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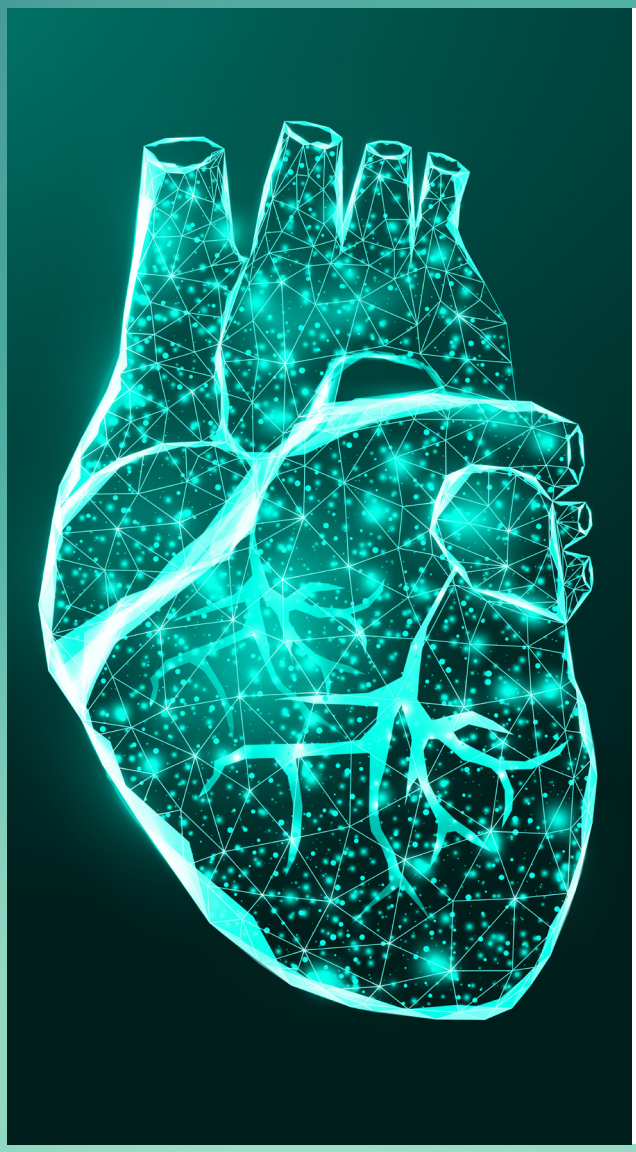

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RESEARCH ARTICLE

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Int J Cardiovasc Acad 2023;9(4):82-87

Assessment of the Extent of Myocardial Injury in Patients Undergoing Transvenous Implantation of a Pacemaker Using Cardiac Troponin I as a Marker of Structural Heart Damage and Its Relation to Different Sites of RV Implantation

Mohamed Mahmoud Ali, Hayam Mohammed Damanhoury, Haitham Abdelfatah Badran, Emad Effat Fakhry

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Abstract

Background and Aim: This study aimed to evaluate the degree of myocardial injury that occurs after transvenous pacemaker implantation using cardiac troponin I (cTnI) as a myocardial injury marker and the relationship between the number of screws, different sites of right ventricle (RV) lead implantation, and myocardial injury.

Materials and Methods: Fifty patients at Ain Shams University Hospitals who underwent transvenous implantation of single- or dual-chamber permanent pacemakers were included in the study. According to the site of RV lead implantation, the study population was divided into 2 equal groups, 25 patients each. In the first group, the RV lead was implanted in the RV apex and in the other group, the RV lead was implanted in the RV septum.

Results: In all patients, the cTnI level was elevated after pacemaker implantation, showing a significant relationship between transvenous pacemaker implantation and the incidence of myocardial injury. Comparing the RV apical pacing group with the RV septal pacing group, a greater rise in cTnI was recognized in the septal RV pacing group, indicating a significant relationship between the site of RV lead implantation and the degree of myocardial injury being more in the RV septum than in the RV apex. Moreover, the higher the number of attempts of screwing the lead in different RV sites caused more rise in cTnI, denoting a significant relationship between the number of screwing attempts and the extent of myocardial injury.

Conclusion: Transvenous pacemaker implantation is associated with an increased incidence of myocardial injury, and septal RV lead implantation is associated with a higher degree of myocardial injury than apical RV lead implantation. In addition, a higher number of screwing attempts of the RV lead into the myocardium is associated with a higher degree of myocardial injury.

Keywords: Apical RV pacing, septal RV pacing, myocardial injury, pacemaker implantation

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***Only the abstract was previously presented at the conference of our university as an obligatory step to register the manuscript in our university.**

INTRODUCTION

Myocardial injury is defined as an elevation of cardiac troponin (cTn) with at least one value above the 99th percentile upper reference limit. It is considered acute if there is a rise and/or fall in cTn values[1]. Cardiac troponin I (cTnI) is a part of the cardiac contractile apparatus, the troponin-tropomyosin complex. It is a very sensitive laboratory marker of myocardial cell necrosis and a gold standard measurement for detecting myocardial injury. Elevated cTnI levels may be associated with a variety of clinical conditions such as myocardial infarction, acute pulmonary edema, ventricular tachycardia, shock, and acute renal impairment[2]. Transvenous insertion of endocardial pacemaker leads is accompanied by cTnI elevation, representing myocardial damage secondary to the direct myocardial trauma elicited by pacing leads[3]. The right ventricular (RV) apex was previously the preferred site for RV lead placement because of its ease of implantation and low risk of lead dislodgement. With the development of active fixation leads, alternative RV pacing sites have been explored, including the RV outflow tract, RV septum, His bundle, and left bundle area. Pacing from these sites is thought to be more physiological, engaging the Purkinje fibers earlier than apical pacing, thus reducing the electric and mechanical dyssynchrony associated with RV apical pacing[4]. In this study, we aimed to evaluate the degree of myocardial injury using cTnI in patients undergoing permanent pacemaker implantation and its relation to different sites of RV lead implantation and the number of RV lead screwing attempts into the RV myocardium.

MATERIALS AND METHODS

This study was approved by the Ain Shams University Faculty of Medicine Research Ethics Committee (no: MS 252/2020, date: 01.04.2020), and all patients signed an informed written consent for participation in the study in accordance with the Declaration of Helsinki. Fifty patients undergoing transvenous implantation of single- or dual-chamber permanent pacemakers were included in the study. According to the site of RV lead implantation, the study population was divided into 2 equal groups, 25 patients each. In the first group, the RV lead was implanted in the RV apex and in the other group, the RV lead was implanted in the RV septum. The study protocol was approved by the scientific and ethical committees. The study included patients above 18 years old, undergoing implantation of single or dual chamber permanent pacemakers with normal pre-procedural cTn values. Those patients were excluded from the study: Patients with previously implanted permanent pacemaker and undergoing re-implantation for any reason, patients with recent active cardiac condition such as myocardial infarction, acute pulmonary edema, cardiogenic shock, and ventricular tachycardia within one month prior to pacemaker implantation, and patients with any form of active infection were excluded from the study. All patients were subjected to 1) Full history taking including age, sex, presence of cardiovascular risk factors such as smoking, hypertension, diabetes mellitus, family history of premature cardiovascular disease, presence or absence of chronic kidney disease, and past history of implantation of a permanent pacemaker. 2) Resting 12 leads surface electrocardiogram: Twelve leads surface electrocardiogram was performed in all patients before and after the procedure. 3) Fluoroscopy data: Fluoroscopy findings of the implantation procedure were obtained to assess the site of implantation of the RV lead and the number of attempts of lead screwing. The lead position was also confirmed via fluoroscopy. 4) Measurement of cTn level: Blood samples for cTnI (using VIDAS® TNHS kit) using were obtained before the procedure and 12 h after the procedure with normal reference ranged from (0-34) pg/mL. Peri-procedural myocardial injury was diagnosed in the presence of elevated cTn values with at least one value above the 99th percentile upper reference limit. 5) Echocardiography: Echocardiography was performed in all patients before the procedure, which showed normal left ventricle (LV) and RV functions and dimensions.

Statistical analysis

Revised data were collected and entered for analysis using the Statistical Package for Social Science (IBM SPSS) version 23. Parametric quantitative data are presented as mean, standard deviation, and range, whereas non-parametric quantitative data are presented as median and interquartile range. Numbers and percentages are used to present qualitative variables. Comparing groups with qualitative data was done using chi-square test, but when the expected count in any cell was less than 5, Fisher's exact test was used. Comparing groups with parametric quantitative data was done by using independent t-test, while comparing groups with non-parametric quantitative data was done by using Mann-Whitney U test. Comparing two paired groups with non-parametric quantitative data was done by Wilcoxon test. The correlation between two quantitative variables in the same group was assessed using Spearman correlation coefficients. The confidence interval was set to 95% and the margin of error accepted was set to 5%; thus, the *P*-value was interpreted as follows: *P*-value >0.05 was non-significant, *P*-value <0.05 was significant, and *P*-value <0.01 was highly significant.

RESULTS

The current study included 50 individuals. All patients underwent pacemaker implantation. Troponin I levels were measured for all patients both before and 12 h after the intervention. The study population was divided equally into two groups: (group A) 25 patients (50.0%) with the RV lead implanted into the RV apex and (group B) 25 patients (50.0%)

with the RV lead implanted into the septum. Their age ranged from 39 to 75 years, with a mean age of 59.56 ± 8.53 years. Among the two groups, there were 39 males (78.0%) and 11 females (22.0%). Regarding smoking habits, 26 patients (52.0%) were smokers and 24 patients (48.0%) were non-smokers.

