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Evaluation of arrhythmia frequency with Holter electrocardiography in pregnants with palpitation complaints

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ABSTRACT

Objectives: The aim of this study was to investigate the underlying etiology and the frequency of arrhythmia by Holter electrocardiography (ECG) in pregnant women with palpitations.

Patients and methods: Between January 2019 and March 2021, a total of 64 pregnant women (mean age: 29.1±5.3 years; range, 20 to 46 years) who were admitted to the cardiology outpatient clinic with the complaint of palpitations and had a Holter ECG were retrospectively analyzed. Data including demographic and clinical characteristics of the patients, Holter ECG records, imaging findings, and biochemical data were recorded.

Results: The mean systolic blood pressure was 118.7±16.4 mmHg and the mean heart rate was 96.2±18.2 bpm. There was an arrhythmia on Holter ECG in 32 (50%) of the patients. The most common arrhythmias were ventricular extrasystoles in 21.9% and supraventricular tachycardia in 14.1% of the patients. The frequency of paroxysmal atrial fibrillation was 4.7%. Non-sustained ventricular tachycardia (VT) was detected in two (3.1%) patients.

Conclusion: Identifying underlying arrhythmias in pregnant women with palpitation is of utmost importance for both the maternal and fetal health. The incidence of arrhythmias that should be treated in this patient group is too high to be ignored.

Keywords: Arrhythmia, holter electrocardiography, pregnancy.

Palpitations are common during pregnancy. Although this condition sometimes affects quality of life, it is usually benign in structurally normal hearts and requires treatment in only rare cases. Physiological changes during pregnancy may be the cause of palpitations. During pregnancy, blood volume increases by an average of 50%.[1,2] This can cause atrial stretch, which may be important for arrhythmogenesis.[1] The hormonal changes that occur during pregnancy can exert a proarrhythmic effect on myocardial tissue. The increased sympathetic outflow that occurs during pregnancy is also likely to contribute to proarrhythmic states.[1,3] Therefore, the risk of supraventricular and ventricular arrhythmias increases during pregnancy. Hormones, atrial stretch, and automatic tone alterations are the main mechanisms of arrhythmia.[2] Thus, pregnant patients can frequently apply to cardiology outpatient clinics with the complaint of palpitations.

In the present study, we aimed to investigate the underlying etiology and the frequency of arrhythmia by Holter electrocardiography (ECG) in pregnant patients with palpitations.

PATIENTS AND METHODS

This single-center, retrospective study was conducted at Silopi State Hospital, Cardiology outpatient clinic between January 2019 and March 2021. A total of 64 pregnant women (mean age: 29.1±5.3 years; range, 20 to 46 years) who applied to our clinic with the complaint of palpitations and had a Holter ECG were included in the study. Data including demographic and clinical characteristics of the patients, Holter ECG records, imaging findings,

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and biochemical data were obtained from the hospital database. Inclusion criteria were as follows: being >18 years old; being pregnant having the complaint of palpitation; having no signs of arrhythmia in baseline ECG; and having a 24-h Holter ECG record. Exclusion criteria were as follows: being younger than 18 years old; having severe kidney or liver failure; having known arrhythmia; being ineligible for an optimal echocardiographic examination; and having no evaluable Holter ECG. A written informed consent was obtained from each patient. The study protocol was approved by the Bakırcay University Non-Invasive Clinical Research Ethics Committee (Date No: 13/10/2021-358). The study was conducted in accordance with the principles of the Declaration of Helsinki.

Definitions

Supraventricular tachycardia (SVT) was defined as a narrow QRS complex rhythm with a rate of >100 bpm originating from or above the atrioventricular node. Paroxysmal atrial fibrillation (PAF) was defined as an atrial origin tachycardia attack with a duration of ≥30 sec on Holter ECG, with a narrow QRS complex and an irregular RR distance. Non-sustained ventricular tachycardia (NSVT) was defined as ventricular-derived tachycardia with a wide QRS complex lasting more than three consecutive beats and less than 30 sec.

Statistical analysis

Statistical analysis was performed using the IBM SPSS for Windows Version 25.0 software (IBM Corp., Armonk, NY, USA). To confirm the normal distribution of the study variables, the Kolmogorov-Smirnov test was used. Continuous variables were expressed in mean ± standard deviation (SD) or median (min-max) values, while categorical variables were expressed in number and frequency.

RESULTS

Of the patients, the mean systolic blood pressure was 118.7±16.4 (range, 90 to 165) mmHg and the mean heart rate was 96.2±18.2 (range, 57 to 138) bpm. Nine (14.1%) of the patients were smokers. Hypertension was present in 11 (17.2%) patients, and seven (10.9%) of these hypertensive patients had gestational hypertension. Ten (15.6%) patients had thyroid disease and three (4.7%) patients had diabetes mellitus. Anemia, one of the etiologies of arrhythmia, was detected in 34.4% of the study population. Demographic and clinical data of the patients are given in Table 1.

Among the biochemical parameters, the mean hemoglobin value was 11.7±1.7 g/dL and the mean thyroid-stimulating hormone was 1.3±0.8 mU/mL. The mean ferritin was 26.4±24.6 ng/mL and the mean vitamin B12 was 250.4±108.7 pg/mL. The mean left ventricular ejection fraction was 63.1±4.3%. Of the patients, three (4.7%) had moderate-to-severe mitral regurgitation, one (1.6%) had moderate-to-severe mitral stenosis, and six (9.4%) had moderate-to-severe tricuspid regurgitation. Laboratory and echocardiographic features of the patients are presented in Table 2.

Table 1
Demographic, clinical, and medication data of the study population (n=64)

Variables	n	%	Mean±SD
Demographic and clinical			
Age (year)			29.1±5.3
Hypertension	11	17.2	
Diabetes mellitus	3	4.7	
Hyperlipidemia	6	9.4	
Asthma	2	3.1	
Anemia	22	34.4	
Thyroid disease	10	15.6	
Parathyroid disease	2	3.1	
Stroke/TIA history	1	1.16	
PTE history	1	1.16	
Smoking	9	14	
Systolic BP (mmHg)			118.7±16.4
Diastolic BP (mmHg)			69.8±10.9
Mean pulse (per min)			96.2±18.2
Medications			
Beta-blockers	15	23.4	
NDH-CCBs	4	6.3	
DHP-CCB	6	9.4	
Iron preparations	15	23.4	
Vitamin B12	12	18.8	

SD: Standard deviation; TIA: Trans ischemic attack; PTE: Pulmonary thromboembolism; BP: Blood pressure; NDH: Non-dihydropyridine; CCB: Calcium channel blocker; DHP: Dihydropyridine.

Table 2
Laboratory and echocardiographic characteristics of the patients (n=64)

Variables	n	%	Mean±SD
Laboratory			
WBC (10^9/L)			10.2±3.3
Hemoglobin (g/dL)			11.7±1.7
Platelet count (10^9/L)			253.0±61.6
Urea (mg/dL)			7.9±2.9
Creatinine (mg/dL)			0.6±0.1
Sodium (mEq/L)			138.9±2.3
Potassium (mg/dL)			4.1±0.3
Fasting blood glucose (mg/dL)			94.7±16.9
AST (U/L)			18.5±7.3
ALT (U/L)			19.5±8.1
Total cholesterol (mg/dL)			175.5±39.4
HDL-cholesterol (mg/dL)			44.2±12.1
LDL-cholesterol (mg/dL)			102.9±26.2
Plasma triglyceride (mg/dL)			157.6±97.1
TSH (mU/mL)			1.3±0.8
Ferritin (ng/mL)			26.4±24.6
B12 (pg/mL)			250.4±108.7
Echocardiography			
LVEF (%)			63.1±4.3
Moderate-severe MR	3	4.7	
Moderate-severe MS	1	1.6	
Moderate-severe TR	6	9.4	

SD: Standard deviation; WBC: White blood cell; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; HDL: High-density lipoprotein; LDL: Low-density lipoprotein; TSH: Thyroid-stimulating hormone; LVEF: Left ventricular ejection fraction; MR: Mitral regurgitation; MS: Mitral stenosis; TR: Tricuspid regurgitation.

Table 3
Types of arrhythmias on Holter ECG (n=64)

Variables	n	%
Ventricular extrasystole	14	21.9
Supraventricular tachycardia	9	14.1
Paroxysmal atrial fibrillation	3	4.7
Premature atrial contraction	3	4.7
Non-sustained VT	2	3.1
Atrial tachycardia	1	1.6

VT: Ventricular tachycardia.

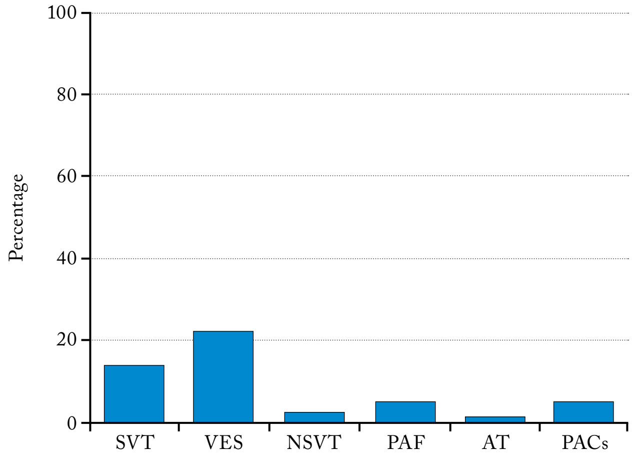

Figure 1. Types and frequency of arrhythmia detected by Holter electrocardiography in pregnant women with palpitation.

Any arrhythmia on surface ECG could be detected in any of the patients. There was an arrhythmia on Holter ECG in 32 (50%) of the patients. The most common arrhythmias detected in Holter ECG were ventricular extrasystole in 21.9% and SVT in 14.1%. The frequency of PAF was 4.7%. Two (3.1%) patients had NSVT. Types of arrhythmias are presented in Table 3 and Figure 1.

Fifteen (23.4%) of the patients were using beta-blocker drugs. The rates of the use of iron and vitamin B12 supplements were 23.4% and 18.8%, respectively.

DISCUSSION

In the current study, we investigated the underlying etiology and the frequency of arrhythmia by Holter ECG in pregnant patients with the complaint of palpitations. Our study showed an arrhythmia in half of the pregnant women as evidenced by the Holter ECG.

Cardiac arrhythmias are one of the most common cardiac complications encountered in pregnancy.[4] Pregnancy may trigger exacerbations of pre-existing arrhythmias in some patients, while in others, arrhythmias may occur for the first time.[5,6] Physiological alterations associated with normal pregnancy, such as increased heart rate, decreased peripheral resistance, increased stroke volume, hormonal changes, and psychological stresses, as well as increased sympathetic activity, are considered the most common triggers of arrhythmias in pregnant women.[6-8]

Ectopic beats and non-sustained arrhythmias are encountered in more than half of pregnant women who undergo examinations for palpitations that are usually benign and do not require treatment.[9,10] Similarly, the frequency of arrhythmia in the current study was 50%.

In previous studies of pregnant women with arrhythmias, the ECG, Holter ECG test, or telemetry was often used to assess the presence of arrhythmias. Holter ambulatory monitors typically record the ECG continuously over a 24- to 48-h period, and document arrhythmias occurring during this time.[11] In the current study, we evaluated the presence and type of arrhythmia by 24-h Holter ECG in pregnant patients who did not have any arrhythmia on surface ECG.

Ventricular tachycardias (VT) may occur as a new-onset arrhythmia during pregnancy or may be exacerbated by pregnancy. This situation is worrisome for both the maternal and fetal health.[12] Ventricular tachycardia can occur at any time during pregnancy. In a study including 11 pregnant women with new-onset of VT during pregnancy, the onset of VT was evenly distributed in the three trimesters and completely disappeared in the postpartum period.[13] In another study including 96 pregnant patients who were referred to the cardiology clinic for palpitations, syncope or dizziness, Holter ECG recordings were obtained in 19 patients, and NSVT was detected in only one patient as severe arrhythmia in the Holter ECG recording.[11] In the current study, there were two patients with NSVT.

It is still unclear whether pregnancy increases the risk of new-onset of SVT.[5,14] Patients with pre-existing SVT may experience exacerbations during pregnancy. Paroxysmal SVT) is the most common tachyarrhythmia in pregnancy presenting with palpitations, dyspnea, and presyncope.[15] In our study, paroxysmal SVT was detected in 14.1% of the patients. It is usually well tolerated, whereas it may cause hemodynamic deterioration and impaired fetal blood flow in patients with structural heart disease.[14,16]

A routine 24- to 48-h Holter monitoring is helpful in detecting frequent paroxysmal arrhythmias.[8,12] Thyroid dysfunction, electrolyte imbalance, anemia, anxiety, toxic drug use, and thromboembolism should be ruled out before the diagnosis of paroxysmal SVT is made.[15] Anemia was observed in 34.4% of the patients in our study, and thyroid disease was observed in 15.6%.

There are several limitations in this study. First, our sample size was relatively small. Second limitation is the retrospective nature of this study. Thirdly, it was not investigated whether arrhythmias in these patients continued after pregnancy.

In conclusion, identifying underlying arrhythmia in pregnant women with palpitation is of utmost importance for both the maternal and fetal health. The incidence of arrhythmias that should be treated in this patient group is too high to be ignored. Even if the ECG is normal, rhythm monitoring with Holter ECG is critical for the detection of silent underlying arrhythmias in pregnant women with palpitations.

Declaration of conflicting interests

The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

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12-Lead electrocardiography training via YouTube: How is it reliable?

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ABSTRACT

Objectives: The aim of this study was to investigate the adequacy and quality of video trainings on electrocardiography (ECG) recording on YouTube for the training of healthcare professionals based on the uploader source.

Materials and methods: Between May 19th, 2020 and May 25th, 2020, a total of 72 videos that were found to be eligible on YouTube were included. Two physicians independently assessed each video and scored the videos. The quality of the ECG recording was determined by giving 1 point for each of the 14 stages determined according to the current guidelines, and a total of 14 points was defined as maximum.

Results: In the first stage, the videos were divided into two groups based on the total score: compatible (≥8 points) and non-compatible (<8 points). Significant differences were found between the groups in the global quality score (GQS) ($p<0.001$) and total score ($p<0.001$). In the next stage, videos were divided into four different categories according to the uploader source: hospital or university, training site for healthcare professionals, physician or non-physician healthcare personnel, and unknown. A significant difference in the total score ($p<0.001$) and GQS ($p<0.001$) among these groups.

Conclusion: In the YouTube database, the scientific quality of the videos for standard 12-lead ECG recording training is highly variable. Training videos uploaded by hospitals and corporate healthcare training sites contain higher quality and scientific data than individual videos and videos of an unknown source.

Keywords: Electrocardiography, training, YouTube.

Electrocardiography (ECG) is an inexpensive, simple, and reproducible non-invasive diagnostic tool that was first developed in 1900 by Willem Einthoven[1] and still occupies an important place in our daily practice. It contains important diagnostic information that is routinely used for clinical assessment and it is the method used to detect the electrical state and instability of the myocardium.[2] Despite the advancement of many other techniques, ECG remains a reference diagnostic tool for some conditions such as transient myocardial ischemia.[3]

Determination of recording standards and the quality of recording are of utmost importance in the correct interpretation of the ECG. It is similarly important that the healthcare professional who is charged with ECG recording has sufficient training and a good command of ECG recording standards.[4]

The Internet has become an easily accessible educational resource in healthcare, as in every field in our era.[5] Training of various health procedures on live cases with video support is used as a very popular training tool. Training on 12-lead ECG recording is also widely used.

