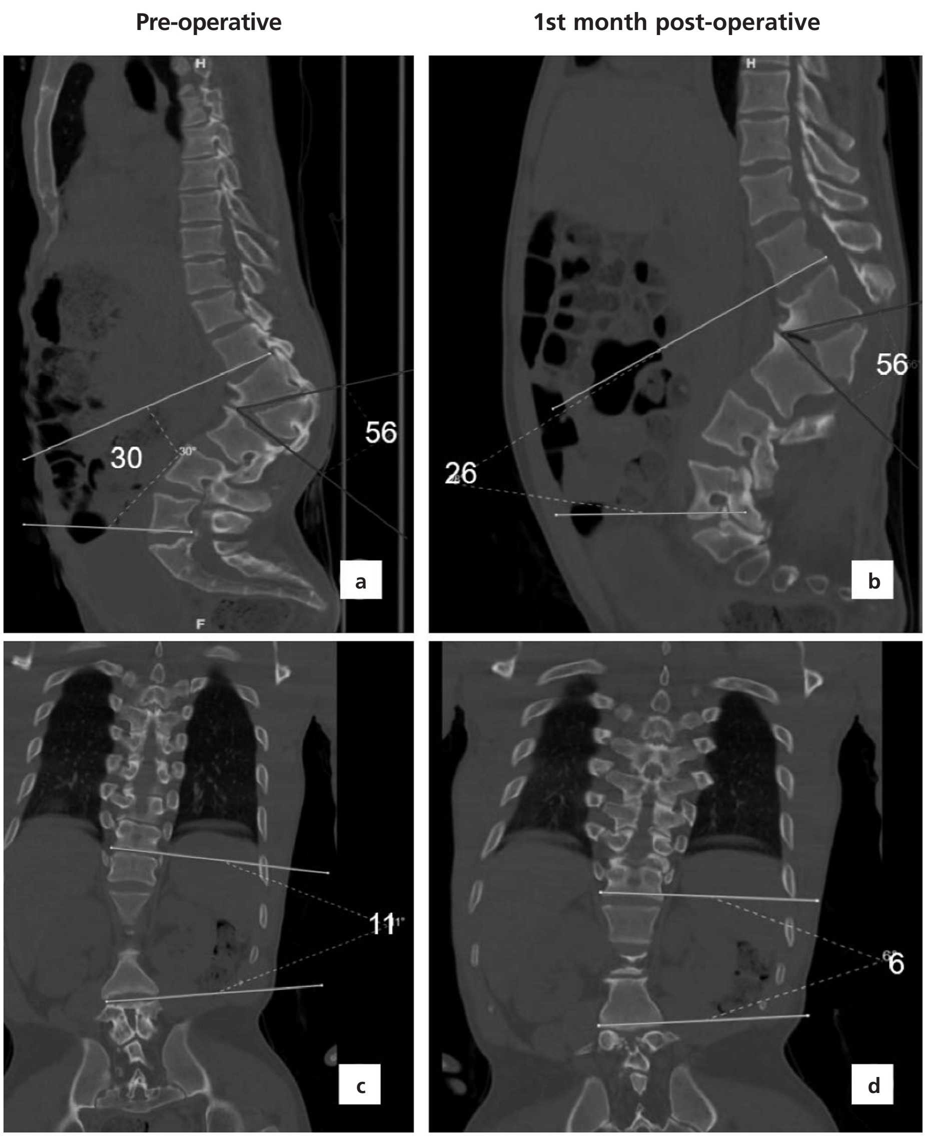

Figure 3. (**a-c**) The measured angles in the pre-operative sagittal and coronal planes. (**b-d**) Angles in the same planes after 1 month.

The occurrence of physiological kyphotic angulation of the thoracic vertebral segment in the lumbar vertebral segment is considered completely pathological.[9] The deterioration of physiological posture of the spine leads to early degeneration of the spine and other structures that provide stabilization. There is a risk of developing kyphosis following extensive laminectomies involving the facet joints during surgery in any segment of the spine. In our case, there was no increase in the kyphosis angle after surgical decompression; there was only a slight decrease in the lumbar lordosis angle (reducing from 26 degrees to 24 degrees) after one year of follow-up. In anaother study, the lumbar lordosis angle was found to be consistent with the mean angle (20.4±10.2°) measured in cases of degenerative lumbar kyphosis.[7] No additional neurological deficits were observed at the end of follow-up. There was also no evidence of back pain suggesting lumbar instability. According to the Roussouly's classification, the lumbar lordosis angle is reported to vary between 52 and 71 degrees.[10] It was observed that the lumbar lordosis angle was much smaller due to the angular effect of kyphotic deformity on the opposite direction.

Cobb's angle is often used to determine the angle of scoliosis in the coronal plane, as well as to grade sagittal plane spinal deformities. It is defined as the angle formed between a line drawn parallel to the superior endplate of one vertebra above the curve and a line drawn parallel to the inferior endplate of the vertebra one level below the curve. Cobb angle is the most important determinant of the severity of kyphoscoliosis. It was reported that Cobb angle above 60° may result in progressive pulmonary and cardiac failure.[5] In our case, the sagittal plane kyphosis angle in the lumbar segment was measured as 65°, and therefore can be considered as severe kyphosis.[5,8,10]

Anterior compression of the spinal cord at the apex of the congenital kyphosis or kyphoscoliosis occurring spontaneously in the patients, neurologically normal previously, leads to progressive spastic paraparesis of the lower limbs.[5] In this case, paraparesis was also observed with a minor trauma, because the spinal canal diameter was narrowed to 4–5 mm. Severe congenital rigid kyphoscoliosis in adults remains challenging for spine surgeons. Kyphoscoliosis is a deformity with fixed spinal vertebrae that does not allow traction, suspension, or side bending of the spine.[11] Surgery is currently the most common treatment, despite being difficult and controversial.[11,12] Vertebral column resection is a technique for correcting rigid severe kyphoscoliosis; posterior vertebral column resection is an effective alternative for moderate to severe deformities with limited flexibility. This is a technically demanding and exhausting procedure with possible major complications.[12] Spinal wedge osteotomy by single posterior approach is a reliable and safe surgical technique for correcting severe rigid angular kyphosis or kyphoscoliosis.[13] Therefore, decompressive minimally invasive surgical methods may be preferred in the presence of neurological deficits. In addition, in the spine anatomy of this patient, spinal biomechanics did not deteriorate after simple surgical decompression.

Conclusion

This case report presents positive results for anatomical and biomechanical changes following surgical treatment of congenital kyphoscoliosis. Long-term radiological follow-up in this patient as well as comparison of the results of surgical treatment in more patients is essential to support these data.

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(**a**) A saccular aneurysm in R-MCA. VA arises through the Type I proatlantal artery, ICA was continued with the left ACA and MCA. From Özgür Ö, Aytaç G, Sindel M, Sindel T. Persistent carotid-vertebrobasilar anastomoses: cases of proatlantal artery Type I and Type II. *Anatomy* 2018;12(2):101–104.

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