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RESEARCH ARTICLE

Diaphragmatic crus indentation to the renal artery: Is it a new etiology for renovascular hypertension in adults?

Dilek Akkurt Acar[1], Atilla Hikmet Çilengir[2], Mehtap Balaban[3], Eren Çamur[1], Betül Akdal Dölek[1], Nilgün Işıksalan Özbülbül[4]

[1]Department of Radiology, Ankara Bilkent City Hospital, Ankara, Türkiye
[2]Department of Radiology, Faculty of Medicine, İzmir Democracy University, İzmir, Türkiye
[3]Department of Radiology, Faculty of Medicine, Ankara Yıldırım Beyazıt University, Ankara, Türkiye
[4]Department of Radiology, University of Health Sciences, Ankara City Hospital, Ankara, Türkiye

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ABSTRACT

Aim: To investigate the relationship between hypertension and the indentation and compression of the diaphragmatic crus in the renal artery.

Material and Methods: Abdominal computed tomography scans of 304 consecutive adult patients performed for any reason were retrospectively analyzed. Patients with crus indentation or compression on the renal artery were identified. Diaphragmatic crus contact was defined as compression if it caused stenosis more than 50% of the renal artery diameter, and indentation if it caused stenosis less than 50%. If the renal artery originated above the level of the L1-2 intervertebral disc, it was considered as a high origin.

Results: The mean age of women was 51±15.29 and the mean age of men was 52±15.38. Hypertension was present in 29.6% (n=74) of the patients. Diaphragmatic crus indentation (DCI) was detected in 8.4% (n=21) of all patients, and 76.2% (n=16) of these were men. Diaphragmatic crus compression (DCC) (n=3) was detected in 1.2% of all patients, and 67% (n=2) of these were women. Hypertension was present in 67% (n=2) of patients with DCC, all of them were women, and the mean age was 65.5 years. Hypertension was present in 38.1% (n=8) of patients with DCI.

Conclusions: DCI and DCC which can be caused by hypertrophic diaphragmatic crus or high origin of the renal artery, should be included in the etiology of renovascular hypertension. In addition to the presence of renal artery stenosis in a patient with hypertension, the relationship between the renal artery and diaphragmatic crus should also be evaluated.

Keywords: diaphragmatic crus, hypertension, renal artery, renovascular

Corresponding author: Atilla Hikmet Çilengir **E-mail:** acilengir@gmail.com
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INTRODUCTION

Diaphragmatic crura are musculotendinous structures that connect the posterior and middle parts of the diaphragm to the lumbar vertebrae. The right crus attaches to the L1-L3 vertebral body, and the left crus attaches to the L1-L2 vertebral body. Since anomalies affecting the diaphragmatic crura are often asymptomatic, they are usually detected incidentally during imaging (1,2).

Direct radiography, fluoroscopy, ultrasonography (US), computed tomography (CT), and magnetic resonance imaging (MRI) are used for imaging the diaphragm. Compared to US, CT is more successful in demonstrating diaphragmatic anatomy and diaphragm-related lung-mediastinum pathologies (3,4). CT is also quite successful in measuring diaphragmatic crus thickness, which is a critical indicator for the evaluation of the pacemaker insertion system in patients with amyotrophic lateral sclerosis, the diagnosis of unilateral diaphragm paralysis, sepsis, and diaphragmatic atrophy due to mechanical ventilator therapy (5-8).

Diaphragmatic crus compression on the renal artery is primarily documented in case reports within the literature, and its incidence remains unclear (9-11). The available studies on renal artery compression have evaluated the origin level of the renal arteries, but have not investigated the incidence of the diaphragmatic crus indentation on the renal artery and accompanying hypertension. We also hypothesized that indentation, unlike compression on the renal artery, may also cause renovascular hypertension. In this analysis, we aimed to investigate the relationship between indentation and compression of the diaphragmatic crus on the renal artery, and hypertension in the adult age group.

MATERIAL AND METHODS

This case-control study was carried out between June – October 2020. The Institutional Review Board approved the survey in terms of ethical suitability (Date: 09.03.2022, Number: E1-22-2407).

Study population

Abdominal CT scans of 304 consecutive adult patients performed for any reason were retrospectively analyzed. Patients with ectopic or solitary kidney (n=10), aortic aneurysm (n=5) or dissection (n=4), severe chronic obstructive pulmonary disease (n=8), and a mass causing invasion/displacement in the diaphragmatic crus (n= 3), rotoscoliosis (n=7), and patients whose images could not be evaluated due to motion artifacts (n=17) were excluded. A total of 250 patients (107 women, 143 men) were included. The clinical and demographic information and CT images of the patients were accessed from the hospital automation system.

