Coronary Artery Aneurysm and a Challenging Inferior ST-Segment Elevation Myocardial Infarction

Raymond Chun-Lin Cheung[1], Ho Lam[1], Chi-Wing Wong[2], Ping-Wa Yam[1]

[1]*Department of Medicine and Geriatrics, Tuen Mun Hospital, Hong Kong, China*

[2]*Department of Medicine, Pok Oi Hospital, Hong Kong, China*

Abstract

Coronary artery aneurysm (CAA) is a focal dilation of a coronary artery that exceeds the reference vessel diameter by at least 50%[1]. It is a rare clinical entity, observed in fewer than 5% of coronary angiograms. The underlying mechanisms of CAA remain elusive; atherosclerosis and Kawasaki disease are the primary etiologies in adults and children, respectively. Currently, there is no established consensus on the optimal management strategy for this condition. We report a unique case of a 54-year-old male who presented with an inferior ST-segment elevation myocardial infarction secondary to a giant coronary aneurysm in the proximal right coronary artery. Angiographic evaluations showed the aneurysm to be too large for conventional coronary stents. Consequently, medical management with anticoagulation therapy was initiated, resulting in a satisfactory patient outcome. The patient reported no chest pain or bleeding events during a 2-year follow-up.

Keywords: acute coronary syndrome, coronary artery aneurysm, percutaneous coronary intervention, ST segment elevated myocardial infarction, thrombosis, anticoagulation

Introduction

Coronary artery aneurysm (CAA) is a focal dilation of a coronary artery that exceeds the size of the adjacent normal vessel segment by 50%. It is considered giant if the diameter exceeds the reference vessel diameter by over four times, or exceeds 8 mm. Morphologically, CAA can be categorized into two types: saccular and fusiform[2]. Saccular aneurysms have a transverse diameter that exceeds their longitudinal extent, whereas fusiform aneurysms exhibit the opposite characteristic. Anatomically, CAA most frequently affects the right coronary artery (RCA), followed by the left anterior descending artery, the left main coronary artery, and the left circumflex artery. The reported incidence of CAA ranges from 0.3% to 5%, with a strong predilection for men.

CAA can lead to various severe complications, including thrombus formation, fistulization, rupture, distal embolization, and the compression of adjacent structures[3]. Typically, it is an asymptomatic condition diagnosed during

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Address for correspondence: Raymond Chun-Lin Cheung

Department of Medicine & Geriatrics, Tuen Mun Hospital; 23 Tsing Chung Koon Road, Tuen Mun, New Territories, Hong Kong, China

imaging procedures such as coronary angiography or computed tomography (CT) angiography. It can also manifest clinically with chest pain or lead to sudden cardiac death from myocardial ischemia driven by the aforementioned complications. The lack of definitive guidelines or consensus regarding CAA management stems from a limited understanding of its underlying mechanisms. Treatment strategies must be highly individualized, taking into account angiographic findings, clinical presentation, and operator preference. This report describes an unusual presentation of an inferior ST-segment elevation myocardial infarction (STEMI) caused by CAA in the proximal RCA. An imaging series revealed the aneurysm to be too large for conventional coronary stents. We chose anticoagulation as the medical treatment strategy due to the significant thrombus load and large aneurysm size. The patient remained stable throughout subsequent follow-ups.

Case Presentation

A 54-year-old male ex-smoker with no significant past medical or family history presented to our hospital with acute-onset chest discomfort. Initial blood tests and physical examinations were unremarkable, and inflammatory markers were normal. The initial troponin level was 218 ng/L (reference limit ≤ 34.2 ng/L). Recent low-density lipoprotein cholesterol was 2.7 mmol/L, and HbA1c was 5.6%. In the emergency department, an electrocardiogram showed ST-segment elevation in the inferior leads. A bedside echocardiogram revealed a preserved ejection fraction of 50% with hypokinesia of the inferior wall. We initiated primary percutaneous coronary intervention (PCI) after discussion with the patient.

Vascular access was established via the right radial artery using a 7-French sheath. Coronary angiography showed minor disease in the left coronary system, but engaging the RCA was technically challenging. Thrombotic occlusion was suspected in the proximal RCA with a giant aneurysm. A standard workhorse wire was initially unable to cross the lesion, but eventually, after escalating to the Sion Black wire (Asahi Intecc, Japan), the RCA was successfully wired.

