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Refractory coronary spasm during emergency PCI: serial IVUS documentation and stent- free management — a case report

Lipeng Mao[1]
Binzhou Central Hospital
Kai Zhang[1]
Binzhou Central Hospital
Ruifeng Jia
Binzhou Central Hospital
Junqiang Cao[1]
zxyycjq@126.com

Binzhou Central Hospital

Case Report

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Abstract

Background

Coronary artery spasm is an important cause of myocardial infarction in patients without obstructive coronary artery disease. Most spasms respond promptly to intracoronary nitroglycerin; however, refractory spasm that progresses despite nitrate therapy is uncommon and can create a vicious cycle of slow flow and recurrent thrombosis during emergency percutaneous coronary intervention (PCI). Serial intravascular ultrasound (IVUS) documentation of this process has rarely been reported.

Case presentation:

A 58-year-old man with no conventional cardiovascular risk factors but a history of recurrent resting chest pain presented with aggravated sudden chest pain. Emergency coronary angiography revealed severe stenosis in the distal right coronary artery. Following intracoronary administration of nitroglycerin, no improvement in the stenosis was observed. IVUS revealed a lumen diameter of 3.22 mm at the origin of the posterior descending artery/posterolateral branch, with a minimum lumen area of 3.19 mm^2. An intraluminal thrombus was present, but no evidence of plaque rupture or dissection was detected. Subsequent IVUS examination revealed further stenosis at the same site (lumen diameter: 2.44 mm; minimum lumen area: 2.75 mm^2), which is consistent with progressive coronary artery spasm. Despite multiple attempts at balloon dilation and intracoronary administration of nitroglycerin, the lumen diameter at the site reached its minimum value (1.87 mm) during the procedure. Intracoronary tirofiban and urokinase were administered, and repeated balloon dilatation was performed. Final IVUS showed adequate lumen expansion with a residual mural thrombus. Stent implantation was withheld because no significant fixed atherosclerotic lesion was present. A repeat angiogram after 8 days showed a patent right coronary artery. The patient remained free of chest pain on continued diltiazem therapy (90 mg twice daily) at 4-month follow-up.

Conclusions

This case demonstrates that coronary spasm can progress despite intracoronary nitroglycerin during emergency PCI, leading to recurrent thrombosis and slow flow. Serial IVUS is valuable for distinguishing functional spasm from structural obstruction and for guiding a stent-free strategy in the absence of a fixed culprit plaque. Recognition of this phenomenon is important in resource-limited settings where intracoronary calcium-channel blocker formulations may not be available.

Background

Coronary artery spasm (CAS) is a transient, reversible vasoconstriction of an epicardial coronary artery that causes myocardial ischemia, and vasospastic angina (VSA; also called coronary spastic angina or

variant angina) is its characteristic clinical manifestation. International diagnostic criteria, standardized by the Coronary Vasomotion Disorders International Study Group, define VSA by rest angina, transient ischemic electrocardiographic changes during spontaneous episodes, and a prompt response to short-acting nitrates, together with a documented coronary spasm [1]. The condition is particularly prevalent in East Asian populations and characteristically occurs at rest, most often between midnight and early morning [2, 3]. Its clinical spectrum extends from asymptomatic or painful transient ischemia to acute myocardial infarction (AMI), life-threatening ventricular arrhythmia, and sudden cardiac death [2, 4].

CAS is now recognized as an important mechanism of myocardial infarction with non-obstructive coronary arteries (MINOCA) and ischemia with non-obstructive coronary arteries (INOCA) [5, 6]. In patients with MINOCA, coronary spasm accounts for a substantial proportion of infarction and often occurs in the absence of conventional atherosclerotic risk factors [5]. The 2023 European Society of Cardiology guidelines for the management of acute coronary syndromes accordingly recommend intracoronary imaging to clarify the mechanism in MINOCA [7]. Both the American Heart Association scientific statement on MINOCA and the European Association of Percutaneous Cardiovascular Interventions (EAPCI) expert consensus document on INOCA identify epicardial spasm and coronary microvascular dysfunction as key categories that require dedicated vasoreactivity assessment [8, 9].