Image acquisition

The dynamic protocol was performed on a 512-slice CT scanner (Revolution, GE Healthcare, Waukesha, WI, USA) using the parameters 120 kV, 100 mAs, pitch 0.9, rotation time 0.6 sec, 80 mm. For contrast-enhanced imaging, 90 ml of contrast agent was administered via an automatic injector at a rate of 5 ml/sec from the left antecubital vein while the patient was lying in the supine position. 40 ml of 0.9% saline was administered after contrast agent injection at the same rate. Images were acquired in the arterial, portal, and venous phases (with fixed delay time or SmartPrep).

Measurements

Diaphragmatic crus thickness was measured on the axial slice at the level of the superior mesenteric artery (Figure 1). The diameters of the renal arteries were measured on the arterial phase axial slices at 1 cm distal from their origins with 100% magnification. Patients with crus indentation or compression on the renal artery were identified. Diaphragmatic crus contact was considered as compression if it caused stenosis greater than 50% of the renal artery diameter (Figure 2), and indentation if it caused stenosis less than 50%. If the renal artery originated from a higher than L1-2 intervertebral disc level, it was considered a high origin. Additionally, accompanying renal parenchymal abnormalities were noted. Measurements were made by an abdominal radiologist (D.A.). The presence of hypertension, and whether there was another cause of hypertension, was determined by the hospital's

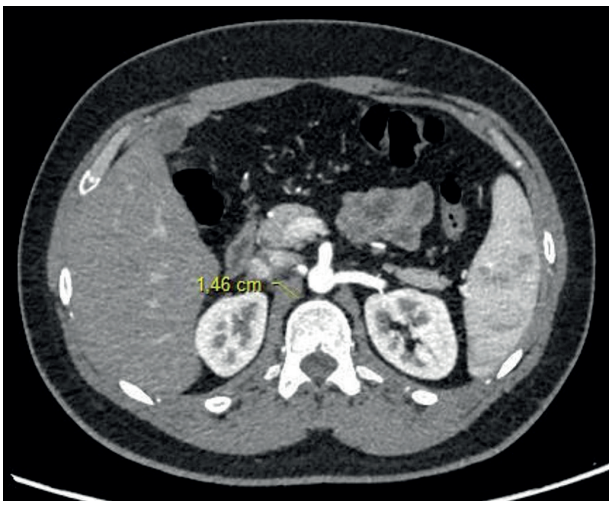

Figure 1. Right diaphragmatic crus thickness measurement on the axial slice at the level of the superior mesenteric artery.

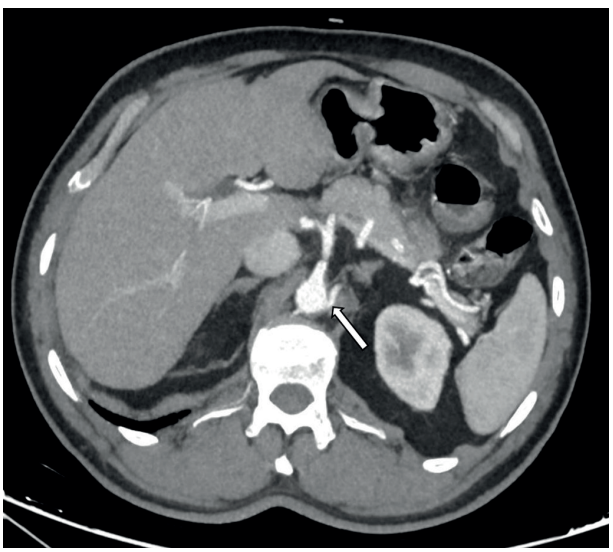

Figure 2. Maximum intensity projection of the axial plane computed tomography image shows the narrowing of the left renal artery orifice (arrow) secondary to the diaphragmatic crus compression.

automated registration system. Blood pressure higher than 140/90 mmHg was considered as hypertension. Estimated glomerular filtration rate (eGFR), serum creatinine level, and accompanying imaging findings were also noted.

Statistical analyses

Data were evaluated using IBM Statistics 22.0 (IBM Corp. Armonk, NY, USA). Frequency tables and descriptive statistics were used to interpret the findings. The relationship between two qualitative variables, and the expected value levels were analyzed by the Fisher-Exact and Pearson-$\chi 2$ test. The Mann-Whitney U test (Z-table value) was used to compare the measurement values of two independent groups. A p-value less than 0.05 was accepted as statistically significant.

RESULTS

The mean age of women was 51±15.29 (range: 18-86 years) and the mean age of men was 52±15.38 (range: 18-84 years). The mean age of patients with diaphragmatic crus compression (DCC) and/or indentation (DCI) was 48.46±15.13 years, while the mean age of those without was 52.04 ±15.33 years (p=0.237). Hypertension was present in 29.6% (n=74) of the patients. There were 45 men (60.8%) and 29 women (39.2%) with hypertension. In contrast, 99 men (55.9%) and 78 women (44.1%) did not have hypertension. The right side was affected in all patients with DCC (n=3).