YouTube is the most popular video sharing site all over the world, and training videos shared from many different sources reach millions of individuals around the world. Freely available video streaming sites such as YouTube are widely used by medical students, practitioners, and all healthcare professionals, and YouTube offers an opportunity for educational use.[6] However, the quality and accuracy of medical information on the Internet is highly variable. Many of the videos on YouTube are based on personal experiences, but some come from professional sources such as universities, hospitals, and healthcare training sites.[4] Unlike rigorously reviewed magazine articles or textbooks, videos uploaded to YouTube are not subject to any control. Therefore, research is necessary to

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determine the accuracy and reliability of these training videos.

In the present study, we aimed to investigate the adequacy and quality of video trainings on ECG recording on YouTube for the training of healthcare professionals based on the uploader source.

MATERIAL AND METHODS

This cross sectional study was conducted at Kartal Koşuyolu Yüksek Ihtisas Training and Research Hospital, Department of Cardiology between May 19th, 2020 and May 25th, 2020. A total of 72 videos that were found to be eligible among 221 videos scanned on YouTube were included. Scanning words were as follows: "how to perform ECG", "how to record ECG", "ECG lead placement", "12-lead ECG", and "how to do ECG". Videos detected in a scan and detected again in a scan with another word were excluded. The study included only videos shot on live cases and models and excluded virtual videos. The study also excluded specific ECG measurements such as posterior ECG, dextrocardia, child ECG, and training videos in languages other than English. No personal YouTube or Google account was used for scanning. Scanning was done by logging out of personal Google and YouTube accounts and deleting account history. Two physicians independently assessed each video and scored the videos. The flow diagram of the videos is shown in Figure 1.

The quality of the ECG recording was determined by giving 1 point for each of the 14 stages determined according to the current guidelines[7] and a total of 14 points was defined as the maximum score. According to the ECG recording criteria, each stage was scored 0 (absent) and 1 (present). The stages determined for ECG scoring are shown in Table 1. One point was given for mentioning the procedure verbally during recording, even if the procedure was not performed. Each video was evaluated statistically based on the total score obtained from these stages. The upload date and duration of the video, total number of views, and the number of likes and dislikes were recorded.

The videos were divided into two groups by educational adequacy based on the total score: compatible (≥8 points) and non-compatible (<8 points) (Table 2).

In the next stage, to evaluate the quality of the videos in the study by their source, the videos were divided into four different categories according to the uploader source: hospital or university: 1, training site for healthcare professionals: 2, physician or non-physician healthcare personnel (individual): 3, and unknown: 4. The videos in four different groups were compared statistically according to the number of views, likes, dislikes, and global quality score (GQS) (Table 3). Finally, linear regression analysis was applied to the videos according to the total ECG score. Detailed findings are shown in Table 4.

Global quality score description

All videos were also rated using GQS that uses a five-point scale to rate the overall quality of the video (Table 5). The GQS is an assessment to ensure that the quality of information and the reviewer decides how useful a particular video would be for a patient.[8]

Statistical analysis

Statistical analysis was performed using the IBM SPSS for Windows version 22.0 software (IBM Corp., Armonk, NY, USA). Descriptive data

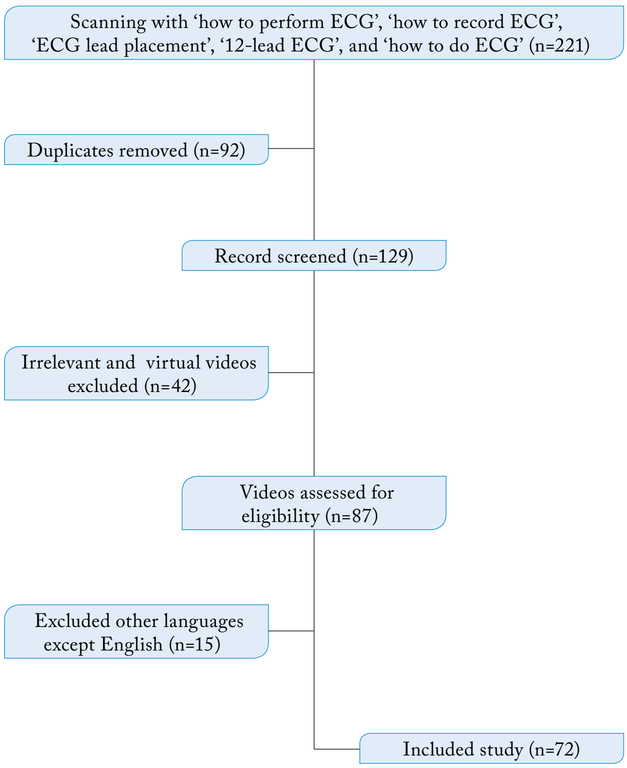

Figure 1. Flow diagram of study videos.
ECG: Electrocardiography.

Table 1
ECG checklist according to consensus reports

	Question	Rating
1	Patient identification	0 1
2	Communication and informed consent	0 1
3	Level of undressed	0 1
4	Patient position (lay down, semi-recumbent)	0 1
5	Skin preparation	0 1
6	Limb electrode position	0 1
7	Chest electrode position	0 1
8	Technique for locating chest electrode positions	0 1
9	Variations of standards (Have the device battery and standard values been checked ?)	0 1
10	Equipment specification	0 1
11	Environmental considerations	0 1
12	Infection control (hand wash ?)	0 1
13	Documentation, processing, storage and confidentiality of ECG recordings	0 1
14	Is the ECG quality checked?	0 1

ECG: Electrocardiography.

were expressed in mean ± standard deviation (SD), median (min-max) or number and frequency, where applicable. The chi-square test and Fisher's exact chi-square test were used to compare categorical variables between the groups. One-way analysis of variance (ANOVA) test was used to compare the means between the groups. In case of non-normal distribution, the Kruskal-Wallis test was used. A *p* value of <0.05 was considered statistically significant.

RESULTS

In the first stage, the videos were divided into two groups by educational adequacy based on the total score: compatible (≥8 points) and non-compatible (<8 points). No statistically significant differences were detected between the groups in the number of likes (compatible: 300±528 point, non-compatible 132.2±295 point; p=0.114), dislikes (compatible: 31.4±57.8, non-compatible: 11.4±22.4; p=0.062) and

Table 2
Features of the videos included in the study

	Non compatible (0-7 points) (n=41)	Compatible (8-14 points) (n=31)	
	Mean±SD	Mean±SD	*p*
Total score	4.7±1.5	10.3±1.8	<0.001
Views (*1000)	36.1±77.7	82.3±149.6	0.117
Duration of video (min)	4.0±2.8	6.7±4.7	0.005
Like	132.2±295	300±528	0.114
Dislike	11.4±22.4	31.4±57.8	0.062
GQS	1.6±0.6	3.4±0.9	<0.001

SD: Standard deviation; GQS: Global quality score.

Table 3
Video properties by uploader source

	Group 1 (n=6)	Group 2 (n=23)	Group 3 (n=13)	Group 4 (n=30)	
	Mean±SD	Mean±SD	Mean±SD	Mean±SD	*p*
Total score	12.2±2.1	8.9±2.8	6.3±2.7	5.2±2.1	<0.001
Views *1000	105.3±9.1	80.1±12.2	129.8±19.7	28.2±5.6	0.069
Duration	6.6±3.3	5.1±3.1	4.5±3.0	4.6±3.1	0.603
Like	452±460	310±563	261±440	37±75	0.057
Dislike	33±35	20±31	42±80	5±10	0.080
GQS	4.3±1.0	3.1±1.0	2.1±1.0	1.6±0.6	<0.001

SD: Standard deviation; GQS: Global quality score; Duration: duration of videos.

Table 4
Effects of predictors in predicting total ECG score presented by linear regression

Predictor	β-coefficient and 95% CI	*p*
Intercept	2.38 (1.12/3.64)	0.001
GQS	2.05 (1.62/2.49)	0.001
Source (unkown *vs.* known)	-1.03(-1.99/-0.07)	0.035
Duration (min)	0.067(-0.04/0.18)	0.244
Like	-0.0001 (-0.0001/0.0001)	0.843
Dislike	0.035 (-0.007/0.07)	0.106
View	-0.0001 (-0.0001/0.0001)	0.115

ECG: Electrocardiography; GQS: Global quality score; Unknown: Unknown source (Group 4); Known: Known source (Group 1, 2, and 3).

Table 5
Global quality scoring

Description of quality	Score
Poor flow, poor quality of the video, most information missing, not at all useful for healthcare professionals.	1
Generally poor quality and poor flow,some information listed but many important topics missing, very limited use of healthcare professionals.	2
Suboptimal flow, moderate quality, some important information is adequately discussed but others poorly discussed, somewhat useful for healthcare professionals.	3
Good flow and good quality. Most of the relevant information is listed, but some topics not covered, useful for healthcare professionals	4
Excellent flow and quality, very useful for healthcare professionals.	5

video duration (compatible: 6.7±4.7, non-compatible: 4.0±2.8; p=0.005). The relationship between video duration and total score is shown Figure 2.

In the next step, the videos were divided into four different groups according to the uploader source.Statistically significant differences were found between the groups in GQSs (compatible: 3.4±0.9 point, non-compatible: 1.6±0.6 point, p<0.001) (Table 2). A significant difference was found between these groups in the total score (group 1: 12.17±2.13, group 2: 8.87±2.71, group 3: 6.31±2.65, group 4: 5.17±2.1, p<0.001) and GQS (group 1: 4.33±1.03, group 2: 3.09±0.99, group 3: 2.08±1.03, group 4: 1.63±0.61, p<0.001). No statistically

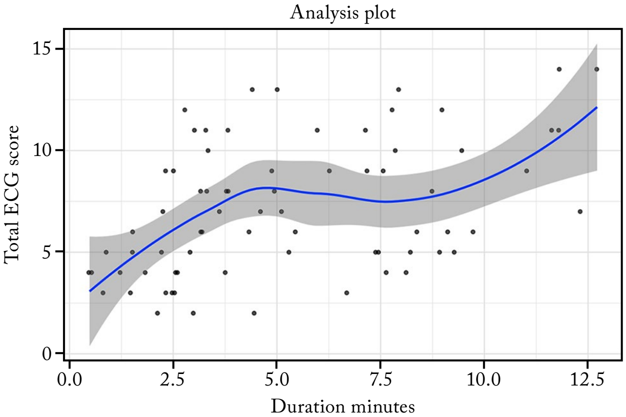

Figure 2. The relationship between video duration and total score.
ECG: Electrocardiography.

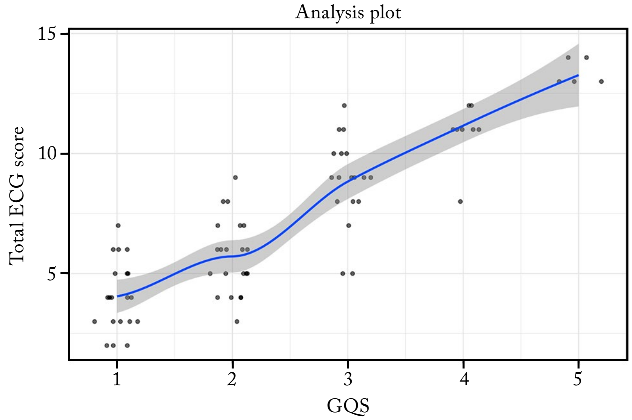

Figure 3. The relationship between total score and GQS.
GQS: Global Quality Score.

significant difference was detected between the four groups in video duration, the number of likes, dislikes, and views. The relationship between the total score and GQS is shown Figure 3.

The Cronbach alpha method was used for inter-rater reliability analysis. Values for GQS and ECG score were found to be 0.896 and 0.796, respectively.

DISCUSSION

In the present study, we investigated the adequacy and quality of video trainings on ECG recording on YouTube for the training of healthcare professionals based on the uploader source. According to the results, GQS was associated with the total ECG score (β-coefficient 2.05, 95% confidence interval (CI): 1.62 to 2.49; $p<0.001$), and videos uploaded from an unknown source were associated with lower total ECG scores (β-coefficient -1.03, 95% CI: -1.99 to -0.07; $p=0.035$ (Table 4).

Electrocardiography is a procedure that requires a long time and repetitive training to learn the recording, understand its physiology and interpret it clinically, and it still presents many unknowns for healthcare professionals.[9] It can only be correctly assessed, if the electrodes are placed in the correct anatomical positions and the recording is of good technical quality. It may be also necessary to compare serial ECGs to identify dynamic ECG changes over time, particularly in ischemic conditions, and it is important that all recordings should be made using a technique consistent with the same standard.[3]

Various studies have shown that differences between individuals and centers in ECG and departure from the standard technique cause clinically significant differences.[10,11] Many factors and technical problems in ECG recording can change the interpretation of ECGs, leading to erroneous diagnoses and putting patients at risk by making therapeutic interventions inappropriate. It is critical for patients and healthcare professionals to perform ECG recording at appropriate standards. Therefore, ECG training programs should include correct electrode placement and differences between normal and pathological patterns, and focus on recognizing ECG patterns derived from electrode misplacement, artifacts, and other technical problems that cause misinterpretation.[12]

There may not always be practical training opportunities for healthcare professionals for ECG recording, and repeated training may be needed. Depending on learning types of individuals, the principles learned from lectures or books can be difficult to apply to real-life situations. It is evident that the closer the educational environment or material is to the real one, the more effective would be the learning tool for the student.[13] In addition, online and distance learning has become much more important in these days of the novel coronavirus 2019 disease (COVID-19) pandemic and the transition to the digital age has accelerated.

In the pre-Internet era, books and articles were the main source of information.[14] As a result of the rapid advancement of technology and the widespread use of the Internet all over the world, training videos have become very popular in healthcare, as in all areas.[15] Currently, sources of access to information in many areas have become almost unlimited and it is often possible to access these information sources even from our home. However, the information age that we live in may bring unique difficulties. Therefore, we should question the accuracy, source and adequacy of the information we have reached. As in many other areas, healthcare training videos are shared a lot on video sharing and training sites; however, there is no specific tool to determine the adequacy and quality of the uploaded video. Some of the tools used to assess websites have been used in YouTube research, but the fact that they are not produced for specific online video media such as YouTube limits the use of these tools.

Founded in 2005, YouTube is the most popular social media platform. In addition to providing unlimited information, some of the videos are loaded from reliable sources (i.e., hospitals, universities, healthcare training sites) and some from uncontrolled non-scientific sources based on personal experiences. Unsupervised videos on YouTube and resource diversity can lead to false or misleading health information.[4] This resource diversity considerably restricts the quality of training and reliability of information and may negatively affect the learning skills of those who are willing to receive training in this field.[13]

Considering the number of views of videos, it is obvious that incorrect or incomplete information would have serious consequences, particularly in healthcare training videos.[9] As shown in our study, individual training videos that do not follow a specific guide or source are quite inadequate than the training videos that follow the current guidelines in hospitals and healthcare training sites. In addition, inadequate videos do not differ in the number of views compared to highly adequate videos. This may point to the risks of incomplete and inadequate videos that reach large audiences. Therefore, healthcare professionals who are trained on these videos should be strongly advised to question the video sources and the identity of the uploader. Encouraging educational institutions and universities to be a part of this education and accessing online ECG teaching videos by medical professionals or students directly from reliable scientific and institutional sources can contribute to the solution of this problem.[4]

Another remarkable finding of this study is that the videos mostly focus on the placement of the chest leads, and the other stages of ECG recording are not paid the same amount of attention. As this is the most important and difficult-to-learn stage of ECG recording, video uploaders probably have focused on this stage most with a similar thought. However, no sufficient emphasis has been placed on the basic steps of ECG recording, such as entering the patient's identity information correctly, reducing the patient's stress by informing them about the procedure, infection control, which we appreciate much better in the pandemic, and determining the recording standards of the device.

