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Evaluation of abdominal vascular structures by multidetector computed tomography in Crimean–Congo hemorrhagic fever patients

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ABSTRACT

Aim: This study aims to determine whether Crimean–Congo Hemorrhagic Fever (CCHF) has effects on abdominal vascular structures.

Material and Method: The study group included 35 patients diagnosed with CCHF. The control group included 35 patients with symptoms such as dyspepsia, pelvic pain, and abdominal pain who underwent multidetector computed tomography (MDCT) and whose MDCT examinations were reported as completely normal. This is a retrospective study and patients admitted to the hospital between May 2016 and April 2022 were included in the study. The patient group and control group were compared in terms of liver size, splenic size, and portal vein (PV), hepatic artery (HA), splenic vein (SV), splenic artery (SA), superior mesenteric vein (SMV) and superior mesenteric artery (SMA) diameters.

Results: The liver size, splenic size, and PV, HA, and SA diameters in the patient group were significantly higher than those in the control group ($p<0.001$). While the SV, SMV, and SMA diameters were higher in the patient group than in the control group, the differences were not significant ($p>0.05$).

Conclusion: Our study shows that abdominal vascular structures are affected in CCHF patients.

Keywords: Crimean–Congo hemorrhagic fever, multidetector computed tomography, portal vein, superior mesenteric vein

INTRODUCTION

Crimean–Congo hemorrhagic fever (CCHF) is a viral zoonotic disease that is characterized by hemorrhage and fever and can be fatal (1-4). It is transmitted to humans through contact with infected secretions and blood of humans or animals or through tick bites (5-7). The pathogenesis of the disease is not completely known. However, it is known to affect some target cells, including endothelial cells and hepatocytes. In particular, endothelial cells are severely affected by the disease (8). Although the destruction of the vascular endothelium by the virus is known, there are insufficient studies in the literature on changes in abdominal vascular structures that may develop due to the disease (9-11). Therefore, our study aims to assess whether any diameter changes are detected on abdominal multidetector computed tomography (MDCT) in CCHF in the portal vein (PV), hepatic artery (HA), splenic vein (SV), splenic artery (SA), superior mesenteric vein (SMV), and superior mesenteric artery (SMA). We think that our study may pave the way for more comprehensive studies on abdominal vascular structures in CCHF.

MATERIAL AND METHOD

Our study was planned retrospectively. The study was carried out with the permission of University Hospital, Noninvasive Clinical Ethics Committee (Date:28.02.2022 Decision No:22-KAEK-140). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

Patients

Our study consists of 35 patients who were admitted to our hospital between May 2016 and April 2022 and were diagnosed with CCHF, and 35 individuals constituting the control group who underwent MDCT with symptoms such as dyspepsia, pelvic pain, and abdominal pain and whose MDCT examinations were reported as completely normal.

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Patients with malignancy, acute or chronic heart or lung failure, blood disease, chronic liver disease, or inflammatory bowel disease in addition to CCHF disease, patients with benign or malignant lesions of the spleen and liver, patients with any chronic gastrointestinal disease, and those who had undergone abdominal surgery or radiotherapy were not included in the patient group. Patients' medical information was obtained from their files.

The control group consisted of patients with nonspecific symptoms such as dyspepsia, pelvic pain, and abdominal pain who underwent abdominal MDCT examination and for whom no radiological findings were detected as a result of the examination. The control group was demographically similar to the patient group and consisted of patients without any additional disease.

MDCT Protocol

The MDCT protocol was applied by using 128-channel MDCT (GE Healthcare, Milwaukee, WI, USA). Consecutive axial sections of 5 mm slice thickness were obtained by abdominal MDCT. Automatic tube modulation at 120– 220 mA. The patient was in the supine position during the examination.The patients were asked to hold their breath during the examination.

Evaluation of MDCT Images

All images were evaluated by a radiologist who had at least 5 years of experience in abdominal radiology. For all patients in the study group, liver and spleen sizes were measured from the mid-clavicular line by creating coronal reformatted images (10). A liver size above 16 cm was defined as hepatomegaly. Spleen size above 12 cm was defined as splenomegaly (10). A gallbladder wall thickness greater than 3 mm was considered as gallbladder wall thickening (10). Measurements of vascular structures were made by taking the largest diameter of the vessel in the axial section of the abdominal window in MDCT of the abdomen. Portal vein and HA diameters were measured at the level of the liver hilum, and SV and SA diameters were measured at the level of the splenic hilum in the axial plane (**Figure**). The diameter of the superior mesenteric vein was measured in the axial plane from the first 2 cm after the origin of the portosplenic junction, and the SMA diameter was measured from the first 2 cm after the origin of the SMA from the aorta in the axial plane (12). The study group was compared with the control group.