Regarding diabetic status, 18 patients (36.0%) were non-diabetic (HbA1C less than 6.5% according to definition of Diabetes Mellitus by American Diabetes Association) and 32 patients (64.0%) were diabetic.[5] Regarding hypertension, 19 patients (38.0%) were non-hypertensive and 31 patients (62.0%) were hypertensive (Table 1). The median number of screws was 2 (1-3) for all patients (Table 2). Twenty patients in group A and 20 patients in group B received dual chamber pacemaker implantation, whereas only five patients in each group received single chamber pacemaker implantation, with no statistically significant difference between the 2 groups (*P*-value: 1). There was no significant relationship between age (*P*-value: 0.233), sex (*P*-value: 0.806), smoking history (*P*-value: 0.353), diabetic status (*P*-value: 0.991), hypertension (*P*-value: 0.701), and incidence of myocardial injury (Table 3, 4). Regarding the relationship between demographic data and post-procedural increase in troponin level in folds, there were no statistically significant differences regarding sex, smoking, presence of diabetes and hypertension, and the increase in troponin level (Table 5). There were no statistically significant differences between age and pre- and postprocedural troponin levels (Table 6). Regarding the relationship between permanent pacemaker implantation and post-procedural rise in troponin level, the median troponin level in the whole study group before implantation was 8 pg/mL (3-10) while median troponin level after implantation was 128 pg/mL (43-227) with a median increase fold of 20.19 (12.25-29.94). Therefore, there was a statistically significant relationship between pacemaker implantation and incidence of myocardial injury (*P*-value >0.001) (Table 7). Regarding the relationship between the two groups regarding myocardial injury in the apex group, the median troponin level before implantation was 3 pg/mL (2-8), while in the septum group it was 9 pg/mL (7-10). In the apex group, the median troponin level after implantation was 43 pg/mL (20-115) with a median increase fold 12 (9.7-21.5), whereas in the septum group, it was 204 pg/mL (135-365) with a median increase fold of 21 (20-30.42). There was a statistically significant difference between the two groups regarding the extent of myocardial injury, which was higher in the septum RV lead implantation group (Table 8, 9) (*P*-value: <0.0001). There was a statistically significant relationship between the number of attempts to screw the RV lead and the degree of myocardial injury (*P*-value: <0.0001) (Table 10) (Figure 1). Multivariate analysis showed no statistically significant correlation between age, sex, hypertension, smoking, diabetes, and the rise in troponin fold in both groups.

Table 1: Demographic data

		Number	%
Sex	Female	11	22.0%
	Male	39	78.0%
Smoker	Non-smoker	24	48.0%
	Smoker	26	52.0%
Diabetic	No	18	36.0%
	Yes	32	64.0%
Hypertensive	No	19	38.0%
	Yes	31	62.0%

Table 2: Number of screwing

Number of screwing	Median (IQR)	2 (1-3)
	Range	1-5

IQR: Interquartile range

DISCUSSION

In this study, 50 patients were scheduled for permanent pacemaker implantation. Troponin I levels were measured in all patients both before the procedure and 12 h after the procedure. They were divided into two equal groups, apical and septal, according to the site of RV lead implantation.

In the current study, there was a statistically significant relationship between the site of RV lead implantation and the degree of myocardial injury. Troponin I was higher in the septal group than in the apical group (*P*-value: <0.0001), which may represent deeper implantation of the RV lead in the septal group. Troponin I elevation was higher with an increased number of attempts to screw the RV lead into the myocardium

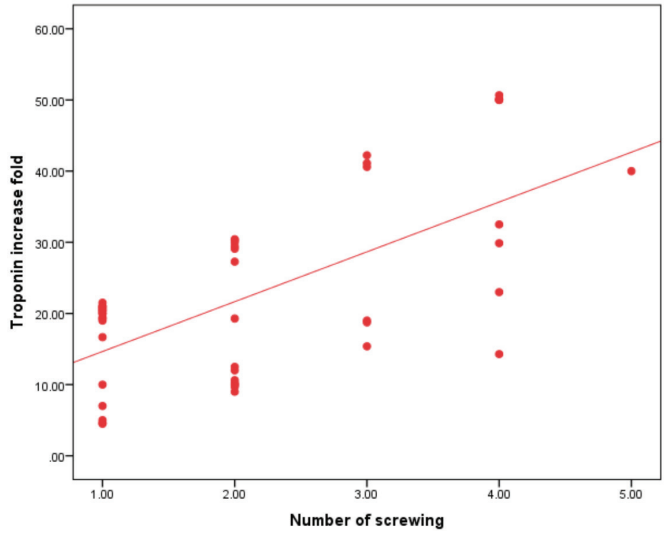

Figure 1: Correlation between number of screwing and troponin increase fold

Table 3: Relationship between demographic data and pre-procedural troponin level

		Troponin before (pg/mL)		*P*-value	Significance
		Median (IQR)	Range		
Sex	Female	7 (2-9)	2-10	0.396	NS
	Male	8 (4-10)	0-13		
Smoker	Non-smoker	7 (2-9)	0-12	0.125	NS
	Smoker	8 (5-10)	0-13		
Diabetic	No	6 (2-8)	0-10	0.062	NS
	Yes	8.5 (5-10)	0-13		
Hypertensive	No	8 (4-10)	0-13	0.265	NS
	Yes	7 (2-9)	0-12		

IQR: Interquartile range, *P*-value >0.05: Non-significant; *P*-value <0.05: Significant; *P*-value <0.01: Highly significant

Table 4: Relationship between demographic data and post-procedural troponin level

		Troponin after (pg/mL)		*P*-value	Significance
		Median (IQR)	Range		
Sex	Female	135 (43-190)	18-380	0.870	NS
	Male	120 (41-239)	5-501		
Smoker	Non-smoker	110 (34-169)	5-500	0.137	NS
	Smoker	175 (65-300)	11-501		
Diabetic	No	117.5 (20-152)	11-500	0.127	NS
	Yes	162.5 (65-282.5)	5-501		
Hypertensive	No	163 (45-303)	11-501	0.294	NS
	Yes	105 (37-225)	5-500		

IQR: Interquartile range, NS: Non-significant, *P*-value >0.05: NS; *P*-value <0.05: Significant; *P*-value <0.01: Highly significant

Table 5: Relationship between demographic data and post-procedural increase fold of troponin level

		Troponin increase fold		*P*-value	Significance
		Median (IQR)	Range		
Sex	Female	20 (10-41.11)	9-50.67	0.806	NS
	Male	20.38 (12.5-29.88)	4.5-50.1		
Smoker	Non-smoker	19.29 (10.63-23)	4.5-50.67	0.353	NS
	Smoker	20.8 (16.67-30)	4.63-50.1		
Diabetic	No	20 (12-30.3)	5-50.67	0.991	NS
	Yes	20.4 (12.5-29.88)	4.5-50.1		
Hypertensive	No	20.19 (15.38-30.3)	4.5-50.1	0.701	NS
	Yes	20.2 (10.63-29.88)	4.63-50.67		

IQR: Interquartile range, *P*-value >0.05; NS: Non-significant; *P*-value <0.05: Significant; *P*-value <0.01: Highly significant

Table 6: Relationship between age and pre, post procedural troponin level

	Troponin					
	Before		After		Increase fold	
	R	*P*-value	r	*P*-value	r	*P*-value
Age	0.097	0.501	0.020	0.888	-0.176	0.233

P-value >0.05: Non-significant; *P*-value <0.05: Significant; *P*-value <0.01: Highly significant

(*P*-value: <0.0001). Of note, there was a statistically significant relationship between pacemaker implantation in all patients regardless of the site of implantation of the RV lead and the incidence of myocardial injury (*P*-value <0.001).

In 2011, Nikolaou et al.[6] studied the effect of implantation of an endocardial permanent pacemaker on myocardial injury using cTnI. During a period of 3 years, 283 patients underwent pacemaker implantation. A normal level of cTnI before pacemaker implantation was required as an inclusion criterion. six hours after the procedure, cTnI was measured in all patients. Elevated cTnI levels were found in 167 patients (59%, 95% CI: 0.53-0.64). There was no clinical evidence of an acute coronary syndrome before or during pacemaker implantation; moreover, coronary angiography showed no significant coronary lesions. They concluded that cTnI elevations following pacemaker implantation may exceed levels that correspond to minimal myocardial damage[6].