The exclusion of online video sites other than YouTube (e.g., Dailymotion) and languages other than English can be counted among the limitations of our study. All 72 videos included in this study only via YouTube may not reflect all the online training videos. In addition, the fact that some tools that measure the quality of the data published on the websites are not suitable for online video sites can be considered as another limitation in our study; however, to avoid this limitation, the GQSs were used.

In conclusion, in the YouTube database, the scientific quality of the videos for standard 12-lead ECG recording training is highly variable. Training videos uploaded by hospitals and corporate healthcare training sites contain higher quality and scientific data than individual videos and videos of an unknown source; therefore, healthcare professionals should question the source and scientific quality of the training videos. Specific tools need to be developed for healthcare training videos uploaded to YouTube; thus, it may be possible to prevent adverse conditions that may arise with online videos that are complimentary, but not alternative to classical training.

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Hemoglobin A1c levels do not predict primary arteriovenous fistula failure in hemodialysis patients

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ABSTRACT

Objectives: In this study, we aimed to assess whether higher preoperative levels of glycated hemoglobin (HbA1c) could predict primary arteriovenous fistula (AVF) failure and to investigate the effect of diabetes mellitus on primary arteriovenous failure.

Patients and methods: Between July 2018 and August 2019, a total of 127 newly created AVFs in 117 patients (67 males, 50 females; mean age: 62.4±12.2 years; range, 18 to 86 years) who underwent primary AVF operation in our cardiovascular surgery clinic were retrospectively analyzed. Medical data were obtained from the institutional database. Arteriovenous fistula failure was evaluated during follow-up.

Results: Primary AVF failure was seen in 24 (18.9%) patients. Patients with diabetes mellitus had a higher ratio of failure compared to those without (62.5% *vs.* 38.8%, respectively; p=0.035). After adjustment, diabetes mellitus was not found to be an independent risk factor for AVF failure (p>0.05). There was no significant correlation between HbA1c levels and fistula failure (p>0.05).

Conclusion: Our study results suggest that diabetes is associated with AVF failure, but it is not an independent risk factor for AVF failure. Higher HbA1c levels fail to predict AVF failure.

Keywords: Arteriovenous fistula, blood glucose, diabetes mellitus, glycated hemoglobin.

Diabetes mellitus (DM) is the leading etiology in chronic kidney disease (CKD), and it is commonly accompanied by vascular complications.[1,2] Other than changes in vascular homeostasis and accompanying medical conditions, prolonged hyperglycemia, itself, also plays a role in the pathophysiology of the vascular damage in these patients.[2] Glycated hemoglobin (HbA1c) is the preferred blood marker of glycemic control in patients with DM and the current guideline of Kidney Disease Outcomes Quality Initiative (KDOQI) recommends a target HbA1c of <7% for DM patients, irrespective of the presence of CKD.[1]

Arteriovenous fistula (AVF) is the recommended vascular access type for hemodialysis according to the recent guideline of KDOQI.[3] In addition to AVF having superior patency and lower re-intervention rates compared to other vascular access types, it has been found to be associated with lower rates of complication, infection, hospitalization, and depression and reduced costs and increased survival benefits.[4-7] Nevertheless, fistula failure is still a challenge. Identifying the factors affecting primary AVF failure is crucial to improve the implementation of optimal hemodialysis therapy. Since DM is the most commonly accompanying disease on CKD, assessment of the effect of DM-related parameters on AVF patency is of particular importance.

Considering that hyperglycemia is one of the reasons of vascular damage in patients with diabetes, we hypothesized that levels of HbA1c could affect AVF maturation. In the present study, we aimed to investigate whether higher preoperative levels of HbA1c could predict primary AVF failure and to

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examine the optimal cut-off value of HbA1c level to predict AVF failure.

PATIENTS AND METHODS

This single-center, retrospective study was conducted at Istanbul Medeniyet University Göztepe Prof. Dr. Süleyman Yalçın City Hospital, Department of Cardiovascular Surgery between July 2018 and August 2019. Initially, a total of 199 primary AVF operations were screened. Exclusion criteria were as follows: age <18 years (n=8), undergoing arteriovenous graft operation and having incomplete data (n=15). In addition, six patients who died before the study was completed and 43 patients who were lost-to-follow-up were excluded. Finally, a total of 127 newly created AVFs in 117 patients (67 males, 50 females; mean age: 62.4±12.2 years; range, 18 to 86 years) were included in the study. A written informed consent was obtained from each patient. The study protocol was approved by the Istanbul Medeniyet University Göztepe Prof. Dr. Süleyman Yalçın City Hospital Ethics Committee (No. 0034-2019). The study was conducted in accordance with the principles of the Declaration of Helsinki.

Data collection

Data including demographic and clinical characteristics of the patients such as age, sex, height, weight and smoking habit; comorbidities (e.g., hypertension, DM, and peripheral artery disease); usage of antiplatelet or anticoagulant medications, history of undergoing hemodialysis through central venous catheter (CVC); and AVF failure were noted. Patients with DM or hypertension were identified as the ones who were under treatment with oral antidiabetic drugs or insulin, and antihypertensive drugs, respectively. Preoperative levels of red blood cells, HbA1c, blood glucose, white blood cells, platelet count, creatinine, and estimated glomerular filtration rate were documented. The tests were done up to two weeks prior to operations. All the values were measured by the hospital laboratory, and there was no considerable change in the measurement methods during the study.

Preoperative Doppler ultrasound mapping was routinely performed to each patient, and vascular diameters were noted. All operated patients had a vein diameter over 2.5 mm under tourniquet and an artery diameter over 2.0 mm. All operations were performed by a single surgeon and, thus, the surgical approach to AVF creation was similar. The primary outcome of this study was primary AVF failure at six weeks. Primary AVF failure was defined as thrombosis or failure to mature at six weeks.[8] Failure to mature was defined as insufficient flow to maintain dialysis or the inability to cannulate an AVF, if required.[9]

Statistical analysis

Statistical analysis was performed using the IBM SPSS version 24.0 software (IBM Corp., Armonk, NY, USA). Descriptive data were expressed in mean ± standard deviation (SD), median (min-max) or number and frequency, where applicable. The Kolmogorov-Smirnov test was applied to test normality. The Pearson chi-square, Mann-Whitney U, Kruskal-Wallis, and binary logistic regression tests were implemented, accordingly. Multiple logistic regression analyses were applied to evaluate independent risk factors for AVF failure. The variables included in the regression models were selected using the forward selection method. Multicollinearity diagnosis tests were performed for both multiple logistic regression models. The receiver operating characteristic (ROC) curve analysis was performed to identify the optimal cut-off value of HbA1c level to predict AVF failure. A p value of <0.05 was considered statistically significant.

RESULTS

The rate of DM and hypertension was 43.3% (n=55) and 76.4% (n=97), respectively. A total of 94 (74.0%) of the operations were distal radiocephalic AVF operations, while there were 23 (18.1%) proximal radiocephalic, four (3.1%) brachiobasilic and six (4.7%) brachiocephalic AVF operations. Primary failure was seen in 24 operations (18.9%). Demographic and clinical characteristics of the patients with and without primary AVF failure are summarized in Table 1.

Diabetes mellitus was found to be associated with AVF failure. Patients with DM had a statistically significantly higher ratio of failure (15/55; 27.3%), compared to those without DM (9/72; 12.5%) (p=0.035). Nevertheless, after adjustment, neither DM nor other parameters were found to be independent risk factors for AVF failure (Table 2). In addition, there was no significant correlation

Table 1
Demographic and clinical characteristics of patients with and without primary AVF failure

	Interventions with primary AVF failure (n=24)			Interventions without primary AVF failure (n=103)			
Variables	n	%	Mean±SD	n	%	Mean±SD	*p*
Age (year)			63.9±9.4			62.0±12.8	0.490
Sex							
Female	11	45.8		39	37.9		0.472
Comorbidities							
Diabetes mellitus *	15	62.5		40	38.8		0.035
Hypertension	17	70.8		80	77.7		0.478
Peripheral arterial disease	4	16.7		12	11.7		0.505
Obesity	9	37.5		30	29.1		0.477
Hemoglobin (g/dL)			10.9±2.0			10.5±1.8	0.319
HbA1c (%)			6.0±0.7			6.3±1.5	0.406
Blood glucose (mg/dL)			130.9±46.6			120.8±52.0	0.394
Platelet count (10^3/µL) *			196.5±66.3			246.8±178.4	0.029
Body mass index (kg/m^2)			28.2±4.1			27.0±5.5	0.318
Smoking status							
Currently smoking	4	16.7		16	15.5		
Ex-smoker	10	41.7		33	32.0		
Never smoked	10	41.7		54	52.4		
History of CVC	14	58.3		44	42.7		0.123
History of hemodialysis	15	62.5		53	51.5		0.249
Usage of antithrombotic drugs	18	75.0		72	69.9		0.621

AVF: Arteriovenous fistula; SD: Standard deviation; HbA1c: Glycated hemoglobin; CVC: Central venous catheter; * $p<0.05$.

between HbA1c levels and primary AVF failure (p=0.406). The ROC curve analysis established no cut-off value of HbA1c level to predict primary AVF failure.

Patients with DM were older (p<0.001), had higher prevalence of hypertension (p=0.035), peripheral artery disease (p=0.001), and antithrombotic drug use (p=0.018), had higher levels of blood glucose (p<0.001) and HbA1c (p<0.001), higher body mass index values (p=0.014) and lower creatinine levels (p=0.020).

In subgroup analysis containing only patients with DM (n=55), mean blood glucose level was

Table 2
Multivariate logistic regression for primary arteriovenous fistula failure

Covariates	Adjusted Odds Ratio	95% CI	*p*
Age	0.988	0.938-1.040	0.640
Diabetes mellitus	2.455	0.488-12.347	0.276
HbA1c (%)	0.782	0.403-1.518	0.467
Blood glucose (mg/dL)	0.999	0.987-1.012	0.933
Hypertension	0.730	0.191-2.789	0.645
Peripheral artery disease	0.926	0.144-5.963	0.935
Platelet count (10^3/µL)	0.995	0.987-1.003	0.245
Antiplatelet usage	1.025	0.327-3.212	0.967

HbA1c: Glycated hemoglobin; CI: Confidence interval.

Table 3
Selected characteristics of the patients with diabetes mellitus

	Interventions with primary AVF failure (n=15)			Interventions without primary AVF failure (n=40)			
Variables	n	%	Mean±SD	n	%	Mean±SD	*p*
Age (year)			67.0±7.7			67.8±10.4	0.783
Sex							
Female	7	46.7		35	87.5		0.912
Comorbidities							
Hypertension	12	80.0		35	87.5		0.482
Peripheral arterial disease	4	26.7		9	22.5		0.746
Obesity	6	40.0		14	35.0		0.731
Hemoglobin (g/dL)			10.7±1.9			10.5±1.8	0.735
HbA1c (%)			6.2±0.7			7.2±1.7	0.054
Blood glucose (mg/dL)			152.7±47.4			158.1±60.8	0.759
Antidiabetic drug type							0.452
Insulin	11	73.3		25	62.5		
Oral antidiabetic	4	26.7		15	37.5		
Platelet count (10^3/µL) *			185.5±66.5			242.9±72.4	**0.016**
Body mass index (kg/m^2)			29.2±3.7			28.4±5.3	0.584
History of CVC	10	66.7		16	40.0		0.051
History of hemodialysis	11	73.3		22	55.0		0.142
Usage of antithrombotic drugs	13	86.7		32	80.0		0.568

AVF: Arteriovenous fistula; SD: Standard deviation; HbA1c: Glycated hemoglobin; CVC: Central venous catheter; * p<0.05.

Table 4
Multivariate logistic regression for primary arteriovenous fistula failure in patients with diabetes mellitus

Covariates	Adjusted Odds Ratio	95% CI	*p*
HbA1c (%)	0.543	0.237-1.244	0.149
Platelet count (10^3/µL)	0.993	0.980-1.006	0.275

HbA1c: Glycated hemoglobin; CI: Confidence interval.

156.7±57.3 mg/dL and mean HbA1c value was 7.0±1.6%. Characteristics of the patients with DM are summarized in Table 3. No significant correlation was detected between blood glucose levels and HbA1c levels in patients with DM (p=0.433). The HbA1c level was not associated with AVF failure (p=0.054). Lower levels of platelet count were observed in patients with AVF failure (p=0.016). However, adjustment tests eliminated the statistical significance of the observations, and analysis revealed no independent risk factor for AVF failure in hemodialysis patients with DM (Table 4). No multicollinearity was detected in both regression models.

DISCUSSION

In the present study, the primary AVF failure was seen in 18.9% of the operations. Our study revealed that patients with DM had a statistically significantly higher ratio of primary AVF failure, but DM was not an independent risk factor for AVF failure. Higher levels of HbA1c failed to predict AVF failure.

A recent meta-analysis demonstrated a higher rate of AVF failure in patients with diabetes,[10] which is consistent with our study. Other than hyperglycemia inducing vascular damage itself, metabolic changes accompanying DM such as endothelial damage and endothelial dysfunction impair venous remodeling and may result in failure to mature.[2,10] Besides, DM is associated with an increased extracellular matrix deposition, deregulated growth factors, and pro-thrombotic activity and it is a known risk factor for atherosclerosis, which all may promote AVF failure.[11]

Our study demonstrated that preoperative levels of HbA1c failed to be a predictor of AVF failure. No consensus has been reached in the literature on whether HbA1c level has an impact on AVF failure. Some authors have advocated that HbA1c ≥7% is associated with higher rates of AVF failure or lower patency rate,[12,13] whereas a recent prospective study has shown that HbA1c level is not related to delayed maturity.[14] Afsar and Elsurer[12] explain their findings mainly based on the assumptions that greater HbA1c levels indicate presence of DM and that greater HbA1c levels suggest poor glycemic control. The authors also suggested that peripheral artery disease, which they found to be associated with the increased HbA1c levels, might affect AVF failure. Singh et al.[14] observed that those with HbA1c <6.5% had higher rates of AVF maturity at six weeks post-creation. Nevertheless, they demonstrated no significant relationship between HbA1c status and delayed maturity. Wu et al.[13] reported a direct effect of hyperglycemia on endothelium, yet underlined their apprehension on whether baseline glycemic status could reflect the severity of underlying diabetes.

Hyperglycemia is known to create tendency to thrombosis via blood flow retardation and platelet aggregation,[10] which may eventually cause AVF failure. Although recent guidelines recommend the same target level of HbA1c for patients with and without CKD, it is a matter of debate whether HbA1c level predicts blood glucose control accurately in CKD patients. It has been suggested by many authors that the correlation between HbA1c and blood glucose is impaired in CKD patients and that HbA1c may not be a reliable indicator of blood glucose control in these patients.[15,16] Reduced red blood cell survival and common use of erythropoietin-stimulating agents increase the rate of young erythrocytes in these patients. These erythrocytes have less exposure time to glucose, which affects HbA1c levels. Several studies have indicated that the measured HbA1c levels of diabetic CKD patients are lower than indicated by their blood glucose levels, and thus HbA1c level mispresents glycemic control.[15,17] Although it is beyond of our scope to assess the predictive value of HbA1c level on blood glucose level in CKD patients, our analysis revealed no significant correlation between blood glucose levels and HbA1c levels in hemodialysis patients with DM. Although hyperglycemia seems to be an important parameter affecting AVF failure, it may be misleading to assess HbA1c as an accurate predictor of blood glucose control in CKD patients. This may explain our finding that HbA1c level is not associated with AVF failure.

