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CASE REPORT

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# Recurrent subacute stent thrombosis after drug-eluting stent implantation: a case report

Yang Yang[1], Jinxin Jiang[1], Zhihao Chen[1] and Changqing Fan[2*]

## Abstract

**Background** While drug-eluting stents (DESs) have revolutionized percutaneous coronary intervention (PCI), early stent-related complications remain challenging. We present a rare case of recurrent subacute stent thrombosis (ST) following DES implantation.

**Case presentation** A 51-year-old female with acute inferior-posterior myocardial infarction underwent primary PCI with everolimus-eluting stent deployment in the left circumflex artery (LCX). Despite dual antiplatelet therapy, she developed recurrent LCX occlusion at 8 days (subacute ST confirmed by intravascular ultrasound) and 25 days post-PCI. Serial interventions included additional stenting and drug-coated balloon angioplasty. Elevated platelet counts and negative autoimmune marker results suggest potential inadequate platelet inhibition or hypersensitivity reactions.

**Conclusions** This case highlights the diagnostic challenges in differentiating ST from restenosis, emphasizes the role of intravascular imaging, and underscores the need for personalized antiplatelet regimens. Hypersensitivity reactions to stent components and Kounis syndrome should be considered in refractory cases.

**Keywords** Stent thrombosis, Drug-eluting stent, Kounis syndrome, Percutaneous coronary intervention, Tissue prolapse

## Background

Primary percutaneous coronary intervention is widely recognized as an effective treatment for patients with acute ST-segment elevation myocardial infarction, including those experiencing cardiogenic shock [1]. Stents have been invented and refined with the purpose of providing structural reinforcement to the artery subsequent to dilation, thereby preventing the occurrence of sudden vessel recoil following angioplasty. The progression of stent technology began with bare-metal stents and advanced to drug-eluting stents (DESs) and bioresorbable vascular scaffolds, aimed at reducing restenosis rates and improving long-term outcomes [2]. Despite these advancements, the complete elimination of acute, severe stent-related complications, which can lead to increased mortality, remains elusive. In this report, we document a case of recurrent subacute stent thrombosis following the implantation of a DES (Table 1). The potential causes behind this phenomenon and recommended therapeutic interventions are also being explored.

*Correspondence:
Changqing Fan
frankdoc@126.com
[1] Department of Cardiology, Xiang'an Hospital of Xiamen University, School of Medicine, Xiamen University, Xiamen 361100, P. R. China
[2] Department of Cardiology, Xiamen Susong Hospital, Xiamen 361100, P. R. China

**Table 1** Timeline of events during hospitalization

| **Admission to Emergency Department** | **Presented with nonspecific intensified retrosternal pain and an ECG registration with acute inferior-posterior ST-elevation myocardial infarction** |
|---|---|
| 40 min after admission | Primary PCI with implantation of DES in the proximal segment of the LCX<br>Resulted in TIMI flow 3 |
| 8 days after admission | Follow-up angiography revealed a subacute stent thrombosis at the proximal stent site in the LCX<br>IVUS-guided implantation of DES in the middle and distal LCX and a drug-coated balloon to address the localized occlusion at the proximal stent site in the LCX<br>Resulted in TIMI flow 3 |
| 12 days after admission | Recurrent inferior ST-segment elevation myocardial infarction |
| 25 days after admission | A third CAG revealed the recurrence of subacute stent thrombosis |

## Case presentation

The patient was a 51-year-old female with a medical history of hypertension and deep vein thrombosis in the lower extremities. She complained of nonspecific retrosternal pain for three days, which intensified significantly three hours before presenting to our emergency department. Her physical examination revealed a blood pressure of 126/78 mmHg, pulse rate of 74 beats/min and respiration rate of 18 breaths/min. A complete blood count revealed thrombocytosis (platelets = 481 × 10^9/L). An electrocardiogram (ECG) initially revealed the presence of acute inferior-posterior myocardial infarction (Fig. 1). Then, 300 mg of aspirin and 180 mg of ticagrelor were loaded, and weight-adjusted unfractionated heparin (60 U/kg) was administered intravenously. Shortly thereafter, the patient experienced sudden unconsciousness, immobility, and urinary incontinence. ECG monitoring revealed evidence of ventricular fibrillation. Normal sinus rhythm was restored through cardiopulmonary resuscitation and electric defibrillation, and emergency catheterization was promptly performed. Coronary angiography (CAG) revealed approximately 70% stenosis in the posterior descending branch and complete occlusion of the proximal segment of the left circumflex artery (LCX) with a Thrombolysis In Myocardial Infarction (TIMI) grade 0 flow (Fig. 2). The LCX was identified as the culprit vessel, and a platinum-chromium alloy everolimus-eluting stent (2.75 mm diameter, 24 mm length, Boston Scientific) was successfully deployed. The patient was then prescribed dual antiplatelet therapy,