Intravascular ultrasound (IVUS) revealed a giant proximal RCA aneurysm with thrombus formation. Its size exceeded the IVUS scan measurement limit and was estimated angiographically to be over 10 mm. We serially dilated the RCA with 2.0 mm and 3.0 mm balloons; however, coronary flow remained poor due to the heavy thrombus load. We administered an intracoronary glycoprotein IIb/IIIa (GP IIb/IIIa) inhibitor and performed mechanical thrombus aspiration using the Penumbra Indigo System CAT RX. Despite these efforts, the RCA remained totally occluded with TIMI 0 flow, and the patient experienced ongoing chest pain accompanied by a drop in blood pressure that required inotropic support. We chose stenting to stabilize the patient's condition, anticipating possible malapposition at the proximal stent edge. We deployed a 5.0 × 30 mm Onyx Frontier (Medtronic, USA) drug-eluting stent (DES) in the proximal-to-mid RCA, which angiographically restored TIMI III flow. IVUS confirmed no stent edge dissection, but the proximal stent edge was mal-apposed, as expected. We optimized the stent using a 6.0 mm non-compliant balloon. The final angiographic result was satisfactory with no immediate complications, and we maintained the intravenous GP IIb/IIIa inhibitor infusion for 24 hours.

A follow-up coronary angiogram performed one week later showed a patent stent. Also, the proximal RCA aneurysm appeared smaller. We rewired the RCA for IVUS, which showed residual intra-aneurysmal thrombus and a patent stent lumen. Once again, IVUS failed to fully define the aneurysm dimensions as these exceeded the imaging limits. Given the optimal flow, we favored anticoagulation treatment over additional stenting of the proximal RCA aneurysm. The patient received clopidogrel 75 mg daily and

apixaban 5 mg twice daily (BD) while awaiting a restudy coronary angiogram to monitor the clinical response. The patient was subsequently discharged and remained free of chest pain.

An outpatient cardiac CT angiography was arranged to determine the size of the aneurysm. This revealed a fusiform aneurysmal dilatation at the proximal RCA, measuring 1.5 × 2.1 × 1.6 cm. The stent was noted in the distal part of the aneurysm and remained patent.

Three months later, a restudy coronary angiogram revealed a patent stent with a large proximal RCA aneurysm. The IVUS catheter initially failed to enter the distal RCA because the wire failed to cross the true stent lumen at the ostium. With the help of a Crusade microcatheter (Kaneka, Japan) for parallel wiring, a second workhorse wire crossed after several attempts. Repeat IVUS showed the proximal stent edge exactly meeting the distal edge of the proximal RCA aneurysm, while the aneurysm remained too large for the IVUS catheter. Otherwise, the stent showed satisfactory expansion and apposition. Aneurysm thrombus remained, but at a lower load than on the previous angiogram. Covered coronary stents commercially available in our region cannot accommodate such a giant aneurysm. Therefore, in view of the stable TIMI III flow, our team recommended lifelong anticoagulation. The antithrombotic regimen consisted of clopidogrel 75 mg daily combined with apixaban 5 mg BD for one year, followed by apixaban 5 mg BD indefinitely. Other discharge medications included atorvastatin 40 mg daily, lisinopril 5 mg daily, bisoprolol 1.25 mg daily, and pantoprazole 40 mg daily. The patient reported no recurrence of chest pain or bleeding events during subsequent clinical follow-ups, and he declined referral for surgical management or further intervention, as he remained asymptomatic.

Discussion

This case illustrates the challenging scenario of an inferior STEMI caused by a giant proximal RCA fusiform aneurysm with diffuse thrombus. Despite balloon dilation, thrombus aspiration, and intracoronary GP IIb/IIIa inhibitors, reflow could not be established. IVUS revealed the aneurysm to be larger than imaging allowed. We performed distal aneurysm stenting to restore flow and stabilize the patient who has since remained stable under anticoagulation treatment.

Pathophysiology

The exact etiology and pathophysiology of CAA remain elusive and are still debated in interventional cardiology[4]. Although the mechanism is unknown, atherosclerosis is the leading cause of adult CAAs. Atherosclerotic processes can compromise vascular structural integrity by causing transmural inflammation across multiple vessel layers, from the tunica intima to the external elastic lamina. Other contributing factors may include inflammation, infection, genetics and connective tissue diseases.