Vascular hyperglycemic memory is a relatively recent definition, describing the persistence of the effects of hyperglycemic stress, even though the blood glucose is normalized.[2] This phenomenon makes the predictive value of the biochemical markers, which can only suggest recent blood glucose control, on vascular outcomes questionable, since recent blood glucose normalization does not rule out previous long-term hyperglycemia. Although the reliability of different markers remains being a matter of debate, it seems that any of these markers would be effective to predict AVF failure.

In our study, lower levels of platelet count were observed in patients with AVF failure. Although it is beyond doubt that thrombosis plays a role in the failure mechanism, an inverse association between platelet count and AVF failure is unexpected. Future studies should be made to analyze the effect of thrombosis-related parameters on AVF failure.

The single-center, retrospective design with a relatively small sample size with AVF failure are the main limitations of our study. Another limitation is that no subgroup analysis was able to be made considering AVF locations. The AVFs with different arteries and veins may have different characteristics which can affect AVF maturation and, thus, AVF failure. However, our study includes only autogenous AVFs, created by a single surgeon, eliminating the effect of surgical approach on fistula failure. Besides, we reviewed an adequate number of primary AVF operations, suggesting that there is no predictive value of preoperative HbA1c on primary AVF failure.

In conclusion, diabetes is associated with AVF failure irrespective of the patients' HbA1c levels. Although hyperglycemia seems to play a role in pathogenesis, there is a growing concern that HbA1c level may not be accurately indicating blood glucose control in CKD patients. In addition, considering that biochemical markers fail to present previous long-term hyperglycemia, predictive value of these markers on vascular outcomes is limited. Based on these findings, the role of preoperative HbA1c is limited in predicting primary AVF failure.

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Why should we perform pulmonary function test before coronary artery bypass grafting?

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ABSTRACT

Objectives: In this study, we aimed to examine the relationship between preoperative pulmonary function test (PFT) parameters and postoperative length of invasive mechanical ventilation (IMV), length of intensive care unit (ICU) and hospital stay in patients who underwent coronary artery bypass grafting (CABG).

Patients and methods: Between October 2017 and July 2018, a total of 100 patients (84 males, 16 females; mean age: 61.1±10.0 years; range, 41 to 85 years) who underwent elective CABG surgery for the first time and who did not have any additional cardiac problems, except for coronary artery disease, had an ejection fraction (EF) of ≥30% and underwent preoperative PFTs were retrospectively analyzed. The percent forced vital capacity (FVC %) predicted from the PFT values and percent forced expiratory volume in 1 sec (FEV_1%) were recorded. An IMV duration of ≤12 h was considered normal and >12 h was considered prolonged. A length of ICU stay for ≤24 h was considered normal and >24 h was considered prolonged. A length of hospital stay for ≤7 days was considered normal and >7 was considered prolonged.

Results: As the predicted FVC (%) value decreased in the preoperative PFTs, the length of IMV, length of ICU and hospital stay increased significantly (p=0.040, p=0.036, p=0.009, respectively). In terms of the predicted FEV_1 (%) value, as the predicted FEV_1 (%) value decreased, the duration of IMV, length of ICU and hospital stay prolonged (p=0.023, p=0.044, p=0.024, respectively).

Conclusion: It is possible to have an idea about postoperative duration of ventilation, ICU and hospital stay based on PFT parameters. Also, the result would be more realistic when adapted to existing scoring systems to assess postoperative complications. Therefore, we believe that scoring systems for evaluating complications after cardiac surgery should include not only chronic lung disease, but also PFT parameters that give a more detailed information.

Keywords: Coronary artery bypass grafting, intensive care unit, pulmonary function test.

Coronary artery bypass grafting (CABG), which was first applied in the 1960s, is still a valuable and effective treatment method in the treatment of coronary artery diseases.[1] However, postoperative pulmonary complications such as atelectasis, respiratory failure, and respiratory tract infections that may develop after open heart surgery are the facts we encounter in our daily practice.[2]

Factors such as median sternotomy, hypothermia applied to protect the myocardium, pleurotomy and internal mammary artery dissection can have a negative effect on lung functions in the postoperative period.[2,3] Besides, CABG disrupts alveolar stability by creating changes in complement system activation, neutrophil increase in the pulmonary vascular bed, formation of free oxygen radicals and alveolar surfactant production.[4] It is inevitable that atelectasis would develop as a result of changes in the amount of alveolar surfactant and a decrease in alveolar stability. Physiological shunts occur in patients during this period and the alveolo-arterial oxygen gradient increases.[5] Pulmonary complications may occur as a result of all them, leading to a serious cost increase by extending the need for intensive care unit (ICU) and the length of hospital stay.[6]

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In scoring systems that are widely used to assess preoperative cardiac surgery risk (Japan Cardiac Operative Risk Assessment System [JapanSCORE], the Society of Thoracic Surgeons [STS] risk model, and the European Cardiac Risk Assessment System [EuroSCORE II], it is roughly questioned whether there is chronic lung disease. Pulmonary function test (PFT) parameters, which is a very easily applicable and accessible method, are not included in any of these scoring systems.[7] However, the need for postoperative ICU and pulmonary complications that may arise can be predicted by the results of PFT in the preoperative period.[8]

In the present study, we aimed to examine the relationship between preoperative PFT parameters and postoperative duration of ventilation, ICU and hospitalization stay.

PATIENTS AND METHODS

This single-center, retrospective study was conducted at University of Health Sciences, Dr. Siyami Ersek Thoracic and Cardiovascular Surgery Training and Research Hospital, Department of Cardiovascular Surgery between October 2017 and July 2018. A total of 100 patients (84 males, 16 females; mean age: 61.1±10.0 years; range, 41 to 85 years) who underwent elective CABG surgery for the first time and who did not have any additional cardiac problems, except for coronary artery disease, had an ejection fraction (EF) of ≥30% and underwent preoperative PFTs were included in the study. The PFT was applied to all patients by the traditional method who were scheduled for elective CABG in our hospital. There was no difference between the patients in terms of anesthesia technique or sedative medication. Predicted forced vital capacity percentage (FVC%) and forced expiratory volume in 1 sec percentage (FEV_1%) were recorded. Patients records were obtained from the hospital database. A written informed consent was obtained from each patient. The study protocol was approved by the Istanbul Yedikule Chest Diseases and Thoracic Surgery Training and Research Hospital Clinical Research Ethics Committee (No: 2021-121). The study was conducted in accordance with the principles of the Declaration of Helsinki.

Patients who were taken to ICU with intra-aortic balloon pump (IABP), extracorporeal membrane oxygenation (ECMO) support devices after the operation, patients who had prolonged ICU stay due to reasons other than respiratory functions during ICU follow-up, patients who were re-transferred to the ICU during hospitalization for non-respiratory reasons and patients with prolonged hospitalization due to reasons other than respiratory functions were excluded from the study. The following parameters were considered in the assessment of data:

- An invasive mechanical ventilation (IMV) duration of ≤12 h was considered normal and >12 h was considered prolonged.
- A length of ICU stay of ≤24 h was considered normal, >24 h is prolonged.
- For the total hospitalization (ICU + ward), a length of stay for ≤7 days was considered normal and >7 days was considered prolonged.

Operation technique

All patients were operated electively. By performing a median sternotomy, arterial cannulation from the ascending aorta and venous cannulation from the right atrium with a two-stage venous cannula was connected to a cardiopulmonary bypass (CPB) device. Moderate hypothermia (28°C) was achieved, cardiac arrest was achieved with 10 to 15 mL/kg cold blood cardioplegia or 5 mL/kg cold crystalloid cardioplegia (St. Thomas II) after insertion of the aortic cross-clamp. After the surgical procedure, reperfusion was achieved with 5 mL/kg warm blood cardioplegia before the cross-clamp was removed. A roller head pump and hollow fiber membrane oxygenator were used in the CPB device. Left internal mammary artery (LIMA) and saphenous vein were used as the graft in all patients.

Statistical analysis

Statistical analysis was performed using the IBM SPSS version 22.0 software (IBM Corp., Armonk, NY, USA). Continuous variables were expressed in mean ± standard deviation (SD) or median (interquartile range [IQR]), while categorical variables were expressed in number and frequency. The compliance with the normality assumption was tested using the Kolmogorov-Smirnov test. In examining the difference between categorical variables with two groups, the independent samples t-test was used for variables suitable for the assumption of normality, and the Mann-Whitney U test was used for variables not compatible with the assumption of normality. The chi-square test was used to examine the relationship between two categorical variables. A p value of <0.05 was considered statistically significant.

RESULTS

Of the patients, 72 had a normal weight (body mass index [BMI] <30 kg/m^2) and 28 had obesity (BMI ≥30 kg/m^2). The mean EF value was 52.6±8.5%. The mean operation time of the patients was 283.0±75.6 min, the mean aortic clamp time was 78.1±52.2 min, and the mean CPB time was 118.0±60.7 min. The mean predicted FEV_1 (%) and FVC (%) values were 90.1±19.4% and 83.1±19.9%, respectively (Table 1).

The mean preoperative laboratory test values and chronic disease status of the patients are shown in Table 2 and Table 3, respectively. None of the patients received blood products and inotropic support in the preoperative period.

Six patients died during the 12-month follow-up period after the operation, 94 of them were still alive. Among deceased patients, the predicted FEV_1 (%) value was 88.3±26.5%. Among survivors, this value was 90.3±19.0%, indicating no statistically difference (p=0.816). The median predicted FVC values were 74.80 (IQR: 70.88-93.66) for non-survivors and 84.10 (IQR: 71.00-93.60) for survivors (p=0.701). Although there was no statistically significant difference, predicted FVC values of survivors were higher.

Table 1
Demographic and baseline characteristics of patients

	n	Mean±SD
Age (year)		61.1±10.0
Sex		
Male	84	
BMI (kg/m^2)		
<30	72	
≥30	28	
IMV stay (h)		
≤12	82	
>12	18	
ICU stay (h)		
≤24	73	
>24	27	
Total hospital stay (days)		
≤7	68	
>7	32	
Procedures done		
CABGx1	4	
CABGx2	28	
CABGx3	45	
CABGx4	19	
CABGx5	4	
Operative time (min)	100	283.0±75.6
Aortic clamping time (min)	100	78.1±52.2
Cardipulmonary bypass time (min)	100	117.950±60.68
Left ventricular ejection fraction (%)	100	52.550±8.54
Predicted FVC (%)	100	83.132±19.91
Predicted FEV_1 (%)	100	90.137±19.40
Mortality*		
Yes	6	
No	94	

SD: Standard deviation; BMI: Body mass index; IMV: Invasive mechanical ventilation; ICU: Intensive care unit; CABG: Coronary artery bypass grafting; FVC: Forced vital capacity; FEV1: Forced expiratory volume in 1 sec. * At postoperative 12 months.

Table 2
The mean preoperative laboratory test results

	Mean±SD
WBC (μg/L)	9.2±3.5
HGB (g/dL)	13.7±1.5
HCT (%)	40.5±4.5
Platelet (μg/dL)	252.3±3.1
Creatine (mg/dL)	0.9±0.2
Urea (mg/dL)	18.1±6.8
AST (u/L)	24.2±17.3
ALT (u/L)	27.2±14.7

SD: Standard deviation; WBC: White blood cell; HBG: Hemoglobin; HCT: Hematocrit; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase.

Table 3
Preoperative chronic disease status of patients

	n
None	28
DM	23
HT	29
COPD	6
DM + HT	11
DM + COPD	1
HT + COPD	1
DM + HT + COPD	1
Total	100

DM: Diabetes mellitus; HT: Hypertension; COPD: Chronic Obstructive Pulmonary Disease.

Duration of ventilation: The relationship between PFT parameters and duration of IMV was evaluated. Accordingly, the median predicted FVC (%) value of the patients with prolonged IMV duration was 64.00 (IQR: 58.20-90.90), while the median predicted FVC (%) value of the patients with normal IMV duration was 85.00 (IQR: 74.30-93.95) (p=0.040) (Figure 1a). The mean predicted FEV_1 (%) value of the patients with prolonged IMV duration was 82.1±22.5, while the mean predicted FEV_1 (%) value of the patients with normal IMV duration was 92.5±17.8 (p=0.023) (Figure 1b).

- The median predicted FVC (%) value of patients with prolonged IMV duration is 64.00 (IQR: 58.20-90.90), the median predicted FVC (%) value of patients with normal IMV duration is 85.00 (IQR: 74.30-93.95) (p=0.040) (Figure 1a).
- The mean predicted FEV_1 (%) value of patients with prolonged IMV duration is 82.1±22.5, the mean predicted FEV_1 (%) value of patients with normal IMV duration is 92.5±17.8 (p=0.023) (Figure 1b).

The patients with low predicted FEV_1% and predicted FVC% values had have statistically significantly longer IMV.

- When the predicted FEV_1 (%) was divided into two groups as ≥80 and <80%, the duration of IMV was found as follows: in patients with predicted FEV_1 (%) ≥80%, the IMV duration was normal in 57 patients, while

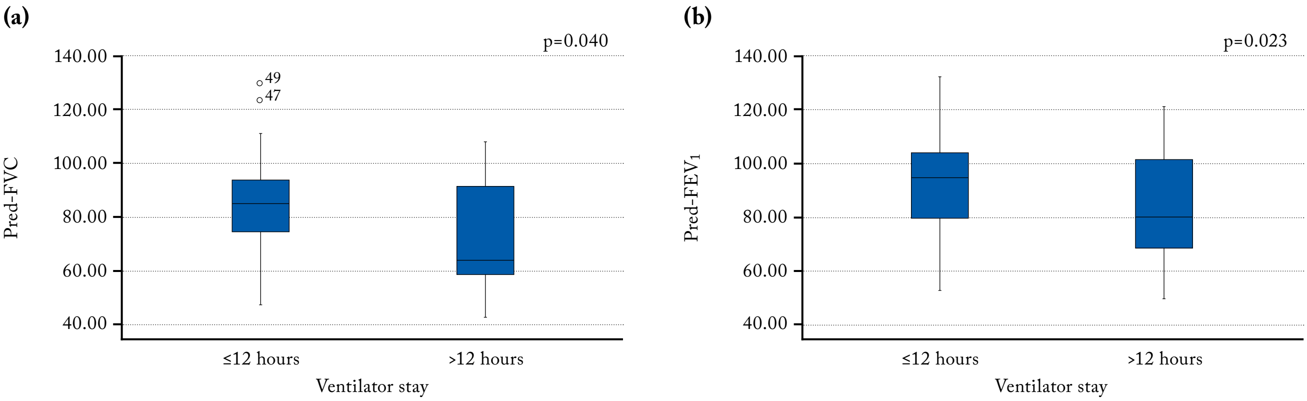

Figure 1. **(a)** Relationship between IMV and predicted FVC (%). **(b)** Relationship between IMV and predicted FEV_1 (%).
IMV: Invasive mechanical ventilation; FVC: Forced vital capacity; FEV_1: Forced expiratory volume in 1 sec.

it was prolonged in 11 patients. In patients with predicted FEV_1 (%) <80%, the IMV duration was normal in 20 patients, while it was prolonged in 12 patients (p=0.035) (Figure 2a). In patients with predicted FEV_1 (%) ≥80%, the IMV duration was normal in 57 patients, while it was prolonged in 11 patients.

