An under-diagnosed caused of myocardial infarction: Coronary embolism

Miyokard enfarktüsünün yeterince teşhis edilemeyen bir nedeni: Koroner emboli

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ABSTRACT

The coronary embolism (CE) is a rare cause of acute coronary syndrome (ACS). The most common cardiovascular disease linked to coronary embolism is atrial fibrillation (AF). Despite the acknowledgment of the entity, it is regarded an under-diagnosed cause of ACS, and there is uncertainty over the short and long term prognosis of these patients. In this case report, we aimed to provide information about the diagnosis and treatment process of a patient who presented with acute inferior myocardial infarction caused by coronary embolism.

Key Words: coronary embolism, acute coronary syndrome, myocardial infarction

ÖZ

Koroner emboli (KE), akut koroner sendromun (AKS) nadir görülen bir nedenidir. Koroner emboli ile ilişkili en yaygın kardiyovasküler hastalık atriyal fibrilasyondur (AF). Varlığının kabul edilmesine rağmen, AKS'nin yeterince teşhis edilmemiş bir nedeni olarak kabul edilir ve bu hastaların kısa ve uzun vadeli prognozu konusunda belirsizlik vardır. Bu olgu sunumunda, koroner emboli nedeniyle akut inferior miyokard enfarktüsü geçiren bir hastanın tanı ve tedavi süreci hakkında bilgi vermeyi amaçladık.

Anahtar Sözcükler: Koroner emboli, akut koroner sendrom, miyokard infarktüsü

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Introduction

The coronary embolism (CE) is an uncommon cause of myocardial infarction (MI). Acute coronary syndrome (ACS) caused by CE has been reported to occur in 3% of cases, while post-mortem investigations reveal an incidence of up to 13% [1]. The most common cardiovascular disease linked to coronary embolism is atrialfibrillation (AF). Despite the acknowledgment of the entity, it is regarded an underdiagnosed cause of ACS, and there is uncertainty over the usual short- and long-term care of these individuals. Understanding the underlying pathophysiology is crucial for optimizing treatment, as it is distinct from plaque rupture and thrombus development [2]. Developing a betterknowledge of coronary embolism and identifying invasive and non-invasive treatment options are the goals of our case report.

Case Report

A 51 year old male patient with typical angina which started 2 hours before admission was admitted to our emergency department. The patient had a history of paroxysmal atrial fibrillation but was not on any regular medication. His vital signs were in normal range and physical examination was normal. The ECG showed ST segment elevation in leads II, III and aVF. The patient was taken into cathetherization laboratory for primary percutanous intervention. The patient's coronary angiography showed thrombus which caused %100 occlusion of distal right coronary artery (RCA). His left main, circumflex and left anterior descending coronary arteries showed no significant lesion (Figure 1). Subsequently, angioplasty was performed using 2.0x15 mm and 2.5x25 mm semi-compliant balloons up to 16 atm with no improvement in flow. The patient underwent manual mechanical thrombus aspiration with a thrombus aspiration catheter which resulted in an large amount of thrombotic material and improvement of distal flow to TIMI 3 without evidence of atherosclerotic lesion or residual thrombus in RCA (Figure 2). Intracoronary tirofiban was administered followed by 24 hours infusion. Transeosophageal and transthoracic echocardiography revealed no thrombus at left atrium, ventricule and left atrial appandage. Rheumatological markers, thrombophilia genetic panel and hemolytic parameters were normal. After exclusion of other causes of arterial thrombus, coronary thromboembolism was proposed as the underlying mechanism in the current patient according to Shibata criteria [3] (Table 1). We diagnosed our patient with coronary embolism with 1 major (angiographic coronary embolism without evidence of atherosclerosis) and 2 minor (atrial fibrillation, stenosis <25% in other coronary arteries other than the target lesion) criteria.

The patient was discharged with clopidogrel 75 mg and rivaroxaban 20 mg daily (acetylcalicilic acid was discontinued after 1 week). At 3-month follow up, the patient remained stable with no reported symptoms.

In this article, we presented a case of acute coronary syndrome caused by coronary embolism.

One misunderstood cause of sudden coronary occlusion is coronary embolism. It nearly disappears following surgery or coronary intervention. CE may be caused by paradoxical emboli, emboli from the left ventricle, or emboli from the left atrial appendage.[1]

Table 1: Shibata criterias [3]

Major Criteria	Minor Criteria
a) Lack of angiographic evidence of atherosclerosis with coronary embolism or thrombüs	a) Stenosis <25% on coronary angiography, except in the culpirit lesion
b) Simultaneous coronary embolization: multiple vessels in the same coronary region or multiple vessels of the coronary tree	b) Evidence of an embolic sources by multimodal imaging
c) Simultaneous systemic embolism without evidence of intracavitary thrombus attributed to acute myocardial infarction	c) Presence of embolic risk factors such as atrial fibrillation, dilated cardiyomyopathy, rheumatic valve disease, presence of valve prosthesis, patent foromen ovale, atrial septal defects, infective endocarditis, or hypercoagulable state