DCI was on the right side in 81% (n=17), on the left in 9.6% (n=2), and bilateral in 9.6% (n=2). Diaphragmatic crura thicknesses in the group with and without diaphragmatic indentation were summarized in Table 1. Ipsilateral crus was found to be thicker in patients with DCI (p=0.004). DCI was detected in 8.4% (n=21) of all patients, and 76.2% (n=16) of these were men. DCC (n=3) was detected in 1.2% of all patients, and 67% (n=2) of these were women. Hypertension was present in 67% (n=2) of patients with DCC, all of them were women, and the mean age was 65.5 years. Hypertension was present in 38.1% (n=8) of patients with DCI, all of them were men, and the mean age was 46.8 years.

The distribution of the patients with DCC – DCI in the high-origin renal artery and hypertension groups is summarized in Figure 3. In 2 patients with DCC and hypertension serum creatinine level was normal,

Table 1. Bilateral diaphragmatic crus thicknesses (in millimeters) at the superior mesenteric artery level in patients with and without diaphragmatic crus indentation or compression

	Diaphragmatic Crus Thickness (Mean ± Standard Deviation)		
	Patients with Crus Indentation (n=21)	**Patients with Crus Compression (n=3)**	**Patients without Crus Indentation or Compression (n=226)**
Right Crus	8.70 ± 2.05	9.90 ± 3.77	7.72 ± 5.96
Left Crus	6.75 ± 2.25	6.26 ± 3.05	6.26 ± 3.07

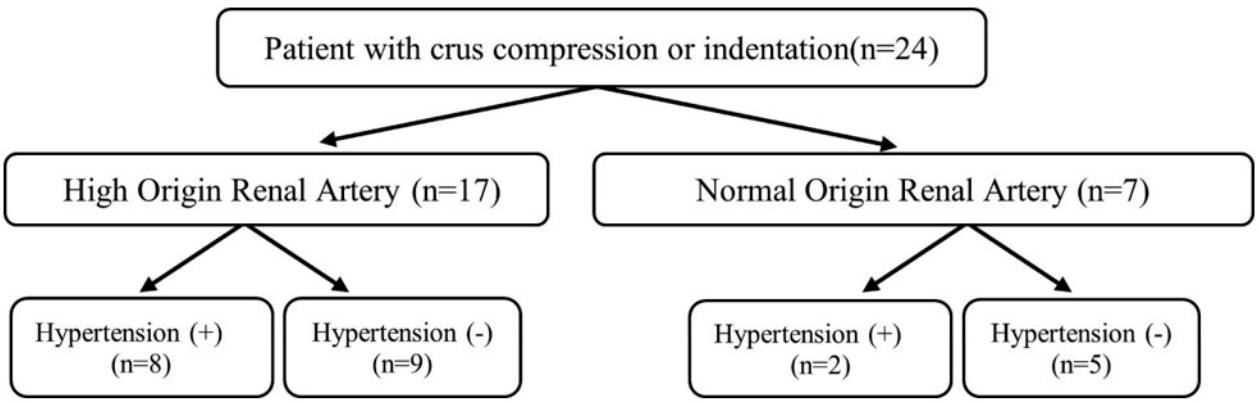

Figure 3. The distribution of the patients with diaphragmatic crus compression or indentation in renal artery origin and hypertension groups.

but their eGFR was 79 ml/min and 80 ml/min which is slightly lower than normal (normal>90 ml/min). While serum creatinine level and eGFR were normal in 4 of 8 patients with DCI and hypertension, serum creatinine level was normal in 2 patients, but GFR was at the lower limit (90 ml/min). There was one patient, whose serum creatinine level was 1.27 mg which is slightly higher than normal, eGFR was 72 ml/min, and there was decreased activity uptake at Tc-99m-DMSA scintigraphy of the right kidney. Additionally, there was one patient, whose serum creatinine level was 5.38 mg and eGFR was 11ml/min which was consistent with renal failure. In 3 patients with DCI without hypertension, serum creatinine levels were normal but eGFR was slightly low (76 ml/min, 89 ml/min, and 82 ml/min).

DISCUSSION

In our study investigating the relationship between DCI and DCC in the renal artery and renovascular hypertension, DCI was detected more frequently than DCC. DCC and DCI were more common on the right side in our study population. The ipsilateral crus was found to be significantly thicker in patients with DCI, and most were men. The frequency of hypertension, female sex, and mean age were higher in patients with DCC.