- In patients with predicted FEV_1 (%) <80%, the IMV duration was normal in 20 patients, while it was prolonged in 12 patients (p=0.035) (Figure 2a).
- *ICU stay:* The relationship between PFT parameters and duration of ICU was examined. Accordingly, the median predicted FVC (%) value of the patients with prolonged ICU duration was 72.65 (IQR: 58.42-93.30), while the median predicted FVC (%) value of the patients with normal IMV duration was 81.10 (IQR: 74.90-94.13) (p=0.036) (Figure 3a). The mean predicted FEV_1 (%) value of the patients with prolonged ICU duration was 82.3±22.0, while the mean predicted FEV_1 (%) value of the patients with normal ICU duration was 93.2±17.5 (p=0.024) (Figure 3b). The median predicted FVC (%) value of patients with prolonged ICU duration is 72.65 (IQR: 58.42-93.30), the median predicted FVC (%) value of patients with normal IMV duration is 81.10 (IQR: 74.90-94.13) (p=0.036) (Figure 3a).
- The mean predicted FEV_1 (%) value of patients with prolonged ICU duration is 82.3±22.0, the mean predicted FEV_1 (%) value of patients with normal ICU duration is 93.2±17.5 (p=0.024) (Figure 3b).

The patients with low predicted FEV_1% and predicted FVC% values had statistically significantly longer ICU stay.

- When the predicted FEV_1 (%) was divided into two groups as ≥80 and <80%, the duration of ICU stay was found as follows: in patients with predicted FEV_1 (%) ≥80%, the IMV duration was normal in 54 patients, while it was prolonged in 14 patients. In patients with predicted FEV_1 (%) <80%, the IMV duration was normal in 18 patients, while it was prolonged in 14 patients (p=0.030) (Figure 2b). In patients with predicted FEV_1 (%) ≥80%, the IMV duration was normal

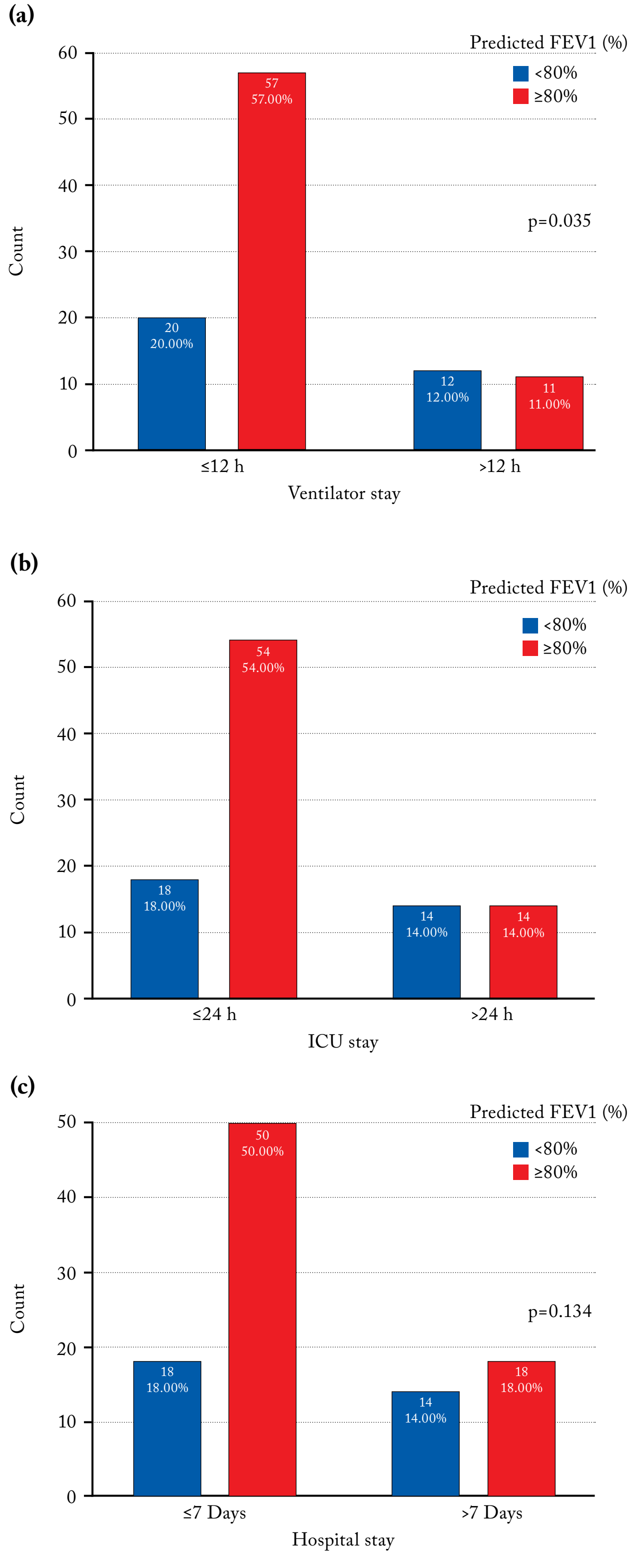

Figure 2. Relationship between ventilator, ICU and hospital stay in patients with predicted FEV1 (%) ≥80 and <80%.
FEV1: Forced expiratory volume in 1 sec.

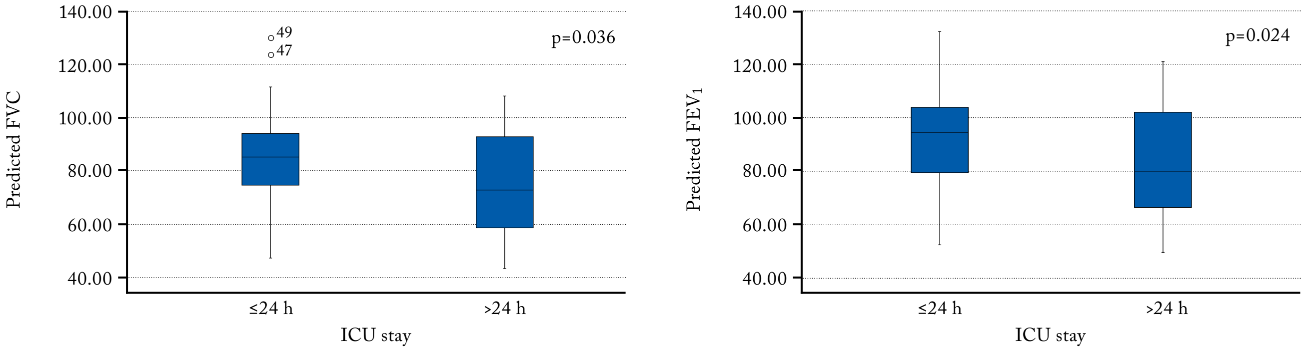

Figure 3. **(a)** Relationship between ICU stay and predicted FVC (%). **(b)** Relationship between ICU stay and predicted FEV_1 (%).
ICU: Intensive care unit; FVC: Forced vital capacity; FEV_1: Forced expiratory volume in 1 sec.

in 54 patients, while it was prolonged in 14 patients.

- In patients with predicted FEV_1 (%) <80%, the IMV duration was normal in 18 patients, while it was prolonged in 14 patients (p=0.030) (Figure 2b).

Hospital stay

The relationship between PFT parameters and duration of hospitalization was examined. Accordingly, the median predicted FVC (%) value of the patients with prolonged duration of hospitalization was 81.65 (IQR: 59.33-88.85), while the median predicted FVC (%) value of the patients with normal duration of hospitalization was 86.50 (IQR: 73.36-96.45) (p=0.009) (Figure 4a). The median predicted FEV_1 (%) value of the patients with prolonged duration of hospitalization was 85.95 (IQR: 70.15-85.95), while the median predicted FEV_1 (%) value of the patients with normal duration of hospitalization was 92.15 (IQR: 79.60-92.15) (p=0.044) (Figure 4b).

- The median predicted FVC (%) value of patients with prolonged duration of hospitalization is 81.65 (IQR: 59.33-88.85), the median predicted FVC (%) value of patients with normal duration of hospitalization is 86.50 (IQR: 73.36-96.45) (p=0.009) (Figure 4a).
- The median predicted FEV_1 (%) value of patients with prolonged duration of hospitalization is 85.95 (IQR: 70.15-85.95), the median predicted FEV_1 (%) value of patients with normal duration of hospitalization is 92.15 (IQR: 79.60-92.15) (p=0.044) (Figure 4b).

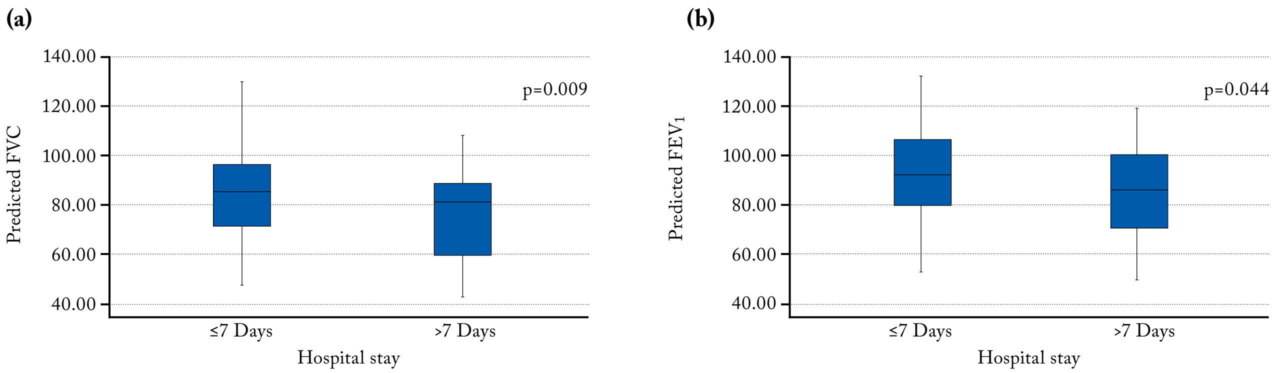

Figure 4. **(a)** Relationship between hospital stay and predicted FVC (%). **(b)** Relationship between hospital stay and predicted FEV_1 (%).
FVC: Forced vital capacity; FEV_1: Forced expiratory volume in 1 sec.

The patients with low predicted FEV_1% and predicted FVC% values had statistically significantly longer hospitalization stay.

- When the predicted FEV_1 (%) was divided into two groups as ≥80 and <80%, the duration of IMV was found as follows: in patients with predicted FEV_1 (%) ≥80%, the hospitalization stay duration was normal in 50 patients, while it was prolonged in 18 patients. In patients with predicted FEV_1 (%) <80%, the hospitalization stay duration was normal in 18 patients, while it was prolonged in 14 patients (p=0.134) (Figure 2c). In patients with predicted FEV_1 (%) ≥80%, the hospitalization stay duration was normal in 50 patients, while it was prolonged in 18 patients.
- In patients with predicted FEV_1 (%) <80%, the hospitalization stay duration was normal in 18 patients, while it was prolonged in 14 patients (p=0.134) (Figure 2c).

DISCUSSION

In the present study, the relationship between predicted FEV_1 (%) and predicted FVC (%) values and duration of ventilation, length of ICU and hospital stay was examined. According to the results, as the preoperative predicted FEV_1 (%) and predicted FVC (%) values decreased, the duration of ventilation, length of ICU and hospital stay statistically significantly increased.

In a study, Alam et al.[8] showed that predicted FEV_1 (%) and FVC (%) were good predictors of postoperative pulmonary complications (e.g., respiratory failure, atelectasis, and pulmonary infections). In the aforementioned study, as the rate of predicted FEV_1 (%) decreased, the rate of respiratory failure and atelectasis increased (p=0.003 *vs.* p<0.001, respectively).

An initial decrease of 40 to 50% in FEV_1 (%) and FVC (%) values on the first and third days after CABG is expected.[9,10] There is a study showing that there is a significant decrease in FEV_1 (%) and FVC (%) values in three-year follow-ups of PFT parameters in patients with CABG compared to the preoperative period.[11] Therefore, in patients with low FEV_1 (%) in the preoperative period, a greater reduction in FEV_1 (%) and hypoxia in the postoperative period would be inevitable. In our study, when we divided and examined patients into two groups as predicted FEV_1 (%) ≥80% and predicted FEV_1 (%) <80%, the group with predicted FEV_1 ≥80% had a shorter ICU stay and ventilation duration than those with predicted FEV_1 <80% (p=0.030 and p=0.035, respectively). However, there was no statistically difference between the two groups in terms of length of hospital stay (p=0.134).

In a study by Kshirsagar et al.,[12] 370 patients who underwent CABG between the ages of 35 and 65 years, the duration of hospital stay increased significantly as FEV_1 (%) decreased. In another study by Saleh et al.[13] including a large number of patients (n=11,217), the patients were divided into three groups according to the severity of chronic obstructive pulmonary disease (COPD) (FEV_1/FVC <70% and predicted FEV_1 ≥80%: mild COPD; FEV_1/FVC <70% and ≤50% predicted FEV_1 <80%: moderate COPD; and FEV_1/FVC <70% and ≤30% predicted FEV_1 <50%: severe COPD). Accordingly, as the severity of COPD increased, many pulmonary disease complications such as inotropic support, acute renal failure, and early mortality increased significantly. Similar to our study, it was shown that there was a negative correlation between FEV_1 (%) and hospital stay. In another study by the aforementioned authors, a reduced FEV_1 (%) (odds ratio [OR]: 0.99; 95% confidence interval [CI]: 0.98-0.99) occurred in 1.96% of CABG patients which was a preoperative predictor of prolonged mechanical ventilation.[14]

Prolonged duration of mechanical ventilation after CABG also extends the length of ICU and hospital stay. It is known to cause a decrease in early and mid-term survival.[7] Hulzebos et al.[15] suggested that preoperative pulmonary muscle physiotherapy could reduce postoperative hospital stay and the incidence of pulmonary complications. In a meta-analysis, the length of ICU stay and hospital stay could be reduced, particularly in elderly patients, with interventions in the preoperative period (e.g., inspiratory muscle physiotherapy, pulmonary exercise physiotherapy).

In the postoperative 12-month follow-up, although there was no statistically difference between the predicted FEV_1 (%) and FVC (%) values of the survivors and non-survivors, the median predicted FVC values of the survivors were higher than those with non-survivors. Patients with moderate and severe airway obstruction (FEV_1 / FVC <70% and FEV_1 (%) <80%) had a 3.2 times higher mortality than patients without airway obstruction in the study of Adabag et al[16] (95% CI: 1.6-6.2, p=001).

In the literature, it has been shown that EuroSCORE is the best scoring system that can predict 30-day and 12-month mortality after cardiac surgery.[17] On the other hand, studies have been carried out on many different parameters that are thought to be missing in this scoring system.[18] McAllister et al.[19] created two scoring groups to determine mortality after cardiac surgery: *(i)* EuroSCORE and *(ii)* FEV_1 (%) values added to the EuroSCORE. As a result, the EuroSCORE scoring system with FEV_1 (%) values was more successful in predicting mortality.

The main limitations of this study include its single-center, retrospective design with a relatively small sample size. In addition, since the study design was retrospective, smoking history and biomass exposure affecting PFT parameters in the preoperative period could not be taken into account.

In conclusion, spirometry is an easily accessible, simple-to-use method that does not require serious costs and is used to evaluate the lung physiological reserve. It is possible to have an idea about postoperative durations of ventilation, ICU and hospital stay based on the PFT parameters. Also, the result would be more realistic when adapted to existing scoring systems to assess postoperative complications. Therefore, we believe that scoring systems for evaluating complications after cardiac surgery should include not only chronic lung disease, but also PFT parameters that give a more detailed information.

Declaration of conflicting interests

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Relationship between the triglyceride glucose index and collateral index in patients with coronary chronic total occlusion

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ABSTRACT

Objectives: This study aims to investigate the relationship between the triglyceride glucose (TyG) index and coronary collateral circulation (CCC) in patients with coronary chronic total occlusion (CTO).

