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A Case of Coronary Artery Rupture Caused by Negative Vessel Remodeling

Negatif Damar Yeniden Şekillenmesinin Neden Olduğu Bir Koroner Arter Rüptürü Olgusu

ABSTRACT

Coronary artery rupture is a rare but potentially fatal complication of coronary procedures. This case report describes a 63-year-old male patient with coronary atherosclerotic heart disease who presented with typical symptoms. However, echocardiography, myocardial injury markers, and electrocardiograms did not indicate an acute myocardial infarction. Coronary angiography (CAG) revealed lesions in two vessels, requiring intervention. During the procedure, a coronary artery rupture occurred. Intravascular ultrasound (IVUS) revealed negative vessel remodeling in the affected arteries. Coronary artery rupture is uncommon in clinical practice and is primarily documented in case reports. Due to the limited information available on its management, early detection and timely treatment are essential.

Keywords: Coronary artery rupture, interventional cardiology, negative vessel remodeling

ÖZET

Koroner arter rüptürü, koroner prosedürlerin nadir ancak potansiyel olarak ölümcül bir komplikasyonudur. Bu olgu sunumunda, tipik semptomlarla başvuran koroner aterosklerotik kalp hastalığı olan 63 yaşında bir erkek hasta bildirilmiştir. Ekokardiyografi, miyokardiyal hasar belirteçleri ve elektrokardiyogramlar akut miyokard enfarktüsünü göstermemiştir. Koroner anjiyografide (KAG), iki damarda müdahale gerektiren lezyonlar saptanmıştır. İşlem sırasında bir koroner arter rüptürü meydana gelmiştir. İntravasküler ultrason (IVUS), etkilenen arterlerde negatif damar yeniden şekillenmesini göstermiştir. Koroner arter rüptürü klinik pratikte nadir görülür ve öncelikle olgu sunumlarında belgelenmiştir. Yönetimi hakkında sınırlı bilgi bulunması nedeniyle erken teşhis ve zamanında tedavi esastır.

Anahtar Kelimeler: Koroner arter rüptürü, girişimsel kardiyoloji, negatif damar yeniden şekillenmesi

CASE REPORT
OLGU SUNUMU

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Coronary artery rupture (CAR) refers to the extravasation of contrast agent or blood from the coronary arteries during or after coronary interventional procedures. The diagnosis of coronary artery rupture is primarily made through angiography and echocardiography. The presence of contrast agent outside the vascular lumen on angiography, or the detection of pericardial effusion on echocardiography, suggests the occurrence of coronary artery rupture. Although rare, it is a potentially fatal complication during coronary artery procedures.[1,2] Studies have reported an increasing incidence, ranging from 0.1% to 0.82%, with a mortality rate between 7% and 19%.[3-6] The incidence of major adverse cardiovascular events (MACE) associated with CAR is also high. Coronary artery rupture most commonly occurs in large vessels, where lesions tend to be complex and the prognosis is often poor. Negative remodeling of the coronary artery is considered one of the key factors contributing to coronary artery rupture. This article presents a case of CAR caused by negative remodeling of the coronary artery.

Case Report

A 63-year-old male patient had a history of hypertension for over five years (peak blood pressure: 180/80 mmHg), managed with Levamlodipine Besylate Tablets. During the same period, he was also diagnosed with diabetes mellitus, which was irregularly treated with metformin and without routine glucose monitoring. Four months prior to admission, he began experiencing chest tightness and pain following

