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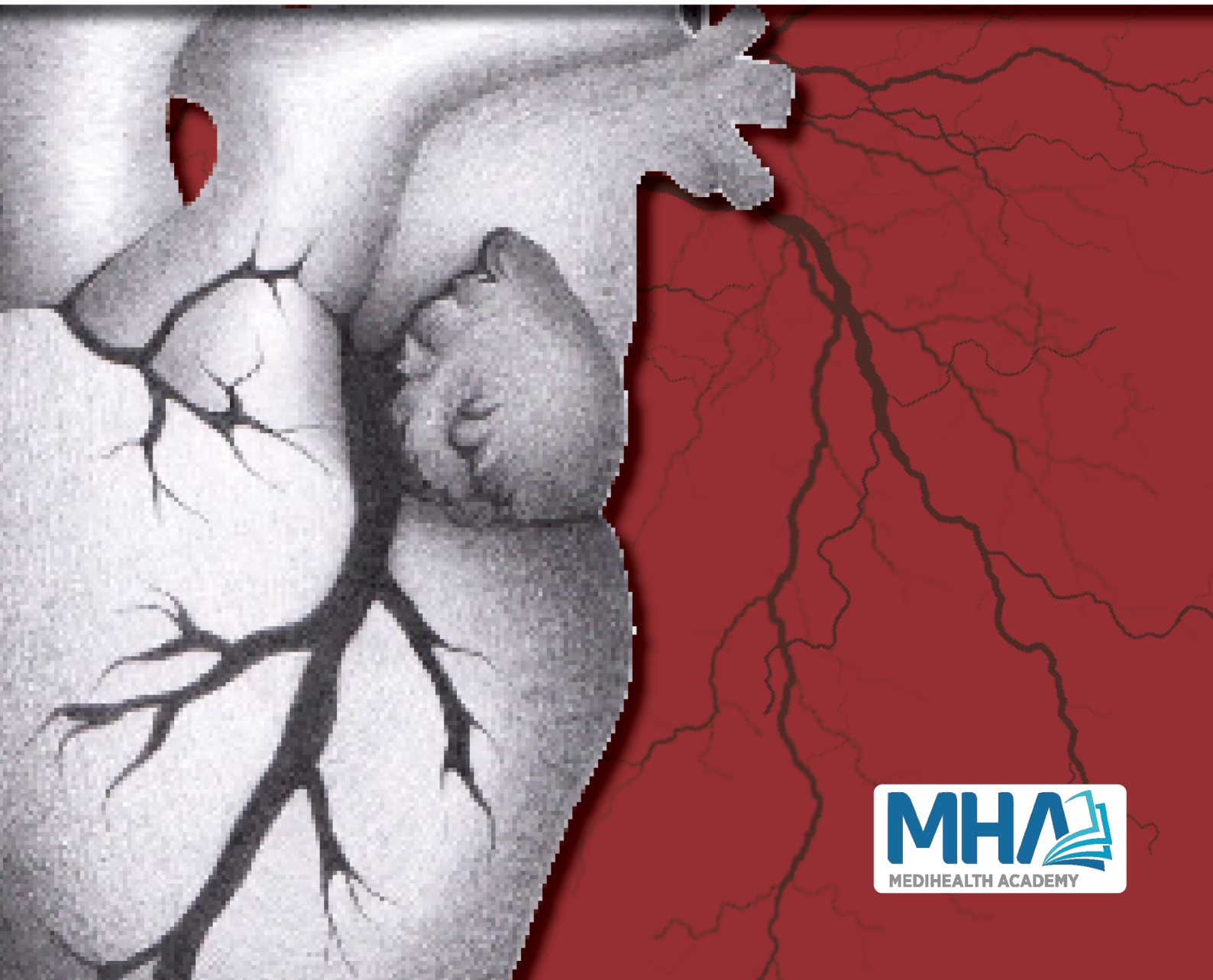

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Dear Colleagues,

I feel excited to welcome you in the March issue of the Journal of Cardiology & Cardiovascular Surgery. This issue included 3 original research articles, 2 interesting cases, and a review.

The first original article involved 1031 patients and investigated the relationship between the ABO blood group and chronic venous disease in terms of deep vein thrombosis. In the second original article, Bayhatun et al. compared the carotid intima media thickness in patients with treatment-regulated hypertension without coronary artery disease with the normal population. They highlighted the importance of effective antihypertensive therapy to slow down the atherosclerotic process. The third original article entitled "May high blood viscosity predict cardiac involvement in COVID-19 patients?" presented the results regarding the effects of whole blood viscosity on prognosis and deterioration in cardiac parameters in COVID-19 patients after recovery using cardiac magnetic resonance imaging and echocardiography.

In the review article, Kabalcı et al. discussed the current approach and tips regarding venous catheterization in hemodialysis patients.

In this issue, the first interesting case entitled "Spontaneous recovery in the early period of cardiac remodeling due to carbon monoxide poisoning" was presented by Tanık et al. The second interesting case entitled "A case report of history of anterior myocardial infarction complicated with left ventricular pseudoaneurysm" was presented by Demirci et al.

I would like to thank the authors, reviewers, editorial team and publisher for their hard work and dedication. We are happy to present you the latest updates in cardiology and cardiovascular surgery and we will strive to mediate all your contributions to science in the way of highest quality.

Sincerely,

İbrahim Halil İNANÇ, MD

Editor-in-Chief

Journal of
Cardiology & Cardiovascular
Surgery

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ORIGINAL ARTICLES

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Original Article

DOI: 10.51271/JCCVS-0024

The relationship between the ABO blood group and chronic venous disease in deep vein thrombosis

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ABSTRACT

Aims: This study aims to investigate the relationship between the ABO blood group and chronic venous disease in terms of deep vein thrombosis (DVT).

Methods: This study was planned as a retrospective case-control study in which 1031 patients were included between February 1, 2022, and July 1, 2022. After systematic sampling, the case group (88) and the control group (113) were analyzed in terms of blood groups. Then the patients were divided into 3 groups. group 1: patients with non-O blood group and chronic venous disease (CVD) (n=75), group 2: patients with non-O blood group and non-CVD (n=73), and group 3: patients with O blood group and CVD patients (n=28). It was analyzed whether there was a difference between these 3 groups and from which group and in what form.

Results: The non-O blood group was significantly higher in the DVT group than the control group (p=0.001). There was no significant difference between the A, B, and AB blood groups (p=0.21, p=0.51, p=0.08, respectively). When group 1, group 2, and group 3 were compared, a significant difference was found (p=0.006). In the posthoc analysis, no difference was found between group 1 and group 2 (p=0.99), a difference was found between group 3 and group 1-group 2, and it was found to be lower (p=0.34, p=0.46, respectively).

Conclusion: We found the non-O blood group was higher in the DVT group compared to the control group. We found that the non-O blood group alone was higher in patients with DVT than CVD alone. While non-O blood type and CVD are important risk factors for DVT, non-O blood type can increase the risk of DVT more than CVD. However, this finding needs to be discussed in future studies.

Keywords: Deep vein thrombosis, ABO blood group, chronic venous disease

INTRODUCTION

Venous thromboembolism (VTE), including deep vein thrombosis (DVT) and pulmonary embolism (PE), is a chronic disease that affects approximately 10 million people every year and is an important health problem.[1]

Commonly known risk factors for VTE include increasing age, inactivity, malignancy, major surgery, and heart failure.[2] A close association has been shown between VTE and the non-O blood group, whereas there is less certainty about the relationship between the non-O blood group and arterial thrombosis, especially myocardial infarction.[3]

There are many studies investigating the relationship between ABO blood group or varicose veins and DVT. However, there are not many studies examining these two risk factors separately and investigating which risk factor alone poses a higher risk for DVT. Therefore, we planned this case-control study. We aimed to investigate the relationship between the ABO blood group and varicose veins in terms of DVT.

METHODS

The study was approved by the Kırıkkale University Faculty of Medicine Ethics Committee (Date: 28.09.2022, Decision No: 2022.09.07). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki. This study was planned as a retrospective case-control study in which all patients who applied to Kırıkkale Yüksek İhtisas Hospital Cardiovascular Surgery outpatient clinic between February 1, 2022, and July 1, 2022, were examined and a total of 1031 patients were included. Those younger than 18 years of age, patients who have previously been diagnosed with DVT but no DVT was found in their examinations, those with a history of cardiac surgery, patients who underwent surgery for chronic venous disease or peripheral artery disease, those who had thrombophlebitis, those who use direct oral anticoagulants (DOAC), for reasons other than DVT, those with chronic kidney disease, those with cancer, patients with a central catheter, pregnant women, and patients with a genetically proven predisposition to thrombosis were excluded.

The basic demographic characteristics, medical histories, laboratory findings, venous clinical severity scores (VCSS), Villalta scores, Clinical, Etiological, Anatomical, and Pathophysiological (CEAP) scores of the patients, drugs administered, and complications were recorded. The patients

were followed up routinely at 1, 3, and 6 months. The medical treatments of the patients were planned by analyzing the risk group, and low molecular weight heparin (LMWH), vitamin K antagonist (VKA), or DOAC were started.

Firstly, the patients were divided into two groups. Case group: patients with DVT detected in color Doppler ultrasonography (DUS) performed during the first examination (n=88), control group: patients without DVT as a result of DUS (n=653). The diagnosis of CVD disease was made with the examination performed in the outpatient clinic and DUS findings. After that, randomized sampling from the control group was performed and 113 patients were evaluated. In the first stage of the study, it was analyzed whether there was a difference between these two groups. Then, to examine the relationship between blood group and CVD in terms of DVT, all patients included in the study were divided into 3 groups as follows; group 1: Those with non-O blood group CVD (n=75), group 2: Those with non-O blood group without CVD (n=73), and group 3: Those with O blood group with CVD (n=73) n=28) (Figure). In the next stage, it was analyzed whether there was a difference between these 3 groups and from which group and in what form.

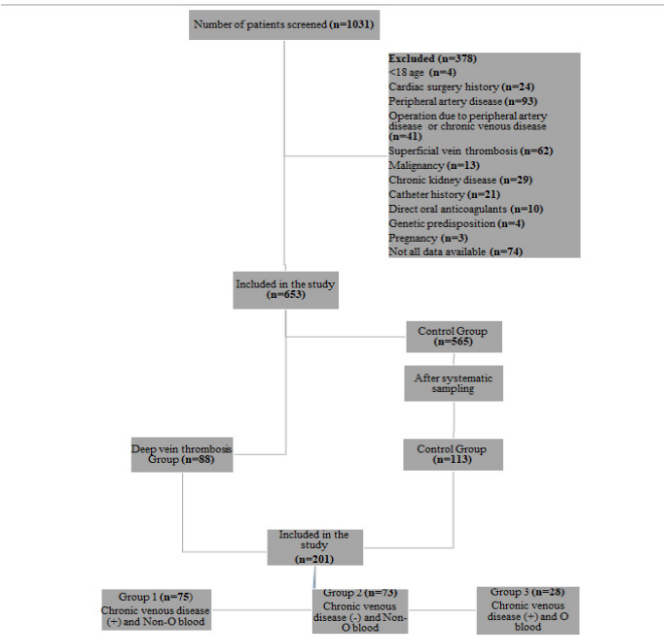

Figure. Flow chart for patient selection

Statistical Analysis

IBM (SPSS) Statistics v.22.0 was used to analyze the data. In descriptive statistics, continuous variables were expressed as min-max, median, and interquartile range and categorical variables were expressed as frequency (n) and percent (%). The normality of the distribution was evaluated with the Kolmogorov-Smirnov test. Student's t-test was used for numerical variables and parametric data, and the Mann-Whitney U test was used for non-parametric data. Pearson $\chi 2$ test and Fisher exact test were used for categorical variables and parametric and non-parametric data. Wilcoxon sign rank test was used to compare scores. The Kruskal-Wallis test was used to make comparisons between groups. Then, post hoc analysis was performed using the Tamhane test. The statistical significance level was accepted as $p<0.05$.

RESULTS

When the basic demographic characteristics of the patients were examined, it was found that chronic obstructive pulmonary disease and cerebrovascular accident were found to be higher in the case group, and female gender in the control group (p=0.001, p=0.02, p=0.001 respectively) (Table 1). There was a similar rate of CVD in both groups (p=0.55). There was no statistical difference between DVT and control groups in terms of CEAP scores among patients with CVD. When compared in terms of blood group, the non-O blood group was found to be significantly higher in the DVT group compared to the control group (n=75, 85.2% and n=73, 64.6%, p=0.001, respectively). There was no significant difference between A, B, and AB blood groups (p=0.21, p=0.51, p=0.08, respectively. Of the patients in the case group, 42 (47.7%) were acute and 44 (50%) were chronic. In the control group, the history of having COVID-19 was lower, and the history of the COVID-19 vaccine was higher (p=0.4, p=0.02, respectively). Although the basic laboratory parameters were similar between the groups, aspartate aminotransferase, and c-reactive protein (CRP) were found to be higher in the DVT group (p=0.03, and p<0.001, respectively). VCSS and Villalta scores decreased significantly in the first month after treatment compared to the first diagnosis (p<0.001, p<0.001, respectively).

To examine the relationship between the non-O blood group and CVD for DVT, the patients were divided into 3 groups and the results of the analysis are shown in Table 2. A statistical difference was found between the groups according to the results of the Kruskal-Wallis test (p=0.006). In the post hoc analysis performed to examine which group the difference originated from, no difference was found between group 1 and group 2 (p=1), a difference was found between group 3 and group 1-group 2, and it was found to be lower (p=0.003, p=0.003, respectively).

Complications observed in the case group during the study period are shown in Table 3. One (1.1%) patient died due to acute massive PE. PE developed in 2 (2.2%) patients, minor bleeding in 1 (1.1%), and residual vein thrombosis in 10 (11.3%) patients. VKA was started in 51 (57.9%) patients, LMWH in 10 (11.3%), and DOAC in 26 (29.5%) patients as initial treatment.

DISCUSSION

DVT is an important cause of morbidity and mortality. Many genetic and acquired risk factors for DVT have been identified so far.[2] The fact that genetic factors cannot be changed is important for patients in this risk group. Many studies have been conducted so far regarding blood type, one of the genetic factors that increase the risk of DVT. Varicose veins, one of the acquired risk factors, have also been shown to have a causal effect on DVT.[4] Yet, as far as we know, there is no study comparing the relationship between these risk factors. Therefore, we planned this case-control study.