Patients and methods: Between July 2018 and December 2019, a total of 228 consecutive patients (186 males, 42 females; mean age: 62.2±9.7 years; range, 18 to 80 years) with stable or unstable angina pectoris who had CCO in at least one coronary artery were retrospectively analyzed. The TyG index was calculated. Coronary collateral circulation was evaluated using the Rentrop grading system. The patients were divided into two groups as low-grade CCC (Group 1, n=101) and high-grade CCC (Group 2, n=127).

Results: There was no significant difference in the body mass index, left ventricular ejection fraction, height, weight, the frequency of dyslipidemia, hypertension, diabetes mellitus, and smoking between the groups (p>0.01). In the multivariate logistic regression analysis, high TyG index (odds ratio [OR]: 1.345; 95% confidence interval [CI]: 1.120-2.184; p<0.001) and uric acid levels (OR: 0.249; 95% CI: 0.105-0.491; p=0.013) were the independent predictors of poor CCC.

Conclusion: Our study results suggest that a high TyG index is related to poor collateral circulation.

Keywords: Atherosclerosis, coronary collateral circulation, coronary occlusion, dyslipidemia, glucose.

Cardiovascular diseases are among the leading causes of mortality and morbidity worldwide.[1] Coronary chronic total occlusion (CTO) is a condition in which the coronary artery is completely occluded for at least three months and there is no blood flow through the occluded vessel.[2] It is approximately detected in 20% of patients with coronary artery disease (CAD).[3] When the blood flow decreases due to stenosis in a coronary artery, the collateral vessels progressively open and begin to transport blood to the ischemic or infracted myocardium.

The presence of coronary collateral circulation (CCC) is important for the prevention of ventricular dysfunction and ventricular aneurysm formation. The mechanism of CCC formation has not been clarified, yet. Vascular growth factors and blood cells such as monocytes, neutrophils, and lymphocytes are implicated in the CCC formation.[4] A good CCC is important for the prognosis of the patient in the long-term. The triglyceride glucose (TyG) index is considered a basic indicator of insulin resistance which can be important for predicting CCC grade. The TyG index, a consequence of triglycerides and fasting plasma glucose (FPG), is a good sign of insulin resistance.[5]

The TyG index is significantly related to a raised risk of developing type 2 diabetes mellitus, arterial stiffness, coronary artery calcification, hypertension, and adverse cardiovascular events.[6-9] In many studies, the relationship between the TyG index and atherosclerosis has been proven.[10,11] Alizargar and Bai[12] reported that the TyG index was significantly related to the total amount of carotid plaque and the increased intima-media thicknesses of carotid arteries in hypertensive and normotensive patients.

In previous studies, higher TyG index levels were related to poor prognosis in patients with acute

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ST-elevation myocardial infarction.[13] However, the relationship between CCC and TyG index in patients with CTO has not been studied in any study to date. In this study, we aimed to investigate the relationship between the TyG index and CCC in patients with CTO.

PATIENTS AND METHODS

This single-center, retrospective study was conducted at Namık Kemal University Health Practice and Research Hospital, Department of Cardiology between July 2018 and December 2019. Our center is a respected health center in the Thrace region where 4,000 coronary angiography procedures are performed annually. Angiography was performed in 3,800 patients in our clinic and CTO was detected in 290 patients. Patients with severe renal insufficiency (creatinine >2 mg/dL), elevated triglyceride levels (≥400 mg/dL), active infection, and malignancy were excluded from the study. Patients taking triglyceride-lowering medications were also excluded. Finally, a total of 228 consecutive patients (186 males, 42 females; mean age: 62.2±9.7 years; range, 18 to 80 years) with stable or unstable angina pectoris were included. Baseline demographic and clinical characteristics of the patients were obtained from the hospital database. A written informed consent was obtained from each patient. The study protocol was approved by the Namık Kemal University, Faculty of Medicine Ethics Committee (Date/No: 25/07/2021/2021,242,10,06). The study was conducted in accordance with the principles of the Declaration of Helsinki.

The fasting blood samples were obtained from all patients during hospitalization after 12 h of fasting. We performed a complete blood count with an automatic blood analyzer and biochemical values were measured with an automatic device via the standard laboratory techniques.

Blood pressure (BP) was measured three times using an automatic BP monitor on both arms, with the arm placed at heart level after a 10-min rest period, and an average of three measurements were taken. Patients with an average of these three measurements >140/90 mmHg or those taking antihypertensive drugs were considered hypertensive. Patients with an FPG level of ≥7.0 mmol/L (126 mg/dL) or hemoglobin A1c (HbA1c) of ≥6.5% or using antidiabetic medication were defined as diabetic. Hyperlipidemia was defined as being on lipid-lowering therapy or having a total cholesterol level above 220 mg/dL. The TyG was calculated as follows: [fasting triglycerides (mg/dL)×fasting glucose(mg/dL)/2].[14]

Coronary angiography

Coronary angiography images of the patients were evaluated by two independent invasive cardiologists who were blinded to clinical data of the patients. Stenosis of ≥50% more in the coronary arteries was considered significant. Each coronary artery was visualized in at least two different projections. The CCC was evaluated according to the Rentrop classification as described previously. The Rentrop classification was as follows: Grade 0= no visible collateral, Grade 1= filling in collateral via collateral vessels without visualizing the side branch epicardial segment, Grade 2= epicardial partial filling coronary artery, and Grade 3= complete filling of the epicardial coronary artery.[15] The patients whose coronary angiography reports were examined were divided into two groups under the Rentrop classification: Group 1 (Grade 0-1) and Group 2 (Grade 2-3). These groups were compared in terms of the calculated TyG index and other biochemical parameters.

Statistical analysis

Statistical analysis was performed using the IBM SPSS version 22.0 software (IBM Corp., Armonk, NY, USA). Continuous variables were presented in mean ± standard deviation (SD) or median (min-max), while categorical variables were presented in number and frequency. The variables were analyzed using the chi-square or Fischer exact test. The distribution of the data was checked using the Kolmogorov-Smirnov test. The Student t-test was used for continuous data conforming to the normal distribution. Non-parametric variables were analyzed using the Mann-Whitney U test. Receiver operating characteristic (ROC) analysis was performed to determine the optimal cut-off value of the TyG index in the prediction of CCC. The correlation analysis between the TyG index and the CCC was performed using the Spearman correlation test. Multivariate logistic regression analysis was used to identify independent predictors of the CCC. A p value of <0.05 was considered statistically significant. coronary collateral index (CCI).

RESULTS

Baseline characteristics and laboratory results of the patients are summarized in Tables 1 and 2,

Table 1
Baseline characteristics of patients

	Group 1 (n=101)			Group 2 (n=127)			Total (n=228)			
Variables	n	%	Mean±SD	n	%	Mean±SD	n	%	Mean±SD	*p*
Age (year)			61.9±9.7			62.4±9.7			62.2±9.7	0.71
Sex										
Male	84	83.2		102	80.3		186	81.6		0.58
Female	17	16.8		25	19.7		42	18.4		0.58
Height (meter)			1.7±0.2			1.7±0.07			1.7±0.2	0.45
Dyslipidemia	70	69.3		94	74		164	71.9		0.43
Hypertension	50	49.5		73	57.5		123	53.9		0.23
Weight (kg)			80.6±10.2			80.3±10.4			78.8±1	0.82
Smokers	65	64.4		70	55.1		135	59.2		0.15
Diabetes mellitus	49	48.5		47	37		96	42.1		0.08†
Body mass index (kg/m^2)			28.1±4.6			28.1±4.7			28.1±4.6	0.92
Ejection fraction (%)			49.2±9.4			50.7±10.0			50.0±9.7	0.24
Vessel with chronic total occlusion										
LAD	64	63.4		83	65.4		147	64.5		0.95
Cx	31	30.7		37	29.1		68	29.8		0.95
RCA	6	5.9		7	5.5		13	5.7		0.95
Medical treatment										
Beta blocker	66	65.3		71	55.9		137	60.1		0.14
Calcium channel blocker	9	8.9		12	9.4		21	9.2		0.88
ACE-I	49	48.5		66	52		115	50.4		0.60
Diuretic	11	10.9		16	12.6		27	11.8		0.69
Acetyl salicylic acid	73	73.3		83	65.4		156	68.4		0.26
Clopidogrel	10	9.9		11	8.7		21	9.2		0.74
Oral antidiabetic	28	27.7		28	22		56	24.6		0.32
Insulin	21	20.8		24	18.9		45	19.7		0.72
Statin	66	65.3		82	64.6		148	64.9		0.90
Number of vessels with coronary artery disease										
One vessel	24	23.8		25	19.7		49	21.5		0.41
Two vessel	61	60.4		80	63		141	61.8		0.41
Three vessel	16	15.8		21	16.5		37	16.2		0.41

SD: Standard deviation; LAD: Left anterior descending coronary artery; Cx: Circumflex coronary artery; RCA: Right coronary artery; ACE-I: Angiotensin-converting enzyme inhibitors.

Table 2
Laboratory data of patients

	Group 1 (n=101)			Group 2 (n=127)			Total (n=228)			
	Mean±SD	Median	Min-Max	Mean±SD	Median	Min-Max	Mean±SD	Median	Min-Max	*p*
Glucose (mg/dL)		134	83-371		90	70-176		106.5	68-371	<0.001
Hemoglobin (g/dL)	13.5±1.4			13.5±1.5			13.5±1.5			0.88
Blood urea nitrogen (mg/dL)		17.28	8-46		15.88	6-40		17.56	6-46	0.56
Creatinine (mg/dL)		1	0.6-1.7		0.9	0.3-1.7		0.96	0.3-1.7	0.34
Uric acid (mg/dL)		8.3	3.4-11		5.9	2.3-7.9		6.48	2.3-11	<0.001
Total cholesterol (mg/dL)	199.2±47.6			183.7±45.9			190.6±47.2			0.01
HDL-cholesterol (mg/dL)		40	23-75.71		42	18-87		41	18-87	0.016
LDL-cholesterol (mg/dL)	148.8±40.4			115.9±43.7			130.5±45.7			<0.001
Triglyceride (mg/dL)	194.3±81.2			113.2±27.8			149.1±70.5			<0.001
White blood cell count ($\times10^3/\mu L$)	8.0±2.2			8.2±2.3			8.1±2.3			0.62
Neutrophil count ($\times10^3/\mu L$)	4.8±1.7			5.1±2.1			5.0±2.0			0.46
Lymphocyte count (×103/µL)	2.2±0.9			2.2±0.9			2.2±0.9			0.92
Monocyte count ($\times10^3/\mu L$)		0.53	0.3-1.44		0.59	0.27-2	0.6±0.3			0.63
Mean platelet volume (fl)	8.5±1.1			8.1±1.1			8.2±1.1			0.10
Platelet count ($\times10^3/\mu L$)	278.4±83.1			274.7±7			276.2±7			0.77
Glomerular filtration rate	81.0±18.1			81.5±17.4			81.3±17.7			0.87
Triglyceride glucose index	9.5±0.5			8.6±0.3			9.0±0.6			0.001

SD: Standard deviation; HDL: High density lipoprotein; LDL: Low-density lipoprotein.

Table 3
Multivariate logistic regression analysis of variables related to CCC

Variables	Odds ratio	95% CI	*p*
Total cholesterol (mg/dL)	1.037	0.929-1.157	0.517
LDL-cholesterol (mg/dL)	0.874	0.753-1.194	0.077
Triglyceride glucose index	1.345	1.120-2.184	<0.001
HDL-cholesterol (mg/dL)	1.060	0.880-1.278	0.539
Uric acid (mg/dL)	0.249	0.105-0.491	0.013

CCC: Coronary collateral circulation; CI: Confidence interval; LDL: Low-density lipoprotein; HDL: High density lipoprotein.

respectively. Of a total of 228 patients, Group 1 consisted of 101 patients (mean age: 63.9±9.9 years) and Group 2 consisted of 127 patients (mean age: 62.1±9.4 years) (p=0.710). The body mass index, left ventricular ejection fraction, height, and weight of the patients were similar in both groups. The frequency of dyslipidemia, hypertension, diabetes mellitus, and smoking were also similar between the groups. The vessels with chronic total occlusion, the number of vessels with CAD, and the medical treatment of both groups were similar.

Furthermore, the laboratory parameters of the two groups were similar, except for the glucose, uric acid, triglyceride, TyG index, and lipid parameters. However, uric acid (p<0.001), glucose (p<0.001), triglyceride (p<0.001), low-density lipoprotein cholesterol (p<0.001), TyG index levels (p<0.001), and total cholesterol (p=0.01) were higher in Group 1 than Group 2. High-density lipoprotein cholesterol was lower in Group 1 than Group 2 (p=0.016).

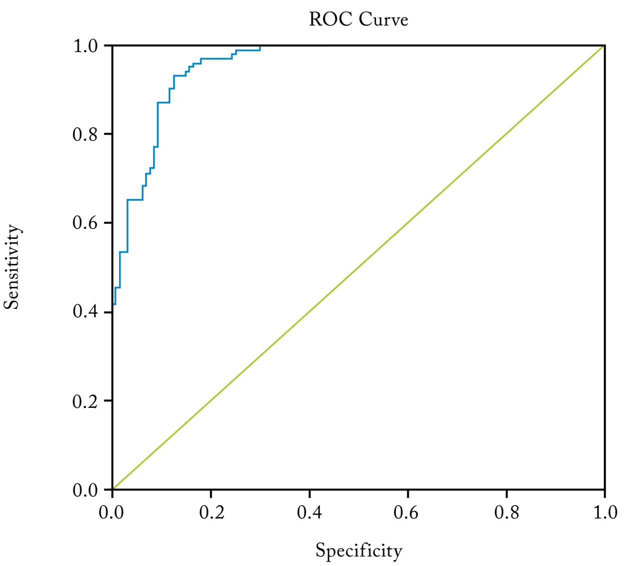

Figure 1. Receiver operating characteristic curve analysis of the triglyceride glucose index levels for the prediction of coronary collateral circulation grade.
ROC: Receiver operating characteristic.

In the multivariate logistic regression analysis, high TyG index (odds ratio [OR]: 1.345; 95% confidence interval [CI]: 1.120- 2.184; p<0.001) and uric acid levels (OR: 0.249; 95% CI: 0.105-0.491; p=0.013) were the independent predictors of poor CCC.

The result of the ROC analysis and area under the curve (AUC) for the TyG index to predict low degree of CCC were as follows: cut-off 8.93, AUC: 0.955, 95% CI: 0.931-0.978, p<0.001 with 88.1% sensitivity and 88.2% specificity (Figure 1).

In the correlation analysis, a high degree of negative correlation was observed between the TyG index and the CCI (r=-0.782, p<0.001).

DISCUSSION

In our study, we examined the relationship between the coronary collateral index and TyG index in patients with stable or unstable angina pectoris who underwent coronary angiography procedures. To the best of our knowledge, there is no study regarding the TyG index and CTO. Early identification of diabetic patients with acute coronary syndrome (ACS) is important to reduce future cardiovascular events. Insulin resistance is an increasingly common metabolic disorder caused by an impaired physiological response to insulin. For many years, insulin resistance and hypertriglyceridemia have been related to metabolic disorders, type 2 diabetes, and atherosclerotic cardiovascular diseases.[16-19] In the onset of diabetes, insulin resistance develops first. Causes of insulin resistance include reduction of glycogen synthesis in skeletal muscles by glucose transporter type-4 (GLUT 4), impaired insulin receptor binding or intracellular signal transduction, as well as the presence of high amounts of circulating free fatty acids. Patients with type 2 diabetes have insulin resistance and decreased β-cell function. Hyperglycemia causes islet cells to be constantly exposed to oxidative stress. Islet cells in the pancreas have a weaker antioxidant capacity. Long-term glucose toxicity and lipotoxicity cause β-cell failure.