Management

CAA's unpredictable natural course makes clinical management difficult[5]. PCI and surgery for CAA-induced myocardial ischemia may be technically difficult due to the altered anatomy. Currently, there is a lack of large-scale randomized controlled trials to address different clinical questions, and current recommendations are based on expert opinions or small case series. While the best time and indications for the management of incidentally discovered CAAs remain unknown, vessel anatomy, clinical presentation, and patient background should inform management strategies.

Medical Treatment

Medical treatment is proposed as the first-line treatment for CAA. Atherosclerosis plays a major role in CAA pathogenesis, so aggressive risk factor modification is necessary. Angiotensin-converting enzyme inhibitors and statins can reduce the vascular inflammation that drives aneurysm progression. While our patient received anticoagulation treatment to address his heavy

thrombus burden, the exact role of anticoagulation or antiplatelet therapy in CAA remains uncertain due to a lack of high-quality evidence.

One retrospective analysis found that warfarin-treated CAA patients experience a statistically significant reduction in composite clinical outcomes (myocardial infarction, unstable angina, and aneurysm thrombosis) compared to those not receiving anticoagulation (8.7% vs. 17.2%; P = 0.01)[6]. Both groups had similar rates of all-cause mortality and hospitalization (39.0% vs. 35.9%; P = 0.47), but the warfarin group experienced more bleeding complications (10.3% vs. 6.2%; P = 0.08). Few case reports have been published on the use and efficacy of novel oral anticoagulants (NOACs) for CAA[7]. After discussing NOAC's ease of administration and lack of INR monitoring, the patient chose NOACs over warfarin.

PCI

Data detailing the long-term outcomes of PCI for CAAs is limited. PCI is typically reserved for symptomatic patients or those with acute ischemic complications. PCI on infarct-related arteries with underlying CAA during acute myocardial infarction shows a lower procedural success rate and a higher incidence of no-reflow phenomena and distal embolization. CAA patients undergoing PCI have higher long-term mortality and an increased risk of target vessel revascularization, stent thrombosis, and recurrent MI during follow-up. Despite PCI often being supported by mechanical thrombus aspiration and GP IIb/IIIa inhibitor injection due to high thrombus burden, no-reflow or distal embolization remains a common complication.

Intervention depends on the anatomy of the aneurysm. Saccular aneurysms without side-branch involvement can often be treated using covered stents, whereas aneurysms involving major side branches may require coil embolization or surgical reconstruction. In our case, the aneurysm was located at the ostium and was enormous, with a diameter reaching 21 mm. This anatomy rendered standard covered stenting unfeasible due to the lack of a suitable proximal landing zone at the ostium. Furthermore, covered stents commercially available in our region have a maximum expandable diameter of only 5 mm, which is inadequate for sealing such a giant aneurysm. Before considering PCI, it is important to assess the size of the aneurysms through imaging to prevent stent migration and thrombosis. Standard coronary angiography may underestimate aneurysm size because of partial thrombosis, and slow coronary flow can degrade overall image quality. IVUS is recommended for its high-resolution imaging, which can accurately delineate the extent of the aneurysm and landing zones necessary for precise stent sizing[8]. It can also provide details of disease morphology, plaque burden, and thrombus load to guide management strategy. However, because the RCA aneurysm in our patient exceeded the imaging limits of IVUS, a CT scan was required to provide precise details of aneurysm size, native vessel diameter, calcification and thrombus burden.

Surgery

Surgical intervention is typically recommended for symptomatic patients whose anatomy is unsuitable for PCI or for those with concomitant cardiac conditions requiring surgery. Various surgical techniques have been described in the literature, including aneurysm ligation, aneurysmectomy (with or without concurrent coronary artery bypass grafting), aneurysm resection, and marsupialization[9]. There is no consensus on the best surgical approach due to limited case numbers, and long-term success rates for each technique are unclear. Ultimately, the surgical strategy adopted must be tailored to the aneurysm anatomy and patient presentation.

Conclusion

CAA represents an uncommon but high-risk etiology for myocardial ischemia, and its clinical management remains highly challenging.