As the first result of our study, we found that the non-O blood group was significantly higher in patients with DVT compared to the control group. However, there was no significant difference between A, B, and AB blood groups. These findings support other studies in the literature. Having a non-O blood type has been associated with an increased

Table 1. Patient demographics. clinical characteristics. and laboratory parameters

	Deep vein thrombosis (n=88)			Control (n=113)			
	Min-max or n (%)	Median	IQR	Min-max or n (%)	Median	IQR	p
Gender female	52 (59.09)			74(65.4)			0.001*
Age (years)	20-99	60	26	21-88	50	23	0.09
Height (cm)	144-189	170	12	150-186	168	11	0.057
Weight (kg)	40-130	78	16	38-110	75	18	0.1
Body surface area (kg/m^2)	1.26-2.36	1.9	0.24	1.32-2.31	1.87	0.99	0.04*
Body mass index (m^2)	17.1-48.2	26.6	6.3	15.2-44.4	26.9	6.4	0.25
Coronary artery disease	5 (5.7)			8 (7.1)			0.69
Heart failure	3 (3.4)			1 (0.9)			0.20
Diabetes mellitus	11 (12.5)			11 (9.7)			0.53
Hypertension	23 (26.1)			20 (17.9)			0.14
Chronic obstructive pulmonary disease	10 (11.4)			1 (0.9)			0.001*
Cerebrovascular accident	6 (6.8)			1 (0.9)			0.02*
Smoking	34 (38.6)			38 (33.6)			0.65
0 blood group	13 (14.8)			40 (35.4)			0.001*
Non-0 blood group	75 (85.2)			73 (64.6)			0.001*
A blood group	42 (47.7)			44 (38.9)			0.21
B blood group	18 (20.5)			19 (16.8)			0.51
AB blood group	15 (17)			10 (8.8)			0.08
Chronic venous disease	43 (48.9)			60 (53.1)			0.55
CEAP 1	23 (26.1)			35 (31)			0.45
CEAP 2	17 (19.3)			20 (17.6)			0.64
CEAP 3	3 (3.4)			5 (4.4)			0.41
COVID-19 vaccination	68 (77.3)			101 (89.4)			0.02*
COVID-19 history	44 (50)			50 (44.2)			0.4
Leukocyte (10^9/L)	1.5-14.6	7.8	4.1	3.2-14.0	7.3	2.2	0.001*
Hemoglobin (g/dl)	8.0-16.9	12.0	3.0	9.0-17.0	12.00	1.00	0.002*
Platelet (10^9/L)	101.0-534.0	254.5	93.8	129.0-423.0	262.00	102.5	0.21
Creatinine (mg/dl)	0.2-2.1	0.7	0.4	0.3-8.7	0.6	0.3	0.8
Aspartate aminotransferase (IU/L)	8-522	20	10.4	7-118	18	7	0.03*
Alanine aminotransferase (IU/L)	4-269	17	9.8	5-198	15	8	0.21
C-reactive protein (mg/dl)	0.02-17	0.59	2.0	0.01-11	0.3	0.5	<0.001*
D-Dimer (ng/mL)	52-3864	358	345				
Fibrinogen (mg/dl)	20-45	24	7				
International normalization ratio	0.8-3.8	1.1	3				
Deep vein thrombosis side							
Right	42 (47.7)						
Left	53 (60.2)						
Bilateral	12 (13.6)						
Deep vein thrombosis time							
Acute	35 (39.7)						
Subacute	16 (18.1)						
Chronic	44 (50)						
Deep vein thrombosis localization							
Common iliac vein	3 (3.4)						
External iliac vein	14 (15.9)						
Common femoral vein	46 (52.2)						
Superficial femoral vein	64 (72.7)						
Popliteal vein	70 (79.5)						
Crural	30 (26.4)						
	Admission			First month			
Venous Clinical Severity Score	2-23	7	4	1-21	4	3	<0.001*
Villalta Score	3-24	9	5	1-22	5	4	<0.001*

CEAP: Clinical. Etiology. Anatomy. and Pathological. IQR: Interquartile range

Table 2. Comparison of groups with the Kruskal-Wallis test in terms of DVT

	N	Mean Rank	χ2	p
Group 1	75	93.09		
Group 2	73	93.10	10,17	0,006
Group 3	28	64.21		

Table 3. Complications in the case group, initial and maintenance treatments

Complications	n=88 (%)
Mortality	1(1.1)
Postoperative pulmonary thromboembolism	2(2.2)
Minor bleeding	1(1.1)
Residual vein thrombosis	10(11.3)
Initial therapy	
Warfarin	52(59)
Low molecular weight heparin	10(11.3)
Rivaroxaban	26(29.5)
Maintenance therapy	
Acetylsalicylic acid	7(7.9)
Warfarin	35(39.7)
Low molecular weight heparin	3(3.4)
Rivaroxaban	42(47.7)
Apixaban	1(1.1)

risk of proximal DVT in the lower extremities.[5] Spavor et al.[6] showed that non-O blood type is associated with the risk of DVT. People with non-O blood type have higher levels of factor VIII (FVIII) and von Willebrand factor (vWF) compared to people with blood type O.[7] Elevated FVIII and vWF are moderate risk factors for VTE.8 Larsen et al.[9] showed that women with blood group A or AB had a 2-fold higher risk of VTE during pregnancy and puerperium than women with blood group O, while women with blood group B did not show an increased risk. Jukić et al.[10] also showed that people with non-OO blood type are at twice the risk of thrombosis than those with non-OO blood type. In addition, carriers of AB and A2 B genotypes showed the highest risk of thrombosis, followed by BB/O1 B/O2 B genotypes and O1 A1/O2 A1 genotypes. Dentali et al.[11] reported that non-O blood type may be associated with residual vein occlusion. It is also reported that O-blood group patients have higher fibrinolytic activity compared to non-O patients. These results and the findings of our study overlap with each other. Blood group, which is a simple and easily accessible genetic risk factor that cannot be changed, can be a guide in determining the patient groups in the risk group for DVT.

Because many factors may pose a risk for DVT and these may affect the results of the study, we had to exclude a significant number of patients from the entire patient cohort to reduce the impact of other risk factors. As a result of our study, no significant difference was found between patients with the non-O blood group and CVD and patients with only the non-O blood group in terms of DVT. However, patients with only CVD were associated with DVT at a significantly lower rate than the other two groups. However, in our study, although there was no difference in CVD rates and CEAP stages in patients in the DVT and control groups, it was observed that the majority of CEAP stages were stages 1 and 2. Since patients who underwent interventional procedures for CVD were excluded from the study, the data of many advanced-stage patients were not analyzed. According to the results of this study, it was found that the non-O blood group poses a higher risk for DVT compared to varicose veins, but future studies with a higher number of patients are required to generalize these results.

DVT is a serious complication of COVID-19.[12] Günertem et al.[13] reported that VTE may adversely affect the prognosis of COVID-19 patients and therefore it is important to conduct a risk analysis for DVT. In the control group, the history of having COVID-19 was lower, and the history of the COVID-19 vaccine was higher. Although we matched patient groups for risk factors as much as possible, this difference may have partially affected the results of the study.

It is important to know beforehand about patients at high risk of DVT. When it comes to the long-term treatment phase after DVT treatment, it is useful to pay attention to the blood group while evaluating the risk status of the patient. This simple information can be helpful in counseling, further testing, and identifying patients at risk. However, future studies with larger numbers of patients are needed to clarify these findings.

Limitations

This study was retrospective. At first, there was a significant difference between the number of patients in the case group and the control group. Therefore, we conducted a completely randomized systematic sampling without appropriate patient selection into groups. Patients in the case group included acute and chronic patients and were not homogeneously distributed. Similarly, the CEAP stages were heterogeneous and most of the advanced CVD patients were excluded because of the interventional procedure. The difference in the rates of COVID-19 between the groups may also have affected the result in terms of DVT risk. In addition, we were not able to analyze the FVIII and vWF levels in our hospital. Although we made a detailed comparison for the case and control groups and found no critical differences in terms of baseline characteristics, we were unable to make an additional comparison for Groups 1, 2, and 3 in the second stage due to small numbers. Finally, we could not perform subgroup analysis for the blood group. By analyzing these, more useful results can be found in the future.

CONCLUSION

The non-O blood group was higher in the DVT group compared to the control group. When the non-O blood group and CVD were evaluated together, we found that the non-O blood group alone was higher in patients with DVT than in CVD alone. While the non-O blood type and CVD are important risk factors for DVT, non-O blood type can increase the risk of DVT more than CVD. However, this finding needs to be discussed in future studies.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was carried out with the permission of Kırıkkale University Faculty of Medicine Ethics Committee (Date: 28.09.2022, Decision No: 2022.09.07).

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.

Financial Disclosure

The authors declared that this study has received no financial support.

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.

REFERENCES

1. Khan F, Tritschler T, Kahn SR, Rodger MA. Venous thromboembolism. *Lancet*. 2021;398(10294):64-77.
2. Anderson Jr FA, Spencer FA. Risk factors for venous thromboembolism. *Circulation*. 2003;107(23 Suppl 1):I9-I16.
3. Franchini M, Mannucci PM. ABO blood group and thrombotic vascular disease. *Thromb Haemost*. 2014;112(6):1103-9.
4. Li R, Chen Z, Gui L, et al. Varicose veins and risk of venous thromboembolic diseases: a two-sample-based mendelian randomization study. *Front Cardiovasc Med*. 2022;9:849027.
5. Spiezia L, Campello E, Bon M, et al. ABO blood groups and the risk of venous thrombosis in patients with inherited thrombophilia. *Blood Transfus*. 2013;11(2):250-253.
6. Spavor M, Halton J, Dietrich K, et al. Age at cancer diagnosis, non-O blood group and asparaginase therapy are independently associated with deep venous thrombosis in pediatric oncology patients: a risk model. *Thromb Res*. 2016;144:27-31.
7. Schleef M, Strobel E, Dick A, Frank J, Schramm W, Spannagl M. Relationship between ABO and Secretor genotype with plasma levels of factor VIII and von Willebrand factor in thrombosis patients and control individuals. *Br J Haematol*. 2005;128(1):100-107.
8. Martinelli I. von Willebrand factor and factor VIII as risk factors for arterial and venous thrombosis. *Semin Hematol*. 2005;42(1):49-55.
9. Larsen TB, Johnsen SP, Gislum M, Moller CA, Larsen H, Sorensen HT. ABO blood groups and risk of venous thromboembolism during pregnancy and the puerperium. A population-based, nested case-control study. *J Thromb Haemost*. 2005;3(2):300-304.
10. Jukic I, Bingulac-Popovic J, Dogic V, et al. ABO blood groups and genetic risk factors for thrombosis in Croatian population. *Croat Med J*. 2009;50(6):550-558.
11. Dentali F, Di Minno MN, Turato S, et al. Role of ABO blood group and of other risk factors on the presence of residual vein obstruction after deep-vein thrombosis. *Thromb Res*. 2014;134(2):264-267.
12. Cai C, Guo Y, You Y, et al. Deep venous thrombosis in COVID-19 patients: a cohort analysis. clin appl thromb hemost. 2020;26:1076029620982669.
13. Günertem E, Akay T, Aliyev A, et al. Treatment and prophylaxis strategies for deep vein thrombosis during COVID-19 outbreak. *Turk J Vasc Surg*. 2020;29(3):203-207.

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Original Article

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Comparison of carotid intima media thicknesses of hypertension patients with normal coronary artery who are under effective antihypertensive therapy with the normal population

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ABSTRACT

Aims: In this study, we aimed to compare the carotid intima media thickness in patients with treatment-regulated hypertension without coronary artery disease with the normal population.

Methods: A total of 62 patients with normal coronary arteries detected after elective coronary angiography; 32 patients without hypertension and 30 patients with hypertension regulated by drug therapy were enrolled in the study.

Results: In this study we conducted in our clinic, no statistically significant difference was found between carotid intima media thickness in the group whose hypertension was regulated by treatment, and in the control group.

Conclusion: The fact that the carotid intima media thicknesses of patients with treatment-regulated hypertension without coronary artery disease were not found to be different compared to the healthy patient group suggests to us that effective antihypertensive therapy may slow down the atherosclerotic process.

Keywords: Carotid intima media thickness, hypertension, normal coronary artery

INTRODUCTION

Hypertension is an important public health problem in all over the world, and it is thought that there are approximately 1 billion hypertension patients worldwide. Hypertension is a common risk factor for coronary and carotid atherosclerosis.[1,2] Intima media thickness, expressed as arterial wall thickness, has been shown to be an early marker of endothelial organ damage and an initial sign of atherosclerotic disease. Increased carotid intima media thickness (CIMT) is closely related to the presence of coronary artery disease and myocardial infarction. About 50% to 60% of those with carotid disease have serious coronary disease, while only 10% of those with coronary artery disease have serious carotid diseases.[3-7] It is not known whether there is carotid atherosclerosis in patients with hypertension without coronary atherosclerosis, that is, it is not known whether there is an increase in CIMT. Although hypertension appears to be associated with increased intima-media thickness in most studies, there are studies that did not yield the same results.[5,6,8-10] Considering that effective antihypertensive treatment prevents cardiovascular complications of hypertension, we aimed to compare carotid intima media thickness (CIMT) in patients whose blood pressure is under control with antihypertensive treatment with the normal population.

METHODS

All patients included in the study were informed about the study and informed consent forms were obtained. The study protocol was approved by the Ethics Committee of İnönü University (Date: 04.02.2009 Decision No: 242972). All procedures performed in this study were done according to the ethical standards of the 1964 Helsinki declaration.

Patients who applied to our cardiology department with chest pain and underwent elective coronary angiography between Dec 2008-2009 were randomized. In patients with normal coronary angiography results, CIMTs of 30 patients who had no additional disease and only known hypertension and were under antihypertensive treatment for at least one year were measured. Those with rheumatic heart disease, kidney failure, hypertrophic cardiomyopathy, Diabetes Mellitus, chronic liver disease, smokers, people with heart failure, people younger than 18 years and older than 75 years were excluded from the study. The blood pressures of the patients who received antihypertensive treatment during hospitalization for coronary angiography were measured every two hours, those with an arithmetic mean above 140/90 mmHg were excluded from the study. As a result, a total of 32 patients with hypertension who had no disease and whose blood pressure was

regulated by antihypertensive therapy were included in the study. Coronary angiography was performed in all patients with 6 french right-left diagnostic cardiac catheters, using the Judkins technique, with a Philips Medical Systems Integris H 3500–5000 device. CIMT measurements were taken with a high-resolution HDI- 5000; ATL (Borhell, Washington- USA) Transthoracic Echocardiography instrument by taking advantage of the characteristic echogenicity of lumen, intima, media, adventitia surfaces in an area of 1 cm of the posterior (far) wall of the internal carotid artery (Figure 1).

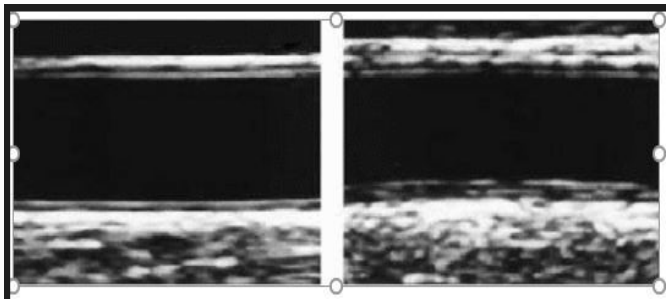

Figure 1: Ultrasonographic representation of intima-media thickness. On the left is the normal intima media thickness of the carotid artery ; on the right, the carotid artery with an increased intima-media thickness.

Statistical Analysis

Continuous variables were expressed as mean±standard deviation and categorical variables as percentage (%). Student t test was used to compare continuous variables, and Chi-square test was used to compare categorical data. A p value of <0.05 was accepted as statistical significance. Statistical analysis was performed using the SPSS statistical software (version 10.0, SPSS, Chicago, IL, USA) program.

RESULTS

In the study, 32 (16 men, 16 women) subjects without hypertension, 30 (15 men, 15 women) subjects with hypertension, who applied to İnönü University Faculty of Medicine Turgut Ozal Medical Center Cardiology Department with atypical chest pain, underwent carotid doppler in the echocardiography unit after elective coronary angiography were taken. The ages of all subjects in the study were between 18 and 75, and the mean age was 51.2±8.3 years in the hypertension group and 52.4±9.0 in the control group without hypertension. Demographic, laboratory and CIMT values of the patients are presented in Table 1.