Most spastic episodes are readily controlled by nitrates and calcium channel blockers (CCBs); however, a subset of patients has refractory or intractable spasm that persists despite adequate medical therapy [10, 11]. When such patients present with AMI and undergo emergency percutaneous coronary intervention (PCI), procedural factors such as catheter and guidewire manipulation and balloon dilation can themselves provoke or aggravate spasms; real-time intravascular imaging has captured severe spasms during PCI [12]. Progressive spasms may produce lumen narrowing, slow flow or no-reflow, and secondary intracoronary thrombus formation, creating a self-perpetuating spasm–slow-flow–thrombosis cycle [12, 13].

Thrombus formation in this setting is not necessarily accompanied by plaque rupture: endothelial erosion, favored by abnormal shear stress and vasoconstriction, exposes thrombogenic subendothelial material and triggers platelet- and fibrin-rich thrombosis [14, 15]. Pathological and intracoronary imaging studies have confirmed that vasospasms can be associated with plaque erosion and intraluminal thrombi [16, 17].

Intravascular ultrasound (IVUS) provides real-time, cross-sectional assessment of plaque morphology, thrombus burden, and luminal dimensions and can distinguish structural obstruction (plaque rupture, dissection, or intramural hematoma) from functional vasoconstriction [12, 18]. Real-time or serial IVUS during spontaneous or provoked spasm has been described [11, 12]; however, serial documentation of spontaneous, nitroglycerin-resistant, progressive spasm with recurrent thrombosis during emergency PCI is rare.

Because stenting within a spastic segment carries risks of stent malapposition, edge spasm, and stent thrombosis [19–22], and drug-eluting stents may themselves provoke vasospasm [21], the management

of spasm-related myocardial ischemia is primarily pharmacological, although stenting has been advocated for selected patients with truly drug-refractory spasm and recurrent events [23]. Here we report a patient with VSA who developed AMI and in whom refractory spasm with recurrent thrombus and slow flow during emergency PCI was documented by serial IVUS. Exclusion of a fixed culprit plaque by IVUS allowed a stent-free strategy. Management was further constrained by a resource-limited (county-level) setting in which acetylcholine/ergonovine and the injectable CCB formulations that are first-line therapy for refractory spasm [24] were unavailable. This case report was prepared in accordance with the CARE guidelines [see Additional file 1].

Case presentation

A 58-year-old man presented to the emergency department with sudden-onset chest pain that lasted 1 h, accompanied by pain in both upper limbs and sweating. His symptoms did not improve after taking Suoxiao Jiu Xin Wan. He had no hypertension, diabetes mellitus, hyperlipidemia, or smoking history. He had been repeatedly hospitalized for resting chest pain over the preceding years, and coronary angiography performed 10 years earlier had shown no significant coronary stenosis. His outpatient medications included aspirin (100 mg/qd), rosuvastatin (10 mg/qn), metoprolol sustained-release (47.5 mg/qd), and diltiazem hydrochloride extended-release capsules (90 mg/qn). Family history was negative for coronary artery disease, sudden cardiac death, and other cardiovascular diseases. The patient was a businessman, denied alcohol use and smoking, lived with his family, and adhered well to his outpatient medications.

On arrival, the patient was hemodynamically stable (blood pressure, 128/88 mmHg; heart rate, 63 bpm). Physical examination revealed scattered, non-pruritic, erythematous skin lesions limited to the bilateral axillae. These lesions were visible both preoperatively and post-coronary angiography; however, the exact onset date was unknown. No hypotension, angioedema, or bronchospasm was observed. The admission electrocardiogram showed sinus rhythm, ST-segment elevation of 0.1 mV in leads III and aVF, and ST-segment depression in leads I and aVL. Preoperative cardiac troponin I levels were normal; however, on postoperative day 2, troponin I was 3.139 ng/mL (high). Transthoracic echocardiography showed a left ventricular ejection fraction of 61%, with no evidence of abnormal ventricular wall motion.