Currently, no universal consensus exists regarding the optimal management approach. Herein, we successfully managed a giant proximal RCA aneurysm with long-term oral anticoagulation, achieving stable, excellent results at 2 years. Future large-scale clinical registries and trials are warranted to bridge knowledge gaps regarding targeted treatment strategies and the optimal timing of interventions for symptomatic and asymptomatic CAA patients.

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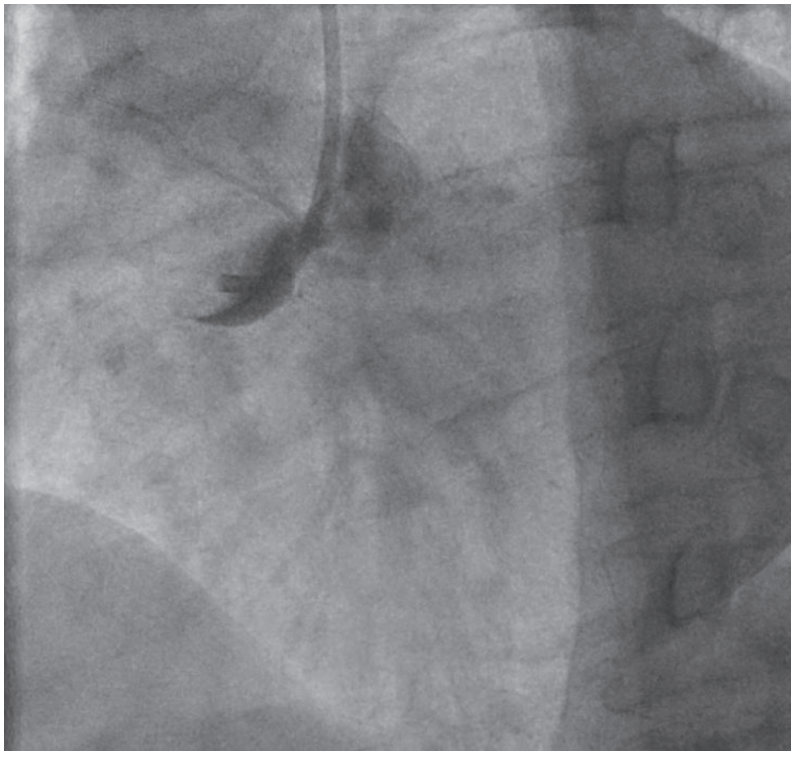

Figure 1. Ostial right coronary artery filled with thrombus, TIMI 0 flow.

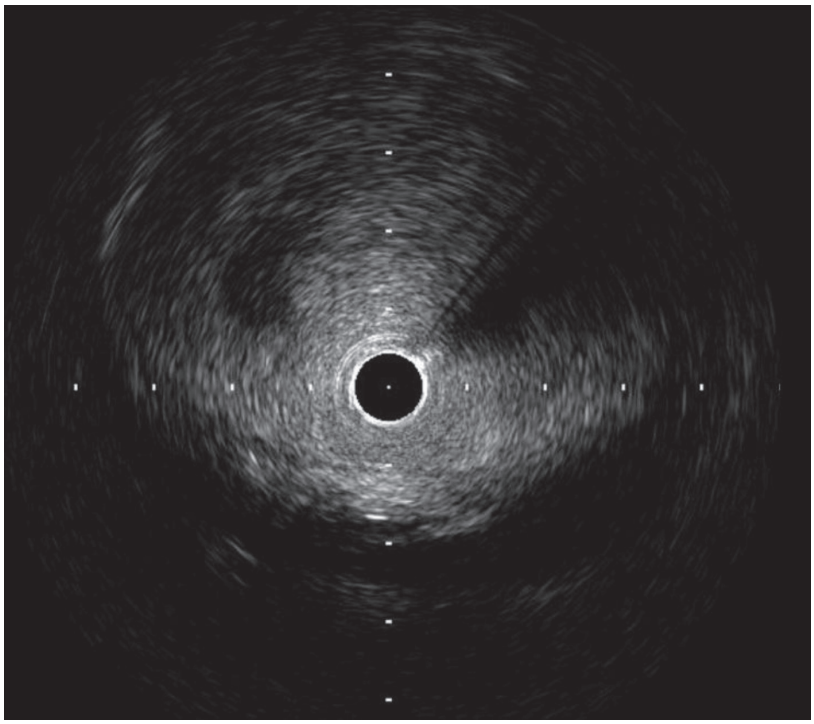

Figure 2. Intravascular ultrasound showed the proximal right coronary artery aneurysm exceeded the scan limit and was filled with thrombus.

