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PROGRESSIVE ANGIOGRAPHIC HAZINESS PRECEDING INTRAPROCEDURAL STENT THROMBOSIS DURING PRIMARY PCI: A CASE REPORT

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ПРОГРЕСИРАЩО АНГИОГРАФСКО ЗАМЪГЛЯВАНЕ, ПРЕДШЕСТВАЩО ИНТРАПРОЦЕДУРНА ТРОМБОЗА НА СТЕНТА ПО ВРЕМЕ НА ПЪРВИЧНА PCI: КЛИНИЧЕН СЛУЧАЙ

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Abstract. **Background:** Angiographic haziness after coronary stent implantation is nonspecific and may represent thrombus, edge dissection, tissue protrusion, intramural hematoma, or stent underexpansion. **Case summary:** A 61-year-old man with anterior ST-segment elevation myocardial infarction underwent primary percutaneous coronary intervention (PCI) of a subtotal mid-left anterior descending artery lesion. One minute after implantation of a 3.0 × 26-mm zotarolimus-eluting stent, new haziness appeared. It was interpreted as an edge dissection and treated with two additional proximal stents, after which the artery abruptly occluded and cardiogenic shock developed. Aspiration retrieved red thrombus, confirming intraprocedural stent thrombosis. Activated clotting time (ACT), first measured 2 min after the onset of shock, was 150 s. Supplemental heparin and eptifibatide restored TIMI grade 2 flow. At staged intravascular ultrasound-guided reintervention on day 4, the vessel was larger than initially estimated and a fluoroscopic balloon waist suggested underexpansion; quantitative imaging data were lost. **Conclusion:** Thrombotic, anticoagulants, and mechanical abnormalities were documented at different time points, but their relative contribution cannot be established. Haziness that persists after a first mechanical correction should prompt verification of anticoagulation and intracoronary imaging before further stenting.

Key words: intraprocedural stent thrombosis; primary percutaneous coronary intervention; angiographic haziness; activated clotting time; stent underexpansion; stent undersizing

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Резюме. **Въведение:** Ангиографското замъгляване след имплантиране на коронарен стент е неспецифично и може да представлява тромб, дисекция по ръба, изпъкване на тъкан, интрамурална хематома или недостатъчно разширяване на стента. **Описание на случай:** 61-годишен мъж с преден миокарден инфаркт с повдигане на ST-сегмента претърпя първична перкутанна коронарна интервенция (ПКИ) поради субтотална лезия в средната част на лявата предна низходяща артерия. Една минута след имплантирането на зотаролимус-покрит стент с размери 3, 0 × 26 mm се появи ново замъгляване. То бе интерпретирано като дисекция по краищата и бе лекувано с два допълнителни проксимални стента, след което артерията внезапно се запуши и се развди кардиогенен шок. При аспирация бе извлечен червен тромб, което потвърди интрапроцедурна тромбоза на стента. Активираното време на съсирване (ACT), измерено за първи път 2 min след началото на шока, беше 150 s. Допълнителното прилагане на хепарин и ептифибатид възстанови кръвопотока от 2-ра степен по скалата на TIMI. При поетапната повторна интервенция под интраваскуларно ултразвуково насочване на 4-ия ден съдът се оказа по-голям от първоначално очакваното, а флуороскопският балон със стеснение в средата подсказа, че разширяването е било недостатъчно данните от количествената визуализация бяха загубени. **Заключение:** В различни моменти бяха документирани;

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тромботични, антикоагулантни и механични аномалии, но относителният им принос не може да бъде установен. Замъгляването, което продължава след първата механична корекция, трябва да е повод за проверка на антикоагулационното лечение и интракоронарното изображение преди по-нататъшно поставяне на стент.