Statistical Analysis

The statistical significance level of p was 0.05. Statistical analysis was performed using commercial software (SPSS 22.0 Chicago, IL, USA). On qualitative variables Chi-square test was used for comparison.

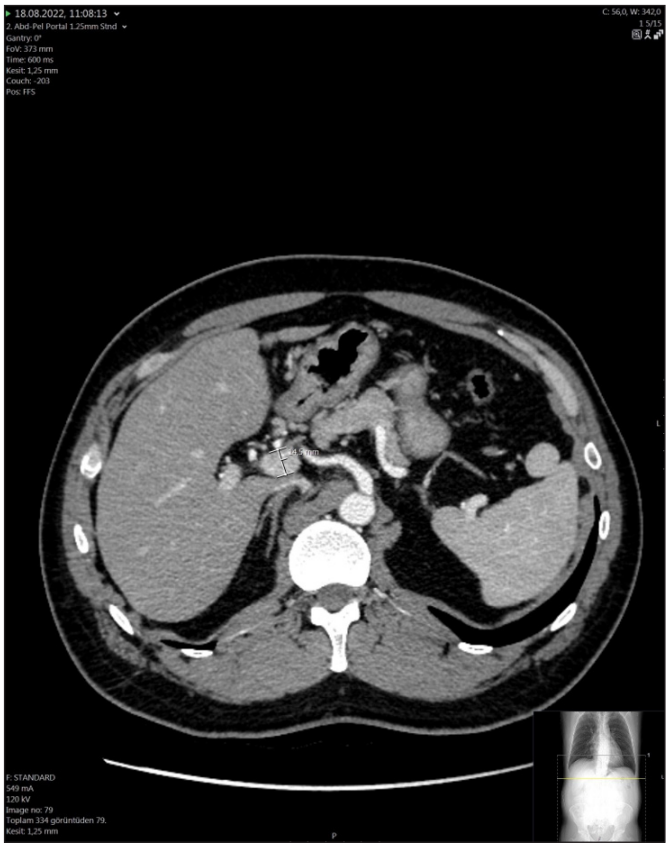

Figure. Measurement of portal vein diameter at the level of the liver hilum in axial multidetector computed tomography

RESULTS

Our study group consisted of 35 CCHF patients (20 males and 15 females) and 35 control patients (18 males and 17 females). The mean age of the CCHF patient group was 57, and the mean age of the control group was 52.63. There were no statistically significant differences between the patients and controls in terms of age and sex (p>0.05) (**Table 1**).

Table 1. Age and gender distribution of the control and patient groups

	Groups		p
	Control	Patient	
	Mean±SD	Mean±SD	
Age	52.63±20.17	57±14.7	0.299
Gender	1.48±0.50	1.41±0.50	0.565

In the evaluation of MDCT examinations of the patients, we found hepatomegaly in 22 (62.8%) patients, pleural effusion in 11 (31.4%) patients, abdominal free fluid in 7 (20%) patients, splenomegaly in 6 (17.1%) patients, mesenteric and omental adipose tissue heterogeneity in 4 (11.4%) patients, gallbladder wall thickening in 3 (8.5%) patients, parenchymal consolidation in 3 (8.5%) patients, pericardial effusion in 2 (5.7%) patients, and a decrease in periportal density detected in the form of a sheath representing periportal edema in 2 (5.7%) patients (**Table 2**).

Table 2. MDCT findings of the 35 patients with CCHF

Findings	Number of patients (n)	%
Hepatomegaly	22	62.8
Pleural effusion	11	31.4
Splenomegaly	6	17.1
Abdominal free fluid	7	20
Mesenteric and omental heterogeneity	4	11.4
Gallbladder wall thickening	3	8.5
Parenchymal consolidation	3	8.5
Pericardial effusion	2	5.7
Decrease in periportal density	2	5.7

The patient group and the control group were compared in terms of liver size, splenic size, and PV, HA, SV, SA, SMV, and SMA diameters. The liver size, splenic size, PV, HA, and SA diameters in the patient group were significantly larger than those in the control group ($p<0.001$). SV, SMV, and SMA diameters were larger in the patient group than in the control group, but no statistically significant differences were found ($p>0.05$) (**Table 3**).