Table 1. Laboratory values, CIMT measurements and demographic characteristics of the patients

	Hypertension group	Control group	P value
Age	51.2±8.3	52.4±9	0.664
Sex (male)	%50	%50	-
BMI	28.2±4.3	26.7±6.5	0.021
Smoker	%65	%60	0.321
Creatinine	0.71±0.18	0.66±0.23	0.211
Total Cholesterol	223±41	209±38	0.344
LDL	123±31	120±28	0.547
HDL	34±12	42±13	0.495
Triglycerides	230±105	199±85	0.197
Glucose	90±11	87±9	0.368
SBP	124±9	120±8	0.448
DBP	79±6	81±7	0.551
TSH	2.18±0.99	2.04±0.88	0.775
CIMT	5.3±1.3	5.3±1.1	0.817

BMI: Body mass index, LDL: Low density lipoprotein, HDL: High density lipoprotein, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, TSH: Thyroid stimulating hormone, CIMT: Carotid intima media thickness

DISCUSSION

This study has shown us that in a group of patients who have been proven not to have coronary artery disease by angiography, the CIMT in patients whose hypertension is under control by treatment is similar compared to those in patients without hypertension. It has been proven in studies that hypertension causes subclinical atherosclerosis with end organ damage.[2,3,11] In addition, studies have shown that hypertension is associated with increased CIMT.[9,10,12] In this study, we found that there was no statistically significant difference between CIMT in patients whose blood pressure was regulated with antihypertensive treatment for more than one year compared to those in the normotensive patient group. This, in turn, suggests to us the preventive effect of effective antihypertensive therapy on the progression of atherosclerosis. In a meta-analysis by Wang et al.[8], similar to our study, they showed that the progression of the atherosclerotic process slowed down with antihypertensive treatment using CIMT measurement. One possible mechanism explaining our findings is that effective anhipertensive treatment may decrease the progression of arterial disease at the level of the carotid arteries by reducing the stress on the vascular wall could lead to structural changes.

Limitations

There are two important major limitations of the study. First, we did not evaluate the efficacy of antihypertensive therapy by measuring 24-hour ambulatory blood pressure, but instead based on blood pressure values measured at two-hour intervals during hospitalization during coronary angiography. Secondly, medial hypertrophy and atherosclerotic background cannot be distinguished in CIMT measured by ultrasound method.

CONCLUSION

As a result, no increase in CIMT was observed in patients with treatment-regulated hypertension without coronary disease when compared to healthy individuals. This makes us think that effective antihypertensive treatment slows down the atherosclerotic process.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was carried out with the permission of the Ethics Committee of İnönü University (Date: 04.02.2009 Decision No: 242972).

Informed Consent

All patients signed and free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

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.

REFERENCES

1. Baysal SS, Pirat B, Okyay K, et al. Treatment-associated change in apelin concentration in patients with hypertension and its relationship with left ventricular diastolic function. *Anat J Cardiol*. 2017;17(2):125.
2. Wright Jr JT, Whelton PK, Reboussin DM. A randomized trial of intensive versus standard blood-pressure control. *New Eng J Med*. 2016;374(23):2294.
3. Naqvi TZ, Lee MS. Carotid intima-media thickness and plaque in cardiovascular risk assessment. *JACC: Cardiovasc Imaging*. 2014;7(10):1025-1038.
4. Şafak KY, Akçiçek M. Kardiyovasküler hastalık risk faktörleri ve karotis arter intima media kalınlığı arasındaki ilişki. *Kafkas Tıp Bil Derg*. 2015;5(2):54-59.
5. Besir FH, Yazgan S, Celbek G, et al. Normal values correlates' of carotid intima-media thickness and affecting parameters in healthy adults/Saglikli eriskinlerde karotis intima-media kalinliginin normal degerleri ve etkileyen parametreler. *Anat J Cardiol*. 2012;12(5):427-434.
6. Youn YJ, Lee NS, Kim JY, et al. Normative values and correlates of mean common carotid intima-media thickness in the Korean rural middle-aged population: the atherosclerosis risk of rural areas in Korea general population (ARIRANG) study. *J Korean Med Sci*. 2011;26(3):365-371.
7. Demirci R, Sevinç C. Kronik böbrek hastalığında ateroskleroz, karotis intima media kalınlığı ve lipid profili arasındaki ilişki. *İst Kanuni Sultan Süleyman Tıp Derg*. 2021;13(2):129-135.
8. Wang JG, Staessen JA, Li Y, et al. Carotid intima-media thickness and antihypertensive treatment: a meta-analysis of randomized controlled trials. *Stroke*. 2006;37(7):1933-1940.
9. Manios E, Tsivgoulis G, Koroboki E, et al. Impact of prehypertension on common carotid artery intima-media thickness and left ventricular mass. *Stroke*. 2009;40(4):1515-1518.
10. Ferreira JP, Girerd N, Bozec E, et al. Intima–media thickness is linearly and continuously associated with systolic blood pressure in a population-based cohort (STANISLAS Cohort Study). *J Am Heart Assoc*. 2016;5(6):e003529.
11. McEvoy JW, Daya N, Rahman F, et al. Association of isolated diastolic hypertension as defined by the 2017 ACC/AHA blood pressure guideline with incident cardiovascular outcomes. *JAMA*. 2020;323(4):329-338.
12. Puato M, Palatini P, Zanardo M, et al. Increase in carotid intima-media thickness in grade I hypertensive subjects: white-coat versus sustained hypertension. *Hypertension*. 2008;51(5):1300-1305.

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May high blood viscosity predict cardiac involvement in COVID-19 patients?

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ABSTRACT

Aims: Assessing the effects of whole blood viscosity (WBV) on prognosis and deterioration in cardiac parameters in COVID-19 patients after recovery using cardiac magnetic resonance imaging (CMRI) and echocardiography is the purpose of this study.

Methods: The study involved 70 patients. Patients who had COVID-19 pneumonia were admitted to the hospital. All patients met the eligibility criteria if they remained symptom-free of respiratory and cardiac symptoms and had negative swab test results at the end of the isolation period, for at least two weeks following the positive swab test result. Transthoracic echocardiography was performed within 24 hours prior to CMRI. WBV was measured in centipoises (cP) at 208 seconds-1 shear rate. The median value of WBV was calculated for the entire study population, which was subsequently utilized to divide the population into two subgroups. These were designated as high WBV and low WBV subgroups.

Results: Elevated levels of hemoglobin, hematocrit, total protein, C-reactive protein, D-dimer, and fibrinogen were observed in individuals with high WBV. Conversely, TAPSE, S', and FAC were notably reduced in those with high WBV. Notably, CMRI revealed significant increases in native T1, native T2 mapping signal, and extracellular volume among patients with high WBV. Furthermore, in individuals with high WBV, there was a significant decrease in right ventricle stroke volume and right ventricle ejection fraction, accompanied by a notable increase in right ventricle end-systolic volume.

Conclusion: WBV values measured during hospital admission may have early and late prognostic importance for COVID-19 infection.

Keywords: Blood viscosity, COVID-19, cardiac MRI, echocardiography

INTRODUCTION

The novel severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) was first appeared in China in 2019 and quickly escalated into a worldwide pandemic. The coronavirus disease 2019 (COVID-19) pandemic has been a major cause of morbidity and mortality on a global scale. Involvement of the respiratory system caused by COVID-19 can range from mild flu to severe pneumonia. Case reports have demonstrated that COVID-19 is also capable of causing cardiac involvement and damage. Proposed pathophysiological mechanisms of cardiac injury include endothelitis, plaque rupture or erosion, stent thrombosis, cardiac stress induced by high cardiac output, myocarditis, heart failure, and arrhythmia.[1,2]

Myocarditis, which encompasses pericarditis, myocardial edema, and myocardial fibrosis, carries a significant risk of adverse outcomes and a poor prognosis.[3-6] The gold standard diagnostic instrument for identifying myocarditis symptoms including inflammation, edema, and fibrosis is cardiac magnetic resonance imaging (CMRI). By employing T1, T2, and extracellular volume (ECV) mapping techniques, CMRI assesses cardiac structure and function and provides a quantitative evaluation of myocardial fibrosis and edema.[7-9] A small study examined patients who recovered from COVID-19 but continued to exhibit cardiac symptoms. In 58% of the patients, CMRI with late gadolinium enhancement (LGE)

detected persistent cardiac involvement, including fibrosis and myocardial edema.[10] An additional investigation carried out on patients who had recently recovered from COVID-19 and did not exhibit any cardiac symptoms revealed that 78% of the patients had cardiac involvement, while 60% of the patients had persistent myocardial involvement.[11]

The acute respiratory distress syndrome (ARDS) is a clinical manifestation of respiratory failure in patients inflicted with COVID-19. Diffuse alveolar injury, edema, and fibrosis are all components of ARDS. Pulmonary thrombosis, which may involve pulmonary microvessels and lead to pulmonary hypertension, is another potential consequence of ARDS. The right ventricle (RV) afterload may then increase, leading to the development of RV dysfunction.[12,13]

It has been established that whole blood viscosity (WBV) is a significant cardiovascular risk factor and that it fluctuates in a variety of cardiovascular disorders. Patients with acute coronary syndrome undergoing percutaneous coronary intervention and a high WBV are associated with adverse outcomes, including an increased incidence of no-reflow phenomenon, stent thrombosis, and apical thrombus. Patients suffering from acute pulmonary embolism and stroke may also experience negative outcomes.[14-19] There is currently no research in the literature to assess the effect of WBV on the prognosis in patients with COVID-19 and its relationship with the deterioration in cardiovascular parameters after the patients recover. The aim of this study is to evaluate the effects of WBV on prognosis and deterioration in cardiac parameters after recovery in COVID-19 patients, with cardiac MRI and echocardiography which are sensitive tools in the detection of cardiac involvement in patients with COVID-19.

METHODS

Ethics

The research received ethical approval from the local committee (Date: 15.03.2021, Decision No: HNEAH-KAEK 2021/KK/75). Prior to participation, all individuals gave their informed assent. The research was conducted in adherence to ethical guidelines, with particular consideration given to the principles outlined in the Declaration of Helsinki.

Study Design and Study Population

A prospective observational cohort study is being conducted. The research was conducted from April to June of 2020. Seventy patients were included in the study whose upper respiratory tract swab tests were positive for SARS-CoV-2 via reverse transcription-polymerase chain reaction. Patients had no history of chronic disease. The patients were initially committed to the hospital due to COVID-19 pneumonia; however, they were ultimately discharged without requiring non-invasive mechanical ventilation support or further observation in the intensive care unit. All patients were deemed eligible for inclusion in the study fourteen days, provided that they had resolved respiratory symptoms, lacked cardiac symptoms, and had obtained negative swab test results by the end of the isolation period. Patients who declined to participate or who had contraindications for a CMRI were excluded from the study. Prior to CMRI, transthoracic echocardiography (TTE) was conducted within twenty-four hours. Upon the initial admission to the hospital, clinical and demographic information and blood test results were collected. All blood values were determined using conventional, standard procedures. Using the following equation, WBV was calculated in centipoises (cP) at a shear rate of 208 seconds-1: WBV=0.12 X hematocrit (%)+0.17 X plasma proteins (g/L). The median value of WBV was determined for the entire study population, and on the basis of that value, the population of the study was divided into two sub-groups. These were designated as high WBV and low WBV subgroups. The clinical demographic characteristics, biochemical, CMRI and TTE parameters of these two subgroups were compared.

An Echocardiographic Examination

TTE was conducted utilizing a GE Vivid E95 device (Vingmed Ultrasound, Horten, Norway; GE Healthcare) equipped with an M5S probe operating within the frequency range of 1.5-4.6 MHz. A thorough conventional transthoracic echocardiographic examination was conducted in accordance with the current guidelines of the American Society of Echocardiography to assess the structure and function of the heart20. For the computation of the left ventricular ejection fraction (LVEF), the modified biplane Simpson method was utilized. Right ventricular fractional area change (FAC) is calculated using a focused apical view of the RV to determine the area difference between the end-diastolic and end-systolic regions.

Cardiac Magnetic Resonance Imaging

A 1.5-T MR scanner (Signa Explorer; GE Medical Systems, Milwaukee, WI, USA) was utilized to conduct the CMRI; it was equipped with a 32-channel phased-array abdominal coil and electrocardiographic gating. Prior to the CMRI, no intravenous sedation was administered. Breathing instructions were provided to all patients prior to the scan. Two technologists and a radiologist and cardiologist with a combined ten years of experience conducted each examination. The acquisition of sagittal, coronal, and axial localizations within the thorax is achieved via an axial black blood stack employing rapid spin echo to visualize the anatomy of the cardiothorax and steady-state free precession. Then, in order to evaluate cardiac function and volume, a steady-state free precession cine film sequence was obtained, which was balanced along the short and long axes. After acquiring each image set using retrospective gating, twenty cardiac phases were reconstructed. Left ventricular tissue was characterised using T1 mapping with long-T1 5(3)3-shortened modified look-locker inversion recovery and T2 mapping with T2-prepared balanced steady-state free precession. Following the administration of the gadolinium-based contrast agent (0.2 mmol/kg), LGE imaging was performed using a phase-sensitive inversion recovery sequence. The post-contrast T1 mapping sequence was acquired 5 minutes after contrast injection. The late gadolinium enhancement sequence for myocardial scar imaging was acquired 10 and 15 minutes after contrast injection. All sequences were acquired over three segments of the short axis of the left ventricle, from its base to its midpoint and apex.

Each CMRI was interpreted by an experienced cardiologist and an experienced radiologist. CMR image analysis of left ventricular morphology and function was performed

using Circle cvi42 software (Circle Cardiovascular Imaging, Calgary, Canada). Automatic calculation of left and right ventricular volume/function parameters was performed using endocardial and epicardial contours. The quality of late gadolinium enhancement images was assessed visually. Endocardial and epicardial contouring of basal, midventricular and apical short-axis slices was performed for native, post-contrast T1 and T2 mapping. Contamination from blood pools was avoided throughout the epicardial and endocardial boundary contouring process.

Statistical Analysis

The statistical analysis was performed using IBM SPSS Statistics version 25 (IBM Corp, Armonk, NY). Categorical variables are presented as frequencies and percentages, while continuous variables are reported as means with corresponding standard deviations. Group comparisons for categorical variables were conducted using the Chi-squared test, and for continuous variables with a normal distribution, Student's t-test was employed. The Mann-Whitney U test was utilized for continuous variables without a normal distribution. A p-value less than 0.05 was considered statistically significant.

RESULTS

70 patients (mean age 42.7±10.4 years; 57.1% male) participated in the investigation. 16.62 was the median value of the WBV. On the basis of their WBV value, patients were separated into two groups: those with a low WBV (n=34 patients) and those with a high WBV (n=36 patients). Table 1 presents the baseline clinic and demographic characteristics, and the laboratory findings, of the patients. The characteristics of patients at baseline did not vary between the study groups. Significantly elevated levels of hemoglobin, hematocrit, total protein, C-reactive protein, D-dimer, and fibrinogen were observed in patients with a high WBV. (12.9±1.5 vs 13.8±1.9 p=0.036, 37.9±4.4 vs 41.3±4.7 p=0.003, 67.5±3.8 vs 75.2±3.5 p<0.001, 1.08±1.39 vs 8.1±4.3 p<0.001, 0.31±0.16 vs 1.04±0.72 p<0.001, 418.9±90.7 vs 653.3±103.1 p<0.001 respectively) The rest of the laboratory results were comparable between the two study groups.

The study participants' TTE results are presented in Table 2. Patients who had a high WBV exhibited substantially reduced values for TAPSE, S', and FAC. (26.6±1.1 vs 22.7±1.5 p<0.001, 0.19±0.03 vs 0.12±0.03 p<0.001, 42.5±6.3 vs 34.8±0.9 p<0.001 respectively) Other echocardiographic parameters were not significantly different between study groups.