Regarding the active fixation of the RV lead into the myocardium and the number of attempts of screwing to achieve the best pacing thresholds, Saxonhouse et al.[7] in 2005 studied whether active fixation leads cause myocardial injury at the time of implantation, indicated by the current of injury that may result in an acute rise in pacing thresholds. Sixty-five patients undergoing pacemaker implantation with active fixation leads were included in this study. The current of injury was defined as the duration of the intracardiac electrogram (EGM) and the amplitude of ST-segment elevation. Pacing parameters were measured up to 10 min after lead fixation. Ninety-six active fixation leads were included in the study, and 76 leads had current of injury. From baseline to the time of lead fixation, the duration of the EGM in ventricular leads increased from 150 +/- 31 ms to 200 +/- 25 ms (*P*-value <0.001), and the ST-segment elevation increased from 1.5 +/- 0.2 mV to 10.0 +/- 2.0 mV (*P*-value <0.001), followed by improvement in pacing thresholds from 1.5 +/- 0.4 V to 0.8 +/- 0.3 V (*P*-value <0.001) at 10 min. Atrial leads with an evident current of injury had

Table 7: Relationship between permanent pacemaker implantation and pre, post-procedural troponin level

		Troponin (pg/mL)
Before	Mean ± SD	6.76±3.66
	Median (IQR)	8 (3-10)
	Range	0-13
After	Mean ± SD	163.94±137.64
	Median (IQR)	128 (43-227)
	Range	5-501
Increase fold	Mean ± SD	50.67±22.68
	Median (IQR)	20.19 (12.25-29.94)
	Range	4.50-50.67
Mean difference	Mean ± SD	157.15±19.10
Wilcoxon signed-rank test		6.154
P-value		<0.001 (HS)

IQR: Interquartile range, SD: Standard deviation, *P*-value >0.05: Non-significant; *P*-value <0.05: Significant; *P*-value <0.01: HS: Highly significant

Table 8: Relationship between site of RV lead implantation and post-procedural troponin level

		Troponin after (pg/mL)		*P*-value	Significance
		Median (IQR)	Range		
Site of RV lead	Apex	43 (20-115)	5-239	<0.0001	HS
	Septum	204 (135-365)	104-501		

IQR: Interquartile range, HS: Highly significant, *P*-value >0.05: Non-significant; *P*-value <0.05: Significant; *P*-value <0.01: HS

Table 9: Relationship between site of RV lead implantation and post-procedural increase fold of troponin

		Troponin increase fold		*P*-value	Significance
		Median (IQR)	Range		
All patients		20.19 (12.25-29.94)	4.50-50.67		
Site of RV lead	Apex	12 (9.7-21.5)	4.50-50.67	<0.0001	HS
	Septum	21 (20-30.42)	16.67-50.10		

IQR: Interquartile range, HS: Highly significant, *P*-value >0.05: Non-significant; *P*-value <0.05: Significant; *P*-value <0.01: HS

Table 10: Correlation between number of attempts of screwing and pre, post procedural troponin level

	Troponin					
	Before		After		Increase fold	
	R	*P*-value	r	*P*-value	R	*P*-value
Number of screwing	0.127	0.380	0.374**	0.007	0.497**	<0.0001

**We evaluate the correlation between the number of attempts to screw the right ventricular lead into the right ventricle and the troponin fold increase after pacemaker implantation, so that the values before and after implantation and their fold increase were correlated with the number of screw attempts

almost similar results. Of the 20 leads with no current of injury, 5 were acutely dislodged and 15 had elevated pacing thresholds in 10 min, necessitating lead repositioning. They concluded that the presence of a current of injury indicates that within 10 min of lead implantation, the pacing threshold will be in an acceptable range even if the initial pacing threshold was elevated. However, without a current of injury, lead fixation is inadequate and must be repositioned. Therefore, they demonstrated that active fixation of the RV lead causes myocardial injury at the time of implantation represented in the current of injury, and without adequate current of injury, the lead must be repositioned until adequate current of injury is achieved[7]. Our results were concordant with the results of this study, but we used cTnI as the biomarker of myocardial injury, which showed a significant increase with increasing number of attempts to lead into the myocardium (*P*-value: <0.0001).

Chen et al.[8] demonstrated that implantation of pacemakers was associated with cTn T elevation in 55.6% of the patients 6 h after the procedure, but this was related to complications and adverse cardiac outcomes at 1 year follow-up. They also showed that gender, NT-pro-BNP at baseline, left ventricle EF, eGFR, and fluoroscopy time were independent predictors of cTn T elevation.[8]

Study limitation

This study was a single-center study with a limited number of patients. We recommend having a larger-scale multi-center study with long-term follow-up. The clinical significance of troponin elevation after pacemaker implantation is unknown, and a follow-up study is needed to assess the clinical significance.

CONCLUSION

Permanent pacemaker implantation is associated with an increased incidence of myocardial injury, and septal RV lead implantation is associated with a higher extent of myocardial injury than apical RV lead implantation.

Increasing the amount of screws during active fixation attempts is associated with a higher degree of myocardial injury.

Although the clinical significance of myocardial injury after pacemaker implantation is unknown, a follow-up study is needed to assess its clinical significance.

Ethics

Ethics Committee Approval: This study was approved by the Ain Shams University Faculty of Medicine Research Ethics Committee (no: MS 252/2020, date: 01.04.2020)

Informed Consent: Informed consent was obtained.

Peer-review: Externally peer-reviewed.

Authorship Contributions

Surgical and Medical Practices: E.E.F., Concept: H.M.D., Design: H.A.B., Data Collection or Processing: M.M.A., H.A.B., E.E.F., Analysis or Interpretation: H.A.B., E.E.F., Literature Search: E.E.F., Writing: E.E.F.

Conflict of Interest: No conflict of interest was declared by the authors.

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RESEARCH ARTICLE

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The Relationship Between SYNTAX II Score and Serum Pleiotrophin Level in Patients with Non-ST-Segment Elevation Myocardial Infarction

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Abstract

Background and Aim: In this study, we aimed to examine the relationship between serum pleiotrophin (PTH) levels at admission and the severity of coronary artery disease (CAD) in patients experiencing non-ST-segment elevation myocardial infarction (NSTEMI).

Materials and Methods: A total of 140 patients with NSTEMI undergoing coronary angiography were consecutively included in the study. The Synergy Between percutaneous coronary intervention with Taxus and Cardiac Surgery (SYNTAX) score was determined based on initial coronary angiography by at least two separate cardiologists.

Results: A positive correlation was found between high SSII and PTH ($r = 0.458$; $P < 0.001$). PTH could anticipate the extremity of CAD with 64.4% sensitivity and 65.3% specificity at 250 ng/mL cut-off value (area under the curve: 0.718, 95% confidence interval, 54.8 - 74.7; $P < 0.001$). Through regression analysis, PTH, hypertension, diabetes mellitus, family history, lymphocyte count, and pro-brain natriuretic peptide levels were found to be independent predictors of SSII.

Conclusion: In patients with NSTEMI, serum PTH levels were significantly associated with higher SSII, an indicator of CAD severity and cardiovascular prognosis. This study obtained positive results that will contribute to our clinical interpretation. More comprehensive studies with PTH will make a more useful contribution to our clinical judgments.

Keywords: Pleiotrophin, NSTEMI, SYNTAX score, acute coronary syndrome, coronary artery disease

INTRODUCTION

Being one of the major public health problems seen worldwide, acute myocardial infarction (MI) is a prominent agent of morbidity and mortality. Atherosclerosis holds a crucial position in the emergence of most cardiovascular (CV) disorders.[1,2] As atherosclerosis is caused by the progressive gathering of fibrous tissue and cholesterol consisting of plaques, it leads to stricture of the arterial lumen and results in non-ST-segment elevation myocardial infarction (NSTEMI). In fact, the atherosclerotic process is frequently followed by inflammation.[3,4] Various studies commonly suggest that each stage of atherosclerosis, including increased plaque instability, is mediated by inflammatory factors and consequently leads to clinical circumstances, as seen in cases of MI, unstable angina, stroke, or sudden death.[5,6]

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Pleiotrophin (PTH) is a secreted multifunctional cytokine with diverse functions in regulating angiogenesis when a tumor emerges and grows around the tissue. High levels of PTH can be experienced in various tumor cell lines as well as in the kidneys, embryonic nervous system, lungs, bones, and intestines. PTH can also play a significant role in physiological angiogenesis, along with tumorous and non-tumorous pathological angiogenesis.s[7] Several experiments conducted on models suggest that PTH provides the creation of operative neovascularization in rat CV tissues *in vivo* and *ex vivo*.[8,9] In the focal cerebral ischemia and reperfusion study on animals, it has been shown that PTH affects neuronal, glial, macrophage, and endothelial cell populations in the brain, and its level increases.[10] In a study by Palmieri et al.[11], it was shown that PTH-secreting cells can regulate proinflammatory and proregenerative changes by modulating PTH tissue levels depending on different stimuli. In the latest study, the PTH level was found to be high in the development of collateral in patients with chronic total coronary artery disease (CAD) after atherosclerosis.[12] However, no research has been conducted to assess the correlation between serum PTH levels and the prevalence of CAD in patients with NSTEMI. In this study, we assessed the severity of CAD and serum PTH rates according to the the synergy between percutaneous coronary intervention (PCI) with Taxus and Cardiac Surgery (SYNTAX) II (SYNergy between PCI with TAXus and cardiac surgery) score (SSII) in patients with NSTEMI.

MATERIALS AND METHODS

Target population

Designed in a cross-sectional and single-center plan, the study included 140 consecutive patients (18-80 years old) who applied to the emergency service with the first incident of NSTEMI from December 2021 to February 2022. SSII was calculated on the basis of angiographic findings. Considering the findings from the scoring system, the target population was separated into subgroups with SSII values of 22 in the low group, those with an SSII value between 23 and 32 in the middle group, and those with an SSII value of 33 in the high SSII group.