Emergency coronary angiography performed 1 h after symptom onset revealed severe stenosis at the distal right coronary artery (RCA), located at the posterior descending artery/posterolateral branch (PDA/PLA) bifurcation, with thrombolysis in myocardial infarction (TIMI) grade 3 flow (Fig. 1). Intracoronary nitroglycerin (200 μg) was administered; however, the stenosis did not significantly improve. Subsequent IVUS examination of the stenotic segment demonstrated a minimum lumen area (MLA) of 3.19 mm^2 and a lumen diameter of 3.22 mm at the PDA/PLA bifurcation. Intraluminal slow flow and thrombosis were observed. No evidence of plaque rupture, intimal dissection, or intramural hematoma was identified.

Balloon angioplasty was performed using a 3.0 × 15 mm balloon at 12 atm. However, repeat coronary angiography demonstrated persistent severe stenosis with slow flow (TIMI grade 3). Repeat IVUS examination at the same site revealed further luminal narrowing compared with the initial assessment, with the lumen diameter reduced to 2.44 mm and the MLA decreased to 2.75 mm^2, consistent with progressive coronary artery spasms. The intraluminal slow flow and thrombus persisted. Despite repeated balloon dilations and intracoronary nitroglycerin administration, the third IVUS examination during the procedure showed further reduction in lumen diameter to a nadir of 1.87 mm, although the MLA increased to 4.13 mm^2, accompanied by recurrent thrombus formation and slow flow signals.

Given the patient's recurrent and progressive vasospastic lesion, intracoronary tirofiban (8 mL, administered via a JR 3.5 guide catheter) and intracoronary urokinase (4.5 mg) were administered, along with repeated balloon angioplasty. Intracoronary diltiazem or verapamil was not used, as these injectable formulations were not available at our institution (a county-level hospital). Following the combined antithrombotic and mechanical interventions, the final IVUS examination showed adequate luminal expansion (MLA, 6.05 mm^2), although a residual mural thrombus remained. Repeat coronary angiography revealed a residual stenosis of 10% with restored blood flow (Fig. 2).

Stent implantation was withheld because IVUS confirmed the absence of a significant fixed atherosclerotic lesion and the luminal changes were dominated by dynamic spasm with secondary thrombosis. Stenting within a severely spastic segment carries risks of stent malapposition, edge spasm, and stent thrombosis.

Post-procedurally, intravenous dexamethasone (5 mg) was administered once; the axillary skin lesions resolved the following day and were considered an incidental finding because the patient refused to undergo further antibiotic susceptibility testing and other relevant tests. The patient had an uneventful hospital course without arrhythmia or hemodynamic instability. Myocardial necrosis was confirmed by elevated cardiac troponin.

A repeat coronary angiogram performed 8 days later revealed a patent RCA with 0% residual stenosis and normal distal flow, without filling defects. An acetylcholine/ergonovine provocation test was not performed because our hospital could not obtain these medications or CCB injections at the county level.

The patient was discharged on diltiazem (90 mg/bid), rosuvastatin (10 mg/qn), metoprolol sustained-release (23.75 mg/qd), aspirin (100 mg/qd), and ticagrelor (90 mg/bid). At 4-month follow-up, the patient remained on diltiazem (90 mg/bid) and reported no recurrence of chest pain (Table 1).