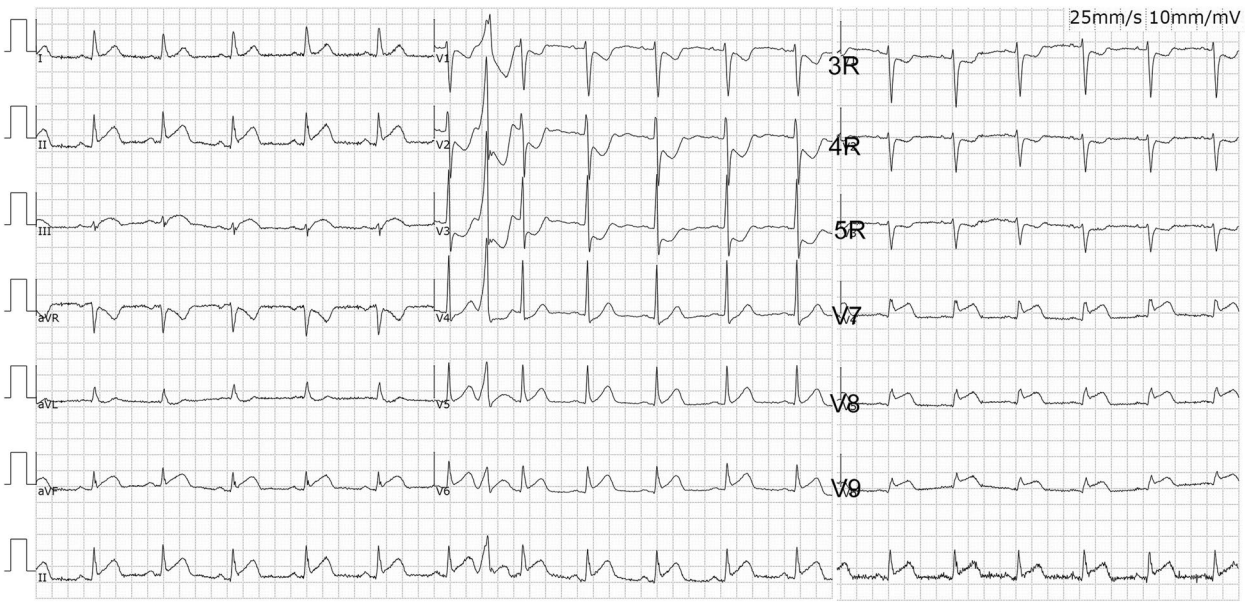


**Fig. 1** Initial presentation of an electrocardiogram showing acute inferior-posterior myocardial infarction

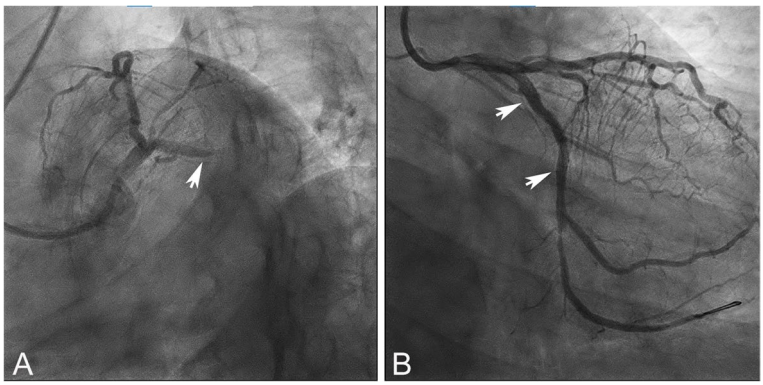


**Fig. 2** **A** CAG in the spider view illustrating complete occlusion of the proximal LCX (arrow). **B** Deployment of a platinum–chromium alloy everolimus-eluting stent (arrows) in the LCX was successfully achieved

comprising aspirin (100 mg once daily) and ticagrelor (90 mg twice daily), metoprolol (47.5 mg once daily), irbesartan (2.5 mg once daily), and rosuvastatin (10 mg once daily). Enoxaparin sodium (30 mg) was administered by subcutaneous injection every 12 h.