We defined exclusion criteria as former PCI or a history of coronary artery bypass grafting, acute liver and kidney disorders, decompensated heart failure, malignancies, autoimmune diseases, hematological disorders, inflammatory or infectious diseases, and intense valvular disorders. The conclusive diagnosis of NSTEMI was defined on the basis of the latest guidelines.[13,14]

The patient's age, gender, CV risk factors, and history of CAD were documented in their records. By conducting at least two calculations or using any antihypertensive medication, we determined hypertension (HT) as systolic blood pressure >140 mmHg and/or diastolic blood pressure >90 mmHg. We also defined diabetes mellitus as fasting plasma glucose level >126 mg/dL or >200 mg/dL by taking into consideration antidiabetic drug usage or relevant measurements. After obtaining a written consent form from all patients and approval of the study protocol by the Ankara City Hospital Ethics Committee, the study protocol (approval number: 2022-2375; date: 09.02.2022) was conducted in accordance with the ethical guidelines of the 1975 Helsinki Declaration.

Laboratory assessment

Initially, we collected peripheral venous blood samples from the patients by atraumatic puncture from the antecubital vein and sent them to the laboratory for cardiac catheterization. Next, we used the Beckman Coulter AU 5800 AutoAnalyzer to measure the ratios of blood biochemical parameters and high sensitivity-C-reactive protein (hs-CRP) urea, along with uric acid, creatinine, sodium, lipid panel, potassium, and pro-brain natriuretic peptide (proBNP). We also calculated the low-density lipoprotein with the Friedewald equationusing an automated blood cell counter (Beckman Coulter LH 750; Beckman Coulter Inc., USA) to analyze variables related to complete blood count.

The blood models obtained for measuring the PTH rates were subjected to centrifugation at 3000 rpm at 4 °C for 10 min. After the process of separation, platelet-poor plasma was preserved at -80 °C for appropriate analysis. Serum PTH levels were calculated using a commercial kit named ELISA (SunRed Biotechnology Campany, Shanghai SunRed Biological Technology Co., Ltd. Hu Tai Road. Baoshan District, Sanghani, China).

All patients underwent transthoracic echocardiography. The left ventricular ejection fraction (LVEF) was calculated using the modified Simpson method.

Angiographic assessment

Coronary angiography was performed using a Siemens Axiom Sensis XP device (Munich, Germany) and Standard Judkins technique (Expo; Boston Scientific Corporation, Natick, Massachusetts, USA). Diameter images of the coronary artery were investigated in at least two perpendicular planes, followed by digital recording of each coronary angiographic image for quantitative analysis. PCI was performed using iopromide (low osmolarity and non-ionic contrast agent) based on clinical practice regulations.

Digital angiograms were evaluated by at least two experienced and independent interventional cardiologists, followed by the calculation of SYNTAX II rates, which were detected to present no difference when compared by the interventional cardiologists. Thus, the online SSII calculator version 2.1 (www.syntaxscore.com) determined ≥1.5 mm in diameter,

each lesion causing ≥50% lumen stricture in the epicardial arteries.

Statistical analysis

We used the SPSS 22.0 Statistical Package Program for Windows (SPSS Inc., Chicago, IL, USA) to perform statistical analysis. Before analyzing the relation of persistent numerical variations for each group, we applied the Kolmogorov-Smirnov or Shapiro-Wilk tests to subject them to normality analysis, taking the number of samples into consideration for each group. Therefore, we determined the continuous variables with a normal distribution as mean ± standard deviation, variations with non-normal distribution as median (minimum-maximum), and categorical variables as percentage and number. In addition, we used the Kruskal-Wallis test or the analysis of variance (ANOVA) test to compare continuous variables based on the SYNTAX scores. Bonferroni analysis was used as a post-hoc test. The chi-square test was used to compare categorical variables. We also defined the independent variations indicated by the high SYNTAX score (≥33) by performing multiple logistic regression analyses. Potential confusing factors for which the unadjusted p-value was detected as <0.20 in univariate regression analysis [the presence of diabetes mellitus and HT, lymphocyte, hemoglobin, proBNP, PTH level, and high-density lipoprotein cholesterol (HDL-C)] were identified as possible risk indicators and evaluated in the multiple logistic regression model. Variables used to calculate the SYNTAX score were excluded from the final analysis. Finally, we performed receiver operating characteristic analysis to determine the cut-off rate of PTH in the calculation of a high SYNTAX score. The significance limit was accepted as $P < 0.05$.

RESULTS

The demographic, basic clinical, and laboratory findings of the study population are shown in Table 1, which statistically presents a remarkable difference among these three groups with respect to age, gender, diabetes mellitus, smoking, CAD, family history, PTH, lymphocyte, proBNP level, and LVEF ($P < 0.05$). However, univariate logistic regression analysis indicated that diabetes mellitus, HT, lymphocyte count, HDL-C, proBNP, and PTH levels were independent predictors of the high SSII group. In multivariate logistic regression analysis, PTH and proBNP were determined as independent predictive values for the high SSII group [odds ratio (OR), 1.003; 95% confidence interval (CI), 1.001-1.005; p<0.001; OR, 1; 95% CI, 1.0-1.001, p=0.031, respectively, Table 2]. Moreover, a positive relationship was detected between PTH and a high SSII score and neutrophil-to-lymphocyte ratio (respectively $r = 0.458$; $P < 0.001$, Figure 1, $r = 0.176$; $P < 0.038$). In addition, we found that PTH could predict the extremity of coronary artery failure with 64.4% sensitivity and 65.3% specificity at 250 ng/mL cut-off value (Area under the curve: 0.718, 95% CI, 54.8-74.7; $P < 0.001$, Figure 2).

DISCUSSION

In this study, we determined a considerable connection between serum PTH levels and the severity of CAD in patients

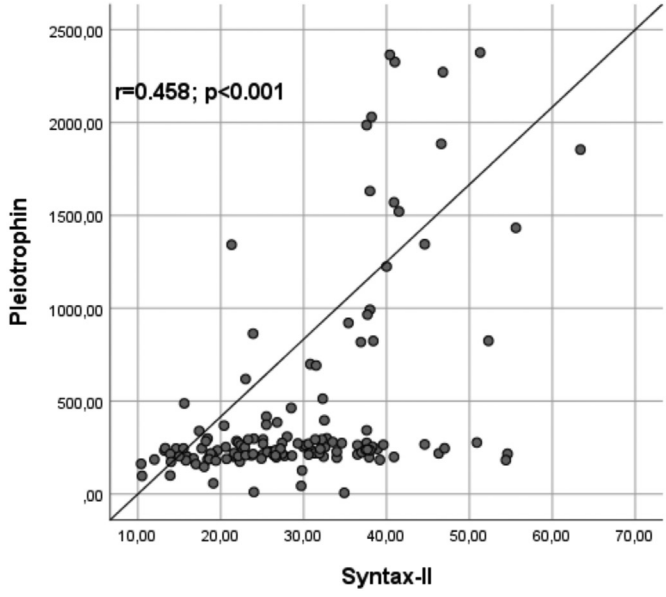

Figure 1: Correlation between pleiotrophin and the SYNTAX II score

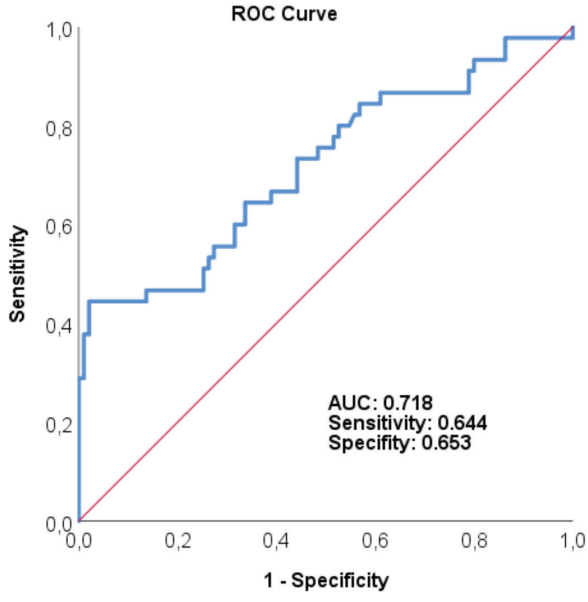

Figure 2: Analysis curve for pleiotrophin to predict coronary artery disease severity

ROC: Receiver operator characteristic, AUC: Area under the curve

Table 1: Demographics, clinical, and laboratory data according to the SYNTAX II score groups