On CMRI, native T1, native T2 mapping signal and extracellular volume (ECV) were significantly increased in patients with high WBV. (1025.9±3.2 vs 1027.0±0.0 p=0.035, 47.0±2.4 vs 52.6±2.9 p<0.001, 19.62±5.81 vs 38.22±14.64 respectively) Patients with high WBV had right ventricle stroke volume (RVSV) and right ventricle ejection fraction (RVEF) were significantly lower (80.9±14.2 vs 72.9±15.5 p<0.001, 57.9±3.4 vs 49.6±2.5 p<0.001 respectively), but right ventricle end-systolic volume (RVESV) was significantly higher (59.3±13.0 vs 73.9±16.2 p<0.001) (Table 3).

Two patients in the low WBV group and four patients in the high WBV group exhibited LGE (Figure).

Table 1. Baseline characteristics of patients, and laboratory results

	Low WBV group (n=34)	High WBV group (n=36)	p value
Patient characteristics			
Age (years)	43.9±10.8	41.5±10.1	0.338
Gender			0.241
Male (n, %)	17 (%50)	23 (63.9%)	
Female (n, %)	17 (%50)	13 (36.1%)	
BMI (kg/m²)	26.8±3.5	25.9±3.9	0.314
SBP (mmHg)	117.6±13.6	120.5±11.0	0.336
DBP (mmHg)	75.2±7.0	77.6±7.6	0.180
Laboratory Data			
Hemoglobin, g/dl	12.9±1.5	13.8±1.9	0.036
Hematocrit (%)	37.9±4.4	41.3±4.7	0.003
WBV	15.68±0.63	17.38±0.49	<0.001
WBC (10^3/µl)	8.0±4.4	7.5±2.6	0.543
Neutrophil (10^3/µl)	5.6±4.0	5.2±2.6	0.618
Lymphocyte (10^3/µl)	1.8±0.9	1.7±0.8	0.480
Platelet (10^3/µl)	318.2±126.8	298.0±97.64	0.474
Serum creatinine (mg/dl)	0.78±0.12	0.80±0.15	0.668
Glucose (mg/dl)	144.8±71.9	156.1±93.0	0.618
Sodium (mEq/L)	133.7±24.8	138.2±2.9	0.315
Potassium (mEq/L)	4.5±0.4	4.5±0.4	0.555
AST (unit/L)	32.2±21.1	28.8±14.0	0.468
ALT (unit/L)	45.1±47.0	52.0±51.8	0.728
Troponin-T (ng/L)	0.70±1.34	0.87±1.48	0.280
Albumin (g/L)	39.5±4.2	38.7±4.6	0.572
Total protein (g/L)	67.5±3.8	75.2±3.5	<0.001
CRP (mg/dL)	1.08±1.39	8.1±4.3	<0.001
D-dimer (ug/ml)	0.31±0.16	1.04±0.72	<0.001
Fibrinogen (mg/dl)	418.9±90.7	653.3±103.1	<0.001
Prokalsitonin (ng/ml)	0.049±0.051	0.380±1.52	0.377
LDH (unit/L)	247.7±65.0	260.6±71.2	0.475
TSH (mlU/L)	1.5±0.7	1.9±0.9	0.294

BMI: Body mass index, SBP: Systolic blood pressure, DBP: Diastolic blood pressure (mmHg), WBV: Whole blood viscosity, WBC: White blood cell count, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, CRP: C-reactive protein, LDH: Lactate dehydrogenase, TSH: Thyroid stimulating hormone

Table 2. Echocardiography results

	Low WBV group (n=34)	High WBV group (n=36)	p value
Left ventricular end-diastolic diameter (mm)	45.2±2.9	45.6±3.2	0.495
Left ventricular end-systolic diameter (mm)	30.2±2.0	30.8±2.7	0.379
Left atrium anterior-posterior diamater (mm)	30.4±4.1	31.2±4.0	0.327
Left ventricular ejection fraction (%)	57.2±5.2	55.6±4.7	0.089
interventricular septum thickness (mm)	9.3±1.1	9.3±1.1	0.765
posterior wall thickness (mm)	9.3±1.0	9.3±1.1	0.736
E/A ratio	1.15±0.64	1.14±0.60	0.690
Em (cm/s)	0.16±0.05	0.15±0.05	0.265
Am (cm/s)	0.14±0.04	0.12±0.04	0.091
IVRT (ms)	176.9±39.1	185.3±38.4	0.535
IVCT (ms)	74.7±6.8	75.3±8.5	0.766
DT (ms)	176.9±39.1	185.3±38.4	0.535
TAPSE (mm)	26.6±1.1	22.7±1.5	<0.001
S' (cm/s)	0.19±0.03	0.12±0.03	<0.001
FAC (%)	42.5±6.3	34.8±0.9	<0.001

E: mitral inflow early diastolic velocity, A: mitral inflow late diastolic velocity, Em: mitral inflow early diastolic tissue velocity, Am: mitral inflow late diastolic tissue velocity, IVRT: isovolumic relaxation time, IVCT: the isovolumic contraction time, DT: left ventricular deceleration time, TAPSE: tricuspid annular plane systolic excursion S': TDI-derived tricuspid lateral annular systolic velocity wave, FAC: right ventricular fractional area change

Table 3. Cardiac magnetic resonance imaging results

	Low WBV group (n=34)	High WBV group (n=36)	p value
Right ventricle end-diastolic volume	140.2±25.6	149.6± 35.0	0.240
Right ventricle end systolic volume	59.3±13.0	73.9±16.2	<0.001
Right ventricle stroke volume	80.9±14.2	72.9±15.5	<0.001
Right ventricle ejection fraction	57.9±3.4	49.6±2.5	<0.001
T1 map native	1025.9±3.2	1027.0±0.000	0.035
T2 map native	47.0±2.4	52.6±2.9	<0.001
Extracellular volume	19.62±5.81	38.22±14.64	<0.001

WBV: Whole blood viscosity

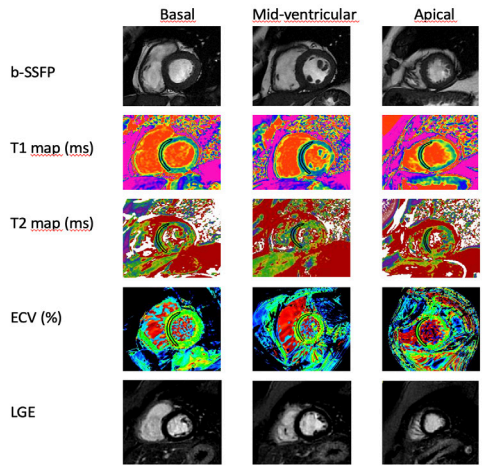

Figure. A 37-year-old male patient diagnosed with COVID-19 underwent cardiac MRI. The same slices of short axis view on basal, mid-ventricular, and apical segment (left to right) for balanced steady state free precession, T1 mapping, T2 mapping, ECV map and LGE images (up-to-down). T1, T2 and ECV mapping values showed increased values for entire ventricle (b-SSFP, balanced steady state free precession; ECV, extracellular volume; LGE, late gadolinium enhancement)

DISCUSSION

COVID-19 has the potential to significantly affect the cardiovascular systems of patients. Morbidity and mortality are more likely to occur in patients with COVID-19 who also have preexisting cardiovascular disease (e.g., coronary artery disease, arterial hypertension, stroke). COVID-19 has been linked to a multitude of cardiovascular complications. COVID-19 has the potential to induce a heightened inflammatory response, upregulate cytokine secretion, and induce a cytokine storm. COVID-19 can induce endotheliitis via vascular inflammation, myocarditis, malign arrhythmias, venous thromboembolism. It is of the utmost importance to comprehend the impact of COVID-19 on the cardiovascular system in order to offer comprehensive medical care to COVID-19 patients and to predict prognoses.[21]

Seventy patients with pulmonary involvement who tested positive for SARS-CoV-2 on swab tests but did not require intensive care unit follow-up or non-invasive mechanical ventilation were included in the study. Patients met the inclusion criteria for the study when they had recovered from COVID-19 infection for a minimum of two weeks. Blood viscosity values were determined using blood samples collected at the time of initial hospital admission. Two categories were formed from the study population based on the median value of WBV. The designations for these two categories are high WBV and low WBV. In addition to CMRI and TTE findings, acute phase reactants over the duration of the disease were compared between these two groups. This is the first report to suggest that cardiac involvement in patients with COVID-19 may be predicted by a high WBV.

Due to the observation of WBV fluctuations in a variety of cardiovascular disorders, it is possible to classify WBV as a significant cardiovascular risk factor. Moreover, high WBV could potentially serve as a risk factor for coronary artery disease. This can occur either indirectly, via its correlation with significant risk factors like arterial hypertension, or directly, by augmenting the resistance generated by the extent of coronary artery constriction. Either way, this would have detrimental consequences for clinical manifestations and maximal myocardial oxygen delivery. Additionally, a high WBV can elevate the probability of stent thrombosis, intraventricular thrombus formation, and no-reflow.[14,22-24] Furthermore, elevated WBV impairs cerebral blood flow and elevates the susceptibility to stroke.[25,26]

An increase in native T2 mapping signal was indicative of myocardial edema, whereas an increase in native T1 mapping signal was indicative of myocardial interstitial fibrosis, according to previous research. The ECV values and native T1 and T2 mapping signals were substantially greater in patients with a high WBV, according to this study. Acute phase reactants, including fibrinogen, D-dimer, and C-reactive protein, were substantially elevated in patients with high WBV. Elevated levels of acute phase reactants may serve as an indicator of an intensified inflammatory response within the body. As a result, our study suggests that diffuse myocardial edema and fibrosis may exist in patients with high WBV.[27-29]

Prior research has demonstrated that RV function may be negatively impacted by ARDS. COVID-19 can induce ARDS

due to the fact that the lungs are among the primary organs affected by SARS-CoV-2; thus, RV may be more susceptible to injury than LV. ARDS can impair the pulmonary circulation through mechanisms other than alveolar injury. These include hypoxic pulmonary vasoconstriction, secretion of vasoconstrictive mediators, extrinsic vascular compression due to interstitial oedema and vascular remodelling. Ultimately, RV failure may result from increased pulmonary vascular resistance and right ventricular afterload.[12,30-32] Patients who were admitted to our study with COVID-19 pneumonia did not develop acute respiratory distress syndrome (ARDS) and did not require follow-up in the intensive care unit. Additionally, we discovered that the RVEDV and RVESV values were greater in the high WBV group on CMRI, whereas the RVEF values were substantially lower. Additionally, RV FAC, TAPSE, and RV S' values, which reflect echocardiographic RV functions, were substantially reduced in the group with a high WBV. An elevation in RV afterload may also occur in patients with COVID-19 pneumonia due to vasoconstriction induced by hypoxia and inflammatory mediators.

Our study demonstrated that WBV measurement at the first hospital admission is associated with higher levels of acute phase reactants values during the course of COVID-19 infection and with impaired right ventricular function parameters with higher T1, T2 and ECV values indicating extensive myocardial fibrosis and oedema when assessed by CMRI and echocardiography after the disease has healed.

Limitations

The results might not be suitable for patients who are younger than 18 years old. Furthermore, these results do not take into account patients with acute COVID-19 infection or those who are asymptomatic at the time of infection.

CONCLUSION

As a result, WBV values measured during hospital admission may have early and late prognostic importance and high WBV may predict cardiac involvement for COVID-19 infection.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was carried out with the permission of Haydarpaşa Numune Training and Research Hospital Clinical Researches Ethics Committee (Date: 15.03.2021, Decision No: HNEAH-KAEK 2021/KK/75).

Informed Consent

All patients signed and free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

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.

REFERENCES

1. Shi S, Qin M, Shen B, et al. Association of cardiac injury with mortality in hospitalized patients with COVID-19 in Wuhan, China. *JAMA Cardiol*. 2020;5(7):802-810. doi: 10.1001/jamacardio.2020.0950
2. Guo T, Fan Y, Chen M, et al. Cardiovascular implications of fatal outcomes of patients with coronavirus disease 2019 (COVID-19). *JAMA Cardiol*. 2020;5(7):811-818. doi: 10.1001/jamacardio.2020.1017
3. Inciardi RM, Lupi L, Zaccone G, et al. Cardiac involvement in a patient with coronavirus disease 2019 (COVID-19). *JAMA Cardiol*. 2020;5(7):819-824. doi: 10.1001/jamacardio.2020.1096
4. Chen L, Li X, Chen M, Feng Y, Xiong C. The ACE2 expression in human heart indicates new potential mechanism of heart injury among patients infected with SARS-CoV-2. *Cardiovasc Res*. 2020;116(6):1097-1100. doi: 10.1093/cvr/cvaa078
5. Varga Z, Flammer AJ, Steiger P, et al. Endothelial cell infection and endotheliitis in COVID-19. *Lancet*. 2020;395(10234):1417-1418. doi: 10.1016/S0140-6736(20)30937-5
6. Zheng YY, Ma YT, Zhang JY, Xie X. COVID-19 and the cardiovascular system. *Nat Rev Cardiol*. 2020;17(5):259-260. doi: 10.1038/s41569-020-0360-5
7. Friedrich MG, Sechtem U, Schulz-Menger J, et al. International Consensus Group on Cardiovascular Magnetic Resonance in Myocarditis. Cardiovascular magnetic resonance in myocarditis: A JACC White Paper. *J Am Coll Cardiol*. 2009;53(17):1475-1487. doi: 10.1016/j.jacc.2009.02.007
8. Ferreira VM, Schulz-Menger J, Holmvang G, et al. Cardiovascular magnetic resonance in nonischemic myocardial inflammation: expert recommendations. *J Am Coll Cardiol*. 2018;72(24):3158-3176. doi: 10.1016/j.jacc.2018.09.072
9. Kammerlander AA, Marzluf BA, Zotter-Tufaro C, et al. T1 Mapping by CMR imaging: from histological validation to clinical implication. *JACC Cardiovasc Imaging*. 2016;9(1):14-23. doi: 10.1016/j.jcmg.2015.11.002
10. Huang L, Zhao P, Tang D, et al. Cardiac involvement in patients recovered from COVID-2019 identified using magnetic resonance imaging. *JACC Cardiovasc Imaging*. 2020;13(11):2330-2339. doi: 10.1016/j.jcmg.2020.05.004
11. Puntmann VO, Carerj ML, Wieters I, et al. Outcomes of cardiovascular magnetic resonance imaging in patients recently recovered from coronavirus disease 2019 (COVID-19). *JAMA Cardiol*. 2020;5(11):1265-1273. doi: 10.1001/jamacardio.2020.3557
12. Paternoster G, Bertini P, Innelli P, et al. Right ventricular dysfunction in patients with COVID-19: a systematic review and meta-analysis. *J Cardiothorac Vasc Anesth*. 2021;35(11):3319-3324. doi: 10.1053/j.jvca.2021.04.008
13. Lazzeri C, Bonizzoli M, Batacchi S, Peris A. Echocardiographic assessment of the right ventricle in COVID -related acute respiratory syndrome. *Intern Emerg Med*. 2021;16(1):1-5. doi:10.1007/s11739-020-02494-x
14. Ekizler FA, Cay S, Tak BT, et al. Usefulness of the whole blood viscosity to predict stent thrombosis in ST-elevation myocardial infarction. *Biomark Med*. 2019;13(15):1307-1320. doi: 10.2217/bmm-2019-0246
15. Yildirim A, Kucukosmanoglu M, Koyunsever NY, Cekici Y, Belibagli MC, Kilic S. Relationship between blood viscosity and no-reflow phenomenon in ST-segment elevation myocardial infarction performed in primary percutaneous coronary interventions. *Biomark Med*. 2021;15(9):659-667. doi: 10.2217/bmm-2020-0772
16. Çınar T, Şaylık F, Akbulut T, et al. The association between whole blood viscosity and high thrombus burden in patients with non-ST elevation myocardial infarction. *Kardiol Pol*. 2022;80(4):429-435. doi: 10.33963/KP.a2022.0043
17. Tekin Tak B, Ekizler FA, Cay S, et al. Relationship between apical thrombus formation and blood viscosity in acute anterior myocardial infarction patients. *Biomark Med*. 2020;14(3):201-210. doi: 10.2217/bmm-2019-0483
18. Aksu E. Is it possible to estimate the mortality risk in acute pulmonary embolism by means of novel predictors? A retrospective study. *Turkish J Vasc Surg*. 2021;30(1):27–34. doi: 10.9739/tjvs.2021.836
19. Antonova N, Velcheva I. Hemorheological disturbances and characteristic parameters in patients with cerebrovascular disease. *Clin Hemorheol Microcirc*. 1999;21(3-4):405-408.
20. Mitchell C, Rahko PS, Blauwet LA, et al. Guidelines for performing a comprehensive transthoracic echocardiographic examination in adults: recommendations from the American Society of Echocardiography. *J Am Soc Echocardiogr*. 2019;32(1):1-64. doi: 10.1016/j.echo.2018.06.004
21. Chang WT, Toh HS, Liao CT, Yu WL. Cardiac involvement of COVID-19: a comprehensive review. *Am J Med Sci*. 2021;361(1):14-22. doi:10.1016/j.amjms.2020.10.002