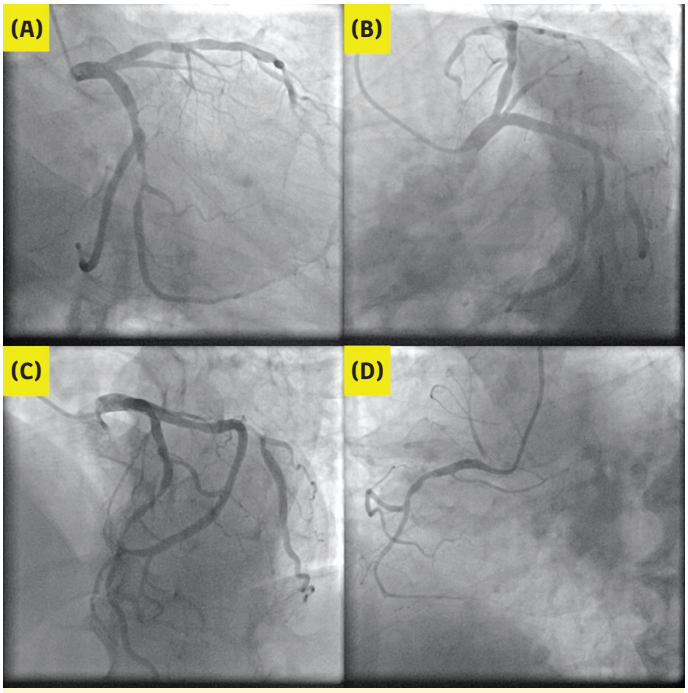

Figure 1. (A–C) Left anterior descending artery (LAD) and left circumflex artery (LCX) coronary angiographic images. (D) Right coronary artery (RCA) coronary angiographic image.

physical activity. The discomfort, localized to the mid-to-lower sternum, lasted for several minutes and was relieved by rest. The episodes were not accompanied by palpitations, dyspnea, or radiating to the shoulders or back. He had previously been admitted to a local county hospital, where he was diagnosed with coronary atherosclerotic heart disease, but no further treatment was provided. His symptoms worsened one week before the current admission. On physical examination, his blood pressure was 152/92 mmHg, and his heart rate was 102 beats per minute. Cardiac auscultation was normal, and there was no peripheral edema. Laboratory and imaging findings are summarized in Table 1.

ABBREVIATIONS

AS%	Area stenosis percentage
BNP	B-type natriuretic peptide
CAG	Coronary angiography
CAR	Coronary artery rupture
CHD	Coronary heart disease
cTn-T	High-sensitivity cardiac troponin-T
ECG	Electrocardiogram
EF	Ejection fraction
IVSD	Interventricular septal thickness at end-diastole
IVUS	Intravascular ultrasound
LAD	Left anterior descending artery
LAS	Left atrium size
LCX	Left circumflex artery
LDL	Low-density lipoprotein
LM	Left main trunk
LVD	Left ventricular diastolic dimension
MACE	Major adverse cardiovascular events
MLA	Minimal lumen area
PA	Pulmonary artery
PCI	Percutaneous coronary intervention
RAS	Right atrium size
RCA	Right coronary artery
RVD	Right ventricular diastolic dimension

In light of the patient's symptoms, clinical signs, and ancillary test results, coronary heart disease was strongly suspected. The patient was started on secondary prevention medications for coronary heart disease, along with antihypertensive therapy. Coronary angiography was performed, and the subsequent treatment plan was developed based on its findings.

Given the patient's long-standing history of hypertension and diabetes, as well as the poor baseline condition of the blood vessels, an intravascular ultrasound (IVUS)-guided percutaneous coronary intervention (PCI) was selected to allow for a clearer and more accurate assessment of the intravascular environment. An extra backup (EBU) 3.5 guide catheter was advanced through the radial artery to the opening of the left coronary artery. The

Table 1. Summary of Laboratory and Imaging Findings

Examination	Result	Normal Reference Value
cTn-T	20.63 ng/L	<14 ng/L
BNP	100 ng/L	<125 ng/L
TC	3.64 mmol/L	<5.2 mmol/L
LDL	2.43 mmol/L	<3.15 mmol/L
Random Blood Glucose	9.3 mmol/L	<11.1 mmol/L
ECG	Sinus tachycardia	
Cardiac Ultrasound	Left heart enlargement; aortic valve irritation; mild mitral regurgitation; mild tricuspid regurgitation.	
CAG	Conclusion: Severe stenosis in the middle and distal segments of the left anterior descending artery and the distal segment of the left circumflex artery.	
	The proximal LAD exhibited 30% stenosis, while the middle and distal segments showed up to 90% stenosis, with a maximum stenosis of 90%, as shown in Figure 1A-C. The distal LCX also demonstrated 90% stenosis (Figure 1A-C). The RCA appeared unremarkable, with no significant stenosis (Figure 1D).	