Eight days postprocedure, follow-up angiography revealed a localized occlusion at the proximal stent site in the LCX (Fig. 3). An Ikari Left 4.0 guiding catheter reached the left coronary artery ostium. The Runthrough NS guide wire was used to cross the occluded lesion but failed because of weak support. A Terumo NC microcatheter was subsequently used to boost support, enabling the wire to reach the distal circumflex branch. Predilation balloons (2.0×2.0 mm, 2.75×15 mm) were advanced along the wire and dilated at 8 atm. After that, a 3.0×15 mm noncompliant balloon was inserted and dilated at 18 atm/6 s. After withdrawal, blood flow was observed in the LCX, with TIMI flow 3. After predilation, intravascular ultrasound (IVUS) examination revealed 81.3% stenosis within the stent, with low-intensity echoes suggestive of a thrombus (Fig. 2B), which was distinct from the homogeneous hypoechoic appearance of neointimal hyperplasia. Additionally, 74.5% stenosis was detected in the middle and distal segments of the LCX, with a minimum lumen area of 2.6 $mm^2$. Following the withdrawal of the IVUS catheter, a 2.5×38 mm stent manufactured by Boston Scientific was carefully advanced along the guidewire to the stenotic area located in the middle-distal segment of the LCX. Once precisely positioned, the proximal portion of the newly introduced stent was deliberately overlapped with the distal end of the previously deployed stent by approximately 1–2 mm. The stent was subsequently dilated at 12 atm/4 s. After the complete withdrawal of the stent-mounted balloon

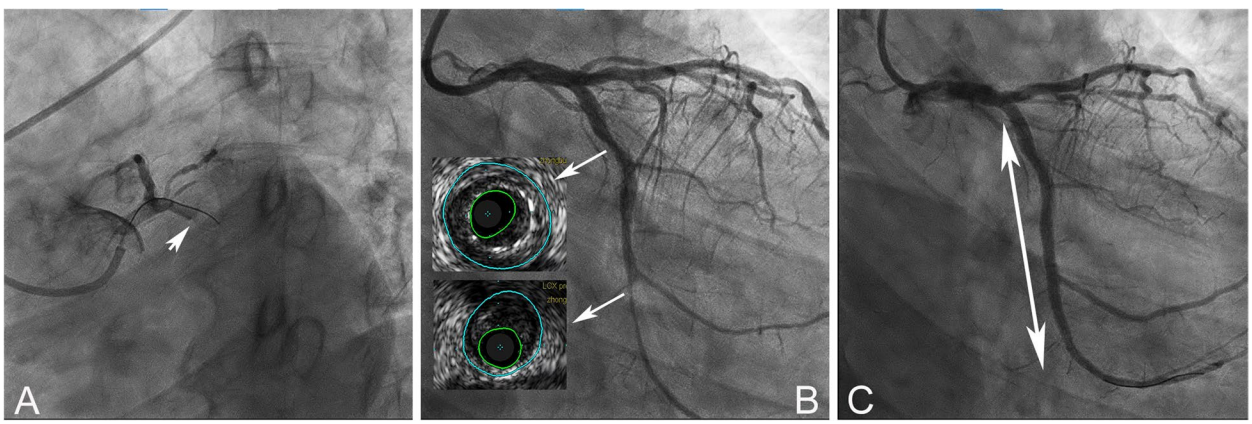


**Fig. 3** **A** Follow-up angiography eight days poststenting showing localized occlusion at the proximal stent site in the LCX (arrow). **B** Predilation was performed, followed by IVUS examination, which revealed complete stent expansion accompanied by thrombotic material (arrows). Additionally, severe stenosis was observed in the middle and distal segments of the LCX. **C** Drug-coated balloon angioplasty and the deployment of a second everolimus-eluting stent successfully restored vessel patency (bidirectional arrows)

from the coronary artery, a Qingzhou drug-coated balloon was reinserted and accurately positioned at the stenosis site within the occluded stent. This drug-coated balloon was then inflated at 24 atm/60 s. Immediately after the removal of the drug-coated balloon, repeat angiography was performed. The results clearly demonstrated that the stent had expanded optimally, and the antegrade TIMI flow had reached Grade III, indicating successful restoration of blood flow. IVUS confirmed the secure attachment of the stents to the vessel wall. The stent apposition was satisfactory, ruling out the possibility of poor stent apposition and dissection. Postoperation, ticagrelor was discontinued, and clopidogrel and tirofiban were added for intensified antiplatelet therapy. Atorvastatin was increased to 40 mg once daily for intensified lipid-regulating therapy.