Ключови думи: интрапроцедурна тромбоза на стента; първична перкутанна коронарна интервенция; ангиографска замъгленост; активирано време на съсирване; недостатъчно разширяване на стента; недостатъчен размер на стента

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Introduction

Intraprocedural stent thrombosis (IPST), the development of new or increasing thrombus within or adjacent to a recently implanted stent before the procedure is completed, is uncommon but is associated with subsequent stent thrombosis and mortality [1, 2]. Its mechanism is frequently multifactorial, with procedural factors such as stent undersizing, underexpansion, or malapposition interacting with the thrombotic milieu and antithrombotic therapy [1]. Angiography can identify abrupt closure or visible thrombus, but an earlier finding such as haziness is less specific and may reflect thrombus, edge dissection, tissue protrusion, intramural hematoma, or incomplete stent expansion [3]. This case is presented not as a novel mechanism of stent thrombosis, but as a clinically instructive sequence in which progressive angiographic haziness was followed by thrombotic vessel closure, while later testing disclosed a low ACT and a mechanical substrate. The case also illustrates an important limitation of retrospective interpretation: findings obtained after hemodynamic deterioration or several days later cannot reliably reconstruct the mechanism present at the first appearance of haziness.

Clinical Case Description

A 61-year-old man with hypertension, dyslipidemia, and poor medication adherence presented 2 hours after the onset of chest pain. Blood pressure was 165/70 mmHg, and heart rate was 60 beats/min. Electrocardiography showed ST-segment elevation in leads V1 to V5, I, and aVL with reciprocal inferior ST depression (Fig. 1A). Coronary angiography demonstrated a subtotal mid-left anterior descending artery (LAD) culprit lesion with TIMI grade 1 flow and severe distal left circumflex stenosis with preserved TIMI grade 3 flow (Fig. 1B). The timing of key events is summarized in Table 1.

The patient weighed 85 kg. Aspirin 325 mg and prasugrel 60 mg (swallowed whole, not crushed) were given 45 minutes before the first stent implantation; he did not vomit, and fentanyl 25 μg was given intravenously for chest pain. An empiric 8000-IU intra-arterial bolus of unfractionated heparin (UFH), approximately 94 IU/kg, was given 25 minutes before stent implantation. ACT was not measured after the initial bolus. Following predilatation with a 2.5-mm semicompliant balloon, a 3.0 × 26-mm zotarolimus-eluting stent (ZES) was deployed in the mid-LAD at 12 atm, sized to a visually estimated reference diameter of 3.0 to 3.5 mm; no postdilatation was performed. Angiographic flow improved to TIMI grade 3, but within 1 min new subtle haziness was visible within the stented segment and near the proximal edge (Fig. 2A).

The proximal abnormality was assessed in two projections (anteroposterior caudal and right anterior oblique cranial). A straight linear radiolucency near the proximal stent edge was interpreted as an intimal flap from an edge dissection. Five minutes after the first stent, an overlapping 3.5 × 8-mm ZES was implanted proximally at 12 atm. Haziness persisted and extended (Fig. 2B). Because residual dissection remained the working diagnosis, a third 3.5 × 12-mm ZES was implanted at 12 atm in the proximal LAD, with its proximal edge approximately 8 mm distal to the LAD ostium, without involvement of the left main. Immediately afterward, the LAD abruptly occluded with TIMI grade 0 flow (Fig. 2C), and cardiogenic shock followed 2 minutes later, with blood pressure 80/60 mm Hg and heart rate 110 beats/min. Intravascular ultrasound (IVUS) was available in the laboratory but was not used during the index procedure because of national reimbursement constraints.

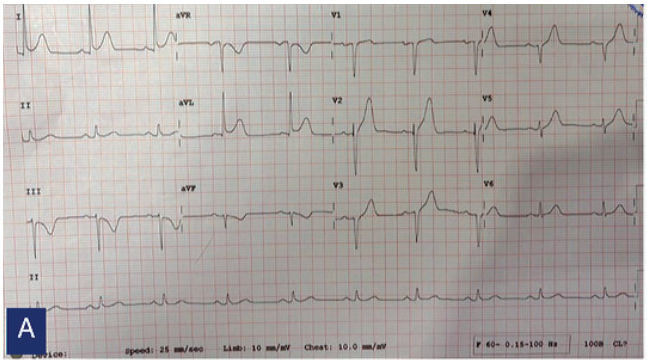
An intra-aortic balloon pump (IABP) and intravenous norepinephrine (0.1 μg/kg/min) were initiated. Point-of-care ACT, obtained for the first time 2 minutes after the onset of shock and 39 minutes after the UFH bolus, was 150 seconds. Bailout aspiration thrombectomy, performed 8 minutes after the onset of shock, retrieved a large red thrombus (Fig. 2D), establishing that substantial thrombus was present at the time of vessel closure. A further 8000 IU of UFH and eptifibatide (180 μg/kg bolus [15, 300 μg], followed by an infusion of 2 μg/kg/min for 12 hours) were administered, and TIMI grade 2 flow was restored. ACT measured at the end of the procedure was 325 seconds. Because the event occurred before the patient left the catheterization table, it falls outside the time frame of Academic Research Consortium-2 (ARC-2) stent thrombosis and is classified as intraprocedural stent thrombosis, with angiographic confirmation of occlusion within the stented segment and retrieval of thrombus [2, 4]. In view of the thrombus burden, recent shock, and newly restored antegrade flow, further mechanical manipulation was de-