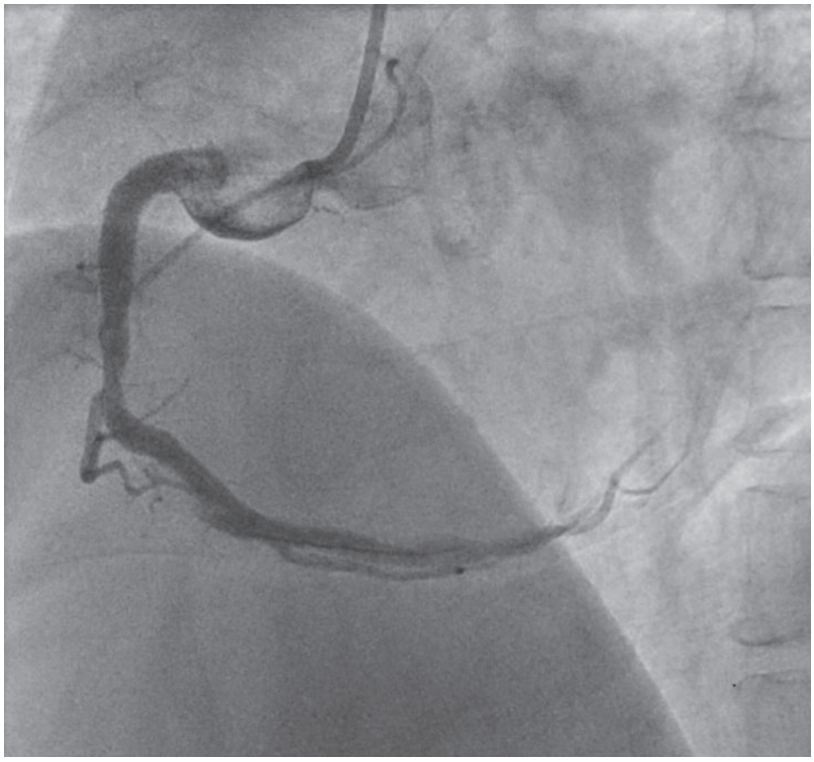

Figure 3. Coronary flow was restored to TIMI III after stenting.

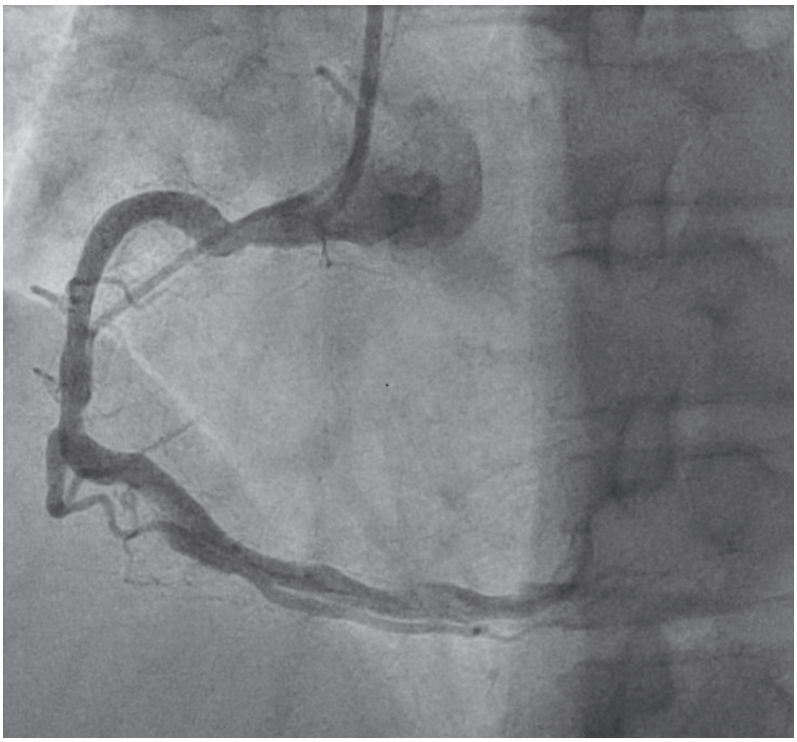

Figure 4. Restudy angiogram one week later showed a patent stent.