Twelve days after admission, the patient experienced a recurrence of chest pain similar to her initial presentation. Blood tests revealed elevated levels of the serum cardiac marker troponin T and an increased platelet count of 530.00×10^9/L. Tests for extractable nuclear antigen antibodies and antineutrophil cytoplasmic antibodies were negative. ECG confirmed the presence of acute ST-segment elevation myocardial infarction (ASTEMI) in the inferior wall (Fig. 4). Consequently, the patient was diagnosed with recurrent ASTEMI, and a tirofiban infusion was initiated. Additionally, ezetimibe was incorporated into the therapeutic regimen. Following intensified medication therapy, the patient achieved symptomatic remission, and the follow-up ECG demonstrated ST-segment resolution compared to prior recordings. Remarkably, twenty-five days post-admission, a third CAG revealed the recurrence of subacute ST, which occurred precisely at the site of the previously implanted stent in the proximal segment of the LCX (Fig. 5).

## Discussion and conclusions

The management of acute myocardial infarction (AMI) has undergone significant advancements over the past three decades, marking a notable achievement in contemporary medicine. For patients with acute ST-segment elevation myocardial infarction (ASTEMI), mechanical

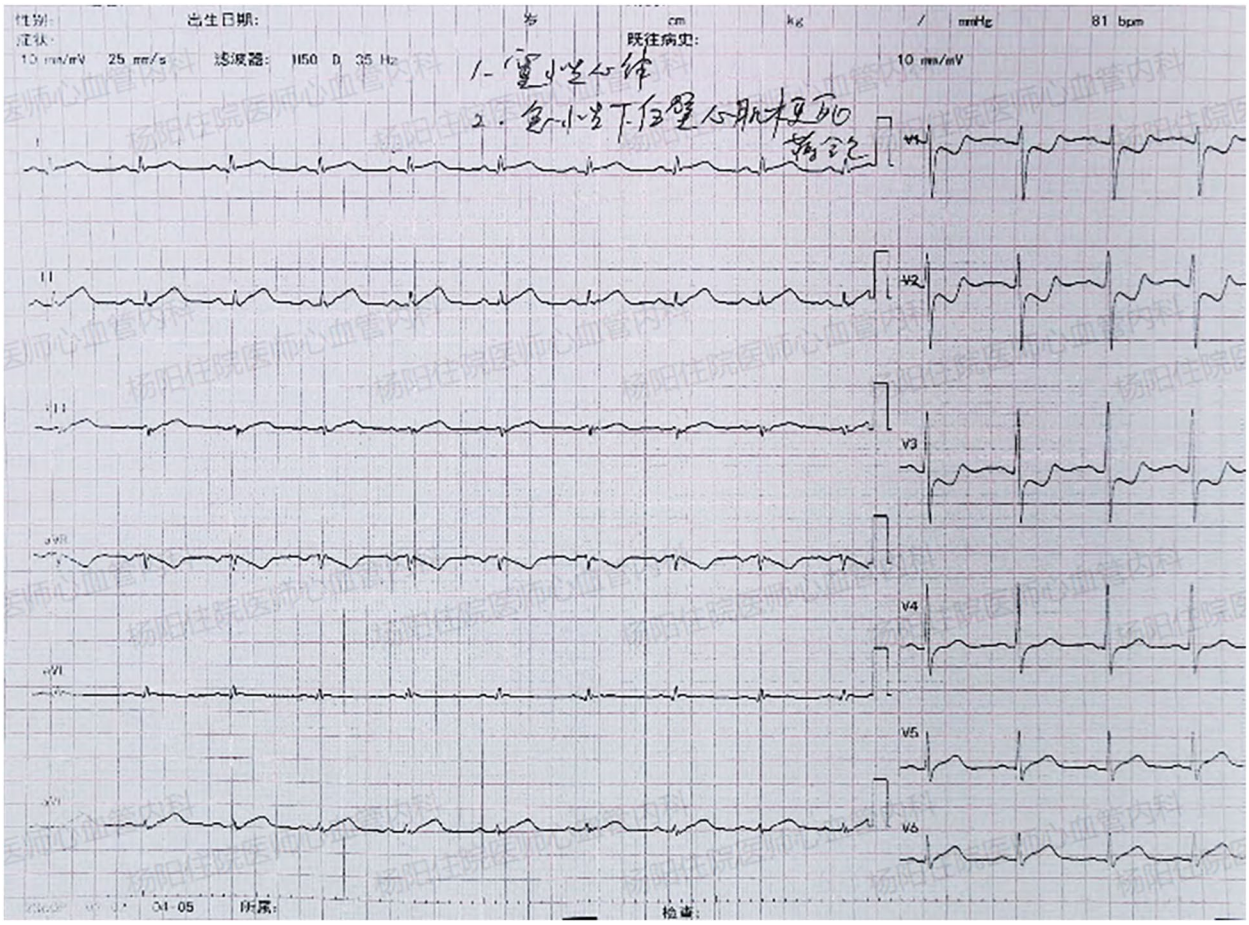


**Fig. 4** Electrocardiogram on day 12 after admission showing inferior ST-elevation myocardial infarction

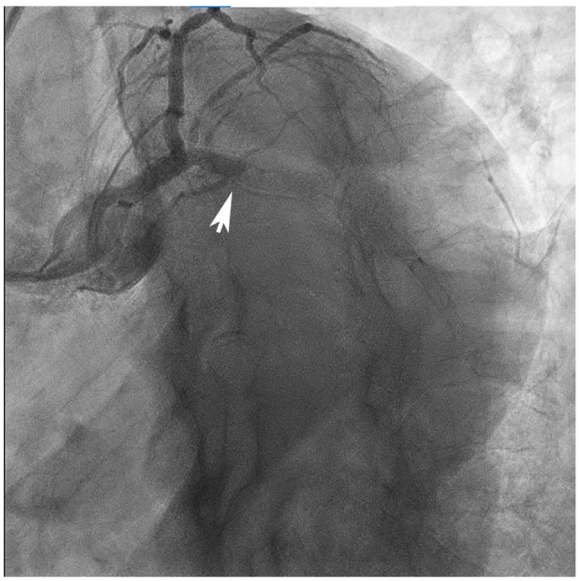

**Fig. 5** A third angiography conducted due to symptom recurrence revealed the recurrence of localized complete occlusion within the LCX (arrow)

reperfusion via primary percutaneous coronary intervention (PCI) is the standard recommendation [1]. However, the optimal strategy