BNP, B-Type Natriuretic Peptide; CAG, Coronary Angiography; cTn-T, High-Sensitivity Cardiac Troponin-T; ECG, Electrocardiogram; LAD, Left Anterior Descending Artery; LCX, Left Circumflex Artery; LDL, Low-Density Lipoprotein; RCA, Right Coronary Artery; TC, Total Cholesterol.

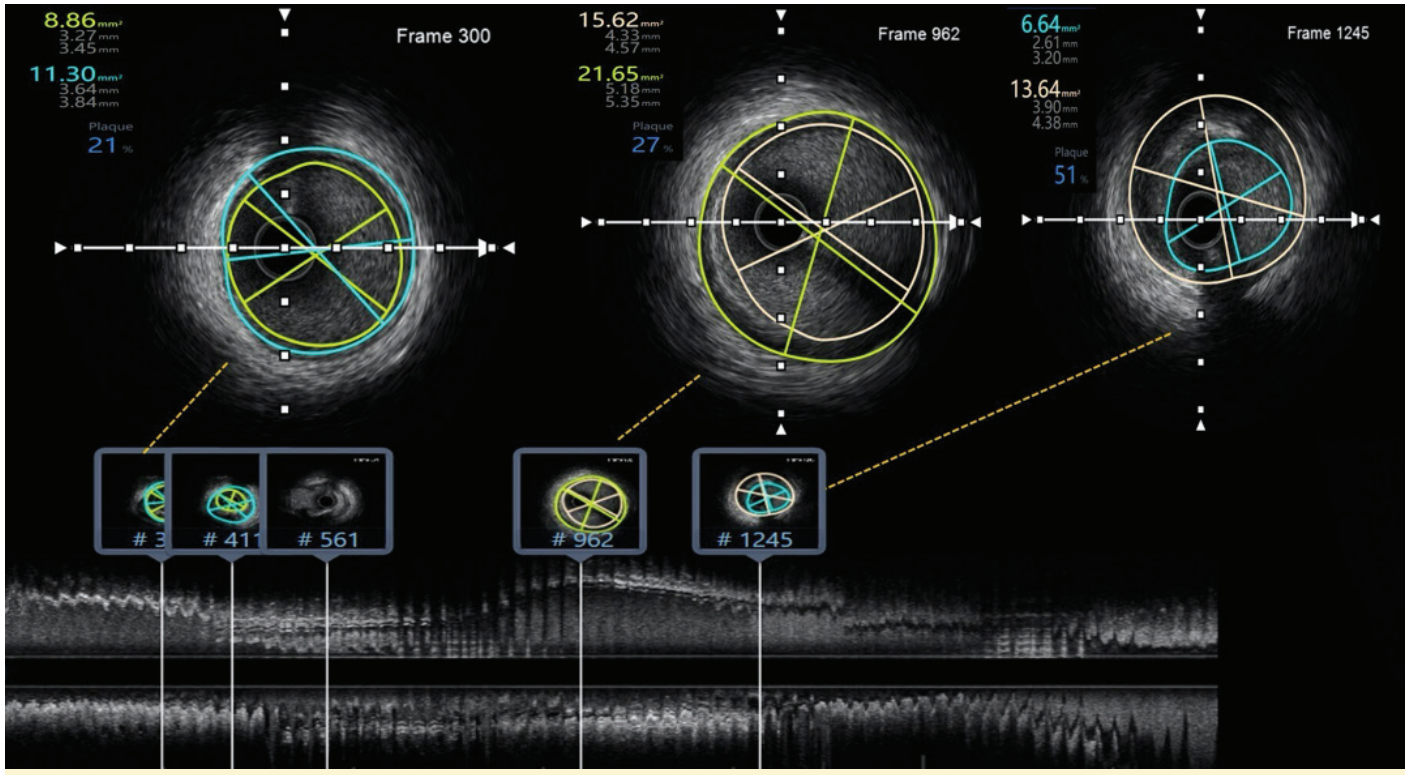

Figure 2. Preoperative examination findings of the left circumflex artery (LCX).