Parameters	SYNTAX score			*P*-value
	Low group (≤22; *n*=39)	Intermediate group (23-32; *n*=56)	High group (≥33; *n*=45)	
Age, years	55.2±9.1[a]	61.1±9.8[a, b]	67.3±8.9[b]	<0.001
Male sex, *n* (%)	32 (22.9)	44 (31.4)	22 (15.7)	0.001
Hypertension, *n* (%)	26 (18.6)	35 (25.0)	15 (10.7)	0.31
Diabetes mellitus, *n* (%)	23 (16.4)	39 (27.9)	21 (15.0)	0.028
Active smoker, *n* (%)	15 (10.7)	15 (10.7)	27 (19.3)	<0.001
Family history of CAD, *n* (%)	30 (21.4)	45 (32.9)	39 (27.9)	<0.001
Creatinine (mg/dL)	0.77 (0.68-0.92)	0.77 (0.62-0.95)	0.88 (0.66-1.0)	0.284
Uric acid (mg/dL)	5.5±1.4	5.8±1.9	5.7±1.6	0.583
Triglyceride (mg/dL)	165 (75-236)	114 (67-162)	95 (67-146)	0.086
LDL-C (mg/dL)	120 (110-143)	124 (105-152)	126 (105-157)	0.943
HDL-C (mg/dL)	42.0 (37.1-46.0)	43.5 (35.0-46.9)	43.5 (39.0-47.8)	0.520
Hemoglobin (mg/dL)	13.9±1.3	14.0±1.5	13.4±1.5	0.05
WBC ($\times10^3$/μL)	10.0 (8.9-11.9)	10.3 (7.8-12.3)	9.8 (8.2-12.2)	0.786
Neutrophil ($\times10^3$/μL)	6.2 (5.0-8.7)	6.8 (5.0-9.4)	7.6 (5.9-9.8)	0.411
Lymphocyte ($\times10^3$/μL)	1.7 (1.3-3.2)	1.8 (1.2-2.4)	1.3 (0.9-2.0)	0.020
Platelet ($\times10^3$/μL)	240 (204-278)	226 (202-318)	240 (206-308)	0.814
proBNP (pg/mL)	165 (97-343)	287 (118-866)	672 (163-2080)	<0.001
hs-C-reactive protein (mg/dL)	0.5 (0.3-0.9)	0.4 (0.3-0.9)	0.4 (0.3-1.0)	0.984
LVEF (%)	50.0 (46.0-55.0)	45.0 (40.0-50.0)	40 (30.0-45.0)	<0.001
Pleiotrophin (ng/mL)	207.7 (185.4-245.6)	248.1 (208.8-293.8)	277.1 (231.9-1476.8)	<0.001

LDL-C: Low-density lipoprotein cholesterol, HDL-C: High-density lipoprotein cholesterol, WBC: White blood cell, BNP: Brain natriuretic peptide, LVEF: Left ventricular ejection fraction, [a]In the post hoc analysis of the Bonferroni test, it was found to be statistically significant in the high group, [b]In the post hoc analysis of the Bonferroni test, it was found to be statistically significant in the low-risk group

with NSTEMI. Thus, it can be considered to be the first study conveyed on humans in the sense of showing a remarkable relationship between high serum PTH levels and the severity of CAD.

In our study, the relationship between hospital admission serum PTH levels and the severity of CAD calculated by SSII was analyzed. Thus, high serum PTH levels are associated with severe coronary atherosclerosis in NSTEMI. In addition to the positive correlation between PTH and SSII, increased PTH levels were found to have an independent predictive value for the severity of CAD.

The most important cause of CAD is coronary atherosclerosis. The triggering mechanism of acute coronary syndrome (ACS) is the emergence of an intracoronary thrombus caused by the erosion or rupture of the atherosclerotic coronary plaque and the inclusion of matrix materials and thrombogenic core from the plaque into the circulation.[15] Within this context, inflammation has a great influence together with numerous other risk agents, especially cardiac inflammation, which results in a life-threatening complication of NSTEMI by causing issues of morbidity and mortality for the general public.[16]

In many studies, patient populations with ACS have been broadly examined for various clinical findings.[17] In the majority of these studies, the SYNTAX score I was assessed, and its relationship with the severity of CAD was revealed. However, the SYNTAX score I is not satisfactory for clinical assessment in the presence of complex CAD. Therefore, the recently developed SSII can enable both clinical and anatomical assessment. In addition to anatomical data, SSII includes clinically remarkable prognostic variations, such as gender, age, and creatinine clearance. Chronic obstructive pulmonary disorder, peripheral artery disease, and LVEF are common independent markers of mortality in patients with stable CAD enrolled in the SYNTAX test.[18,19] Studies conducted with SYNTAX have reported that SSII predicts mortality better.[20,21] Therefore, the SSII scores can effectively demonstrate the individualized chance of mortality associated with each revascularization procedure. In addition, it has been recently demonstrated by research that SSII can

Table 2: Univariate and multivariate logistic regression analysis for the assessment of independent predictors of high SYNTAX scores

	Univariate		Multivariate	
	***P*-value**	**OR (95% CI)**	***P*-value**	**OR (95% CI)**
Diabetes mellitus	0.038	2,147 (1,043-4.42)	0.897	1,068 (0.392-2,908)
Hypertension	0.001	3,588 (1,698-7,584)	0.166	2,025 (0.745-5,503)
Family history of CAD	0.339	0.615 (0.227-1,666)		
Uric acid	0.879	0.984 (0.795-1,217)		
Sodium	0.271	0.944 (0.852-1,046)		
WBC	0.503	0.959 (0.85-1,083)		
Neutrophil	0.364	1,059 (0.936-1,197)		
Lymphocyte	0.010	0.574 (0.376-0.877)	0.823	0.946 (0.581-1,54)
Hemoglobin	0.017	0.733 (0.568-0.947)	0.391	0.867 (0.626-1,201)
MCV	0.108	1,055 (0.988-1,127)		
Platelet	0.536	1,002 (0.996-1,007)		
Triglyceride	0.163	0.998 (0.995-1,001)		
Total cholesterol	0.486	0.998 (0.991-1,004)		
LDL-C	0.825	1,001 (0.992-1,011)		
HDL-C	0.071	1,035 (0.997-1,074)	0.222	1,029 (0.983-1,078)
proBNP	0.006	1 (1-1,001)	0.031	1 (1-1,001)
hs-CRP	0.965	0.997 (0,866-1,147)		
Pleiotrophin	<0.001	1,003 (1,001-1,004)	<0.001	1,003 (1,001-1,005)

CAD: Coronary artery disease, WBC: White blood cell, MCV: Mean corpuscular volume, HDL-C: High-density lipoprotein cholesterol, LDL-C: Low-density lipoprotein cholesterol, pro-BNP: Brain natriuretic peptide, hs-CRP: High-sensitivity C-reactive protein, OR: Odds ratio, CI: Confidence interval

independently predict the incidence of all-cause deaths in patients suffering from 1-or 2-vessel failures.[22]

PTH, an 18-kDa secretory protein, is involved in various biological functions, such as migration, survival, and growth of cells, angiogenesis, neurite outgrowth, and tumor growth.[23,24] As a multifunctional cytokine, it is also known as a heparin-binding growth-associated molecule, osteoblast-specific factor-1, heparin-binding growth factor-8, or neurite outgrowth promoting factor, and is remarkably conserved across several species.[25]

The *PTH* gene is deactivated in the postnatal period and is in small amounts in most adult tissues. Upregulation of PTH in adults supports tumoral and non-tumor pathological angiogenesis and physiological angiogenesis. PTH has been observed in microglia and macrophages in neovascular endothelial cells and neovasculature around the infarcted rat brain secondary to ischemic injury. However, the expression of PTH was remarkably reduced in injured neurons that were unlikely to survive, highlighting the regulative role of PTH in postischemic renewal.[10] An important controversy regarding the therapeutic role of proangiogenic agents is their protumorigenic potential, which can lead to undesirable consequences. In another study, it was shown that *PTH* gene delivery has a functional effect in a severe ischemic hindlimb model in mice with low tumorigenic potential.[26] The effect of PTH on cardiac tasks has not yet been entirely elucidated. Research conducted *in vitro* suggests that PTH is upregulated when cardiomyocytes begin to grow out of pluripotent stem cells.[27] Research conducted on a rodent heart revealed that strong PTH staining can be seen in peri-infarction and infarct regions, similar to cerebral infarction. Moreover, it has been stated that there is an increase in PTH value during the removal of cellular residues, remodeling in heart failure, and scar formation.[28] PTH can be assessed for potential future benefits in vascular bioprosthesis engineering.[11] In a study, it was reported that serum PTH levels in patients with angina pectoris were associated with maintaining coronary collateral circulation. However, serum PTH levels were not detected in patients with ACS.[12] Başyiğit et al.[29] It was determined that the serum concentration of PTH in the patients was related to ACS. Despite animal models, few studies are available on human cells; therefore, this research can be considered as the first attempt to support the therapeutic impact of this effective molecule in humans. This study confirms the score of PTH as a biomarker showing the relationship between CAD severity and PTH.

In our study, in addition to PTH, proBNP, hs-CRP, lymphocyte, hemoglobin, HDL-C level, diabetes mellitus, HT, and family

history of CAD were assessed. Statistically significant levels were obtained by univariate logistic regression analysis, except for hs-CRP. In addition, proBNP was found to be an independent predictor of SSII in multivariate regression analysis. The fact that these variables were statistically significant supports our hypothesis.

Although hs-CRP was found to be insignificant in demonstrating the severity of CAD in our study, it is an independent risk agent for CAD.[30] Numerous studies have revealed that high levels of hs-CRP can be linked with a high atherosclerosis progression rate in the carotid vessels and the risk of stroke.[31] This indicates that hs-CRP may be associated with inflammation. However, a wide variety of controversial articles are available on hs-CRP and its effect on the treatment, diagnosis, and prevention of all primary and secondary CAD cases. The main reason the hs-CRP level was insignificant in our study may be that we did not reach a sufficient number of patients.