Insulin resistance is related to the chronic increase of plasma glucose and triglycerides.[19] The TyG index, which is an indicator of insulin resistance, is related to cardiovascular mortality and morbidity in patients with and without diabetes in many studies.[20] The sensitivity and specificity of the TyG index for determining insulin resistance were 84.0% and 45.0%, respectively, in the study reported by Simental-Mendia et al.[21] Zhao et al.[22] revealed that the TyG index could be a better predictor of cardiovascular risk than FPG or HbA1C for patients with ACS.

In their study, da Silva et al.[23] found that the TyG index could be used as a marker in determining the intensity of atherosclerosis in patients with symptomatic CAD. Mao et al.[24] revealed that the TyG index could be a detached predictor of CAD severity as assessed by the SYNTAX score in patients with ACS. Luo et al.[25] also observed that a TYG increase index could be a potent indicator of the worst prognosis in patients with acute ST-segment elevation myocardial infarction cured with percutaneous coronary intervention for one-year follow-up. In a cross-sectional study, patients with type 2 diabetes, but without a CAD history were found to have a higher TyG index related to an increased risk of significant coronary artery stenosis.[26] In another study involving Korean adults, the TyG index was associated with the progression of coronary artery calcification.[27]

Recent studies have revealed that insulin resistance takes part in macrophage, endothelial, and vascular smooth muscle cell destruction, which contributes to plaque progression.[28] Impaired glucose tolerance, increased insulin resistance increases oxidative stress in the long-term and damages vascular endothelial cells. Moreover, insulin resistance, hyperglycemia, and dyslipidemia increase plasminogen activator inhibitor-1 levels, resulting in reduced fibrinolytic activity and raised thrombotic events.[29,30] Again, in several studies, insulin resistance causes both structural and functional deterioration such as increased intima-media thickness, coronary artery calcification, and arterial stiffness in the vessel wall.[31]

In different studies, cardiovascular diseases and complications were found to be higher in diabetic patients.[32,33] On the other hand, a higher level of TyG index can expedite atherosclerosis in patients with CABG that causes graft failure, as insulin resistance has a proinflammatory and procoagulatory effect, and TyG index is related to endothelial dysfunction. In previous studies, increased insulin resistance levels were associated with the decreased circulation at the microvascular level.[34]

Insulin resistance was associated with an increased infarct area of single-photon emission computed tomography and myocardial perfusion imaging in a study that included non-diabetic patients with ST-segment elevation myocardial infarction.[35] In our study, the TyG index was found to be higher in the group with poor CCC. This situation may have been the result of endothelial dysfunction as a result of the proinflammatory and procoagulatory effects of IR.[36,37]

The main limitation to this study is that it was conducted with a small group of patients in a single center. In addition, the retrospective nature of our study, including a small group of patients, reduces the power of the study. The findings may not cover other demographic groups. The TyG index of patients was calculated only once during hospitalization. Also, we could not reach the HbA1c results of all patients. Therefore, we did not include HbA1c results in the table as the statistical data. Calculating the changes in the TyG index during follow-up may be better in predicting the prognosis. Further multi-center, large-scale prospective studies are required to confirm our results.

In conclusion, high TyG index levels are related to poor collateral circulation in patients with CTO. A high TyG index is an important predictor of a low CCC grade. Based on these findings, TyG index can be a beneficial marker for prognosis in patients with type 2 diabetes and ACS undergoing percutaneous coronary intervention. For CTO interventions, these and similar markers can be a guide in the patient selection.

Declaration of conflicting interests

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Cardiovascular Surgery and Interventions

Case Report Open Access

Improvement in hearing functions after successful carotid body tumor resection

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ABSTRACT

Carotid body tumors (CBTs), also known as paragangliomas, are rare neuroendocrine hypervascularized neoplasms. They are slowly growing pulsatile masses, usually benign, near the carotid arteries. Besides the endocrinological effects of the tumor, compression and hypervascular invasion effects can be also seen. A 63-year-old female patient who had hearing loss in the right ear was admitted to our clinic. Coil embolization was performed to the major arteries feeding the tumor before total excision of the tumor. A decrease in the hearing loss was observed after the operation which might be due to compression and hypervascular steal effects of the tumor. In conclusion, hearing functions can be improved with success CBT resection.

Keywords: Carotid artery, carotid body tumor, glomus tumor, hearing loss paraganglioma.

Carotid body tumors (CBTs), also known as paragangliomas, are rare neuroendocrine hypervascularized neoplasms that arise on the glomus derived from the embryonic neural crest within the carotid bifurcation. The incidence is 1 to 2/100,000, and they have usually a benign character. Less than 10% of the tumors are malignant.[1]

Carotid body tumors are painless, slowly growing pulsatile masses, usually lateral to the hyoid bone. Symptoms may vary depending on the site of the invasion and the compression. Tinnitus and hearing loss can be seen, particularly in paragangliomas with tympanic and jugular extension.[2]

In this report, we present a case with a CBT that caused hearing loss for two years whose symptoms relieved after surgical excision of the tumor following preoperative coil embolization.

CASE REPORT

A 63-year-old female patient who had no chronic disease other than hypertension and hearing loss in the right ear was admitted to our clinic suffering from swelling in the right neck region. The physical examination revealed a palpable pulsatile mass in the neck extending laterally and superiorly from the bifurcation level of the common carotid artery. On magnetic resonance imaging, we detected a hypervascular hyperintense mass approximately 55×45 mm in size with smooth borders causing displacement and extrinsic compression to the parotid gland. No abnormalities were found in the patient's catecholamine and other laboratory tests. On carotid angiography, major feeding branches of the tumor were visualized (Video 1, 2). The branches close to the bifurcation arising from the temporal artery were embolized to decrease bleeding during surgical excision of the tumor and it was confirmed that the tumor blood supply was minimized by re-imaging. The patient underwent surgery for 36 h after embolization. During the operation, we observed that the tumor arose from the carotid bifurcation and covered the proximal internal carotid artery, external carotid artery, and the distal common carotid artery completely with the extensions to the mastoid bone toward the skull base, mandibular region, and the thyroid gland (Video 3). The tumor was evaluated as a Shamblin type 3 tumor. It was totally excised using the bipolar cautery and scissors (Video 4). A total of 110 mL bleeding was detected during the operation.

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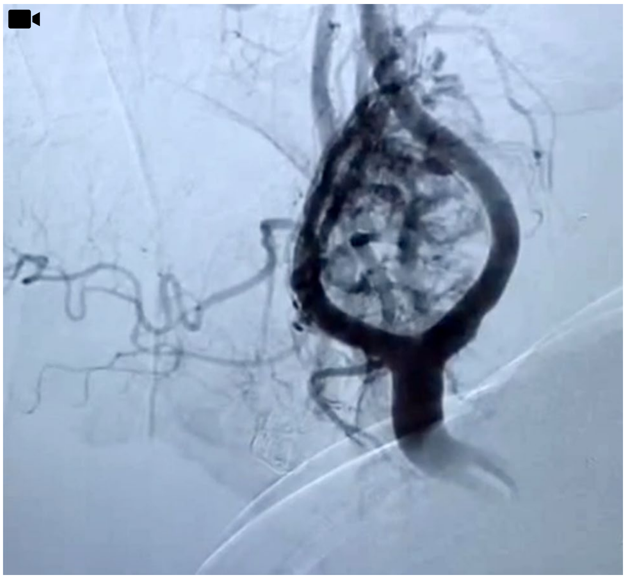
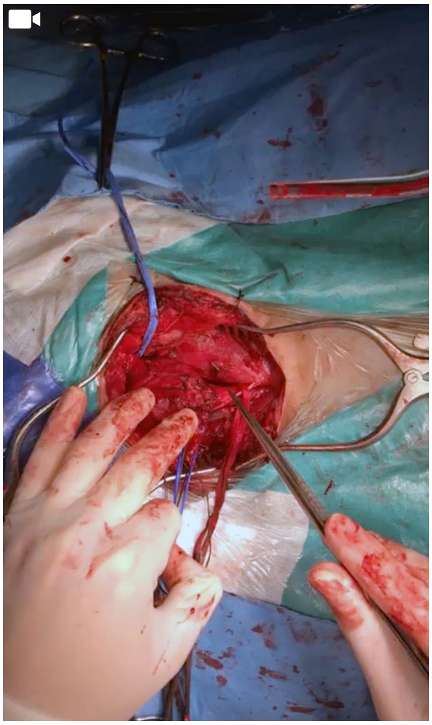
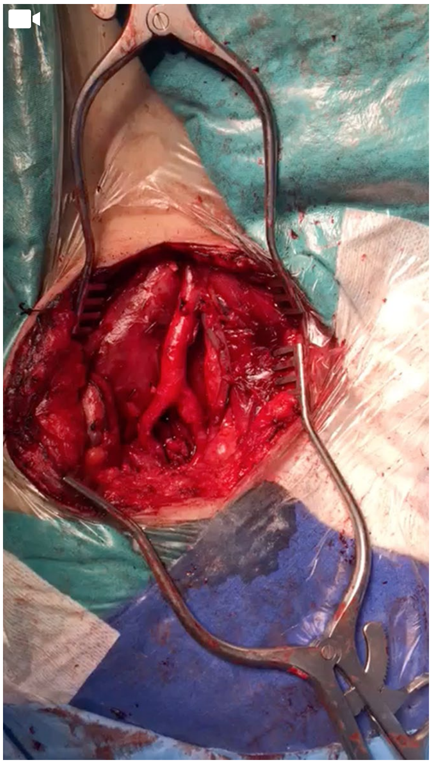


Video 1. Location of the tumor relative to the carotid arteries in angiographic imaging.



Video 3. The relation of the tumor with other tissues in surgical exposure.

Video 2. Angiographic imaging of the vessels feeding the tumor.



Video 4. Tumor totally excised carotid arteries.

No neurological deficit was seen after surgery. The patient who used a hearing aid due to severe hearing loss in the right ear for two years before the operation and could not hear normal speech sounds in his right ear without the device reported that she could hear rough sounds without the hearing aid after the operation. Compared to the previous hearing test, the patient's hearing loss decreased clinically and 15 dB in the audiogram. The patient was discharged on Day 4 after the operation. The pathology report revealed a unifocal paraganglioma with solid and alveolar growth patterns; without mitosis, composite tumor elimination, transcapsular and lymphovascular invasion. The specimen had a diffuse strong staining with synaptophysin and the surgical margins were negative. A written informed consent was obtained from the patient.

DISCUSSION

Conventional surgery, endovascular embolization and rarely radiotherapy can be used in the treatment of CBTs. Endovascular embolization before classical surgery is controversial. Hypervascularization of the tumor is associated with an increased risk of bleeding during the operation and increased blood transfusion. To prevent this, endovascular embolization prior to surgery may reduce bleeding by closing the major vessels feeding the tumor. However, the risk of cerebrovascular events and vascular rupture increases with this procedure. In a study of 29 patients with CBTs conducted by Zhang et al.,[3] significantly less bleeding during the operation and shorter operative times were observed in the patients who underwent preoperative endovascular embolization; however, no relationship was found with the nerve damage. In our case, we performed an endovascular embolization before surgery and we had a total bleeding of 110 mL during the surgical dissection of the tumor with more than one feeding artery. We did not encounter any cerebrovascular diseases after endovascular or surgical procedures. Hearing loss due to CBT is not common in the literature. There is a limited number of studies and case reports related to this. Temmel et al.[4] presented a case with decreased neurosensory hearing loss after CBT excision, but they could not reveal a clear pathophysiological condition to explain this situation. However, they focused on two possible considerations. First, the mass with neurosecretory features could affect the sympathetic ganglia via neurotransmitters. The second and more effective, the sympathetic ganglia were affected by the compression effect of the tumor. In a study, Lord and Chambers[5] reported that familial genetically inherited (autosomal dominant) CBTs with hearing loss were transferred to the lower generations by genetic inheritance and the source of hearing loss was more frequently accompanied by genetically inherited CBT. In our case, we did not consider genetic inheritance, as there was no CBT or any other relatives with hearing loss in her family. As a result of total surgical excision, we removed the pressure on neighboring tissues and increased the perfusion to other tissues and organs in this area, as the excessive blood flow to the tumor in the neck area was removed.

In conclusion, we operate patients with CBT in our clinic. However, hearing loss is extremely rare in these patients. In general, the main complaints are pulsatile masses in the neck and tinnitus. This case is the first in our clinic presenting with CBT accompanied by hearing loss. We believe that the mass has the effect of compression on the sympathetic ganglia in this region rather than the neuroendocrine effect of the tumor, resulting in both a decrease in neural conduction pathways and vasoconstriction in the vertebrobasilar system. Another possible explanation is that the hypervascular tumor has a steal effect on cochlear perfusion. If we consider that there is a mass (particularly located superiorly) and steal effect in non-familial CBT cases, hearing loss may be improved in these patients after surgical operation. However, further studies are needed to establish a definite conclusion on this subject.

Declaration of conflicting interests

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How to do it? Open Access

Double-patch repair of left ventricular rupture after mitral valve replacement

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ABSTRACT

Left ventricular rupture is a rare, but catastrophic complication of mitral valve surgery. Herein, we describe the double-patch repair technique that can be applied in case of left ventricular rupture after mitral valve replacement. Using this technique, the first pericardial patch is positioned inside the endocardium, and the second pericardial patch is positioned outside the epicardium creating a double-layer closure of the defect.

Keywords: Double-patch repair, left ventricle, mitral valve surgery, rupture.

Left ventricular rupture after mitral valve replacement was first reported by Roberts and Morrow[1] in 1967 in a postmortem study. Since then, many surgeons have sought a solution to this mortal surgical approach.[2-7] Left ventricular ruptures after mitral valve replacement are rare, but fatal complications. The incidence varies between 0.24 and 2% in the literature.[5-8] Surgical mortality is very high up to 50 to 100%).[5,7,9] Late ruptures result in pseudoaneurysms and, therefore, treatment of late ruptures is more favorable. Herein, we describe the double-patch repair technique to manage this difficult situation.

SURGICAL TECHNIQUE

A 53-year-old male patient underwent mitral valve replacement with a 29-mm bioprosthesis for mitral valve stenosis and single coronary artery bypass for right coronary artery stenosis. At the end of the operation, while closing the sternum, massive bleeding started from the posterior of the heart. The patient was immediately re-cannulated and cardiopulmonary bypass (CPB) was re-initiated. Bleeding was detected from the posterior wall of the left ventricle. The digital examination revealed that the strut of the bioprosthesis ruptured the posterior wall of the left ventricle. After cross-clamping the aorta, diastolic cardiac arrest was induced using antegrade blood cardioplegia. The mitral bioprosthesis was left *in situ*, and an external repair technique was chosen.

The posterolateral portion of the heart was exposed by two hanging sponges. We prepared two autologous elliptical pericardial patches of 2×3 cm in dimensions. Pericardial patches were treated with 0.9% glutaraldehyde solution for 2 min. To adjust the size of the pericardial patches, they should be larger than the defect of the ruptured area. In this manner, the patches are healthy sutured avoiding tears of the fragile ruptured ventricular area.