22. Ceyhun G, Birdal O. Relationship between whole blood viscosity and lesion severity in coronary artery disease. *Int J Angiol*. 2021;30(2):117-121. doi:10.1055/s-0040-1720968
23. Lowe GD, Drummond MM, Lorimer AR, et al. Relation between extent of coronary artery disease and blood viscosity. *Br Med J*. 1980;280(6215):673-674. doi: 10.1136/bmj.280.6215.673
24. Cekirdekci EI, Bugan B. Whole blood viscosity in microvascular angina and coronary artery disease: significance and utility. *Rev Port Cardiol (English Edition)*. 2020;39(1):17-23. doi: 10.1016/j.repc.2019.04.008
25. Hudak ML, Koehler RC, Rosenberg AA, Traystman RJ, Jones Jr MD. Effect of hematocrit on cerebral blood flow. *Am J Physiol*. 1986;251(1):H63-H70. doi: 10.1152/ajpheart.1986.251.1.H63
26. Uzunget SB, Sahin KE. Another possible determinant for ischemic stroke with nonvalvular atrial fibrillation other than conventional oral anticoagulant treatment: the relationship between whole blood viscosity and stroke . *J Stroke Cerebrovasc Dis*. 2022;31(9):106687. doi: 10.1016/j.jstrokecerebrovasdis.2022.106687
27. Li H, Zhu H, Yang Z, Tang D, Huang L, Xia L. Tissue characterization by mapping and strain cardiac MRI to evaluate myocardial inflammation in fulminant myocarditis. J Magn Reson Imaging. 2020;52(3):930-938. doi:10.1002/jmri.27094
28. Dolan RS, Rahsepar AA, Blaisdell J, et al. Multiparametric cardiac magnetic resonance imaging can detect acute cardiac allograft rejection after heart transplantation. *JACC Cardiovasc Imaging*. 2019;12(8 Pt 2):1632-1641. doi: 10.1016/j.jcmg.2019.01.026
29. Hinojar R, Varma N, Child N, et al. T1 mapping in discrimination of hypertrophic phenotypes: hypertensive heart disease and hypertrophic cardiomyopathy: findings from the international T1 multicenter cardiovascular magnetic resonance study. *Circ Cardiovasc Imaging*. 2015;8(12):e003285. doi: 10.1161/CIRCIMAGING.115.003285
30. Park JF, Banerjee S, Umar S. In the eye of the storm: the right ventricle in COVID-19. *Pulm Circ*. 2020;10(3):2045894020936660. doi:10.1177/2045894020936660
31. Grasselli G, Tonetti T, Protti A, et al. Pathophysiology of COVID-19-associated acute respiratory distress syndrome: a multicentre prospective observational study. *Lancet Respir Med*. 2020;8(12):1201-1208. doi: 10.1016/S2213-2600(20)30370-2
32. Bleakley C, Singh S, Garfield B, et al. Right ventricular dysfunction in critically ill COVID-19 ARDS. *Int J Cardiol*. 2021;327:251-258. doi: 10.1016/j.ijcard.2020.11.043

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Current approach to venous catheterization in hemodialysis patients and important points

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ABSTRACT

Hemodialysis procedure is commonly performed in hospitals. It is typically conducted due to renal dysfunction, elevated levels of urea and serum creatinine, or excessive volume overload. Additionally, emergency hemodialysis may be implemented in cases of certain poisonings. Emergency hemodialysis procedures are usually performed through a temporary venous catheter. The majority of patients undergoing continuous hemodialysis therapy access dialysis through arteriovenous fistula and permanent hemodialysis catheter. This procedure can be carried out with a very low risk of complications when performed by experienced individuals.

Keywords: Venous catheterization, catheterization, hemodialysis

INTRODUCTION

The prevalence of chronic kidney disease (CKD) is increasing worldwide, driven by the rising rates of obesity, diabetes, extended life expectancy, and the surge in chronic conditions such as hypertension. This trend has led to a growing frequency of hemodialysis (HD) requirements, with HD also being utilized in cases of certain poisonings. While peritoneal dialysis (PD) is common in some countries (such as Mexico), hemodialysis remains more prominent in many others. In our country, there are approximately 80,000 dialysis patients, with around 70,000 receiving HD treatment. The number of HD patients in our country was around 60,000 in 2017, and as of 2023, this figure has exceeded 70,000 (Figure 1). Emergency dialysis treatments, particularly for patients undergoing dialysis for the first time, are predominantly conducted through venous catheterization. The majority of patients undergoing continuous HD therapy receive dialysis through arteriovenous fistula (AVF) and permanent HD catheter (Figure 2). Venous catheterization procedures, when performed by experienced hands, carry a very low risk of complications.[1-4]

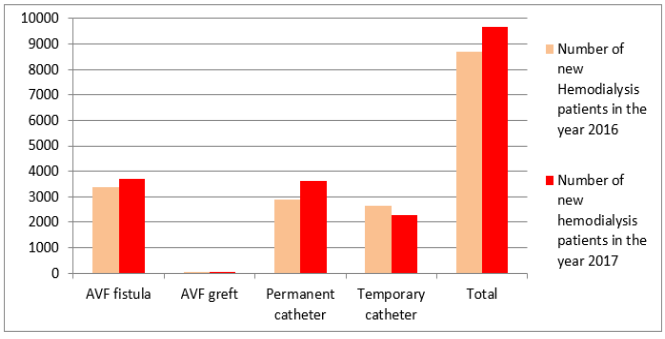

Figure 1. Distribution of new hemodialysis patients based on the currently used vascular access route as of the end of 2016-2017[2]

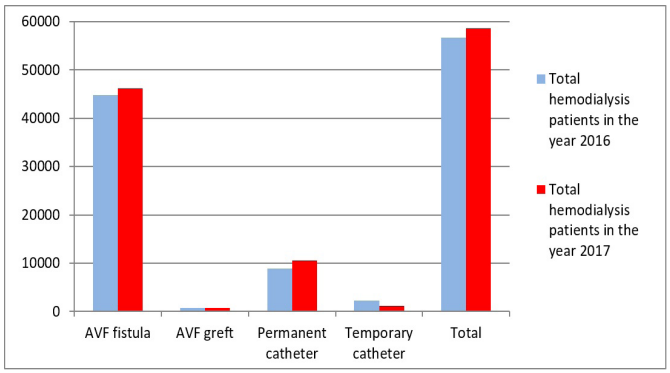

Figure 2. Distribution of hemodialysis patients based on the currently used vascular access route as of the end of 2016-2017[2]

HEMODIALYSIS: WHAT IS IT?

Hemodialysis (HD) is the process of removing various toxic products such as urea, creatinine, potassium, phosphorus, and excess water from the body due to kidney insufficiency. This is achieved through the use of a semi-permeable membrane located outside the body, which cleans the blood.

WHO UNDERGOES HEMODIALYSIS AND HOW IS IT PERFORMED?

As kidney function declines, the levels of toxic products such as urea, as well as various electrolytes like potassium and phosphorus, become uncontrollable. Conditions such as uremia, hyperkalemia, and hypervolemia can lead to severe complications and, if left untreated for an extended period, may result in death. While the primary goal in acute kidney failure is to address these complications, the objective in

chronic kidney failure is to reintegrate the individual into daily social and work life.[3-5]

In the process of HD, the fundamental technique involves passing the patient's blood through an external filter to eliminate toxic substances. Simultaneously, the depleted electrolytes are replenished before returning the blood back into the body. Essential components such as proteins and shaped elements of blood, including vital elements like electrolytes and water, are retained in the blood, while toxins and excess water are removed through micro-pores. The HD solution also possesses a replacement feature, normalizing the electrolyte and water balance. During treatment, anticoagulants such as heparin may be administered to prevent blood clotting.[4-7]

VASCULAR ACCESS ROUTE

To transport blood from the body to the machine and back to the patient, a vascular access route is essential. The choice of vascular access route depends on the urgency of the HD requirement. Catheters, associated with both infectious and non-infectious complications, contribute to increased morbidity and mortality. In cases of urgent HD need arising from acute kidney failure (AKF) or chronic kidney disease (CKD), catheters are the initial preference. The ability to initiate HD treatment immediately upon catheter insertion is the reason for this preference, eliminating the need for waiting. Further planning is made based on the clinical course.

If the anticipated duration of HD need is short-term (a few weeks), a temporary catheter may suffice. If a prolonged need for this treatment is foreseen, an arteriovenous fistula (AVF), also known as AVF, is planned as soon as possible. This topic is explained in more detail under the relevant heading. If there is an obstacle to creating an AVF, a "permanent tunneled catheter" can be placed after a temporary catheter. Moreover, if the need for such access is foreseeable from the beginning, placing a permanent catheter instead of a temporary one in a single procedure may be more appropriate.[1-4,6,8]

CATHETER

A central catheter is a type of central venous vascular access route with one end located outside the body and the other advanced towards the cardiac cavities.[3,8]

While this vascular access route has various applications, this discussion will primarily focus on its use in HD.

Catheter Usage

The use of catheters comes with both advantages and disadvantages (Table 1, 2).[1-3,4-8]

Table 1. Advantages of catheter usage

No maturation time is required, as in shunts (arteriovenous fistula).
Cost-effective.
Applicable in every center and easily implemented.
Offers numerous anatomical options.
Depending on the type, can be used for weeks to years.
Catheter blockage is often a correctable condition.

Table 2. Disadvantages of catheter usage

Increased risk of catheter thrombosis or thrombotic occlusion in the vessel where it is placed.
Short lifespan of temporary catheters.
Directly accessing central vascular structures increases the risk of infection and sepsis.
Complicates hygiene practices.
Disrupts patient comfort.

Catheter Types

There are two different types of catheters based on duration of use: permanent and temporary. If the planned duration of use is between a few days and a few weeks, temporary catheters are initially preferred. However, even within this period, especially to prevent complications such as infection, it may be necessary to remove the catheter every 72 hours and replace it with a new one from a different site. This practice becomes even more crucial in areas with high vulnerability and increased incidence of resistant infections, such as intensive care units. A temporary catheter enters the central vein just below the skin entry point and reaches the heart. On the other hand, a permanent catheter, after entering the skin, is advanced a certain distance (usually around 10 cm) under the skin before entering the central vein and reaching the heart. These catheters are referred to as tunnelled catheters because a tunnel-like space is formed as they progress under the skin. Additionally, to firmly anchor in the area where it will remain for an extended period, there is Dacron felt outside the catheter, and this felt ensures adhesion to the surrounding tissue a few days later. It should be noted that due to catheter dysfunction and dysfunction-related issues, approximately 52% of tunnelled HD catheters need to be removed within one year. Therefore, if vascular access is required for more than three weeks, a tunnelled catheter should be preferred.[4-8]

Catheter Placement

Different techniques are used for catheter placement for each central vein except for the external jugular vein, which is superficial. Almost all central venous catheters are placed deep below the fascia. Therefore, vascular tracing is done by creating reference points using the vein trajectory, surrounding muscles, and the artery. Prior to the procedure, the patient's cardiac rhythm is monitored with electrocardiogram (ECG) leads, blood pressure is monitored with a cuff placed near the central area, and oxygen saturation is monitored with a pulse oximeter. The area is sterilized with betadine, covered with a sterile drape with small holes, and local anesthetic is applied. Before starting the procedure, it is confirmed that the patient does not have bleeding diathesis and has not used any medication that may disrupt hemostasis. For this purpose, the intrinsic pathway (aPTT), extrinsic pathway (PT/INR), and platelet count are checked with a complete blood count. In very specific cases, catheter placement may be performed despite unfavorable conditions, but this situation carries significant risks and should be managed with awareness of potential complications. Particularly in such cases, performing the procedure under ultrasound guidance can reduce complications. In the standard method, a small amount of saline is drawn into a syringe first. A puncture needle is attached to the tip, and the air is expelled. The needle is advanced toward the target point with negative pressure created in the syringe.