Runthrough guidewire was then navigated through the lesion into the distal left anterior descending (LAD) artery, while the Sion guidewire was directed through the lesion into the distal left circumflex (LCX) artery. Subsequently, IVUS examinations of both the LAD and LCX arteries were performed individually. The IVUS revealed mixed fibrocalcific plaques with distal calcified nodules and negative remodeling in the LCX (minimal lumen area [MLA] 2.38 mm^2; area stenosis [AS%] 77%), and fibrotic plaques in the LAD (MLA 2.99 mm^2, AS% 68%) (Figure 2).

Based on the IVUS findings, interventional treatment was initiated for the LAD lesions. The lesions were first dilated using a 2.5 × 20 mm balloon, followed by the implantation of two drug-eluting stents (3.0 × 29 mm and 3.5 × 29 mm) in the distal and proximal segments of the LAD, respectively. A 3.5 × 15 mm non-compliant balloon was then used to further expand and shape the stents and their junction.

For the LCX lesion, a 3.0 × 10 mm cutting balloon was used for dilation at a pressure of 10–12 atm (Figure 3A). This was followed by the deployment of a 3.5 × 21 mm drug-eluting stent at 10 atm pressure in the LCX lesion (Figure 3B). Angiography revealed contrast extravasation consistent with an Ellis III coronary perforation (red arrow, Figure 3C). To prevent adverse events such as pericardial tamponade, myocardial infarction, or stroke, immediate intervention was undertaken. A 3.5 × 15 mm non-compliant balloon was first used to seal the proximal LCX. Subsequently, an Abbott 3.5 × 19 mm covered stent was implanted at the site of the rupture in the LCX (Figure 3D). However, follow-up angiography showed persistent contrast extravasation. Given the severity of the rupture and the inadequate response to balloon occlusion, a self-made 3.5 × 12 mm covered stent was deployed (Figure 3E). No extravasation of contrast medium was observed upon re-examination with angiography (Figure 3F).

The operation was successful, and the patient reported no discomfort. During the procedure, two drug-eluting stents were implanted in the LAD lesions, while one drug-eluting stent, one covered stent, and one self-made covered stent were implanted in the LCX lesions.

Discussion

The cause of the patient's LCX rupture is believed to be related to negative remodeling of the vessel and the selection of an oversized stent during the procedure, as illustrated in Figure 4. Coronary remodeling refers to the compensatory thickening of coronary lesion segments in response to the progression of coronary artery disease, helping to preserve blood flow in affected arteries.[7] The degree of coronary artery remodeling is quantified by the Remodeling Index (RI), defined as the ratio of the minimal vessel area within the lesion to the average vessel area of the reference segments located proximally and distally to the lesion. When the Remodeling Index is less than 0.95, it is classified as negative vascular remodeling.[8,9] Negative remodeling is typically characterized by severe stenosis, smaller plaque burden, and more stable plaque features. These lesions are often the result of plaque healing and intimal repair following earlier rupture.[10] As coronary atherosclerosis progresses to its later stages, the deposition of calcium salts and intimal fibrosis within plaques further exacerbates luminal narrowing, leading to altered hemodynamics. In a case report of Ellis III coronary artery perforation following PCI by Watabe et al.,[11] a review of IVUS and optical coherence