Table 3. Liver size, splenic size, portal vein, hepatic artery, splenic vein, splenic artery, superior mesenteric vein and superior mesenteric artery diameters in the control and patient groups

	Groups		p
	Control	Patient	
	Mean±SD	Mean±SD	
Liver size	16.08±1.40	17.36±1.76	0.001
Splenic size	8.93±1.61	10.36±1.96	0.001
PV	12.53±1.3	13.61±2.04	0.010
HA	4.2±0.63	4.86±0.98	0.001
SV	7.93±1.52	7.99±1.57	0.852
SA	4.21±0.77	5.83±1.1	0.001
SMV	10±1.46	10.75±1.9	0.066
SMA	7.01±1.13	7.46±1.69	0.200

DISCUSSION

Crimean-Congo Hemorrhagic Fever begins with the virus entering the body through mucous membranes, or by inhalation. The virus can have direct toxic effects on the liver, spleen, and endothelial cells. This toxic effect stimulates the release of cytokines and chemokines from defense cells and may lead to an intense inflammatory response as well as endothelial damage. Considering the pathophysiological mechanisms at the molecular level, this process, which is caused by many inflammatory factors, is associated with increased endothelial damage and vascular permeability. Due to this toxic effect of the disease on endothelial cells, some studies have been conducted on changes caused by the disease in vascular structures (7,13-18). Two previous studies evaluated Doppler findings in the carotid and vertebral arteries in CCHF disease (19,20). Karavas et al. (19) and Salk et al. (20) found that peak end systolic and diastolic flow rates of bilateral carotid arteries and vertebral arteries were elevated in CCHF patients. These studies have shown that there are alterations in the vascular system in CCHF patients.

Aktas et al. (7) found a significant increase in the diameter of both main pulmonary arteries and pulmonary trunks in CCHF patients in their study on the thoracic findings in the disease. Karavas et al. (19) conducted a study on the abdominal findings of the disease with Doppler US. In their study, they found a significant increase in portal vein flow velocity in CCHF patients. They also found that liver size, spleen volume, and portal vein and splenic vein diameters were significantly larger in CCHF patients. In our study using MDCT, we evaluated liver size, spleen size, and PV, HA, SV, SA, SMV, and SMA diameters in CCHF patients. In our study, we found that liver and spleen sizes were significantly larger in the patient group compared to the control group ($p<0.01$). There was also a significant increase in PV, SA, and HA diameters in the patient group compared to the control group ($p<0.01$). Although SV, SMV, and SMA diameters were larger in the patient group compared to the control group, these differences were not statistically significant. The results of our study are compatible with the literature. A significant increase in diameter is observed in CCHF patients in the majority of abdominal vascular structures. These vascular structures especially concern the liver and spleen. In our previous studies (10,11), the most common findings in CCHF disease were hepatomegaly and splenomegaly. The increase in diameter, especially in PV, HA, and SA, in our study may be the cause of hepatomegaly and splenomegaly. The involvement of these vascular structures is thought to be due to the intense inflammatory response and endothelial damage caused by the pathophysiological mechanisms we mentioned before. The absence of a significant increase in SMV and SMA diameters in our study may explain that the changes seen in mesenteric and omental fatty tissue and intestinal loops are relatively less than the changes seen in liver and spleen in CCHF patients. Although the structures affected by the disease in the abdomen and the resulting findings are quite different, it is thought that the common point of all findings is endothelial damage and systemic inflammation.

The Most Important Limitations of Our Study

The study was conducted retrospectively on MDCT data. Therefore, only diameters of vascular structures were evaluated, but no comment can be made on vascular flow. Our study was conducted with a small group of patients. The reason for this is that while CCHF disease is endemic, the disease shows mostly thoracic findings, and abdominal MDCT is deemed necessary only in a small group of patients.

CONCLUSION

Our study shows that abdominal vascular structures are affected by the disease in CCHF patients. It is thought that these changes in the vascular structures may be responsible for the complications seen in the abdomen.

ETHICAL DECLARATIONS

Ethics Committee Approval: The study was carried out with the permission of University Hospital, Noninvasive Clinical Ethics Committee (Date:28.02.2022 Decision No:22-KAEK-140).

Informed Consent: Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

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Author Contributions: All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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