Some complications may arise at this stage. Attempting to enter one of the veins around the thorax, especially when air is aspirated into the syringe, may indicate entry into the thoracic cavity. Pneumothorax is very rare when the needle is rapidly withdrawn with negative pressure, as long as there is no injury to the lungs. Alternatively, entering one of the arteries adjacent to the target vein in any region may result in bright-colored and pressurized blood. In both cases, the needle is immediately withdrawn, and pressure is applied to the relevant area for about 5 minutes. Then, the procedure continues. It should be kept in mind, especially in patients with COPD or respiratory distress at that moment, with low oxygen saturation, that arterial blood color may be dark like venous blood, and there may be a false vascular puncture. Similarly, considering the patient profile, patients with poor cardiac function, low ejection fraction, and inadequate blood pressure, pulse, and circulation may also have pressureless arterial blood, leading to a potential false vascular puncture. Once the relevant vein is punctured correctly, the needle is advanced without any movement, and the guide wire is threaded from the needle into the vein and then towards the heart. In a patient without cardiac risk, the ECG trace is monitored from the monitor as the guide wire is advanced to the heart, and the change in heart rhythm at the moment the guide wire touches the atrial tissue is considered an indicator that the guide wire has reached the heart. However, especially for elderly patients with rhythm problems, such verification is a significant risk and may not be necessary.[1,2,4-8]

Venous Selection for Hemodialysis Catheterization

Choosing the appropriate vein for intervention is crucial, taking into account the individual patient's condition, as each venous access site has various advantages and disadvantages. The most commonly preferred veins include:[4-8]

1. Vena jugularis interna (Internal jugular vein)

2. Vena subclavia (Subclavian vein)

3. Vena jugularis externa (External jugular vein)

4. Vena femoralis (Femoral Vein)

1. Vena jugularis interna: This is the most commonly chosen route for short-term central venous catheterization. The relatively lower complication rate and the wide diameter of the vein make it a primary choice. It runs within the carotid sheath in the neck, passing anteriorly and laterally to the carotid artery. It joins the subclavian vein at the level of the clavicle, forming the brachiocephalic vein that enters the thorax. The patient lies supine with arms secured at the sides, and the head is slightly turned in the opposite direction during the procedure. The Trendelenburg position (head down) is useful to protect the brain from potential air embolism during the procedure and to increase venous filling, thereby expanding the vein diameter. In cases of insufficient venous filling, additional volume replacement from another peripheral vascular access may be performed. The Valsalva maneuver or maintaining a brief inspiratory hold on a mechanical ventilator can increase venous filling. Gentle rotation of the head in the opposite direction of the puncture site can ease the procedure but excessive rotation may increase the risk of arterial puncture. The puncture site is usually the apex of the triangle formed by the clavicular and sternal heads of the sternocleidomastoid muscle. Palpate the carotid artery at the level of the cricoid cartilage, then puncture the skin at a 30-40 degree angle towards the nipple, maintaining slight negative pressure in the syringe. Generally, the vein is accessed at a depth of 2-3 cm; going deeper increases the risk of complications.[4-8]

2. Vena subclavia: It is chosen when catheter placement in the neck veins, the primary preference area, is not feasible. While the advantage lies in the wide diameter of the vein, entering the thorax increases the risk of complications such as pneumothorax/hemothorax and arterial injury, and their repair can be challenging. Subclavian vein catheters have the highest risk of stenosis (30-50%), a concern that is particularly significant in patients with bleeding diathesis. It is a continuation of the axillary vein. The subclavian vein is located in the lower part of the supraclavicular triangle, between the posterior edge of the sternocleidomastoid muscle, the middle of the clavicle, and the anterior surface of the trapezius muscle. It joins the internal jugular vein behind the sternoclavicular joint and extends below the first rib, passing beneath and in front of the artery. The pleura is immediately below the vein. The patient lies supine with arms secured at the sides, and the head is slightly turned in the opposite direction during the procedure. The Trendelenburg position (head down) is important to protect the brain from potential air embolism during the procedure and to increase venous filling, expanding the vein diameter and volume. In cases of insufficient venous filling, additional volume replacement from another peripheral vascular access may be performed. The Valsalva maneuver or maintaining a brief inspiratory hold on a mechanical ventilator can increase venous filling. Puncture is made 1 cm below and lateral to the midpoint of the clavicle, directed towards the sternal notch. If the needle tip touches the clavicle, it is slightly withdrawn and redirected to go deeper. The needle tip should not pass the sternal alignment. Generally, the vein is accessed at a depth of 2-3 cm; going deeper increases the risk of complications.[4,5,7,8]

3. Vena jugularis externa: Especially in cases of short-term and urgent central venous catheterization needs, when catheter placement in the internal jugular vein or subclavian vein is not possible for any reason, and there is a visibly prominent external jugular vein structure, it may be preferred. Although not always prominent in every patient, it is easily visible and palpable in a position accessible in the neck, leading to fewer complications associated with puncture. However, even if successful vein puncture and advancement of the guidewire are possible, the course, diameter, and valvular structure of the vein may not allow the catheter to advance. It descends down from the mandible, crossing the sternocleidomastoid muscle, and joins the subclavian vein at the level of the clavicle. Similar to the internal jugular vein, the patient lies supine with arms secured at the sides, and the head is slightly turned in the opposite direction during the procedure. The position is given to facilitate easy visualization and palpation, and the puncture is performed as if opening a peripheral vascular access, with the guidewire and then the catheter advanced. The Trendelenburg position (head down) is important to protect the brain from potential air embolism during the procedure and to increase venous filling, expanding the vein diameter and volume. In cases of insufficient venous filling, additional volume replacement from another peripheral vascular access may be performed.

The Valsalva maneuver or maintaining a brief inspiratory hold on a mechanical ventilator can increase venous filling. While a slight rotation of the head in the opposite direction of the puncture site can ease the procedure, excessive rotation may increase the likelihood of arterial puncture.[4-8]

4. Vena femoralis: It may be preferred, especially in cases of short-term and urgent central venous catheterization needs, when catheter placement in the internal jugular vein or subclavian vein is not possible for any reason. Since it is adjacent to the easily palpable femoral artery, complications associated with puncture are minimized. However, prolonged use poses a risk of deep vein thrombosis and can be a significant source of infection due to the difficulty in keeping the area clean at all times. Additionally, in mobilized patients, the catheter in this region can easily become obstructed when folded in a sitting position. Located just distal to the inguinal ligament, in the femoral triangle area, it is in proximity to the medial side of the femoral artery. The patient lies supine, and the vein is punctured at an angle of 20-30 degrees to the skin, just distal to the inguinal ligament and immediately medial to the femoral artery. Puncture is performed as if opening a peripheral vascular access, guided by palpation, and then the guidewire and catheter are advanced. If the needle is advanced too laterally, arterial puncture may occur. Inappropriately lateral entries can lead to femoral nerve damage. Achieving adequate hygiene, especially due to proximity to the genital area, is often challenging and carries a much higher risk of infection. Therefore, long-term use is not recommended.[4-8]

Catheter Complications

Improper catheter placement has the potential to cause life-threatening complications, including pneumothorax, arterial puncture, bleeding, and central circulation infections associated with vital structures.[1,3,4]

During catheter placement, the following complications may arise:

1. Arterial/venous injury
2. Bleeding
3. Pneumothorax
4. Emboli
5. Cardiac arrhythmias
6. Obstruction

During hemodialysis:

1. Collapse
2. Obstruction
3. Bleeding
4. Emboli
5. Cardiac arrhythmias
6. Hypotension

Late Complications:

1. Infection: Treatment of catheter-related bacteremia often necessitates the removal of the catheter in most patients.[6-7]

2. Catheter thrombosis: While vascular wall injury typically occurs during catheter placement, any manipulation or repositioning of the catheter can lead to further injury. This triggers turbulent blood flow around the catheter, stimulating coagulation and inflammatory cascades, resulting in thrombosis.[7,8]

3. Stenosis in central vein: Stenosis in the central vein is one of the most common complications, occurring in around 40% of patients undergoing HD despite all precautions. The development of collateral veins can compensate for the narrowing, allowing asymptomatic patients without venous return problems to be monitored. However, if the return is impaired and venous hypertension develops, percutaneous transluminal angioplasty is the preferred treatment method.

4. Perforation

Catheter Obstruction and Treatment

For the HD machine to perform adequately, the device usually requires a blood flow rate of 300-500 ml/min to come into the machine, and after the procedure, it should be able to be given back to the patient.[8,9] In hemofiltration devices, although this need drops to levels around 100 ml/min, it is still important that the vascular access pathway operates at full capacity. When there is a decrease in inflow or outflow to the device, the machines usually give warnings. The possibility of the catheter being folded should be considered first in this situation. Both the catheter itself and all connections should be checked. Catheter clamps can easily cause folding in this area due to constant compression. Also, considering the possibility of thrombosis, prophylactic heparin administration during the procedure can be preventive for such occlusion.[9,10] Additionally, after flushing the catheter line with sterile saline at the end of the procedure, the space inside the catheter line is filled with a diluted solution in temporary catheters and with pure heparin in permanent catheters. The volume of the relevant space is indicated on the catheter. It should be remembered that filling the catheter line only with the amount of heparin indicated on the catheter, without giving any heparin to the patient, is essential. For example, giving 2 ml of heparin to a space indicating 1.7 ml means administering 0.3 ml (1500 units) of heparin to the patient, which can lead to bleeding, especially in a patient with bleeding diathesis. It is crucial to precisely fill the catheter line with the amount of heparin written on the catheter using a precise measurement. If the catheter is blocked for any reason, several maneuvers can be attempted to salvage the situation. A classic sign is the absence of both blood coming in and going out through the catheter. Using the thinnest possible syringe (2 cc or even an insulin syringe), pressure is applied to the catheter with a solution diluted to 1/10 with heparin. Since the syringe tip can pop out of place during this pressure, the connection point should be held firmly. Using a syringe with a screw tip is even more secure. The highest possible pressure is achieved with the thinnest syringe. Moreover, since only a small volume of 1-2 cc is delivered to the vessel during the opening of the blockage, vascular damage is prevented. Although some thrombus may fall into the vessel during the clearance of the blockage, this amount is usually small enough not to cause a problem. Nevertheless, to prevent possible complications, heparinization of the patient is recommended. If thrombosis cannot be removed despite all these efforts, the catheter is replaced with a new one.[8-11]

Another significant reason is the rapid blockage of catheter holes due to contact with the vessel or atrium walls during rapid suction. The HD machine creates a vacuum at the catheter tip, pulling tissue towards itself along with blood,

similar to an electric vacuum cleaner. If the region where the catheter tip is located is not in a wide space or there is not enough fluid in the area, the walls near the catheter holes are pulled towards the catheter, instantly blocking itself, stopping the blood flow, and locking the system. The classic sign of this problem is that when blood is slowly withdrawn from the catheter with a syringe, it flows smoothly, but when withdrawn rapidly, it gets blocked. Before intervening in the catheter, several basic techniques can be tried to still operate the system. Firstly, changing the patient's position or making them cough may resolve the situation. The patient may be hypovolemic, and venous filling can be increased by placing them in the Trendelenburg position or using the Valsalva maneuver. Excessive fluid removal from the patient during dialysis can also cause hypovolemia. The system working very well at the beginning of dialysis but inadequate flow towards the end is a typical sign of this condition. Hypotension, a common complication during HD, can also indicate preload reduction due to hypovolemia. In this case, fluid removal can be stopped, and fluid replacement (including blood products if necessary) can be considered.[4-7,9,11]

If the problem of flow inadequacy cannot be corrected with these maneuvers in the dialysis room, a chest X-ray can provide important information regarding the location of the catheter. Especially for catheters placed in the chest or neck area, the tip must have reached the atrial space. As the venous diameters and venous filling are not very high at this level, when the system creates a vacuum, venous structures can collapse, and the system can lock. If the catheter is too advanced and touches the heart valves or bends in the atrium, the system can also collapse. If the catheter is too inside, it can be pulled out a bit surgically or with controlled small manipulations. However, the opposite is dangerous and difficult. That is, pushing the catheter from the outside to advance it to reach the atrial space is both difficult due to the catheter's softness and its partial adherence to the surrounding tissues, and it brings an additional risk of sepsis by inserting a catheter region that cannot be completely sterilized into the vessel. Generally, in this case, the catheter is replaced with a new one. As a convenience, a guidewire can be inserted into the old catheter, the catheter can be withdrawn, and then a new catheter can be placed over the same guidewire. However, this method also carries an additional risk for sepsis as the sterility of the area cannot be guaranteed during this procedure. Another problem we can see with chest X-ray is the folding of especially tunneled permanent catheters inside the body. Often this folding is at the level just before entering the vessel in the tunnel. Sometimes this folding can be corrected by manipulating the skin from the outside, but many times surgical manipulation or sometimes replacement of the catheter is needed.[7-11]

Catheter Care

Every invasive procedure carries various risks. Therefore, reducing the need for the same process to be repeated is crucial. Appropriate care and usage techniques from the moment of placement extend the catheter's lifespan. This care and dressing changes should be performed by trained healthcare personnel.[7-11]

- The entry site of the catheter should be checked before and after each hemodialysis session. Temporary catheters are held in place with stitches, and if these stitches have become loose or broken, they should be repaired, and if necessary, new stitches should be applied. Permanent catheters, on the other hand, are held in place from a few days onwards by the adherence of the Dacron cuff around them to the skin. Until this adhesion is achieved, a stitch also helps keep the catheter in place. If the stitches holding the catheter in place have broken or been damaged without sufficient adhesion, they should be repaired or new stitches should be applied. The cuff of the catheter must be within the skin. If the cuff is visibly protruding (malposition), it should be referred to a center that can correct the situation through surgical intervention. In such a center, the cuff can be pushed a bit under the skin as much as possible in sterile conditions, or a cut can be made in the skin to bury the cuff, and then it can be covered or a new catheter can be placed.
- Before and after each hemodialysis session, the catheter site and catheter caps should be sterilized with a solution (2% chlorhexidine or 10% povidone iodine or 70% alcohol) and covered with a sterile gauze after the procedure.
- During bathing, the catheter site should be covered with a waterproof drape that completely covers the entire catheter. Applying a small amount of antibacterial ointment around the catheter entry hole can contribute to protection by providing a liquid seal feature.

PERMANENT CENTRAL VENOUS PORTS

This is a method commonly used to administer agents sequentially to the patient that could potentially damage the vascular lumen. The most frequent application is for the administration of chemotherapeutic agents. One end of the catheter is placed in a central vein with high blood flow, similar to HD catheters, allowing the medication to quickly dilute in the blood. Unlike HD catheters, the other end does not exit the body; it is subcutaneous and not visible from the outside. This significantly reduces the risk of infection. The other end is left in the body as a small reservoir. The silicone surface is left in the front for needle puncture. It can remain in the body for months to years. Using a special needle, the silicone membrane is pierced to access the reservoir, and the needle is removed at the end of the treatment. Another difference from HD catheters is that the catheters that continue as a reservoir have much smaller diameters. This reduces the risk of causing venous stenosis.[7-11]

CONCLUSION

The need for venous catheterization is increasing due to various reasons, especially in hemodialysis patients. When performed with certain principles and in experienced hands for appropriate indications, the risk of this procedure is minimal.

ETHICAL DECLARATIONS

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

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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.

REFERENCES

1. Vats HS. Complications of catheters: tunneled and nontunneled. *Adv Chronic Kidney Dis*. 2012;19(3):188-194.
2. Türk Nefroloji Derneği Registry Raporları. http://www.nefroloji.org.tr/icerik.php?gid=88 Accessed December 11, 2018.
3. Miller LM, MacRae JM, Kiaii M, et al. Hemodialysis tunneled catheter noninfectious complications. *Can J Kidney Health Dis*. 2016; 3:2054358116669130.
4. Shingarev R, Barker-Finkel J, Allon M. Natural history of tunneled dialysis catheters placed for hemodialysis initiation. *J Vasc Interv Radiol*. 2013;24(9):1289-1294.
5. Osman OO, El-Magzoub AR, Elamin S. Prevalence and risk factors of central venous stenosis among prevalent hemodialysis patients, a single center experience. *Arab J Nephrol Transplant*. 2014;7(1):45-47.
6. US Renal Data System: USRDS 2012 Annual Data Report: Atlas of Chronic Kidney Disease and End-Stage Renal Disease in the United States, Bethesda, MD, National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases, 2012.
7. Chan MR. Hemodialysis central venous catheter dysfunction. *Semin Dial*. 2008;21(6):516-521.
8. Vascular Access Work Group. Clinical practice guidelines for vascular access. *Am J Kidney Dis*. 2006;48(Supplement 1):S248-S273.
9. Moran JE, Ash SR. Locking solutions for hemodialysis catheters; heparin and citrate—a position paper by ASDIN. *Semin Dial*. 2008;21(5):490-492.
10. NKF-K/DOQI Clinical Practice Guidelines UpDate 2000. *Am J Kidney Dis*. 2001;37(1):S137-S181.
11. Yamamoto Y, Orii T, Yoshimura M, Kitahara H, Karasawa Y. Is there any correlation between venipuncture sites and complications of central venous port placement in the chest wall? *Int J Surg Open*. 2018;14:15-18.