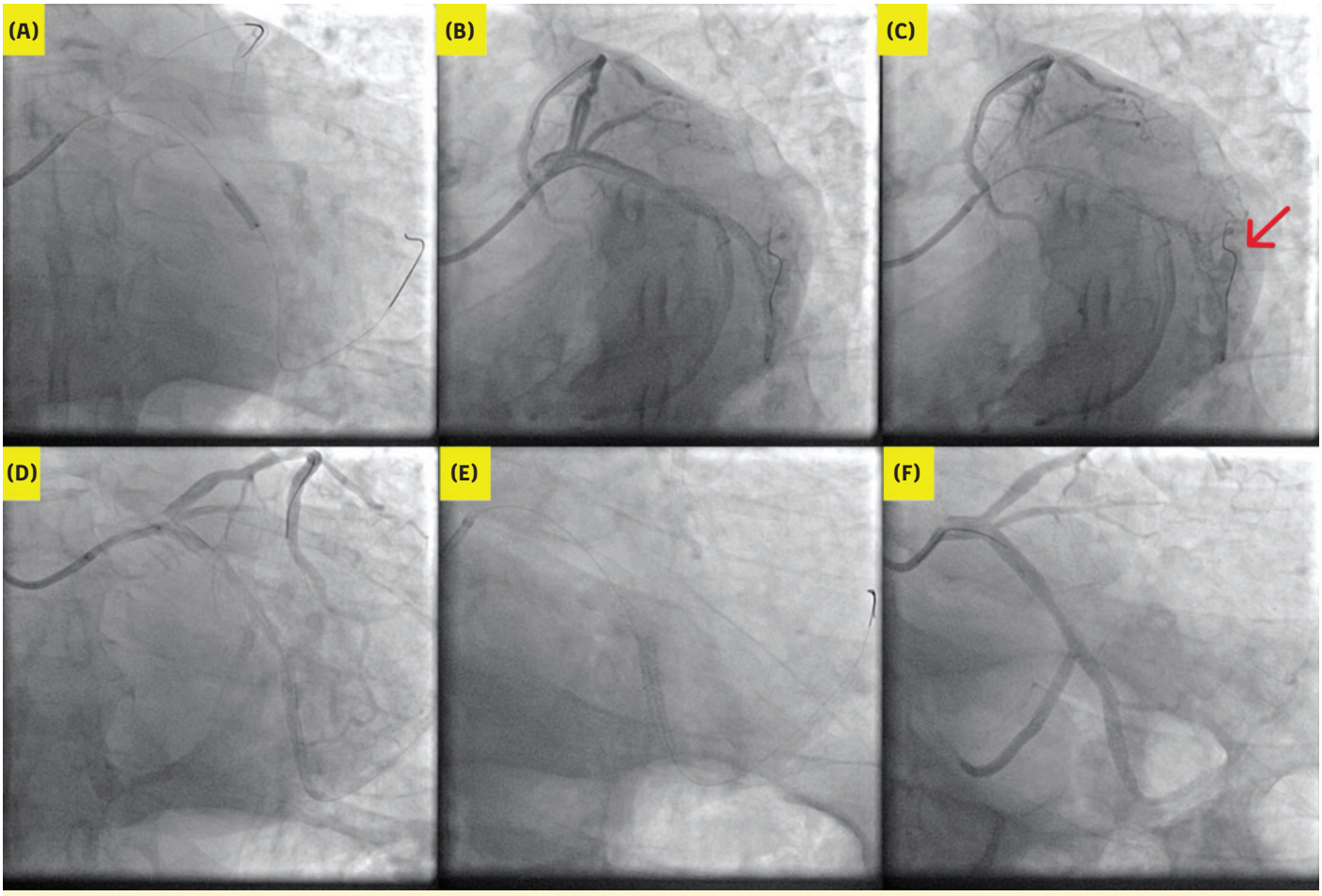

Figure 3. (A) 3.0 × 10 mm cutting balloon dilation in the left circumflex artery (LCX) lesion. (B) Implantation of a 3.5 × 21 mm drug-eluting stent. (C) Contrast medium extravasation at the LCX stent implantation site (red arrow). (D) Implantation of a 3.5 × 19 mm covered stent. (E) Implantation of a 3.5 × 12 mm self-made covered stent. (F) Final angiographic image.