Hypertension is arterial blood pressure that is consistently 130/80 mmHg or higher (12). Hypertension caused by any pathology in the renal artery is called renovascular hypertension. Color Doppler US, CT angiography, and magnetic resonance angiography (MRA) are used to diagnose renovascular hypertension. Color Doppler US is the first diagnostic tool for detecting the etiology of hypertension because it is accessible and cheap. Peak systolic velocity in the renal artery>180-200 cm/sec and/or reno-aortic ratio>3 indicate renal artery stenosis (RAS). Indirect findings of RAS are prolongation of acceleration time (>70msec) in segmentary branches of the renal artery and tardus parvus waveform distally from the stenosis (13). Obesity, the patient's breathing noncooperation, and abdominal bowel gas are limitations of this

method. Contrast-enhanced MRA has a sensitivity and specificity of 90% in detecting RAS (14,15). Due to the renal toxicity of the iodinated contrast agents, MRA is often preferred in patients with renal failure for detecting RAS. CT angiography perfectly reveals the vascular anatomical structure with multiplanar images. The sensitivity and specificity of CT angiography in the diagnosis of RAS vary between 67-100% and 77-98%, respectively (16,17). It provides excellent anatomical orientation, visualization of the surrounding tissues of the renal artery, and 3-dimensional reconstruction.

The incidence of hypertension secondary to RAS is around 1% (18). The most common cause of RAS in children and young adults is fibromuscular dysplasia, on the other hand, the most common cause in middle-aged adults is atherosclerosis (19). Other causes of RAS include aortic dissection, arteritis, congenital malformations, extrarenal masses compressing the renal arteries, diaphragmatic crus compression/ indentation, and thromboembolism.

DCI and DCC have been reported in case reports in the literature, and their exact frequency is not known (9-11). DCC was first described by D'Abreu in two surgical cases in 1962 (20). There is a case series of three cases of renal artery compression by the fibromuscular band. It was reported in this study that one of the three bands originated from the diaphragm, while the other two were from the sympathetic aortorenal plexus (21). Rather than mechanical compression, sympathetic ganglion compression on the renal artery may be effective in the hypertension cascade (21). In line with all this information, we found the incidence of DCI was 8.4% among 250 patients and hypertension was accompanied by 38.1% of them. The fact that the DCI ratio is less than the hypertension ratio supports that the indentation of the diaphragm crus on the renal artery is not only related to the renal artery but may also be related to the compression of the sympathetic plexus.

Thony et al. (9) detected DCC in 15 patients in their study. They reported that 73% of these cases were on the right side, 53% had crus hypertrophy, and 40% had high-origin renal artery. In our study, we found that the crus on the same side was thicker in patients with DCI ($p=0.004$).

The high origin of the renal artery has been reported as an important cause of DCC in the literature and is more common on the left side (9,22-25). It is thought to be due to an anomaly during the migration of the kidney (10). In our study, the majority of patients (n=8) (87.5%) with DCI had a high origin of the renal artery. Four patients were on the right side, one patient was on the left side, and two patients had a bilaterally high origin. Ozkan et al. (26) revealed that 98% of renal arteries originate from any level between the L1 vertebra superior end plate and the L2 vertebra inferior end plate in their study. In our study, 71.4% of the patients with DCI had a high origin of the renal artery, and the origin of the renal artery was above the L1-L2 intervertebral disc.

Since CT scans of our patients were not performed to detect the etiology of hypertension, the laboratory data of some patients are not available in the automation system. Therefore, we could not make a detailed evaluation of renovascular hypertension in terms of laboratory findings. Other limitations of our study include the possibility of exaggerated stenosis, and the lack of color Doppler US to correlate stenosis.

The present study has several limitations. The first limitation was the retrospective design and relatively small number of patients. Due to the retrospective design, we could not perform any additional diagnostic tests for the etiology of the renovascular hypertension. Since CT scans were performed in the supine position with deep inspiration, there might have been an exaggeration of the indentation or compression.

In conclusion, DCI and DCC which can be caused by hypertrophic diaphragmatic crus or high origin of the renal artery, should be included in the etiology of renovascular hypertension. Although hypertension was detected more frequently than DCI in patients with DCC, the frequency of DCI was higher in our study. Therefore, even if there is no compression of the renal artery, the interaction of the renal arteries with the diaphragmatic crus should be kept in mind for patients with hypertension.

Ethical approval

This study has been approved by the Ankara City Hospital Review Board (approval date 09.03.2022, number E1-22-2407). Written informed consent was obtained from the participants.

Author contribution

Concept: DAA, NIÖ; Design: DAA, NIÖ; Supervision: AHÇ, MB, NIÖ; Materials: DAA, EÇ, BAD; Data Collection and/or Processing: DAA, EÇ, BAD; Analysis and/or Interpretation: DAA, EÇ, BAD; Literature Search: DAA, EÇ, BAD; Writing Manuscript: DAA, AHÇ, MB, EÇ, BAD, NIÖ; Critical Review: MB, AHÇ, NIÖ. All authors reviewed the results and approved the final version of the article.

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Conflict of interest

The authors declare that there is no conflict of interest.

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