In our study, we hypothesized that PTH, an anti-inflammatory factor, may be correlated with the existence of NSTEMI and the extremity of CAD. Thus, we examined the relationship between high PTH rates, the incidence of NSTEMI, and the severity of CAD. Lab findings showed that PTH rates in NSTEMI patients were positively and remarkably correlated with high SSII. In addition, it was determined that there was an independent predictive variable for high SSII in the regression analysis. Nevertheless, further studies are required to clarify the prognostic effect of PTH rates in patients with ACS.

Study limitations

Some limitations of this study are as follows: the presence of a single center and therefore a small sample size. Because the study was designed in a cross-sectional plan, no follow-up data could be obtained. Second, we could only calculate PTH rates at admission, but not convey serial measurements owing to financial issues. In fact, measuring PTH levels after the acute phase of infarction could reveal additional information. Third, other possible inflammatory and pro-angiogenic factors have not been assessed simultaneously in the same population. If the interaction between PTH and proangiogenic factors is demonstrated with simultaneous studies, it will provide more precise results.

CONCLUSION

In patients with NSTEMI, serum PTH levels were significantly associated with higher SSII, an indicator of CAD severity and CV prognosis. It also has an independent predictive value for high SSII. This outcome could pave the way for a more aggressive pharmacoinvasive treatment strategy in patients with high SYNTAX scores. However, larger prospective examinations are required to detect the effect of PTH rates in patients with ACS.

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Ethics

Ethics Committee Approval: After obtaining a written consent form from all patients and approval of the study protocol by the Ankara City Hospital Ethics Committee, the study protocol (approval number: 2022-2375; date: 09.02.2022) was conducted in accordance with the ethical guidelines of the 1975 Helsinki Declaration.

Informed Consent: Informed consent was obtained.

Peer-review: Internally peer-reviewed.

Authorship Contributions

Surgical and Medical Practices: S.G.N., O.K., F.B., B.Ş., M.C., Concept: S.G.N., O.K., F.B., B.Ş., M.C., Design: S.G.N., O.K., F.B., B.Ş., M.C., Data Collection or Processing: S.G.N., O.K., Analysis or Interpretation: S.G.N., O.K., F.B., B.Ş., M.C., Literature Search: S.G.N., O.K., F.B., B.Ş., M.C., Writing: S.G.N., O.K., B.Ş., M.C.

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CASE REPORT

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Successful Retrieval of Stripped Coronary Stents Using the Twisted Guide Wire Technique

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Abstract

Stent stripping during percutaneous coronary intervention is a rare but serious complication. The patient was admitted to the emergency department with chest pain. Electrocardiography revealed ST elevations in the anterolateral leads. The patient was urgently taken to the catheter laboratory. A guiding catheter without a hole was used to imagine the left coronary system. To avoid dumping, two holes were manually drilled into the distal region of the catheter with the needle tip. Occlusion was observed in the proximal region of left anterior descending artery. It was decided to implant a stent. However, when the stent arrived distal in the catheter, strain was felt. When the stent was advanced to the left main coronary artery (LMCA), it was observed that the stent detached from its balloon in the LMCA. It was planned to remove the stent using the twisted guide wire technique. In this technique, the guide wire inside the stripped stent lumen was not retracted. A second guide wire was advanced distally outside the stent lumen. Subsequently, these two parallel wires were twisted 10-15 times until the stent was trapped by the guide wires with the help of the torquer device. Twisted wires were pulled back. While the wires were retrieving, the trapped stent between guide wires was pulled back. Subsequently, a new stent was successfully implanted into the lesion. After the procedure, the damaged catheter and the removed stent were investigated. The distal inner lumen of the guiding catheters not smooth at the edges of the manually opened side holes because of protrusions. It was thought that the stent was stuck to these inner protrusions of the holes and detached from the balloon. Manually drilling a hole in the guiding catheter or any intervention disrupting the structure of the catheter should be avoided. The twisted guide wire technique is an alternative and effective method to retrieve a stripped stent.

Keywords: Complication, stent stripping, twisted guide wires

INTRODUCTION

Stent stripping during percutaneous coronary intervention is a rare but serious complication. This can lead to systemic or coronary embolization, which can result in severe morbidity and mortality.[1] Stripping of the stent can lead to emergency coronary bypass surgery, coronary thrombosis, myocardial infarction, cerebrovascular accident, and death. Calcified and angulated lesions increase the risk of stent stripping and embolization.[2] Various devices and techniques are used to remove the stripped stent. There is no standard method for removing the embolized stent, and different methods can be applied depending on the case and the operator. In this case report, we presented a stripped stent due to manual puncture of the back-up guiding catheter and its removal using the twisting guide wire technique. In this case report, we highlight the risk of complications that may arise from manual puncture of the back-up guiding catheter and emphasize the effectiveness of the twisting guide wire technique in retrieving a stripped coronary stent. This case serves as a reminder that even small manual interventions on equipment during a procedure can lead to significant and potentially life-threatening complications.

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CASE REPORT

A 68-year-old female patient with a past medical history of hypertension without any cardiac history was admitted to the emergency department with chest pain. Electrocardiography revealed ST elevations in the D1-AVL and V1-V6 leads and reciprocal ST depression in the inferior leads (Figure 1). The patient's blood pressure was found to be 140/85 mmHg, heart rate was 105 beats/min, and physical examination findings were normal. The patient was urgently taken to the catheter laboratory with a diagnosis of acute anterolateral myocardial infarction for primary percutaneous intervention. Ticagrelor (180 mg), acetyl salicylic acid (300 mg), and unfractionated heparin (100 u/kg) were administered during the procedure. The patient was cannulated with a 7 French (Fr) sheath through the right femoral artery, and a 6 Fr diagnostic right Judkins 4 catheter was advanced. No critical stenosis was observed in the right coronary artery. Later, a 7-Fr extra backup (EBU) 3.5 guiding catheter without a hole was used to visualize the left coronary system. To avoid dumping, two holes were manually drilled into the distal region of the catheter with the needle tip. There was no significant stenosis in the circumflex artery. Occlusion was observed in the proximal region of the left anterior descending artery (LAD). The lesion was crossed with a floppy guide wire (0.014 Asahi). Coronary flow was provided after 2.0x15 mm balloon predilatation. Then, it was decided to implant a 3.5x16 mm drug-eluting stent (DES) (Firehawk; Microport, Shanghai, China). However, when the stent arrived distal in the catheter, strain was felt. When the stent was advanced to the left main coronary artery (LMCA), the stent was detached from its balloon in the LMCA. It was planned to remove the stent using the twisted guide wire technique. In this technique, the guide wire inside the stripped stent lumen was not retracted. A second guide wire was advanced distally outside the stent lumen. Subsequently, these two parallel wires were twisted 10-15 times until the stent was trapped by the guide wires with the help of the torquer device. The twisted wires were then pulled back. While the wires were retrieving back, the trapped stent between guide wires was pulled back (Figure 2). Figure 3 shows the step-by-step twisted guide technique. Subsequently, a new not perforated EBU 3.5 guiding

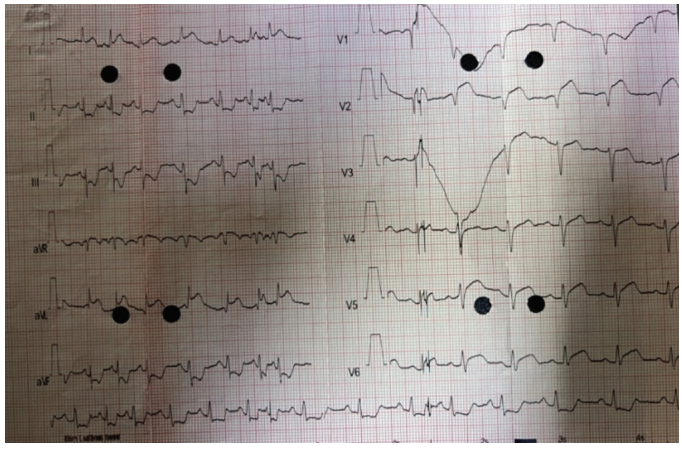

Figure 1: Electrocardiography at admission showing ST-segment elevation in D1-AVL, V1-V6 leads

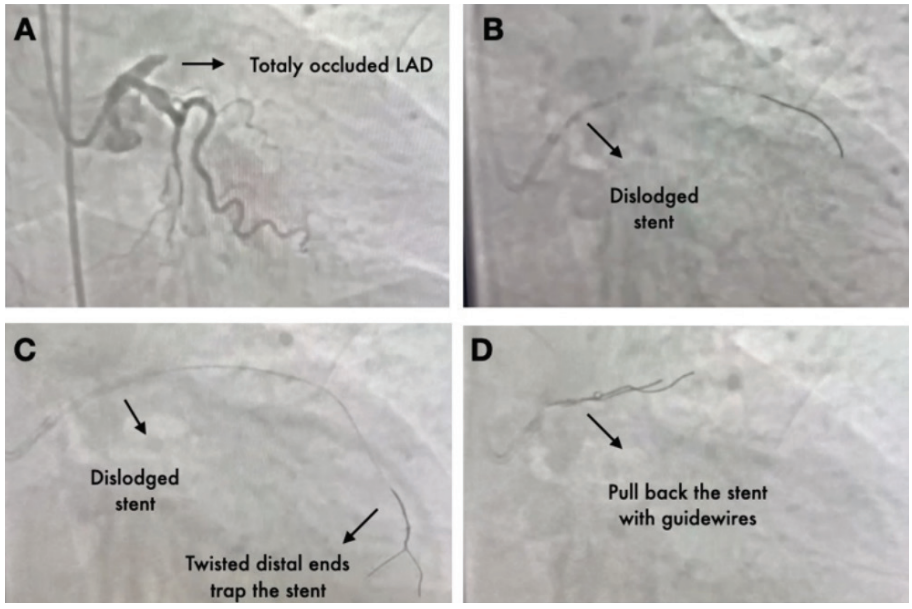

Figure 2: Angiographic views **A.** Totally occluded LAD, **B.** Dislodged stent in LMCA, C. Second guide wire was advanced outside the stent lumen and two parallel wires were twisted around each other 10-15 times until the stent was trapped by the guide wires. D. While the wires were retrieving back, trapped stent between guide wires was pulled back.