Two or more major criteria, one major and more than two criteria, or all three minor criteria make a definitive diagnosis of coronary embolism, and a probable diagnosis would be made if one major criterion or two minor criteria are present
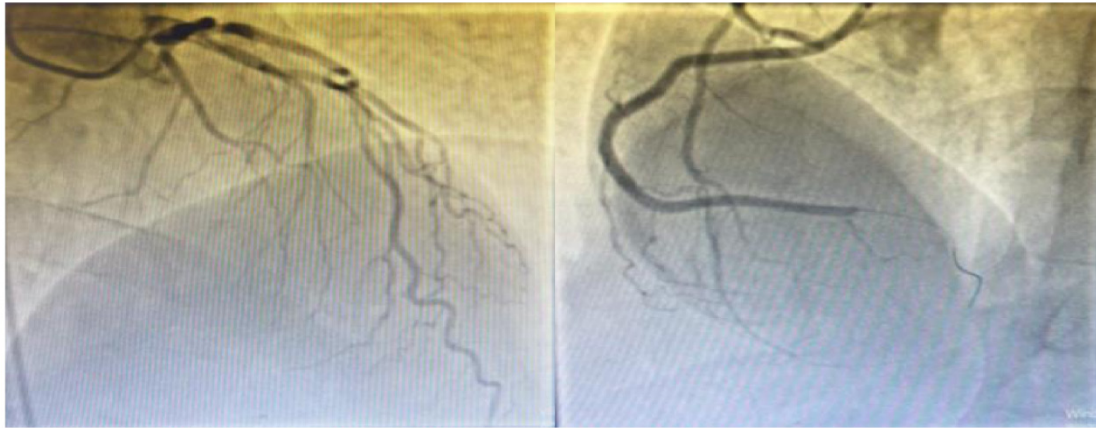
Figure 1. A) thrombus which caused %100 occlusion of distal right coronary artery (RCA). **B)** Left main, circumflex and left anterior descending coronary arteries showed no significant lesion
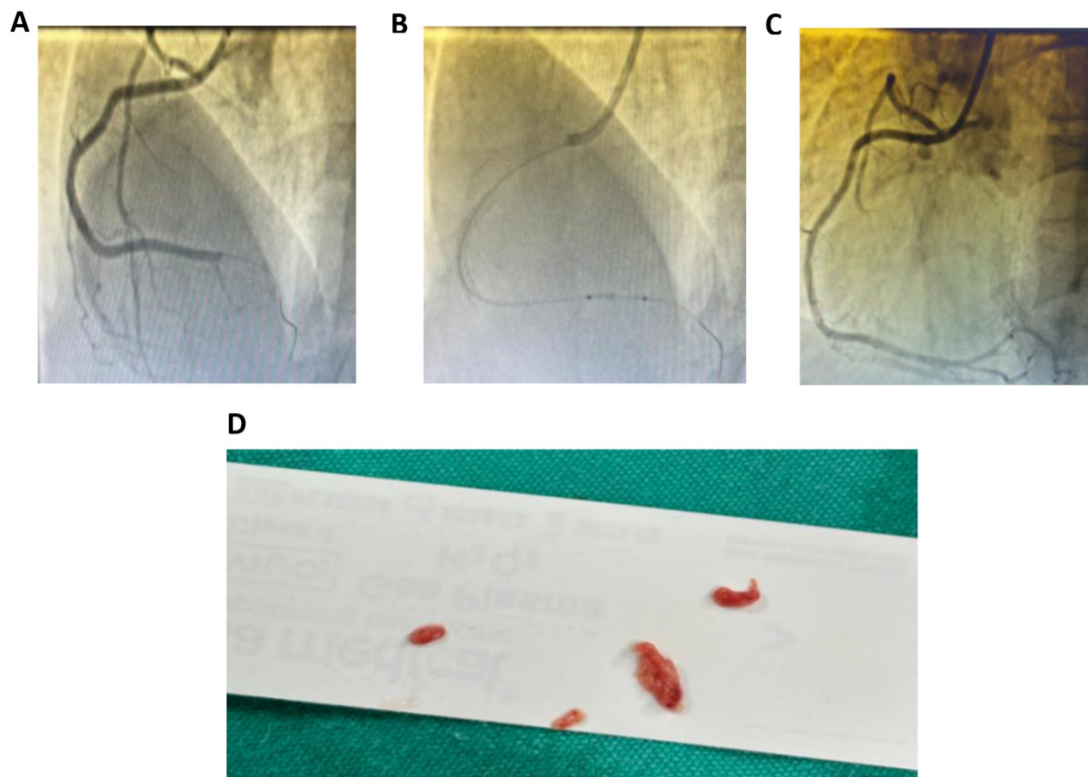

Figure 2. A. Initial Coronary Angiography showing thrombotic occlusion in the right coronary artery. **B.** Balloon angioplasty with 2,0 X 15 mm, 2,5 X 25 mm semi-compliant balloons up to 16 atm **C.**Postmechanical thrombus aspiration with improvement of distal flow **D.**Mechanical thrombus aspiration obtaining abundant thrombus material

The most frequent cause is atrial fibrillation; other reasons have been identified as malignancy, antipholipid syndrome, and cardiac tumors [4]. Coronary embolism could go unrecognized, because parodoxical embolism is harder to detect. [1]For additional research, imaging techniques like transtoracic and transeosefageal echocardiography might be required. Aspiration thrombectomy may be utilized if the coronary artery has a significant thrombus load. Only anticoagulation was used to treat dİstal CE. If there is no evidence of atherosclerosis following aspiration, anticoagulation of the patients may be sufficient for treatment.

The usual short-term and long-term care of individuals with CE is unclear [1,3,5]. Triple treatment or a single antiplatelet medication

combination with oral anticoagulation, such as clopidogrel and warfarin, might be used for long term management. According to Raphael et al., oral anticoagulation should be used for three months in the absence of continuous procoagulation risk factors, such as immobilization, active malignancy, antiphospholipide syndrome, pregnancy or postpartum, use of oral contraceptives, estrogen or progesterone therapy, etc. Long-term oral anticoagulation should be taken into consideration for people with persistent risk factors [1]. According to Shibata et al., the recurrence rate for patients with atrial fibrillation was 10% [3].

So, in this case we discharged the patient with P2Y12 inhitors and direct oral anticoagulan therapy were given to the patient. We planned using oral anticogulan for a life time.

The lack of agreement in the literature regarding the origins of the illness and CE treatment necessitates more research.

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