Table 1
Timeline

Time	Event
10 years earlier	Recurrent resting chest pain; coronary angiography showed no significant stenosis
1 h before presentation	Sudden-onset chest pain radiating to both upper limbs, with sweating
On admission (0 h)	Hemodynamically stable (BP, 128/88 mmHg; HR, 63 bpm); non-pruritic erythematous axillary skin lesions noted; ECG: ST elevation in leads III and aVF
~ 1 h after symptom onset	Emergency coronary angiography: severe distal RCA stenosis (TIMI grade 3 flow); intracoronary nitroglycerin (200 μg) was ineffective
During PTCA (IVUS #1)	MLA, 3.19 mm^2; lumen diameter, 3.22 mm; intraluminal thrombus; no plaque rupture or dissection
During PTCA	Balloon angioplasty (3.0 × 15 mm, 12 atm);
During PTCA (IVUS #2)	Lumen diameter, 2.44 mm; MLA, 2.75 mm^2 (progressive spasm)
During PTCA	Balloon angioplasty (3.0 × 15 mm, 12 atm)
During PTCA (IVUS #3)	Lumen diameter nadir, 1.87 mm; recurrent thrombus and slow-flow signals
During PTCA	Intracoronary tirofiban + urokinase + repeated balloon dilatation; balloon angioplasty (3.0 × 15 mm, 12 atm)
During PTCA (IVUS #4)	Final IVUS MLA, 6.05 mm^2; residual stenosis, 10%
Post-procedure	Intravenous dexamethasone, 5 mg; axillary skin lesions resolved the following day
Postoperative day 2	Troponin I, 3.139 ng/mL (myocardial necrosis confirmed)
Day 8	Repeat angiography: patent RCA, 0% residual stenosis, normal distal flow
4 months	Follow-up: no recurrent chest pain on diltiazem (90 mg twice daily)

Discussion and Conclusions

Case summary

This case had several instructive features. A middle-aged man without conventional cardiovascular risk factors had a long history of recurrent resting chest pain and a normal coronary angiogram 10 years earlier, and he presented with an inferior ST-elevation myocardial infarction (STEMI)-like syndrome. Although angiography showed what appeared to be a fixed critical stenosis of the distal RCA, serial IVUS

demonstrated a dynamic, progressive reduction in lumen dimensions with an intraluminal thrombus but no plaque rupture, dissection, or intramural hematoma. The lesion resolved after antithrombotic and antispasmodic treatment. Notably, the patient had already been taking diltiazem before admission, indicating a breakthrough spasm despite CCB therapy [24]. Therefore, a stent-free strategy was adopted and was followed by complete angiographic resolution at 8 days and freedom from symptoms at 4 months.

Epidemiology and clinical features

CAS shows a striking geographic predilection for East Asian populations and characteristically affects patients at rest, with a nocturnal or early-morning peak [2, 3]. The clinical spectrum is broad: vasospasm may present as a STEMI mimic [25], as the mechanism of an acute coronary syndrome [26, 27], or as recurrent infarction complicated by ventricular fibrillation [28] or aborted sudden cardiac death [11], underscoring its potential lethality. A proportion of patients, as in our case, continue to have breakthrough or refractory symptoms despite standard therapy [10, 24].

Pathophysiology

The pathophysiology of CAS is multifactorial [3]. Endothelial dysfunction—reduced nitric oxide bioavailability with increased endothelin-1 and von Willebrand factor activity—shifts vascular tone toward vasoconstriction and favors platelet adhesion [3]. Enhanced vascular smooth-muscle contractility, mediated largely through the Rho-kinase/Rho-associated kinase pathway, is a central mechanism. Circulating leukocyte Rho-kinase activity correlates with spasm severity and prognosis, making it a potential therapeutic target (for example, the Rho-kinase inhibitor fasudil) [3, 29]. Adventitial and perivascular adipose tissue inflammation has been demonstrated in patients with VSA, providing an inflammatory substrate that may explain spasm in individuals without classical atherosclerotic risk factors [30]. Autonomic imbalance and coronary microvascular dysfunction frequently coexist with epicardial spasm [3, 24]. Allergic or mast-cell–mediated mechanisms (the Kounis spectrum) can also provoke spasms through histamine, leukotrienes, and related mediators [31]. In our patient the transient, non-pruritic axillary rash that resolved after a single dose of dexamethasone raised this possibility. However, allergen and mast-cell testing were not performed; thus, the association remains speculative.