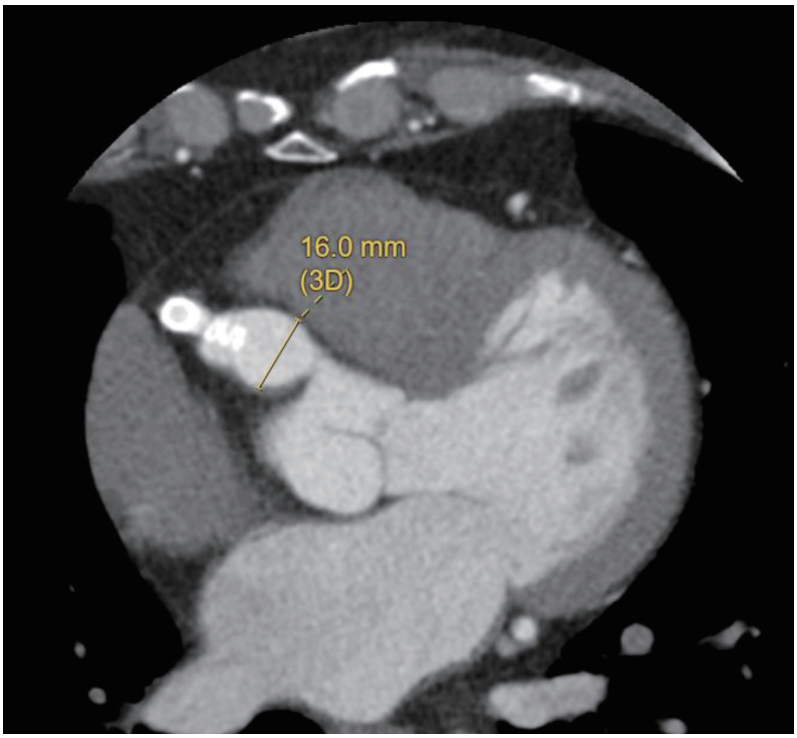

Figure 5. Regarding the aneurysm, representative computed tomography (CT) image showed a 16 mm measured diameter; full CT assessment measured 1.5 × 2.1 × 1.6 cm.

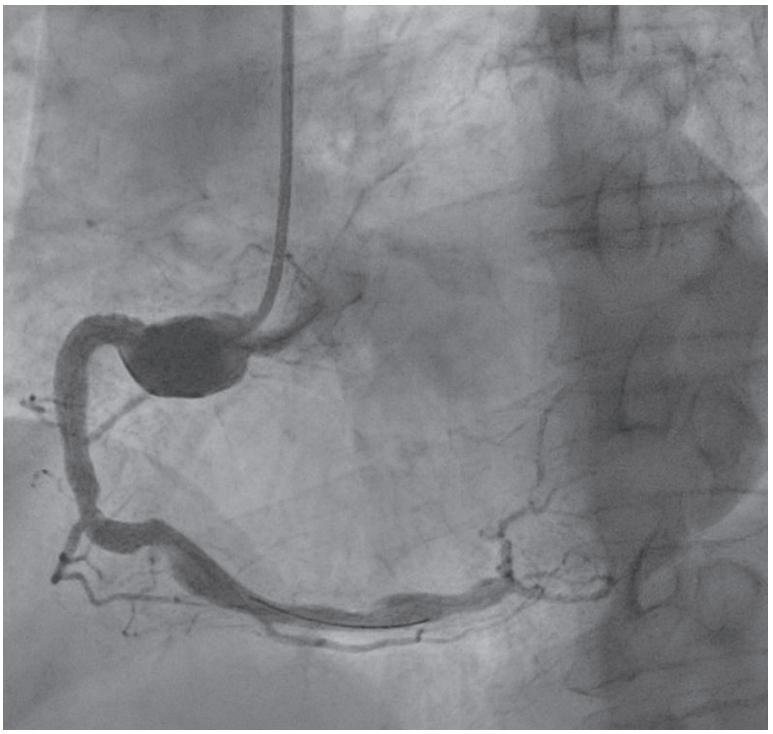

Figure 6. Restudy angiogram 3 months later showed a patent stent with TIMI III flow. The proximal right coronary artery aneurysm remained angiographically very large in size.