LAD: Left anterior descending artery, LMCA: Left main coronary artery

catheter was advanced to LMCA. Predilatation was applied to the lesion this time with a 2.5x15 mm balloon. Subsequently, a new 3.5x16 mm DES (Firehawk; Microport, Shanghai, China) was successfully implanted into the lesion. Instent postdilatation was performed using a 4.0x12 mm noncompliant balloon, and the procedure was successfully terminated. After the procedure, the damaged catheter and the removed stent were investigated. The distal inner lumen of the guiding catheters not smooth at the edges of the manually opened side holes because of protrusions (Figure 4). It was thought that the stent was stuck to these inner protrusions of the holes and detached from the balloon. During follow-up, the patient had no complaints, the ejection fraction was 55%, and no wall motion abnormality was observed on echocardiography. The patient was discharged after adjusting the optimal medical treatment, and the patient had no complaints at the next outpatient visit. Written informed consent was obtained from the patient for publication of the case report and the accompanying images.

DISCUSSION

Stent stripping usually occurs for 3 reasons.[1] These are;

1. The stent can be detached from the balloon while the stent is being advanced to the not well predilated coronary artery segment.

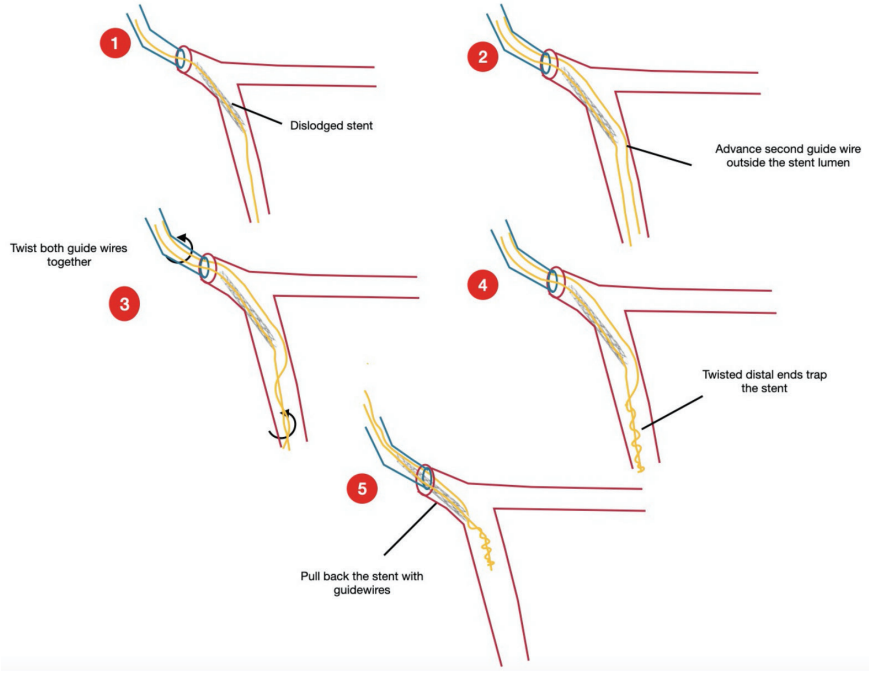

Figure 3: Step by step twisted guide wires technique

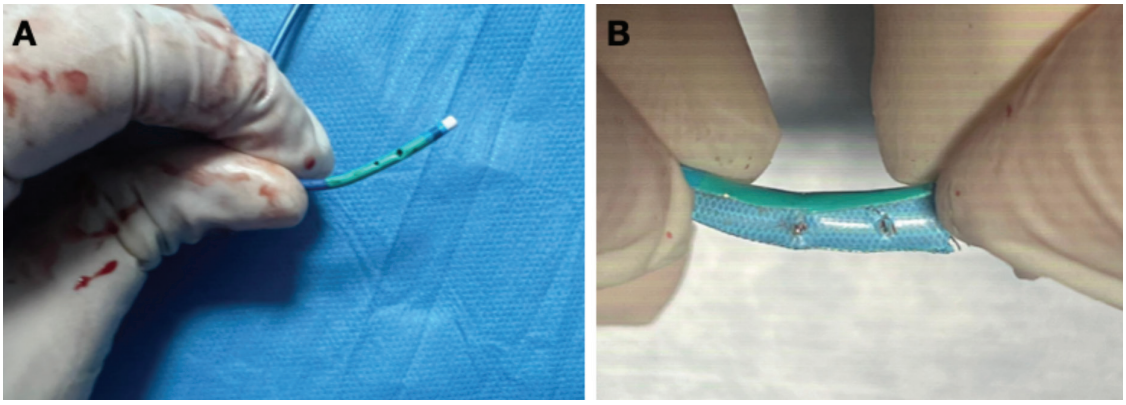

Figure 4: Images of the catheter **A.** Manually drilled holes on the extra-back up guiding cat heter, **B.** The inner side of the catheter showing protrusions at the edges of the holes

2. The stent may become stuck in the lesion when advancing from the coronary lesion and may be stripped from its balloon when retreating.

3. If the stent was deformed proximally during the procedure or if the catheter was not coaxially cannulated, the stent may be stripped as it was pulled into the catheter. This is why it occurs most often.

However, in our case, because of the unnoticed damage to the inner lumen of the distal end of the catheter when opening a hole in the catheter in a way not included in the literature, the stent was stripped due to inner protrusions of the holes in the catheter while it was entering the main coronary.

When a stent is detached from the balloon in the coronary artery it is necessary to decide what can be done based on the position and condition of the stent. It should be planned to remove the stent back first, but if this is not possible, it should be considered to implant it in a suitable place in the artery. If the stent is to be removed, there are many methods available to retrieve the stripped stent, including gooseneck snare, balloon inflation distal to the stent in a low atmosphere, and the twisted guide wire technique.[3,4] In this case, we preferred the twisted guide wire technique, where one of the guide wires is inside the lumen of the stripped stent, while the other guide wire is advanced outside the stent.[5,6] The same torque device is attached to the ends of the two guide wires and rotated in the same direction until they twist each other.[7] Then, the two wires are pulled back together, and the stent is retrieved using the guide wires.[8] The operator should have previous experience with this twisted guidewire technique to decrease the risk of coronary artery damage. The twisting guide wire technique may cause coronary artery dissection. The operator has previous experience with this maneuver to avoid coronary artery dissection because we seek to palliate damage without increasing it. In cases where the stent cannot be retrieved and implanted where it is, it can be attached to the vessel wall with a new stent as a bail-out procedure, but this will increase the metal load. In addition, coronary bypass is an alternative to stent stripping in multi-vessel coronary artery disease involving LAD.[9]

CONCLUSION

Stent stripping is a rare but difficult complication of percutaneous procedures that can cause serious cardiovascular problems. The operator should not force the stent, especially through an angulated and severe calcified coronary artery. Moreover, manually drilling a hole on the guiding catheter or any intervention disrupting the structure of the catheter should be avoided. The twisted guide wire technique is an alternative and very effective method to retrieve a stripped stent. The technique to be used in the case of an emboldized stent should be decided according to the experience of the operator and the patient's condition. It is extremely important to keep the laboratory equipped with equipment that may interfere with stent stripping.

Ethics

Informed Consent: Written informed consent was obtained from the patient for publication of the case report and the accompanying images.

Peer-review: Internally peer-reviewed.

Authorship Contributions

Surgical and Medical Practices: E.K., T.Ş., Concept: E.K., T.Ş., Design: E.K., T.Ş., Data Collection or Processing: E.K., T.Ş., Analysis or Interpretation: E.K., Literature Search: E.K., Writing: E.K., T.Ş.

Conflict of Interest: No conflict of interest was declared by the authors.

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CASE REPORT

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Innovative Technique for Evacuating Side Branch in Bifurcation Lesion

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Abstract

Treatment of bifurcation lesions changes according to lesion characteristics and the patient's clinical diagnosis, including acute or chronic coronary syndrome. Treatment of bifurcation lesions in patients with acute coronary syndrome (ACS) is more difficult. We presented an innovative treatment for a bifurcation lesion in a patient with ACS.