Interaction between spasm and thrombosis

Vasospasm and thrombosis can reinforce one another. Spasms alter local shear stress and may injure or erode the endothelium, exposing the thrombogenic subendothelial matrix and promoting platelet adhesion and fibrin deposition. The resulting thrombus and slow flow prolong blood–vessel contact and further aggravate vasoconstriction [14, 15]. Pathological and optical coherence tomography (OCT) studies confirm that spasm-associated thrombi frequently arise from plaque erosion rather than rupture [16, 17, 32], and residual vasospasms are common in patients with erosion or rupture [33]. Our IVUS

findings—intraluminal thrombosis without plaque rupture, dissection, or hematoma—are consistent with this erosion-type, non-rupture mechanism of acute coronary syndrome [14, 15], and they support a strategy based on antithrombotic therapy rather than plaque ablation or stenting.

Diagnostic value of IVUS

IVUS was decisive in this case. It excluded plaque rupture, dissection, intramural hematoma and severe calcification, thereby identifying the culprit as functional rather than structural [12, 18]. By repeating the imaging at four time points, we could track the dynamic behavior of the segment: the MLA decreased from 3.19 mm^2 to 2.75 mm^2, while the minimum lumen diameter decreased from 3.22 mm to 1.87 mm, and both recovered after antithrombotic and mechanical therapy (final MLA, 6.05 mm^2). This serial in-vivo documentation is the main original contribution of our report because most previous reports described imaging at a single time point [11, 12]. However, IVUS has limitations relative to OCT: its lower axial resolution means it cannot reliably identify superficial plaque erosion or characterize thrombus composition, whereas OCT provides superior definition of the superficial plaque surface at the cost of limited penetration and the need for contrast flushing [32, 33].

Interventional strategy: why stenting was withheld

Stent implantation was withheld for several reasons. First, IVUS showed that the luminal narrowing was dominated by dynamic spasms and that no significant fixed atherosclerotic plaque was present; therefore, no structural lesion required scaffolding. Second, stenting within a spastic segment is associated with stent malapposition [19], in-stent and edge vasospasms [20], and stent thrombosis [21]. Drug-eluting stents may themselves induce or aggravate vasospasms, and PCI in patients with documented vasospasms has been linked to adverse long-term outcomes [22]. Third, medical and mechanical antispasmodic therapy restored flow, and angiography at 8 days showed 0% residual stenosis, confirming that the lesion was reversible. Nevertheless, stenting is reasonable in selected patients with truly drug-refractory spasms or recurrent life-threatening events [23], or when imaging demonstrates a fixed, progressive culprit plaque [18]. Two further reports have shown how spasms may mimic or coexist with structural diseases such as spontaneous coronary artery dissection, and emphasize that intracoronary imaging is required before attributing a lesion to spasms alone [34, 35]. Conversely, medical therapy alone has been successful when spasms are accompanied by dissection [36]. Our case complements these reports by demonstrating that when IVUS excludes a fixed culprit lesion, a stent-free approach can be safely pursued.

Pharmacological therapy

CCBs are the cornerstone of therapy for VSA and should be titrated toward the maximum tolerated dose to suppress spasms [4, 24]. In our patient, the diltiazem dose was increased to 90 mg twice daily. Long-acting nitrates and nicorandil are useful second-line or add-on agents [24]. Rho-kinase inhibition with

fasudil is a recognized option for refractory spasms [3, 29]. Statins improve endothelial function and are appropriate for long-term risk reduction [4]. The value of beta-blockers in VSA is debated: non-selective beta-blockade may worsen spasms, whereas cardioselective agents may be continued cautiously, which is why the metoprolol dose was reduced from 47.5 mg to 23.75 mg daily. Because the index event involved a spasm-related thrombus, dual antiplatelet therapy with aspirin and ticagrelor was administered for secondary prevention, complemented in the acute phase by intracoronary glycoprotein IIb/IIIa inhibition (tirofiban) and fibrinolysis (urokinase). Antiplatelet therapy is central to the management of MINOCA and erosion-type acute coronary syndromes [8, 14].