ferred, and the patient left the catheterization laboratory with an unoptimized angiographic result.

Norepinephrine was discontinued after 12 hours, and the IABP was maintained for 4 days. UFH was continued as an infusion of 12 IU/kg/h for 4 days, titrated to an activated partial thromboplastin time of 1.5 to 2.0 times control, according to our local practice during prolonged IABP support and, additionally, because of the thrombus burden and the unoptimized stent result pending staged reintervention. We acknowledge that prolonged UFH after PCI is not standard practice.

On day 4, staged coronary angiography with IVUS guidance was performed. IVUS was interpreted by the operator as showing underexpansion of the mid-LAD stented segment and undersizing of the proximal stents, with reference vessel diameters of approximately 3.5 to 3.75 mm in the mid-LAD and 4.0 to 4.25 mm in the proximal LAD. The IVUS recording could not be retrieved because of a storage failure of the imaging console, and no quantitative measurements were saved; minimum stent area is therefore not available. High-pressure dilatation with a 3.5-mm noncompliant balloon at 18 atm produced a distinct fluoroscopic waist (Fig. 3A), consistent with mechanical resistance to expansion but not permitting quantitative assessment. Subsequent dilatation of the proximal-to-mid LAD with a 4.0-mm noncompliant balloon at 18 atm resulted in a widely patent LAD with TIMI grade 3 flow (Fig. 3B). As a procedural complication of the staged intervention, the second diagonal branch occluded after aggressive postdilatation. The patient remained free of chest pain, the electrocardiogram showed established Q waves in V1 to V3 without new dynamic ST-segment changes, and high-sensitivity troponin T (99th percentile upper reference limit, 14 ng/L) was 230 ng/L 14 hours after the procedure compared with 210 ng/L before it, a relative increase below the 20% threshold required for procedure-related myocardial infarction when baseline values are elevated [5]. After the index procedure, high-sensitivity troponin T measured at 20 hours was 897 ng/L.

The patient was discharged on day 6 on aspirin 81 mg and prasugrel 10 mg daily, which were continued at 30-day follow-up. At that visit, he was free of angina in functional class I. Echocardiography showed mild anterior hypokinesia and a left ventricular ejection fraction of 45%. He declined intervention for the residual distal left circumflex lesion.



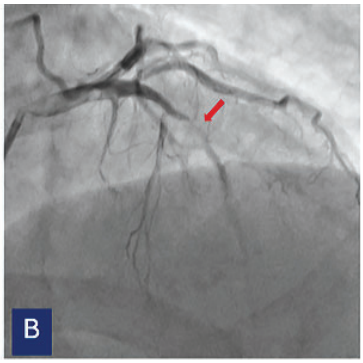


Fig. 1. Presentation. (**A**) Twelve-lead electrocardiogram showing ST-segment elevation in leads V1 to V5, I, and aVL with reciprocal inferior ST depression. (**B**) Baseline coronary angiography demonstrating a subtotal mid-LAD culprit lesion with TIMI grade 1 flow and severe distal left circumflex stenosis. LAD = left anterior descending artery

Table 1. Chronology of key events. Times are as recorded in the procedural log; intervals are calculated from the initial UFH bolus