and timing for revascularization, particularly in patients with multivessel disease (MVD) during ASTEMI, remain contentious [3]. Recent guidelines and randomized clinical trials suggest that achieving complete revascularization before hospital discharge, whether immediate or staged, is now considered a Class IIa recommendation [4]. Drawing insights from the Culprit Lesion Only PCI versus Multivessel PCI in Cardiogenic Shock trial, as well as the present case, it is evident that primary PCI targeting only the culprit lesion, with the option for staged revascularization of nonculprit lesions, offers a safer and more effective approach than immediate multivessel PCI in ASTEMI patients with MVD [5]. Staged multivessel PCI strategies during ASTEMI can effectively mitigate additional risks, particularly the induction of further ischemia, stent failure, and subsequent restenosis as well as renal impairment induced by the use of a greater quantity of contrast material [6]. This approach ensures focused and controlled intervention, allowing for better management of each lesion and reducing the overall procedural burden on the patient.

PCI remains a cornerstone for treating coronary artery disease, yet stent thrombosis (ST) and in-stent restenosis (ISR) persist as critical complications that impact long-term outcomes. Distinguishing these entities through mechanistic understanding, precise diagnostics, and tailored management is essential for optimizing patient care (Table 2).

ST, characterized by thrombus-induced stent occlusion, is classified temporally by the Academic Research Consortium: acute (0–24 h post-PCI), subacute (24 h–30 days), late (31–360 days), and very late (>360 days) [7]. Angiographic confirmation requires visualization of the intraluminal thrombus (e.g.,