Comparison with previous reports

Compared with earlier reports, our case has three distinctive features. First, four sequential IVUS measurements captured the entire course of the progressive spasm with secondary thrombosis and its subsequent resolution in vivo, whereas most published observations are single-time-point images [11, 12]. Second, the decision to avoid stenting was explicitly justified by imaging evidence that the lesion was functionally dominated, in contrast to reports in which spasms overlying a progressive plaque were stented [18, 23] and consistent with data on the hazards of stenting spastic segments [19–22]. Third, the antithrombotic regimen combined intracoronary tirofiban with intracoronary urokinase and repeated balloon dilation, a combination that, to our knowledge, has rarely been described.

Resource-limited setting

Management was also shaped by the resource-limited setting. Acetylcholine and ergonovine for provocation testing and injectable CCB formulations for intracoronary use are unavailable in county-level hospitals [9, 24]. Therefore, diagnosis relied on the clinical picture and dynamic IVUS evidence obtained during PCI, rather than on functional provocation testing as recommended in expert consensus documents [9]. Similarly, refractory spasms were managed with repeated intracoronary nitroglycerin, intracoronary tirofiban and urokinase, and mechanical dilation instead of intracoronary CCBs. This pragmatic pathway produced a favorable result and may serve as a reference for operators working in similar settings.

Limitations

This study had several limitations. First, the diagnosis of VSA was not confirmed by acetylcholine or ergonovine provocation testing, which remains the reference standard [1, 9]; however, it relied on the clinical course and intraprocedural imaging findings. Second, coronary reactivity and microvascular function were not assessed [6]. Third, the axillary rash was not investigated with allergen testing or eosinophil counts; therefore, an allergic (Kounis-type) mechanism could only be hypothesized [31]. Fourth, IVUS could not reliably detect superficial plaque erosion, which OCT would have assessed better [32, 33]. Finally, this was a single case with only 4 months of follow-up, and the stent-free strategy described here requires confirmation in larger series.

Conclusions

This case shows that refractory coronary spasms can progress despite intracoronary nitroglycerin during emergency PCI and can generate recurrent thrombi and slow flow. Serial IVUS is the key tool for distinguishing this functional process from structural obstruction and for guiding a stent-free strategy when no fixed culprit plaque is present. Operators, particularly those in resource-limited settings, should be aware of this entity and consider deferring stenting when IVUS excludes a fixed lesion, while ensuring adequate antispasmodic and antithrombotic therapy.

Abbreviations

CCB
calcium-channel blocker
IVUS
intravascular ultrasound
MLA
minimal lumen area
MI
myocardial infarction
PCI
percutaneous coronary intervention
RCA
right coronary artery
TIMI
Thrombolysis In Myocardial Infarction
VSA
vasospastic angina

Declarations

Ethics approval and consent to participate

This case report was approved by the Ethics Committee of Binzhou Central Hospital (approval number □kyl1-2026112□). Written informed consent was obtained from the patient for participation in this study.

Consent for publication

Written informed consent for publication of the clinical details and images was obtained from the patient. A copy of the consent form is available for review by the Editor of this journal.

Competing interests

The authors declare that they have no competing interests.

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Author Contribution

LM. performed the PCI procedure and contributed to data collection and manuscript drafting. KZ., RJ. collected and analysed the clinical and imaging data. JC. conceived the case report, performed the PCI procedure, and revised the manuscript. All authors read and approved the final manuscript.

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Availability of data and materials

All data generated or analysed during this study are included in this published article. The imaging data are available from the corresponding author on reasonable request.