Time	Minutes after UFH bolus	Event
20:30	–	Aspirin 325 mg and prasugrel 60 mg (uncrushed)
20:50	0	UFH 8000 IU intra-arterial bolus (≈94 IU/kg); ACT not measured
21:15	25	Predilatation; 3.0 × 26-mm ZES in mid-LAD at 12 atm; no postdilatation; TIMI 3 flow
21:16	26	New haziness within stent and near proximal edge; linear radiolucency interpreted as edge dissection
21:21	31	3.5 × 8-mm ZES overlapping proximally at 12 atm; haziness persists and extends
21:25	35	3.5 × 12-mm ZES in proximal LAD at 12 atm; abrupt occlusion, TIMI 0 flow
21:27	37	Cardiogenic shock (80/60 mm Hg, 110 beats/min); IABP and norepinephrine started
21:29	39	First ACT: 150 s
21:35	45	Aspiration thrombectomy (large red thrombus); UFH 8000 IU and eptifibatide bolus followed by 12-h infusion; TIMI 2 flow restored
22:20	90	End of procedure; repeat ACT 325 s
Days 0–4	–	UFH infusion (12 IU/kg/h; aPTT 1.5–2.0 × control) and IABP support; norepinephrine for 12 h
Day 4	–	IVUS-guided staged PCI; balloon waist on 3.5-mm NC balloon at 18 atm; 4.0-mm NC balloon at 18 atm in proximal-to-mid LAD; TIMI 3 flow; second diagonal branch occlusion
Day 6	–	Discharge on aspirin 81 mg and prasugrel 10 mg daily
Day 30	–	Free of angina; LVEF 45%

ACT = activated clotting time; aPTT = activated partial thromboplastin time; IABP = intra-aortic balloon pump; IVUS = intravascular ultrasound; LAD = left anterior descending artery; LVEF = left ventricular ejection fraction; NC = noncompliant; UFH = unfractionated heparin; ZES = zotarolimus-eluting stent.

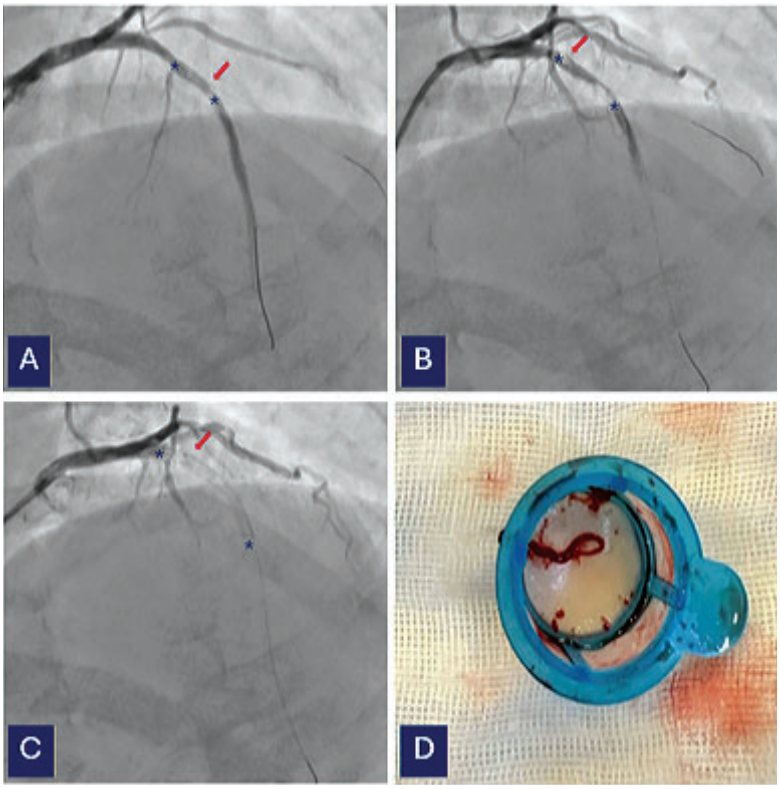


Fig. 2. Progressive angiographic haziness and intraprocedural stent thrombosis. (**A**) One minute after implantation of the first 3.0 × 26-mm zotarolimus-eluting stent (ZES), subtle haziness is visible within the stented segment and near the proximal edge (arrows), with TIMI grade 3 flow. (**B**) After implantation of the overlapping 3.5 × 8-mm proximal ZES, the angiographic haziness persists and extends (arrow). (**C**) Abrupt LAD closure with TIMI grade 0 flow immediately after implantation of the third 3.5 × 12-mm proximal ZES. (**D**) Large red thrombus retrieved by bailout aspiration thrombectomy. Panels A to C are shown in the same projection and magnification. TIMI = Thrombolysis In Myocardial Infarction. Asterisks indicate the stent edges