**Table 2** The key differences between acute stent thrombosis (ST) and in-stent restenosis (ISR)

| Feature | Stent Thrombosis (ST) | In-Stent Restenosis (ISR) |
|---|---|---|
| Definition | Blood clot within/adjacent to the stent, causing partial/complete vessel occlusion | Luminal narrowing (≥ 50% stenosis) within the stent/margins due to neointimal hyperplasia |
| Timeline | - **Acute:** 0–24 h<br>- **Subacute:** 24 h–30 days<br>- **Late:** 31–360 days<br>- **Very late:** > 360 days | Peaks at **3–6 months** (drug-eluting stents); chronic process |
| Pathophysiology | - Early ST (≤ 30 days): Stent malapposition, underexpansion, residual dissection<br>- Late ST (> 30 days): Neoatherosclerosis, delayed endothelialization | Excessive smooth muscle cell/fibroblast proliferation → neointimal hyperplasia |
| Angiographic Features | - Thrombus (filling defects, contrast staining)<br>- Abrupt vessel occlusion | - Smooth, concentric narrowing<br>- Patterns: focal, diffuse, proliferative, or total occlusion (no thrombus unless secondary) |
| IVUS Findings | - Stent malapposition/underexpansion<br>- Thrombus: echolucent/heterogeneous masses | - Homogeneous neointimal hyperplasia (concentric/eccentric hyperechoic layer) |
| OCT Findings | - Thrombus (low-signal, irregular masses)<br>- Malapposition, plaque rupture, thin-cap fibroatheroma | - Homogeneous neointima (high-signal tissue)<br>- Subtypes: homogeneous vs. heterogeneous hyperplasia |
| Clinical Presentation | - Acute coronary syndrome (STEMI/NSTEMI)<br>- Requires urgent revascularization | - Recurrent angina/stable symptoms<br>- Biomarkers (e.g., troponin) less elevated |
| Management | - Antithrombotic therapy (antiplatelets/anticoagulants)<br>- Urgent revascularization | - Mechanical/interventional approaches (balloon angioplasty, cutting balloon, new stent) |

contrast staining, filling defects). In contrast, ISR—defined as ≥ 50% luminal narrowing within or adjacent to the stent—represents a chronic process driven by neointimal hyperplasia or vascular remodeling, typically presenting without thrombus unless complicated secondarily [8]. Early ST (≤ 30 days) predominantly arises from procedural factors: stent malapposition, underexpansion, or residual dissection creates thrombogenic surfaces by exposing stent struts or disrupting vessel integrity [9]. Underexpanded stents create microenvironments of low shear stress and incomplete endothelialization, promoting platelet adhesion and therefore increasing the risk of early ST. Late ST (> 30 days) correlates with delayed endothelialization and neoatherosclerosis, where atherosclerotic plaque rupture within the stent triggers thrombosis [10]. ISR pathophysiology diverges, and involves excessive smooth muscle cell proliferation and extracellular matrix deposition, culminating in neointimal hyperplasia. Despite drug-eluting stents (DESs) mitigating hyperplasia, the ISR incidence peaks at 3–6 months because of delayed endothelial repair from antiproliferative agents [11].

Intravascular ultrasound (IVUS) and optical coherence tomography (OCT) are pivotal for differentiation [12]. In STs, IVUS identifies malapposed/underexpanded stents and echolucent thrombi, whereas OCT delineates thrombi as low-signal masses and detects plaque rupture or thin-cap fibroatheroma [13]. For ISR, IVUS reveals homogeneous hyperechoic neointima, whereas OCT reveals high-signal proliferative tissue, distinguishing focal from diffuse subtypes and excluding thrombotic components [14].

ST frequently manifests as acute coronary syndrome (ACS), necessitating urgent revascularization and intensified antithrombotic therapy (dual antiplatelet agents ± anticoagulants) [15]. Probable STs include unexplained death ≤ 30 days post-PCI or target-vessel myocardial infarction without angiographic confirmation. ISR typically presents with exertional angina; biomarkers remain normal unless severe ischemia ensues [16]. Management prioritizes mechanical interventions—balloon angioplasty, cutting balloons, or repeat stenting—to address neointimal obstruction.

Accurate discrimination between the ST and ISR hinges on the integration of the clinical context, imaging findings, and temporal patterns. IVUS/OCT enhances diagnostic precision, guiding targeted therapies: antithrombotic strategies for ST versus mechanical interventions for ISR. Further research is warranted to refine risk stratification and therapeutic approaches, ultimately improving PCI-related outcomes.