First, 3/0, 25 mm double-needle non-absorbable Ticron® polyester U sutures (Sherwood-Davis & Geek, MI, USA) were passed through the first pericardium (Figure 1). The same sutures were, then, passed through the endocardial, myocardial, and epicardial layers by appropriate intervals, in an inside-out manner, 1.5 cm distant to the ruptured area (Figure 2). The first pericardial patch was positioned to the endocardial face of the ventricle leaving the needles outside the pericardium (Figure 3). The needles outside the epicardium were passed from a second pericardial patch with the same dimensions

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and this second patch was positioned on the epicardial surface of the ventricle creating a double-layer repair of the defect (Figure 4). In between the two patches, an absorbable collagen hemostatic sponge (Helistat®, Integra Life Sciences Inc., NJ, USA) was placed and 1 mL tissue adhesive (Preveleak®, Baxter Healthcare Corp., CA, USA) was injected for reinforcement and prevention of leakage. Immediately after the adhesive dried, all the sutures were tied (Figures 5 and 6).

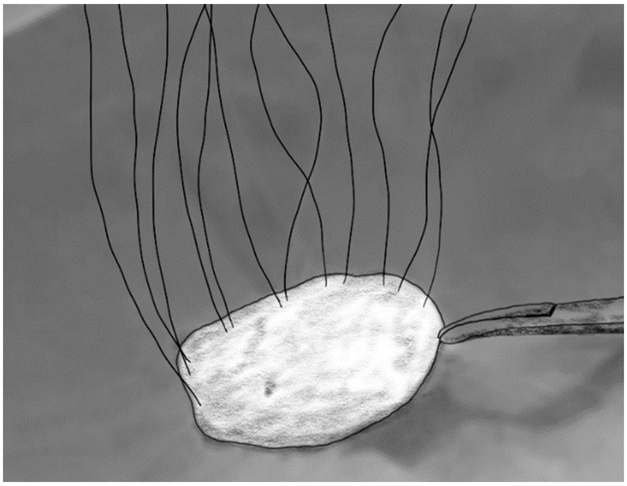

Figure 1. Double-needle U-sutures were passed through the first pericardial patch.

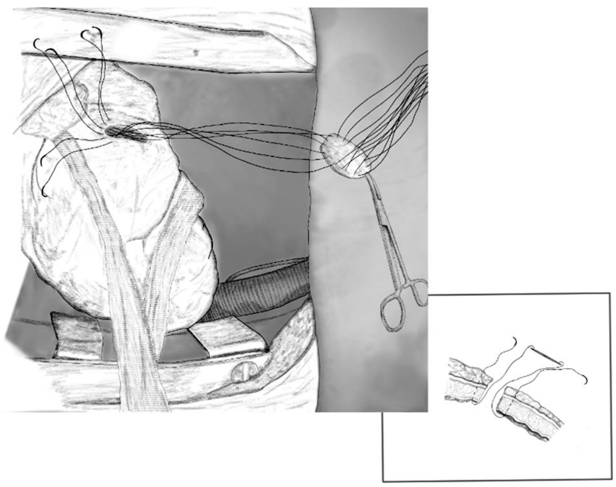

Figure 2. Double-needle U-sutures were, then, passed through the myocardium in an inside-out manner.

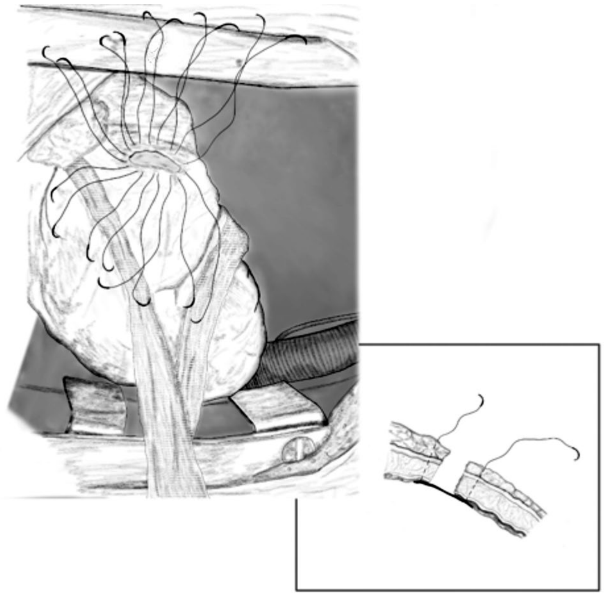

Figure 3. The first pericardial patch was positioned to the endocardial face of the ventricle, leaving the needles outside the epicardium.

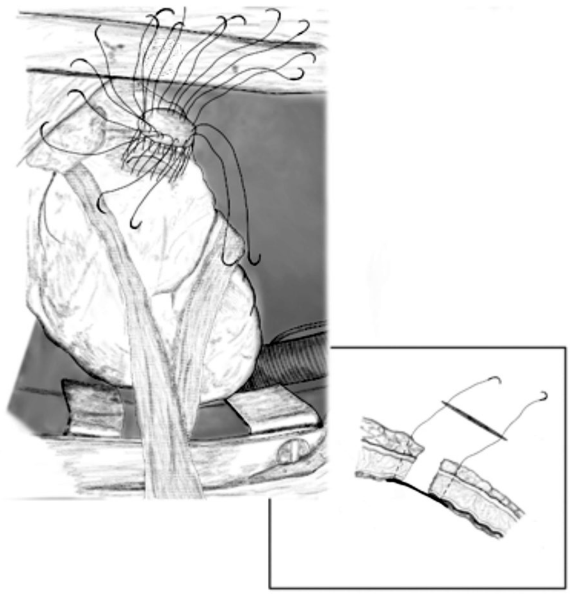

Figure 4. The needles outside the epicardium were passed from a second pericardial patch positioned on the epicardial surface of the ventricle creating a double-layer repair of the defect.

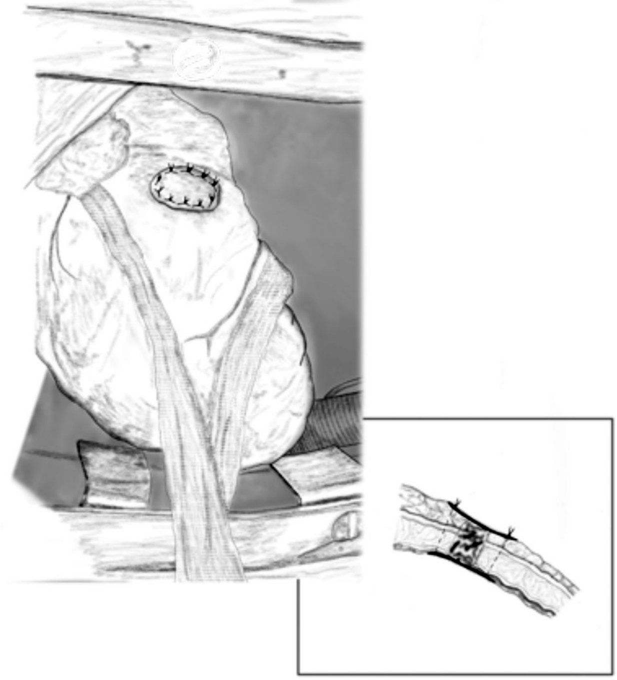

Figure 5. All the sutures were tied and the defect was securely closed.

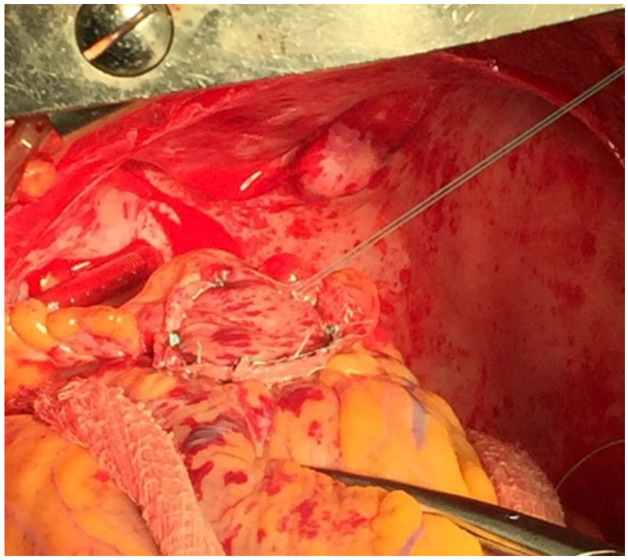

Figure 6. Final image of the double-patch repair.

The patient was weaned from CPB with intra-aortic balloon and inotropic support and discharged on Day 11, after intensive care unit (ICU) stay for four days.

Types of ventricular rupture after mitral valve surgery

Classically, there are three types of ventricular rupture after mitral valve surgery. Type 1 lesion originates between posterior atrioventricular sulcus along the posterior mitral annulus. These are the most common lesions.[2,6,7] Type 2 lesion originates from the roots of papillary muscles due to aggressive excision of the anterolateral and posteromedial papillary muscles,[10] in case of mitral valve replacement, chordal transfer, or excess and deep sutures in tissue. The primary reason for this complication is the chords' deep excision and the discordance between the artificial valve and the annulus.[11] Eventually, ventricular region cannot withstand systolic contractions, and the small tear in the epicardium grows gradually. Type 3 lesion occurs between the type 1 lesion, atrioventricular groove, and type 2 lesion.[7] This rupture can be caused by traumatic reasons, as well as by the misuse of scissors and scalpel, or traumatic effect of aspirator tips. Beside these three classical sites, the rupture of the left ventricular outflow tract, most probably due to the struts of the mitral bioprosthesis, was also described in some cases.[6]

Risk factors for the formation of left ventricular wall ruptures are mainly ischemic heart disease, valve replacement based on annular abscess in case of infectious endocarditis, advanced age, chronic renal failure, left ventricular diastolic diameter less than 50 mm,[6,7,12] posterior annular calcification,[12] previous interventional cardiology applications for the mitral valve, redo heart surgery surgical dissection of the posterolateral wall, emergency intervention, calcified annulus,[13] rigidity of the vent cannulas to be used to decompress the left heart and its' placement from the left ventricular apex,[6] female sex, low body mass index, mitral stenosis, small left ventricular cavity,[14] excessive resection in operations, the use of a larger frame heart valve, high-profile valves, high-profile sharp-tipped struts, the technique of using deep sutures, creating excessive annular tension, unintentional injuries, and extreme stresses due to insufficient diastolic arrest.[6,7]

DISCUSSION

Left ventricular wall ruptures occur in the posterior and posterolateral wall and, classically, there are three different types according to the location of the rupture. Reports of posterior wall ruptures caused by strut positioning and their high incidence in the small left ventricular cavities[14] lead the companies to reduce the strut profiles and lead the surgeons to pay attention, while positioning bioprosthesis struts with good results.[4] The concept of “untethered loop” was introduced by Cobbs et al.[15] The suture technique applied to the posterior annulus is crucial. Sutures from both the annulus and prosthetic valve should be positioned equally spaced, and the sutures should be reciprocal. Asymmetrical and uneven spaced suture positioning may cause either leakage or rupture in the later period.[4]

In the injured area, first dissection and hematoma develop in the myocardium and, then, rupture.[9,12] In redo cases, the main reason is the deep cut of tissue through the annulus during the removal of the valve.[12] However, in redo cases, this area is very adherent, and the attached posterior wall may be partially protective. Although acute left ventricular ruptures are not seen in these cases, pseudoaneurysms may develop in the late period. The repair of late pseudoaneurysms is easier, and the results are more satisfactory.

In the publication of Cabrol in 1984, Lillehei reported that none of the 2,100 cases had ventricular wall rupture for 20 years due to the protection of the posterior leaflet.[16] In the series of Deniz et al.,[13] no ventricular wall rupture was observed in patients with preserved posterior leaflet. Karlson et al.[14] also agreed with this opinion. In our practice, we also preserve the posterior leaflet in primary cases and bioprostheses. If cardiopulmonary resuscitation is needed in a patient with mitral valve prosthesis, we recommend a soft cardiac massage to avoid the rupture of left ventricle. It is not possible to preserve the posterior leaflet in all cases. However, it should be preserved in all eligible patients. Deep excision of the posterior annulus and deep hard decalcification should be avoided. The appropriate size of valves should be selected, sutures from both annulus and prosthetic valve should be equally spaced and reciprocal.

Treatment of left ventricular rupture is challenging in many ways. The left ventricular muscle tissue easily splits and does not support the sutures.[6] Regardless of the type of rupture, it is difficult to access and vision is limited.[6] Surgical suturing and positioning of the heart is also very difficult, when repair under a beating heart is chosen. Repair techniques on the beating heart can lead to elongation of the tear widening the ruptured area. Repairing the ventricle by decompressing the heart under CPB and cross-clamping the aorta is the most reliable way.[5]

The reasons that make the situation more difficult are sutures extending to the ventricle's endocardium that may damage the mitral bioprosthesis and the circumflex artery and its branches, inducing acute myocardial infarction (MI).[17] Sutures may occlude the coronary sinus or cause stenosis. Malignant arrhythmias may develop in the following period. The surgical technique aims to cover the ruptured area with patches and pass the sutures through the intact myocardial tissue by a wide external patch covering the laceration and hematoma area.[10] Also, the major branches of the circumflex artery must be protected.[6] In type 1 tears, the great vein of the coronary sinus should not be damaged. In the double-patch repair or sandwich technique, sutures must pass through non-damaged tissue. If there is a doubt about the coronary arteries injury, revascularization of the suspected vessel is needed. Successful cases have been described in the literature using the posterior leaflet as support in type 1 ruptures.[6]

Repair with a buttressed suture technique using a felt strip is recommended in type 1 ventricular rupture. Both internal and extracardiac patching techniques are suggested for type 2, 3, and mixed ruptures.[6] Another method is to support the surgical repair of the torn region with adhesives such as bio-glue.[7,9] To create a bloodless environment in the 2-min period in which adhesives are used, the total circulatory arrest may be needed. Adhesives alone do not form the basis of repair, but support the surgical technique.[9] The chance of encountering left ventricular wall rupture varies between clinics and even between surgeons in a single clinic.

If the rupture occurs in the operating room, it is easier to re-start CPB, and the problem is easier to manage. However, if it occurs during the postoperative period, the diagnosis is complex and there may not be enough time for stabilization.

Two main routes of repair the left ventricular rupture have been defined as internal and external.[5,14] The method we describe has an external approach, and it differs from the sandwich method 2 in terms of suture technique and placement of the pericardial patches.

In the internal repair, the lesion is exposed through atriotomy. However, if mitral valve repair is applied, it is impossible to perform the internal repair without removing the valve. The results of the external technique with CPB are better than the internal technique.[5,14] The results of repair without CPB are even worse.[5,12] We do not recommend autotransplantation, which is the last step of the algorithm created by Sersar and Jamjoom[6]

In the present case, the patient underwent bioprosthetic valve implantation, and injury of the left ventricle due to the bioprosthesis struts occurred. The ventricle was repaired with the double-patch repair technique. Eventually, the patient was discharged on postoperative Day 11 after ICU stay for four days. We also used this technique in two more patients previously. Both of them were left ventricular free wall rupture due to ischemic MI. After the operation, bleeding stopped and both patients were discharged. All three patients weaned from the CPB with inotropic support. Two patients needed intra-aortic balloon support.

We also suggest the double-patch repair with a similar technique in patients with post-MI ventricular septal defect.[18]

In conclusion, preventive measures should be taken to avoid the occurrence of left ventricular rupture after mitral valve replacement, particularly in patients with predisposing factors. In case of left ventricular rupture, repair of the defect on CPB with cross-clamping the aorta protects the left ventricular functions, thereby avoiding excessive blood loss. The double-patch repair technique secures the suture lines and strengthens the jeopardized area with better results in this difficult situation to handle complication.

Declaration of conflicting interests

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