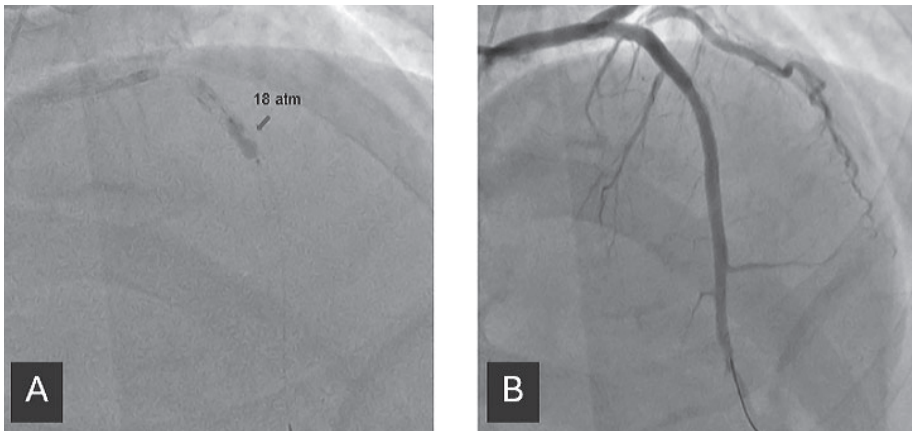


Fig. 3. Staged IVUS-guided reintervention on day 4. (**A**) Distinct fluoroscopic waist (arrow) on a 3.5-mm noncompliant balloon inflated at 18 atm within the stented LAD segment, consistent with mechanical resistance to expansion. (**B**) Final angiographic result after dilatation of the proximal-to-mid LAD with a 4.0-mm noncompliant balloon at 18 atm, with TIMI grade 3 flow

Discussion

The central feature of this case is not the occurrence of intraprocedural stent thrombosis itself, which is well recognized, but the diagnostic uncertainty surrounding the progressive haziness that preceded abrupt closure. Angiographic haziness is a descriptive sign rather than a diagnosis. In the setting of primary PCI, it may represent thrombus, dissection, tissue protrusion, intramural hematoma, or an abnormality related to incomplete stent expansion [1, 3]. When the appearance persists or progresses despite a treatment directed at one presumed mechanism, the differential diagnosis should be reconsidered before further irreversible intervention.

There is direct evidence that thrombosis was present at the time of abrupt vessel closure: aspiration retrieved a large red thrombus. However, the ACT of 150 seconds was measured 39 minutes after the UFH bolus and only after cardiogenic shock had developed. A value this low after a bolus of approximately 94 IU/kg is unexpected and could reflect heparin resistance, incomplete intravascular delivery of the bolus, or consumption during active thrombosis; these possibilities cannot be distinguished retrospectively. The measurement therefore indicates an inadequate

anticoagulant effect at that time but cannot establish the anticoagulant state 13 minutes earlier, when haziness first appeared, or prove that insufficient UFH effect was the principal precipitating mechanism. UFH response during PCI is variable, and contemporary ACS guidance supports monitoring of anticoagulant effect during PCI rather than assuming that a weight-based dose guarantees a therapeutic response [6, 7]. Oral P2Y12 inhibition may also have been incomplete: the uncrushed prasugrel loading dose was given only 45 minutes before stent implantation, platelet inhibition after oral loading is delayed in STEMI, and opioids such as fentanyl can further delay absorption [7, 8]. No platelet-function testing was performed, so this contribution remains uncertain.

A mechanical substrate was suggested at the staged procedure. Staged IVUS was interpreted as showing stent underexpansion, and the visible waist during high-pressure noncompliant balloon dilatation supports the presence of mechanical resistance. All three stents had been deployed at 12 atm without noncompliant postdilatation, which increases the likelihood of underexpansion. Stent underexpansion is a recognized substrate for stent thrombosis and an important target for intracoronary imaging-guided optimization [1, 9]. Nevertheless, IVUS was performed four days after the acute event. It cannot establish whether the initial haziness represented underexpansion, thrombus protrusion, dissection, malapposition, or a combination of findings. In addition, the IVUS recording and quantitative measurements were lost, so the mechanical findings rest on the operator's interpretation together with the fluoroscopic balloon waist rather than on a complete reconstruction of the acute mechanism.