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Figures

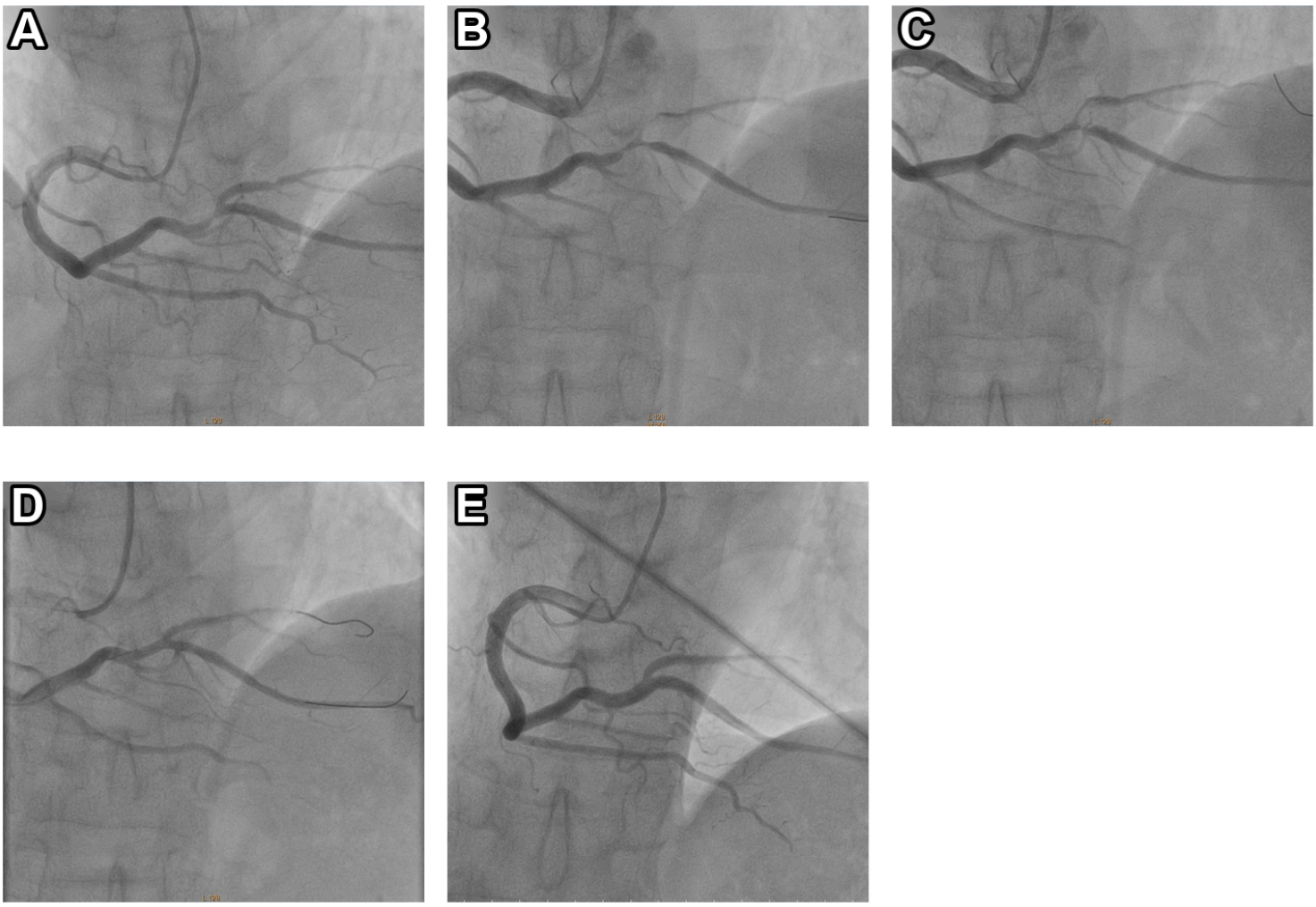


Figure 1

Coronary angiography of the right coronary artery (RCA). (A) Severe stenosis of the distal RCA at presentation. (B) Persistent severe stenosis after intracoronary nitroglycerin. (C) Slow flow after the first balloon angioplasty. (D) Final angiographic result after repeated balloon dilatation combined with intracoronary tirofiban and urokinase. (E) Follow-up angiography at day 8 showing a patent RCA with normal distal flow.