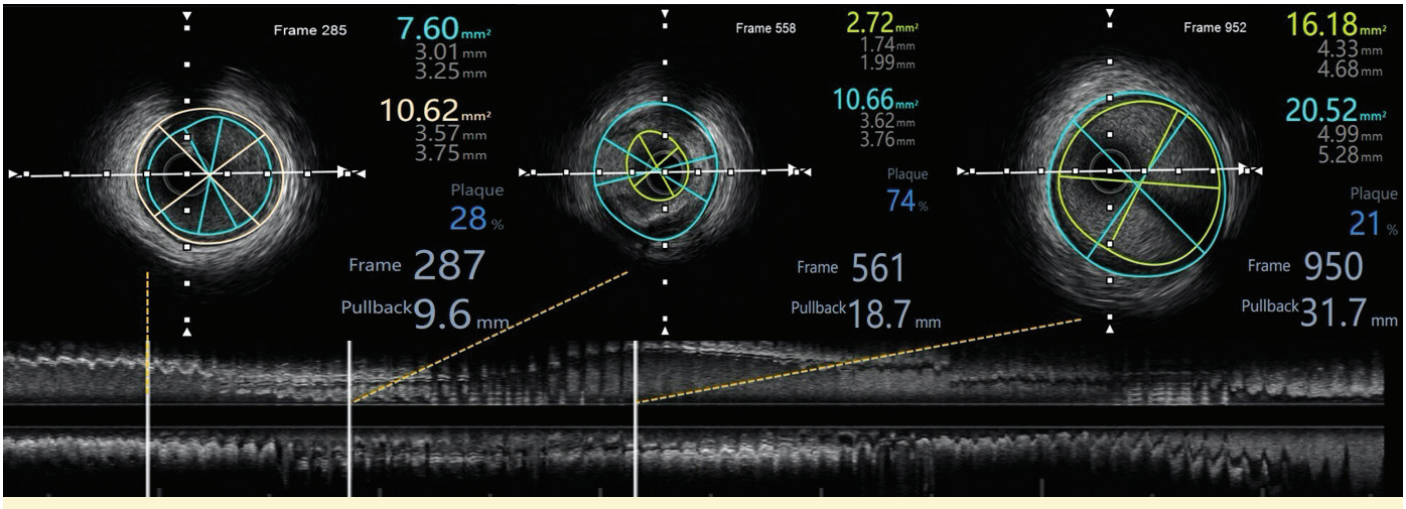

Figure 4. Remodeling Index (RI) = 10.66 / (10.62 + 20.52) = 0.68, which is less than 0.95, indicating negative vascular remodeling.

tomography (OCT) findings revealed focal negative remodeling and eccentric plaques with superficial intimal calcification in the culprit vessels. In another case involving a type III perforation of the mid-left anterior descending coronary artery after stent implantation, OCT imaging demonstrated focal negative remodeling, which may have contributed to the coronary artery rupture.[12] IVUS evidence also highlights the vulnerability of these lesions to external elastic membrane (EEM) and adventitial overstretch during dilation.

Based on these cases, special attention should be given to coronary arteries exhibiting negative remodeling. The symmetry and characteristics of the plaque should be carefully evaluated. Prior to balloon dilation and/or microcatheter advancement, multi-angle projections should be used to confirm that the guidewire is positioned within the lumen. Intravascular imaging should be utilized appropriately to determine vessel size, lesion length, and plaque type. Balloon and stent sizes should be selected with consideration of the external elastic membrane of the remodeled vessel. Reducing pressure and minimizing the use of stents and balloons can help further reduce vascular injury caused by the procedure. In cases of severe CAR, a covered stent is typically effective for sealing perforations in large vessels, especially in arteries with a diameter greater than 2.5 mm and without major branches.[13]

Conclusion

Coronary artery rupture is a potentially fatal complication that requires prompt diagnosis and effective intervention. Medical personnel may consider the impact of negative remodeling in coronary interventional therapy, as illustrated by this case. However, this report represents a single case and is not broadly generalizable. Further research is needed to expand knowledge in this area, enabling more patients with complex coronary heart disease to avoid such complications or receive timely treatment, ultimately improving clinical outcomes.

Ethics Committee Approval: This is a single case report, and therefore ethics committee approval was not required in accordance with institutional policies.

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

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