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Case Report

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Spontaneous recovery in the early period of cardiac remodeling due to carbon monoxide poisoning

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ABSTRACT

Carbon monoxide can cause cardiac injuries, including transient left ventricular dysfunction, by causing hypoxia at the tissue level. In this case, we present a case that developed serious global left ventricular dysfunction (ventricular remodeling) as a result of carbon monoxide poisoning and recovered very early. Although we could not use any cardioprotective agents in the treatment of the patient without hyperbaric oxygen, we experienced that the left ventricular functions completely recovered on the 7th day of hospitalization.

Keywords: Carbon monoxide poisoning, acute left ventricular dysfunction, hypoxia

INTRODUCTION

Carbon monoxide (CO) occurs as a result of carbon-based fuels not burning properly. It can cause acute and chronic poisoning in humans (alives).[1,2] CO's affinity for hemoglobin (Hb) is 220 times higher than oxygen. By binding to Hb, CO reduces the oxygen-carrying capacity of the molecule (it both competes with oxygen and causes structural changes in Hb).[3,4] Exposure to high concentrations of CO can have fatal consequences and is one of the most common causes of death due to poisoning worldwide. CO can cause cardiac injuries by causing hypoxia at the tissue level.[5] Cardiac symptoms such as myocardial ischemia, heart failure, and arrhythmia have been reported after exposure to CO.

While 4 of the 45.000/500 million people affected by COP die every year in the USA, but these data are not clear in our country.[6] COP can affect all systems, including the heart, brain and nervous system, muscle, gastrointestinal system, and skin. Since neurological and cardiac functions are affected in the early period, the first symptoms are usually seen in the neurological and cardiovascular systems.[7] Clinical severity depends on the amount of CO inhaled, its duration, and the current health status. There is no specific antidote. The most commonly applied treatment methods are normobaric and hyperbaric oxygen therapy.[8]

In this article, we will present a case of severe left ventricular dysfunction in a young female patient, which developed in the early period after COP and resolved spontaneously.

CASE

A 29-year-old woman was found unconscious in her patient room and was taken to an emergency room at another medical center.

The patient, whose Glasgow coma scale (GCS) was evaluated and found to be low, was intubated (GCS: 3). Since the room she was staying in was heated with a stove and the CO levels in her blood were between 20% and 30%, she was diagnosed with COP and sent to our hospital for treatment and admitted to the general intensive care unit.

In the history taken from the patient's relatives, it was stated that he had no known chronic disease and did not use cigarettes, alcohol or drugs. During the cardiovascular system examination, blood pressure arterial measurement was evaluated as 70/56 mmHg and pulse rate was 110/min. S1 and S2 heart sounds were natural, and no murmurs were heard. In the respiratory system examination, respiratory rate was evaluated as 30/min and blood oxygen saturation was evaluated as 98% with ventilator support. Lung sounds were natural bilaterally and both hemithoraxes participated equally in breathing. No pathological appearance was observed on electrocardiography other than sinus tachycardia (Table 1) (Figure 1). No obvious pathology was seen on the posterior-anterior chest radiography when the intubation tube was broken. Abdominal examination was normal. The laboratory results of the intensive care unit admission were aspartate aminotransferase (AST)/alanine aminotransferase (ALT):45/33 creatine kinase (CK):3113, creatine kinase isoenzyme MB (CK-MB):85, aminoterminal part of B-type natriuretic peptides (NT-Pro BNP):1887 and changes in these patient outcomes throughout treatment are summarized in Table 2. Laboratory results other than these were evaluated to be within the normal range. General supportive treatment (fluid support, daily electrolyte replacements, antiemetic treatment, respiratory support, etc.) and hyperbaric oxygen therapy were planned for the patient. Cardiology consultation

was requested from the patient because he was hypotensive on the day of admission and his cardiac troponin I values were high. To evaluate the level of cardiac involvement, transthoracic echocardiography was performed with a device with a 3.5 MHz transducer (Vivid 5 GE Medical System, Horten, Norway)). In echocardiography, LV ejection fraction was evaluated as 30-35% and global hypokinesia was detected in left ventricular wall movements (Figure 2). No pathological findings were detected in other echocardiographic measurements and valve evaluations. Since the patient's general condition was not good and inotropic support continued, classical LV remodeling and/or LV insufficiency drug treatments such as B-blockers, angiotensin-converting enzyme inhibitors, etc. could not be given. The patient was given hyperbaric oxygen therapy for 4 days. In the control echocardiography performed on the seventh day of the patient's hospitalization, it was observed that the LVEF improved (55%) (Table 1). The patient, who had no additional problems, was discharged from the general intensive care unit on the 15th day without any cardiac medical treatment planned. Service monitoring was continuing at the time this article was written.

Table 1. Echocardiographic and electrocardiographic results of the patient throughout the treatment process

Days	Echocardiography		Electrocardiography
	(Ejection fraction %, Simpson metod)	Wall motion	
1	30-35	LV global hypokinetic	Sinus tachycardia
3	35	LV global hypokinetic	Sinus tachycardia
5	40-45	All walls slightly hypokinetic	Sinus rhythm
7	55	-	Sinus rhythm

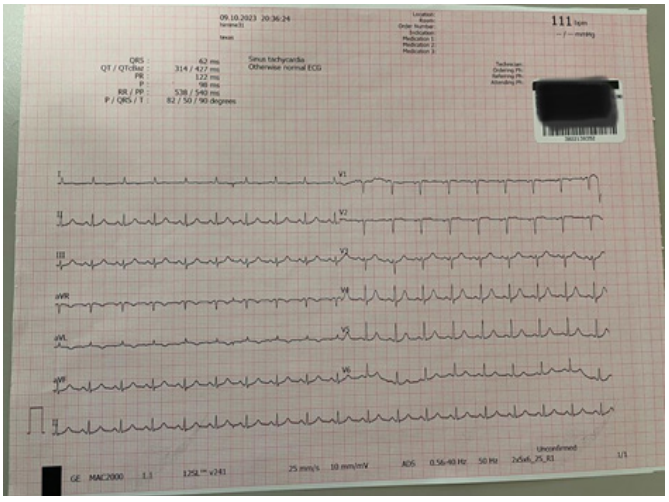

Figure 1. Electrocardiography performed when she was admitted to the general intensive care unit

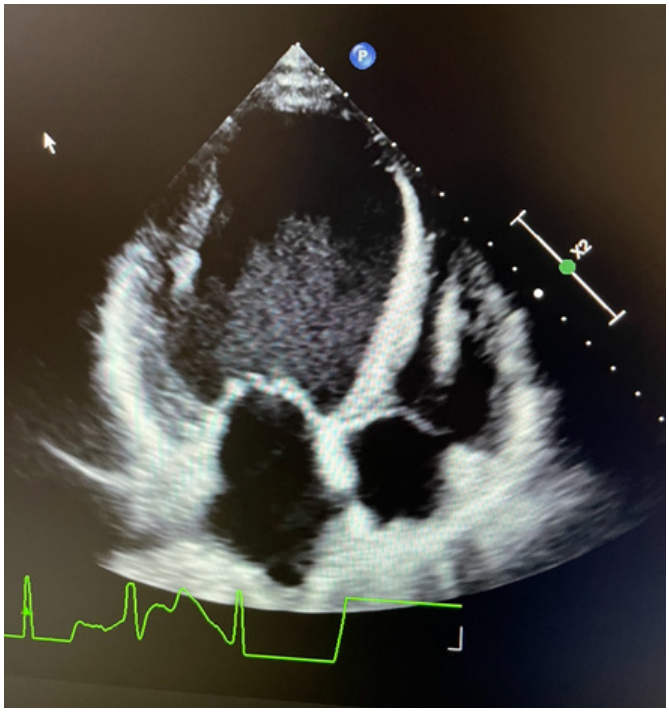

Figure 2. Echocardiography performed when she was admitted to the general intensive care unit

DISCUSSION

In this article, a case of left ventricular remodeling that occurs in the early period after COP and resolves spontaneously is presented. In our case, the recovery time for left ventricular remodeling was 7 days.

In COP, hypoxia due to inadequate oxygen delivery is the cause of the basic symptoms and signs. In most cases, cardiovascular findings may be missed or diagnosed late due to respiratory and neurological symptoms that predominate.[9] While myocardial damage and fibrosis may be observed at low dose exposures, it has been previously reported in the literature that fatal arrhythmias may occur at high dose exposures. In addition to hypoxia due to COP cardiotoxicity, CO gas also has a direct toxic effect by inhibiting cytochrome oxidase on myocyte mitochondria.[10]

In 1865, CO-induced heart failure and myocardial ischemia were described for the first time.[11,12] In later years, various cardiac clinical diseases such as CO-induced arrhythmias, electrocardiographic changes, acute myocardial infarction, pulmonary edema, and cardiogenic shock were reported.[11,13,14] Those with a previous history of cardiovascular disease are more sensitive to CO-induced cardiotoxicity.[15] Tachycardia is the most common cardiovascular finding.[16] Chest pain occurs regardless of the presence of coronary artery disease due to myocardial ischemia or necrosis.[16,17] Shortness of

Table 2. The laboratory results of the patient throughout the treatment process

Days	Tro I (ng/ml)	CK (U/L)	CK-MB (U/L)	AST/ALT (U/L)	Cre (mg/dL)	GFR (ml/dk)	Sodium (mEq/L)	Potassium (mEq/L)	Calcium(mg/dL)
1	541	3113	85	45/33	0.66	120	140	4.5	7.4
3	423	2809	59	675/1183	0.92	84	151	4.1	7.2
5	230	2222	37	170/997	0.64	121	150	3.7	7.1
7	164	473	22	58/303	0.55	127	137	3.7	7.7

Abbreviations; Tro I; Troponin I, CK; Creatine kinase, CK-MB; creatine kinase isoenzyme MB, AST/ALT; Aspartate aminotransferase/alanine aminotransferase, Cre; Creatine, GFR; Glomerular filtration rate

breath may be due to hypoxia or to depression in heart function.[18] Shortness of breath and tachycardia were detected in this patient at the time of admission, and these symptoms suggested the presence of cardiac dysfunction.

Diffuse or segmental wall motion abnormalities may occur in patients exposed to CO. Echocardiography may be considered, especially in cases of unexplained hypotension, myocardial damage detected by laboratory tests, and in patients with shortness of breath without lung pathology. Satran et al.[19] showed in a study that the left ventricle was affected by 57% after CO exposure. Additionally, it was determined that the decrease in LV systolic functions was proportional to the amount of CO exposure. Park et al.[20] found dysfunction in the LV in 29% of the patients in their study and reported global involvement in only 5% of the patients. Information about the recovery period of this depression, which is generally described as reversible, is very limited. Jang et al.[5] reported in their case report that the general condition was better and that severe left ventricular systolic dysfunction improved on the 4th day by receiving heart failure treatment. Lee et al.[21] reported that 18 of 21 people who had follow-up echocardiography recovered within 3 days by receiving heart failure treatment. However, in this series, both the patients' ages were older and their LVEF was around 53% on average. Although our case is similar to the cases of Jang et al.[5], in our case the general condition of the patient was much worse (probably the CO exposure time was longer) and although he could not take heart failure medications due to hemodynamic compromise, we still detected an improvement in a short time.

This study gives an idea that left ventricular dysfunction after COP can improve in a very short time, there is no need to pass for a long time for control echocardiography, and perhaps there may be no rush to start heart failure treatment. However, case-controlled clinical studies are needed for more definitive results.

CONCLUSION

This study suggests that left ventricular dysfunction after COP can improve quickly, there is no need to wait a long time for control echocardiography, and there may be no rush to begin heart failure treatment. More definitive results, however, will require case-controlled clinical studies.

ETHICAL DECLARATIONS

Informed Consent

All patients signed and free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

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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.

REFERENCES

1. Committee of the Environmental and Occupational Health Assembly of the American Thoracic Society. State of the art. Health effects of outdoor air pollution. *Am J Respir Crit Care Med*. 1996;153:477-498.
2. Kandiş H, Katırcı Y, Karapolat BS. Karbonmonoksit zehirlenmesi. *Düzce Med J*. 2009;11(3):54-60.
3. Kandis H, Katırcı Y, Çakır Z, Aslan Ş, Uzkeser M, Bilir Ö. Acil servise karbonmonoksit entoksikasyonu ile başvuran olguların geriye dönük analizi. *Akad Acil Tıp Derg*. 2007;5(3):21-25.
4. Piantadosi CA. Carbon monoxide poisoning. *Undersea Hyperb Med*. 2004;31:167-177.
5. Jang W, Park JH. Transient left ventricular systolic dysfunction associated with carbon monoxide toxicity. *J Cardiovasc Ultrasound*. 2010;18(1):12-15.
6. Weaver LK. Carbon monoxide poisoning. *N Engl J Med*. 2009;360(12): 1217-1225.
7. Brown SD, Piantadosi CA. In Vivo binding of carbon monoxide to cytochrome c oxidase in rat brain. *J Appl Physiol*. 1990;68(2):604-610.
8. Jain KK. Textbook of Hyperbaric Medicine. In: Jain KK, ed. Carbon monoxide and other tissue poisons. 6th ed. Springer International Publishing: 2016:131-154.
9. Fiorista F, Casazza F, Comolatti G. Silent myocardial infarction caused by acute carbon monoxide poisoning. *G Ital Cardiol*. 1993;23(6):583-537.
10. Peers C, Steele DS. Carbonmonoxide: a vital signalling molecule and potent toxin in the myocardium. *J Mol Cell Cardiol*. 2012;52(2):359-365.
11. Gandini C, Castoldi AF, Candura SM, et al. Carbon monoxide cardiotoxicity. *J Toxicol Clin Toxicol*. 2001;39(1):35-44.
12. Marius-Nunez AL. Myocardial infarction with normal coronary arteries after acute exposure to carbon monoxide. *Chest*. 1990;97(2):491-494.
13. Anderson RF, Allensworth DC, DeGroot WJ. Myocardial toxicity from carbon monoxide poisoning. *Ann Intern Med*. 1967;67(6):1172-1182.
14. McMeekin JD, Finegan BA. Reversible myocardial dysfunction following carbon monoxide poisoning. *Can J Cardiol*. 1987;3(3):118-121.
15. Allred EN, Bleecker ER, Chaitman BR, et al. Short-term effects of carbon monoxide exposure on the exercise performance of subjects with coronary artery disease. *N Engl J Med*. 1989;321(21):1426-1432.
16. Thompson N, Henry JA. Carbon monoxide poisoning: poisons unit experience over five years. *Hum Toxicol*. 1983;2(2):335-238.
17. Ebisuno S, Yasuno M, Yamada Y, et al. Myocardial infarction after acute carbon monoxide poisoning: case report. *Angiology*. 1986;37(8):621-624.
18. Diltoer MW, Colle IO, Hubloue I, et al. Reversible cardiac failure in an adolescent after prolonged exposure to carbon monoxide. *Eur J Emerg Med*. 1995;2(4):231-235.
19. Satran D, Henry CR, Adkinson C, Nicholson CI, Bracha Y, Henry TD. Cardiovascular manifestations of moderate to severe carbon monoxide poisoning. *J Am Coll Cardiol*. 2005;45(9):1513-1516.
20. Park JS, Seo KW, Choi BJ, et al. Various echocardiographic patterns of left ventricular systolic dysfunction induced by carbon monoxide intoxication. *Cardiovasc Toxicol*. 2016;16(4):361-369.
21. Lee JH, Kim HS, Park JH, et al. Incidence and Clinical course of left ventricular systolic dysfunction in patients with carbon monoxide poisoning. *Korean Circ J*. 2016;46(5):665-671.