Stent sizing warrants separate consideration. At the index procedure, the reference diameter was visually estimated at 3.0 to 3.5 mm and 3.0-mm, and 3.5-mm stents were implanted, whereas IVUS on day 4 indicated reference diameters of approximately 3.5 to 3.75 mm in the mid-LAD and 4.0 to 4.25 mm in the proximal LAD. In primary PCI, reduced antegrade flow, thrombus, and adrenergic vasoconstriction commonly lead to underestimation of true vessel size, and reference dimensions distal to the culprit lesion may increase substantially within days [10]. In this patient, sizing was performed by visual estimation alone while flow was TIMI grade 1. Undersizing may therefore have contributed through malapposition as well as through underexpansion at the lesion itself; the available data do not allow these mechanisms to be separated.

In retrospect, when haziness persisted after the second stent, several options available in the laboratory could have tested competing mechanisms before a third stent was implanted: point-of-care ACT measurement, aspiration thrombectomy or a glycoprotein IIb/IIIa inhibitor if thrombus was suspected, and intracoronary imaging to distinguish dissection, tissue protrusion, and thrombus, although the last was limited by reimbursement constraints. None of these was used before the third stent. We state this not as a judgement made with hindsight certainty, but because it is the most transferable lesson of the case: an unexpected angiographic finding that does not respond to a first mechanical correction should prompt a pause to test alternative explanations rather than a second mechanical correction based on the same assumption.

The most defensible interpretation is therefore multifactorial. A thrombotic event was proven at bailout, an inadequate anticoagulant effect was documented after deterioration, and a mechanical substrate of stent undersizing and probable underexpansion was suggested at staged reassessment. What cannot be determined is the relative contribution or temporal order of these abnormalities at the first appearance of angiographic haziness. This distinction is clinically important because retrospective knowledge can make a complex intraprocedural situation appear more certain than it was in real time.

Following internal review of the case, our laboratory adopted three local safeguards. ACT is now checked after the initial UFH bolus and repeated when an unexpected angiographic change occurs. Persistent or progressive haziness prompts an explicit reassessment of thrombotic and mechanical alternatives and intracoronary imaging when feasible before additional stent implantation. Finally, the operator and team are encouraged to state the competing diagnosis before escalating an irreversible mechanical strategy. These measures represent a local quality-improvement response to this case rather than evidence of a universally validated algorithm.

The principal limitations of this report are the absence of intracoronary imaging during the index procedure, the delayed timing of the first ACT measurement, the absence of platelet-function testing, visual rather than quantitative vessel sizing at the index procedure, and the loss of the staged IVUS recording with no quantitative measurements. Accordingly, the report cannot establish a single causal pathway. Its educational value lies instead in sequential angiographic evolution and in demonstrating why progressive post-implantation haziness should be approached as a diagnostic problem with potentially interacting thrombotic and mechanical contributors.

Conclusions

Progressive angiographic haziness during primary PCI may precede intraprocedural stent thrombosis but

does not identify a single mechanism. In this patient, a large, aspirated thrombus, a low ACT measured after hemodynamic deterioration, and later findings suggesting undersized and underexpanded stents provided complementary but temporally separated clues. When haziness persists or worsens despite attempted correction, reassessment of the differential diagnosis, verification of anticoagulant effect, and intracoronary imaging when feasible may help avoid further intervention based on an untested assumption.

Declarations

Ethics approval. The requirement for formal ethics review and approval for this single-patient case report was waived by the Human Research Ethics Committee of Bhumibol Adulyadej Hospital, Royal Thai Air Force. The report was prepared in accordance with the Declaration of Helsinki.

Consent for publication. Written informed consent was obtained from the patient for publication of the case details and accompanying images.

Competing interests. The authors declare no competing interests.

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Data availability. All data relevant to the case are contained within the manuscript. Original clinical images are retained by the authors subject to institutional and patient-privacy requirements.

AI disclosure. During preparation of this manuscript, the authors used generative AI tools (ChatGPT, OpenAI; and Claude, Anthropic) for language editing and structural refinement. All clinical facts, interpretations, and cited references were independently reviewed and verified by the authors, who take full responsibility for the final manuscript. No AI tools were used to generate or modify clinical figures.

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