The present case of recurrent subacute ST in the left circumflex artery raises critical questions regarding antiplatelet regimen optimization. Following the second angiography, IVUS-guided stent optimization excluded mechanical causes, prompting the evaluation of pharmacodynamic limitations. The decision to switch from ticagrelor to clopidogrel, while seemingly counterintuitive given guidelines favoring ticagrelor for its rapid and potent platelet P2Y12 inhibition, may be justified in this context. Ticagrelor resistance, although rare, has been reported in patients with recurrent ST despite adequate adherence, potentially due to impaired enteral absorption or epigenetic differences [17]. Clopidogrel, despite its slower onset and greater interindividual variability, may bypass ticagrelor-specific resistance mechanisms. However, this strategy necessitates caution, as the efficacy of clopidogrel is heavily influenced by CYP2C19 polymorphisms, which are prevalent in Asian populations and associated with reduced active metabolite generation. The lesson we learned from this case is that, in an attempt to minimize potential catastrophe, the first consideration should be to adjust to the administration of ticagrelor powder on an empty stomach. If it still proves ineffective, consideration can be given to adjusting to intravenous administration of cangrelor [18]. The absence of platelet function testing or CYP2C19 genotyping in this case limits definitive conclusions but underscores the importance of personalized antiplatelet therapy guided by pharmacodynamic or genetic profiling.

In the present case, the persistent increase in the peripheral platelet count in the patient despite dual antiplatelet therapy (DAPT) raises critical considerations regarding two potential mechanisms: inadequate platelet inhibition and drug-induced hypersensitivity reactions. With respect to antiplatelet failure, both ticagrelor and clopidogrel carry risks of a hyporesponse. Aspirin resistance, though less common, cannot be excluded without thromboxane testing. For patients with dual resistance, prasugrel—a third-generation thienopyridine with more consistent platelet inhibition—or adjunctive cilostazol (a phosphodiesterase inhibitor) could be considered. In hypersensitivity scenarios, desensitization protocols or novel agents (e.g., vorapaxar) may offer alternatives, although bleeding risks must be weighed. Importantly, the combination of low-dose rivaroxaban with DAPT has shown promise in reducing thrombotic events in high-risk PCI patients, a strategy meriting consideration in refractory cases.

Tissue prolapse (TP) refers to the intraluminal protrusion of tissue through stent struts, and is frequently visualized poststent implantation via IVUS or OCT [19]. While IVUS provides foundational insights into stent apposition and tissue morphology, its limited resolution complicates the definitive differentiation between thrombus and prolapse. IVUS cannot exclude

superimposed microthrombi or fully characterize the microstructure of the prolapsed tissue. In contrast, OCT, with its superior axial resolution (~ 10–20 μm vs. 100–200 μm for IVUS), enables precise discrimination of TP subtypes and thrombotic components. OCT delineates thrombi as irregular, low-signal protrusions with dorsal shadowing, whereas plaque prolapse typically manifests as smooth, signal-rich tissue herniation through stent struts [20]. OCT further enhances microstructural assessment by identifying features associated with thrombogenicity, such as plaque rupture, thin-cap fibroatheroma, or macrophage infiltration within prolapsed tissue. According to OCT examinations, the occurrence rate of TP following stent insertion is 100% in patients with unstable conditions [21]. Several IVUS studies have shown that TP is a significant determinant of adverse short-term outcomes, including early stent thrombosis and increased myocardial necrosis [22, 23]. In AMI patients, OCT-defined TP results in greater thrombus adhesion due to an exposed necrotic core or disrupted fibrous caps, increasing the risk of thrombosis. Despite these advantages, the clinical adoption of OCT remains limited by cost and procedural complexity, including contrast-induced complications [24]. In this case, OCT was not performed due to institutional resource constraints; however, its application could have clarified the presence of the thrombus and the composition of the prolapse, particularly given the ambiguous IVUS findings.

Given the diagnostic uncertainty inherent to IVUS in distinguishing thrombus from prolapse, future cases may benefit from protocolized OCT use in high-risk scenarios. Moreover, identifying biomarkers that are predictive of TP remains critical. The Apo B/A1 ratio, an independent correlate of TP volume and plaque vulnerability on OCT [25], represents a promising noninvasive tool for stratifying patients warranting advanced imaging follow-up.

Cardiac stents play a pivotal role in the management of cardiovascular diseases, providing essential support through stability, flexibility, and durability. However, the very materials that confer these properties can also trigger distinct pathological mechanisms: hypersensitivity-related restenosis driven by immune activation versus thrombotic events caused by mechanical or hemodynamic factors [26]. Polymer coatings, such as permanent polymers in first-generation DESs (e.g., polyurethane or polyethylene-co-vinyl acetate), have been associated with allergic reactions because of their prolonged exposure to vascular tissues, leading to eosinophilic infiltration, mast cell degranulation, and chronic inflammation [27–29]. In contrast, thrombotic mechanisms primarily involve platelet aggregation due to stent malapposition, delayed endothelialization, or neoatherosclerosis, which are often unrelated to immune hypersensitivity [29].