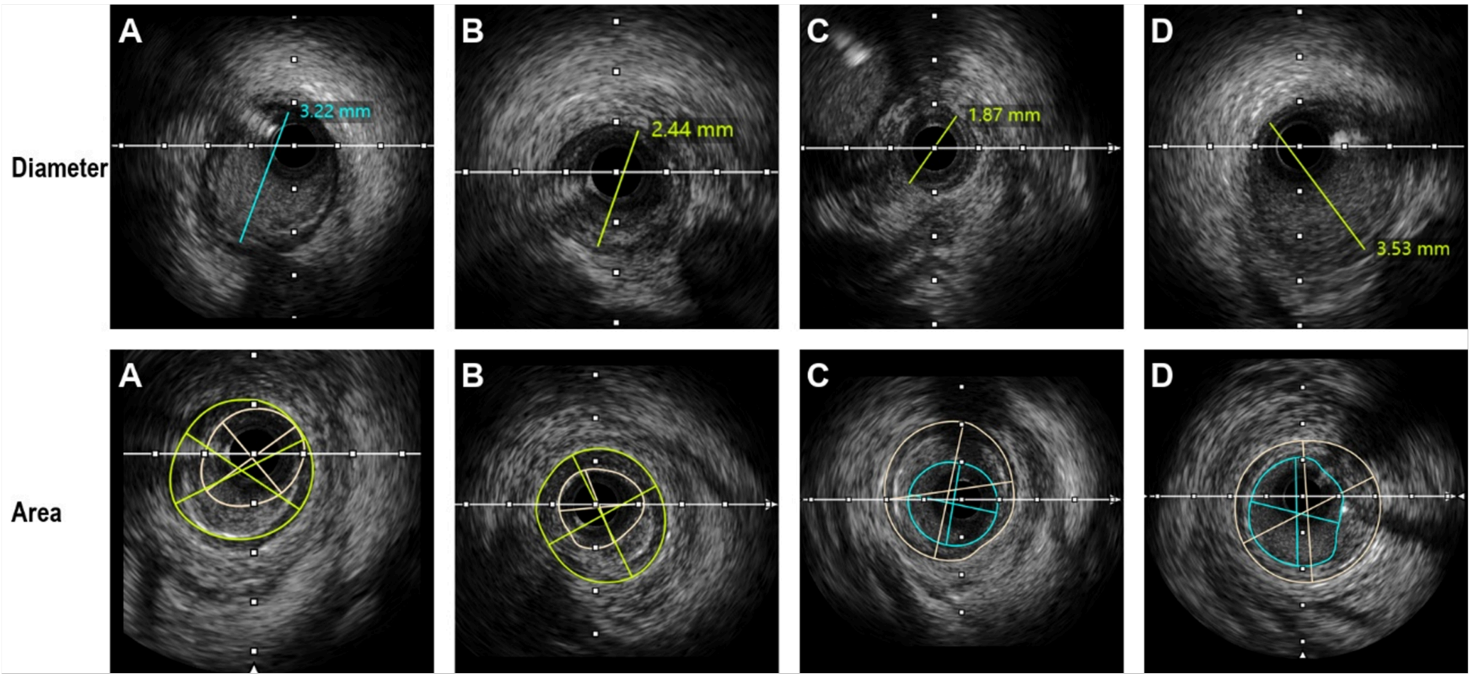


Figure 2

Serial intravascular ultrasound (IVUS) images of the right coronary artery (RCA) obtained at the stenosis site. The upper row shows the minimum lumen diameter and the lower row shows the minimum lumen area (MLA) at four time points: at presentation (A), after the first balloon angioplasty (B), at the nadir during the procedure (C), and the final result after repeated balloon dilatation combined with intracoronary tirofiban and urokinase (D). The minimum lumen diameter progressively decreased from 3.22 mm to 2.44 mm and 1.87 mm despite repeated intracoronary nitroglycerin and recovered to 3.53 mm at the end of the procedure, whereas the MLA was 3.19 mm^2, 2.75 mm^2, 4.13 mm^2, and 6.05 mm^2, respectively. An intraluminal thrombus is indicated by arrows, and residual mural thrombus remained on the final image. MLA, minimum lumen area.

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