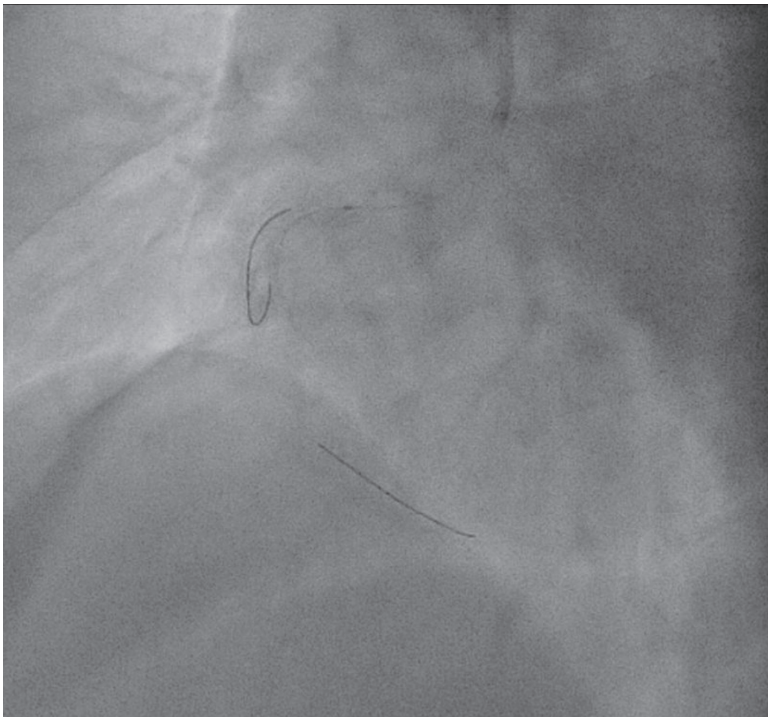

Figure 7. Crusade microcatheter was used for parallel wiring to ensure entry of the coronary wire into the true stent lumen at the proximal stent edge.

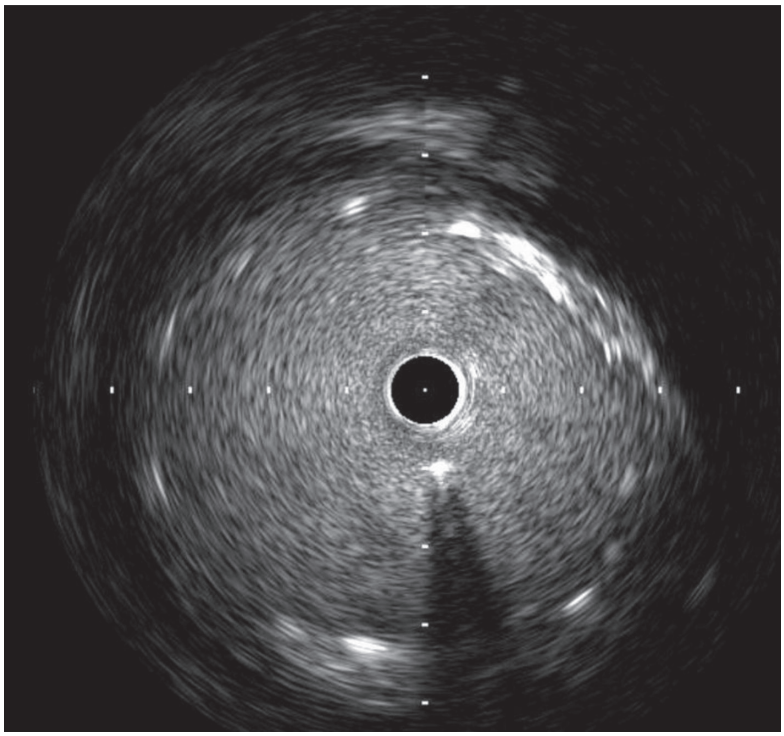

Figure 8. Intravascular ultrasound showed the proximal stent edge just landed on the distal edge of the aneurysm.