Keywords: Coronary bifurcation, thrombus aspiration catheter, acute coronary syndrome

INTRODUCTION

Bifurcation lesions are considered to be a serious problem in invasive cardiology. Treatment of bifurcation lesions in patients with acute coronary syndrome (ACS) is more difficult. We presented an innovative treatment for a bifurcation lesion in an ACS patient.[1]

CASE REPORT

A 59-year-old male patient was admitted to the emergency department with two hours of chest pain. He was diagnosed with hyperacute anterior myocardial infarction in the emergency department. The cardiac risk factor for the patient was smoking. He was transferred urgently to the catheterization laboratory to perform percutaneous coronary intervention (PCI). We performed coronary angiography via the right femoral access. The left anterior descending coronary artery (LAD) was occluded from the mid-portion (Figure 1a). The completely occluded part of LAD was passed with a soft wire (Choice floppy - Boston Scientific) gently. Consecutive pre-dilatation was performed using a 2.0*20 balloon (Sprinter - Medtronic). After balloon inflation, we took consecutive films to evaluate the lesion and plan the interventional strategy.

There was a serious lesion in the mid-portion of LAD before diagonal 1 (D1) bifurcation. We preferred provisional LAD stenting. Also, D1 was wired (Choice floppy - Boston Scientific).

Then a 3.0*24 drug-eluting stent (DES) (Promus - Boston Scientific) was inserted into the mid-LAD. We performed proximal optimization (POT) via a 3.5*12 non-compliant (NC) balloon (Sprinter - Medtronic) without passing the carina after stent implantation. The total occlusion of LAD was completely resolved. However, serious plaque shifting developed into D1 (Figure 1b).

We decided to make a kissing balloon to restore plaque shifting into D1 without disturbing LAD flow. We rewired D1 (Choice floppy - Boston Scientific) and performed kissing balloon inflation with two NC balloons (3.0*20 Sprinter - Medtronic for LAD and 2.75*20 Sprinter - Medtronic for D1) (Figure 1c). After

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kissing balloon inflation, we saw no reflow in the D1 artery. We thought dissection was the main cause of no reflow in the D1 artery (Figure 1d). We inserted a 2.75*28 DES (Promus - Boston Scientific) into the D1 artery using the T and small protrusion technique (Figure 1e). No reflow was observed in the D1 artery after stent implantation (Figure 1f). We did not get a response to 500 ug intracoronary adenosine.

If you were the operator in this case, what would you do?

We decided to evaluate the distal part of the D1 stent via a thrombus aspiration catheter (TAC). We passed the distal part of the D1 stent via 6F TAC (Medtronic, Minneapolis, MN, USA). Blood was drawn back, and 2 cc opaque was given to ensure that the catheter was in the artery lumen. There was serious dissection in the distal part of the stent (Figure 2). We decided to insert another stent to overlap the first stent. Finally, we restored the D1 flow and completed the procedure with final kissing and POT (Figure 1g).

We took control of angiography one month later. LAD and D1 stents were patent, and thrombolysis in myocardial infarction III flow was observed in both arteries (Figure 1h). The study complied with the Declaration of Helsinki and informed consent has been obtained from the participant.

DISCUSSION

There are some techniques for treating bifurcation lesions, including provisional, mini crush, double kissing, and T stent. Treatment of bifurcation lesions changes according to lesion characteristics and the patient's clinical diagnosis, including acute or chronic coronary syndrome (CCS). Operators are more comfortable in treating bifurcation lesions in CCS patients than in ACS patients. Because they have more time and are ready for treatment techniques with planning before.[1]

If operators perform PCI in bifurcation lesions in ACS patients, they may encounter additional ACS problems not only in bifurcation lesions. Because ACS patients have unstable clinical situations due to thrombotic unstable plaque, TAC was developed for thrombus aspiration, especially in ACS patients. However, we used TAC in a bifurcation lesion in an ACS patient to evaluate side branch flow for discrimination thrombus,

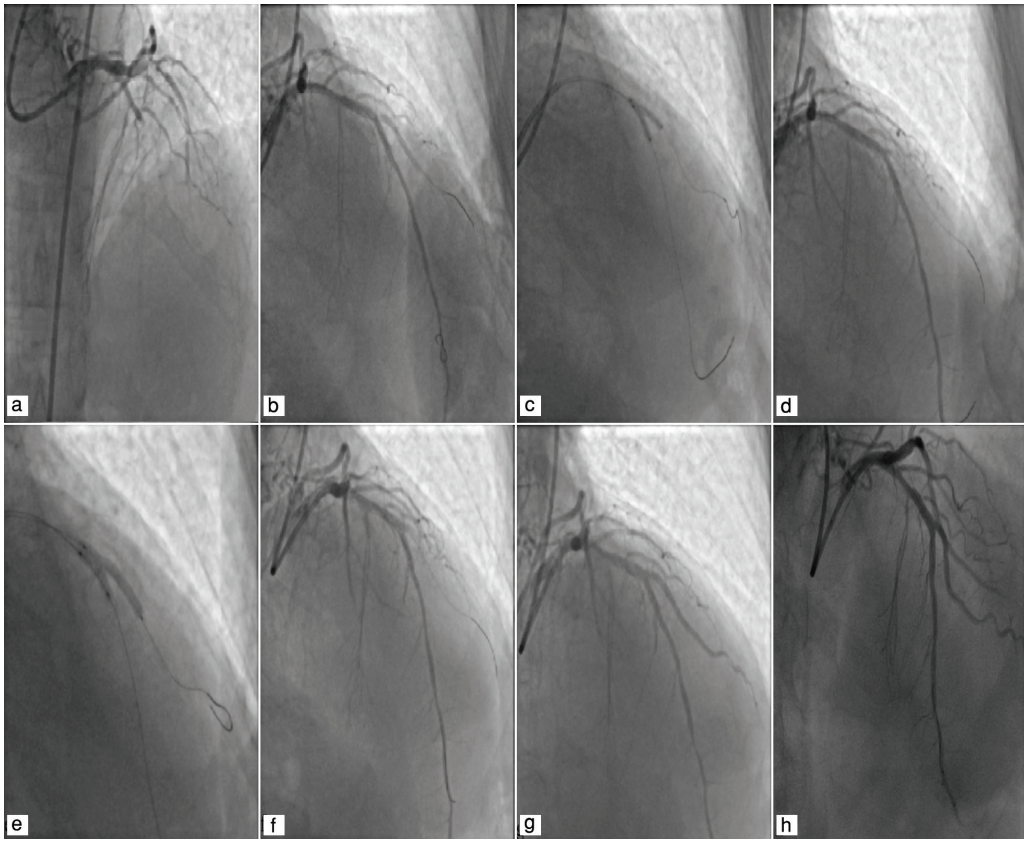

Figure 1: a) Totally occluded LAD artery. **b)** Serious plaque shift into the D1 artery. **c)** Kissing balloon inflation in bifurcated LAD-D1 arteries. **d)** No reflow in the D1 artery. **e)** Insertion of a DES into the D1 artery using the T and small protrusion technique. **f)** No reflow in the D1 artery after stent implantation. **g)** Restored flow in the D1 artery. **h)** Thrombolysis in myocardial infarction III flow in LAD and D1 arteries after one month later

LAD: Left anterior descending coronary artery, D1: Diagonal 1, DES: Drug-eluting stent

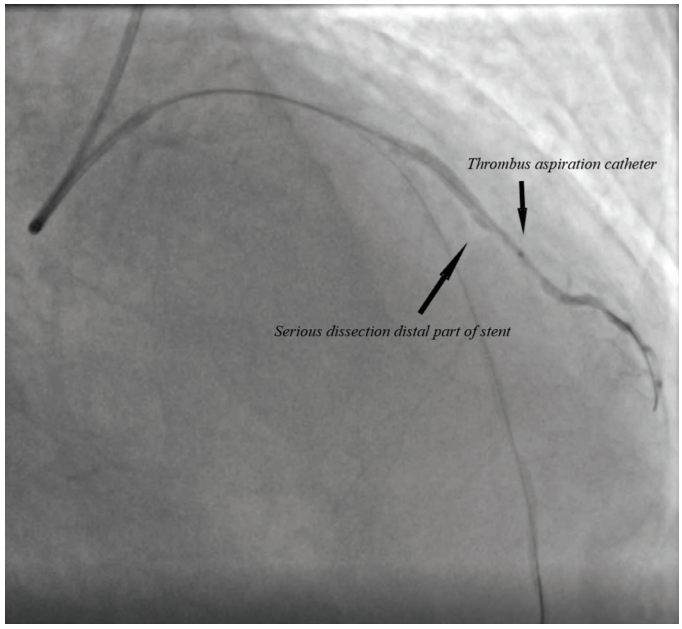

Figure 2: Serious dissection of the distal part of the stent in the D1 artery

D1: Diagonal 1

dissection, and no-reflow. First, TAC was used to visualize the occluded distal part of the side branch vessel in this case. Second, we do not need to change the wire in the side branch, contrary to the microcatheter. However, the optimal method to evaluate the distal part of the side branch is intravascular ultrasonography (IVUS) or optic coherence tomography (OCT) in this case. However, we did not manage to use this equipment due to technical reasons in an emergency. TAC was used to visualize the occluded distal part of the stent.[2-4]

CONCLUSION

If imagining techniques such as IVUS or OCT are unavailable, TAC can be used in emergencies to evaluate the distal part of the stent in case of no reflow, as in our case.

Ethics

Informed Consent: Informed consent has been obtained from the participant.

Peer-review: Externally and internally peer-reviewed.

Authorship Contributions

Surgical and Medical Practices: R.G., B.S.Y., Concept: B.S.Y., Design: R.G., Data Collection or Processing: B.S.Y., Analysis or Interpretation: S.Ö., Literature Search: R.G., B.S.Y., Writing: R.G.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

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