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Case Report

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A case report of history of anterior myocardial infarction complicated with left ventricular pseudoaneurysm

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ABSTRACT

Pseudoaneurysm, which is a fatal complication of myocardial infarction and occurs when left ventricular rupture is limited by the pericardium and hematoma, is a mortal complication that allows life to continue compared to rupture. In this case report, we tried to discuss the successful management of a 61-year-old male patient who applied to our clinic with a complaint of chest pain and dyspnea, from diagnosis to surgery, within the literature.

Keywords: Left ventricle, pseudoaneurysm, echocardiography

INTRODUCTION

Left ventricular pseudoaneurysm (LVP) is a rare but catastrophic complication of myocardial infarction (MI). In addition, it could develop after infection, trauma, and valvular or ventricular surgery. LVP occurs when cardiac rupture is limited by the pericardium and scar tissue.1 The incidence of left ventricular pseudoaneurysm after MI is around 0.2-0.3%.2 Patients with left ventricular pseudoaneurysm may be asymptomatic or present to the clinic with a wide range of symptoms, including congestive heart failure (CHF), chest pain, dyspnea, arrhythmia, tamponade, syncope and sudden cardiac death.[1,2]

Ventricular pseudoaneurysm after inferior MI is twice as common as pseudoaneurysm after anterior MI.[3] If LVPA occurs after MI, it may have an insidious course and the patient may present to outpatient clinics and emergency departments with atypical findings. The rupture risk of pseudoaneurysm is inversely proportional to the time elapsed after acute myocardial infarction. This period is important in the classification and treatment management of pseudoaneurysms. In pseudoaneurysm, as time passes after MI, the cavity stabilizes and the risk of rupture gradually decreases. Otherwise, the risk of rupture depends directly on the size of the ischemic necrotic area and the wall stress in the aneurysm sac.[3]

LVPA, which should be considered especially in post-MI patients with unexplained clinical symptoms such as dyspnea or angina, can be evaluated with transthoracic echocardiography. Although it is known that surgical intervention is still superior to medical treatment, its mortality is high with a rate of 7-30% (Inayat F, Ghani AR, Riaz I, et al. Left ventricular pseudoaneurysm: An overview of diagnosis and management. J Investig Med High Impact Case Rep. 2018;6:2324709618792025). The indication for surgery in the treatment of pseudoaneurysm is determined by heart failure, syncope, ventricular arrhythmia, the possibility of thromboembolic complications, and the risk of rupture.[4]

In this case report, we discussed the successful management of a 61-year-old male patient who applied to our clinic with a complaint of chest pain and dyspnea, from diagnosis to surgery.

CASE

A 61-year-old male patient was admitted to the cardiology outpatient clinic with complaints of chest pain and dyspnea. In the assessment, it was learned that the patient had a stent applied to the left descending artery (LAD) three years ago with the diagnosis of acute anterior MI and that he was diagnosed with hypertension and chronic obstructive pulmonary disease. It was learned that the patient was using acetylsalicylic acid 81 mg, nebivolol 5 mg, ramipril 5 mg, spironolactone/hydrochlorothiazide 25/25 mg, and atorvastatin 20 mg. During the physical examination of the patient, his blood pressure was measured as 100/70 mmHg and his heart rate was 84 beats/min. In the cardiovascular auscultation of the patient, a grade 2 systolic murmur was detected in the apical area as well as S1 and S2. The electrocardiogram showed normal sinus rhythm at 100 beats/min and biphasic T waves with 1 mm aneurysmatic ST elevation along with loss of R progression in leads V2-6.

In the echocardiography performed on the patient, the ejection fraction was measured as 40%. An aneurysmatic area

with hypoechoic fluid collection was observed in the apical region, with an appearance compatible with hyperechoic thrombus (Figure 1). In the patient's thorax computed tomography, a 50x35 mm cardiac apical partially thrombosed aneurysm with chronic infarction sequelae was observed (Figure 2 and Figure 3).

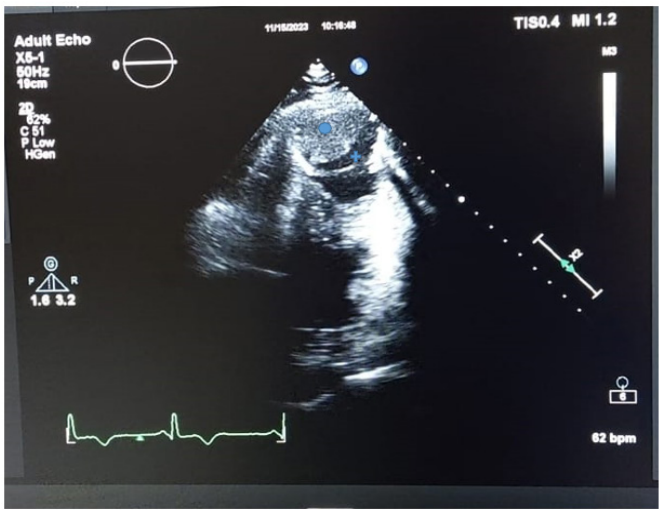

Figure 1. Two-chamber echocardiography image shows an aneurysmatic area with hypoechogenic fluid

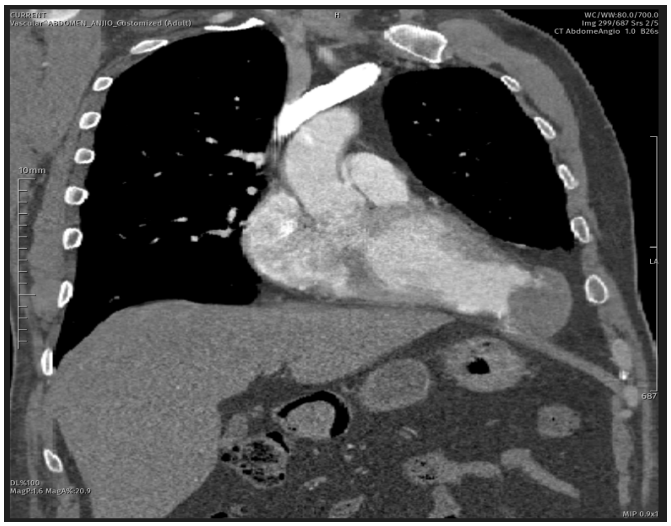

Figure 2. Pseudoaneurysm in the left ventricular apical region on contrast-enhanced thoracic computed tomography

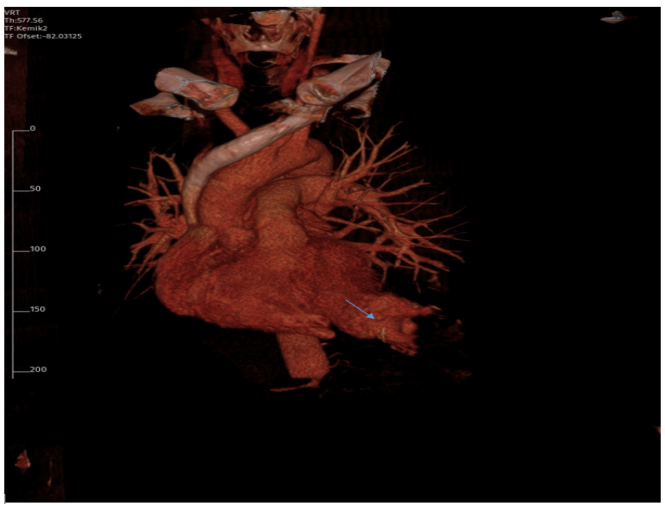

Figure 3. Three-dimensional view of left ventrilular pseudoaneurysm on three-dimensional thoracic computed tomography

Coronary angiography was planned for the patient who had class 2 angina according to the Canadian Heart Association angina classification. In the invasive angiography performed on the patient, the stent was open in the proximal region of the left descending artery and 90% stenosis was observed in the distal region (Figure 4).

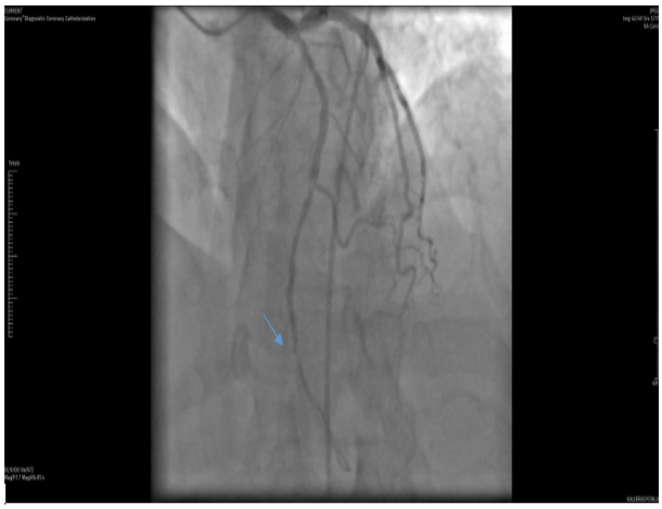

Figure 4. Critical stenosis after distal LAD stenting

The patient was evaluated by the council, which also included cardiovascular surgery. Since the LAD distal lesion was not suitable for bypass, it was decided to apply a stent to the LAD. A 2.5*15 mm stent was implanted in the distal region of the left descending artery (Figure 5).

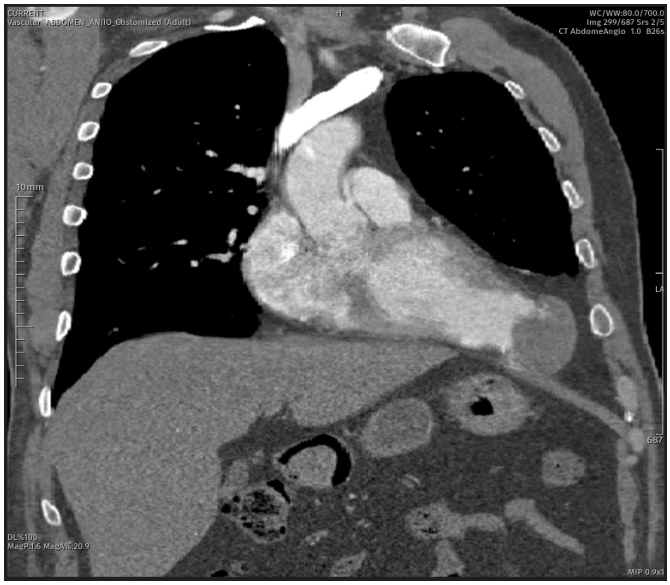

Figure 5. View after 2.5*15 mm stent implantation to the distal LAD

It was decided to follow the patient with medical treatment and to add anticoagulant therapy to his medication due to a thrombus in the left ventricular region. The patient's medication was arranged as warfarin 5 mg, clopidogrel 75 mg, nebivolol 5 mg, ramipril 5 mg, spironolactone/hydrochlorothiazide 25/25, atorvastatin 20 mg.

Since the patient's dyspnea continued at the first check-up after stent implantation, the patient was re-evaluated by the council and a decision was made for aneurysmectomy. The patient is still being followed up asymptomatically by the cardiology clinic after surgery.

DISCUSSION

True ventricular aneurysm is characterized by a ventricular aneurysm involving all myocardial layers, including the endocardium. LVP, on the other hand, is a structure that connects the ventricle to a larger aneurysmatic area with a narrow neck, contains blood and thrombus, and terminates with pericardial fibrous tissue rather than myocardial tissue. It occurs when the free wall rupture of the ventricle heals with an organized thrombus along with pericardial scar tissue. Rupture is rare after fibrosis and scar tissue have formed. The risk of rupture is higher in the first year with a rate of 30-45%.[5]

Although there is no consensus on the treatment of left ventricular pseudoaneurysm, if it occurs within the first three months after acute myocardial infarction and its diameter is larger than 3 cm, emergency surgery is the treatment method.[6] However, it is also possible to follow up patients with LVP with conservative treatment. In a study conducted on patients followed with medical treatment in this way, the one-year and four-year survival rates of the patients were found to be 89% and 74%. The basis of medical treatment is blood pressure management and anticoagulant treatment due to the high risk of thromboembolism.[7] While the surgical mortality rate of left ventricular pseudoaneurysms is 23%, the mortality rate of patients followed with medical treatment is 48%.[3] In LVP surgery, there are risks such as adhesion, increased fragility, and systemic embolization of thrombosed material in the pseudo aneurysmatic area during ventricular dissection as a result of the healing of ruptured tissue by fibrosis.8 Percutaneous closure is not a frequently used method; The experience of surgeons remains limited at this point. It limits this method to patients who are considered to be at high risk for surgery.[9]

In the current case, surgery (CABG and aneurysmectomy), percutaneous closure, and medical treatment were among the possible options. However, in the presented case, the fact that no objective distinction could be made as to whether the symptoms were caused by LVP or ischemia, and the fact that the risk of possible rupture was thought to be low since the patient was in the third year of post-MI suggested that this patient could be followed up with medical treatment. However, since the patient was symptomatic despite the medical treatment after the stent, the council decided on surgery. Each option chosen is associated with increased mortality. There is no consensus as to which method would be more appropriate for which patient. All these reasons make the management of patients with LVP more difficult.[9,10]

CONCLUSION

LVP can be seen in the early post-MI period, or it can be seen after three years, as in our case. Early diagnosis of LVP is of vital importance in determining prognosis and treatment methods. It is a diagnosis that can be easily missed unless evaluated with high clinical suspicion of two-dimensional echocardiography. Although echocardiography is the first evaluation method in diagnosis, methods such as invasive imaging methods, contrast-enhanced computed tomography, and cardiac MRI allow more efficient imaging.

In a patient with left ventricular pseudoaneurysm, determining the indication for surgery and ensuring that the right patient undergoes surgery, as well as diagnosis by imaging, are important due to the high risk of intraoperative mortality.

ETHICAL DECLARATIONS

Informed Consent

All patients signed and free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

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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.

REFERENCES

1. Dachman AH, Spindola-Franco H, Solomon N. Left ventricular pseudoaneurysm. Its recognition and significance. *JAMA*. 1981;246(17):1951-1953.
2. Csapo K, Voith L, Szuk T, Edes I, Kereiakes DJ. Postinfarction left ventricular pseudoaneurysm. *Clin Cardiol*. 1997;20(10):898-903.
3. Frances C, Romero A, Grady D. Left ventricular pseudoaneurysm. *J Am Coll Cardiol*. 1998;32(3):557-561.
4. Komeda M, David TE. Surgical treatment of postinfarction false aneurysm of the left ventricle. *J Thorac Cardiovasc Surg*. 1993;106:1189-1191.
5. Alapati L, Chitwood WR, Cahill J, Mehra S, Movahed A. Left ventricular pseudoaneurysm: a case report and review of the literature. *World J Clin Cases*. 2014;2(4):90-93.
6. Eren E, Bozbuga N, Toker ME, et al. Surgical treatment of post-infarction left ventricular pseudoaneurysm: a two-decade experience. *Tex Heart Inst J*. 2007;34(1):47-51.
7. Moreno R, Gordillo E, Zamorano J, et al. Long-term outcome of patients with postinfarction left ventricular pseudoaneurysm. *Heart*. 2003;89(10):1144-1146.
8. Sakaguchi G, Komiya T, Tamura N, Kobayashi T. Surgical treatment for postinfarction left ventricular free wall rupture. *Ann Thorac Surg*. 2008;85(4):1344-1346.
9. Dudiy Y, Jelnin V, Einhorn BN, Kronzon I, Cohen HA, Ruiz CE. Percutaneous closure of left ventricular pseudoaneurysm. *Circulation: Cardiovasc Intervent*. 2011;4(4):322-326.
10. Hulten EA, Blankstein R. Pseudoaneurysms of the heart. *Circulation*. 2012;125(15):1920-1925.