Metallic platforms (e.g., stainless steel or cobalt–chromium alloys) and eluted antiproliferative drugs (e.g., paclitaxel) promote antigenic sensitization by acting as mast cell-activating triggers, thereby exacerbating coronary intimal inflammation and predisposing patients to hypersensitivity-driven ACS, such as Kounis syndrome, a condition characterized by concurrent myocardial ischemia, vasospasm, and ST mediated by mast cell-derived mediators such as histamine and tryptase [30]. The diagnosis of Kounis syndrome hinges on detecting these mediators, as serum tryptase levels peak within 1 h of symptom onset and histamine becomes undetectable within minutes owing to rapid clearance. However, in this case, the absence of serum tryptase or histamine measurements during recurrent ischemic episodes—critical biomarkers for confirming mast cell activation—precluded definitive differentiation of Kounis syndrome from conventional ACS etiologies, underscoring the diagnostic challenges in hypersensitivity-associated coronary events.

To mitigate hypersensitivity-driven complications, bioabsorbable polymer stents (e.g., poly-L-lactic acid-based scaffolds) offer a promising alternative by eliminating persistent antigenic stimuli after complete degradation. Recent studies have demonstrated that bioabsorbable stents reduce long-term inflammation and restenosis rates compared with permanent polymer DESs, particularly in patients with preexisting metal allergies [31]. Additionally, newer-generation DESs containing anti-inflammatory agents, such as sirolimus derivatives or novel mammalian target of rapamycin inhibitors (e.g., everolimus), have shown efficacy in suppressing local immune responses [32, 33]. For example, the XIENCE V stent, which combines a thin strut design with an everolimus-eluting fluoropolymer, significantly reduces both thrombotic and hypersensitivity-related adverse events. Notably, the Firehawk stent provides patients with safer and more effective treatment options by optimizing drug release and reducing inflammatory reactions [34].

Systemic pharmacological interventions, such as corticosteroids or leukotriene receptor antagonists, may complement stent-based strategies in high-risk patients with documented hypersensitivity. However, long-term corticosteroid use poses significant risks, necessitating personalized risk–benefit assessments. Prospective biomarkers, including serum eosinophil counts or immunoglobulin E levels, could aid in stratifying patients for targeted therapies.

This case highlights the importance of individualized risk stratification and multidisciplinary management for high-risk patients facing early stent-related

complications. Rigorous post-PCI monitoring, especially in DES recipients, should incorporate advanced intravascular imaging, such as IVUS or OCT, for timely interventions. Risk-adapted protocols are crucial: high-risk patients, such as those with hypercoagulable states or allergic diathesis, need customized antiplatelet regimens, enhanced anticoagulation, and preemptive biomarker screening. Imaging-guided surveillance helps differentiate thrombosis, restenosis, and tissue prolapse, enabling early intervention. Technological innovations, including bioabsorbable stents and immune-modulating platforms, may offer solutions for hypersensitivity-related complications in the long term, especially for patients unresponsive to conventional therapies. Future research should focus on understanding the interactions among stent materials, immune responses, and thrombotic pathways, and standardized protocols integrating imaging, biomarkers, and personalized treatments are needed to improve outcomes for this vulnerable group.

**Abbreviations**

| | |
|---|---|
| ACS | Acute coronary syndrome |
| AMI | Acute myocardial infarction |
| ASTEMI | Acute ST-segment elevation myocardial infarction; |
| CAG | Coronary angiography |
| DAPT | Dual antiplatelet therapy |
| DES | Drug-eluting stent |
| ECG | Electrocardiogram |
| IVUS | Intravascular ultrasound |
| LCX | Left circumflex artery |
| MVD | Multivessel disease |
| OCT | Optical coherence tomography |
| PCI | Percutaneous coronary intervention |
| ST | Stent thrombosis |
| TIMI | Thrombolysis in myocardial infarction |
| TP | Tissue prolapse |

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**Authors' contributions**
YY and JJ cared for the patient and wrote and revised the manuscript. ZC and CF revised the manuscript and figures. All authors reviewed and approved the final manuscript.

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**Data availability**
No datasets were generated or analysed during the current study.

## Declarations

**Ethics approval and consent to participate**
Not applicable.

**Consent for publication**
Written informed consent was obtained from the patient for the publication of this case report and any accompanying images.

**Competing interests**
The authors declare no competing interests.

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