


Acute coronary syndrome due to native coronary occlusion proximal to a patent bypass graft: a case report

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Received 10 July 2020; first decision 18 August 2020; accepted 2 December 2020

Background

Primary percutaneous coronary intervention (PCI) for acute coronary syndrome has significantly contributed to improvements in overall outcomes. However, clinical challenges exist when performing urgent PCI for patients with a history of coronary artery bypass grafting (CABG).

Case summary

An 83-year-old man with a history of CABG presented with an inferior ST-elevation myocardial infarction (STEMI). Emergent coronary angiography showed an occlusion of the right coronary artery that had been previously grafted with the right gastroepiploic artery. Primary PCI for the native coronary artery was performed on the assumption that the bypass graft had been occluded. We were unable to attain angiographic antegrade flow after balloon angioplasty, and intravascular ultrasound revealed a ruptured plaque with a thrombus proximally and a patent bypass graft with complete recanalization distally. These findings suggested that the plaque rupture with resultant thrombus formation proximal to the anastomosis eventually overlay the patent bypass graft. Subsequent stent implantation covering only the culprit site with a residual stenosis proximal to the anastomosis was performed, resulting in good patency of both the native coronary artery and bypass graft for more than 3 years.

Discussion

This is the first documented case of a patient with STEMI due to proximal native coronary artery occlusion with a thrombus overlying a patent bypass graft. Intravascular ultrasound was helpful to recognize the distal patency and guide optimal stent implantation. This case illustrates the complexity of treating a patient with a history of CABG and the importance of a multifaceted approach in such an urgent situation.

Keywords

Acute coronary syndrome • Native coronary artery • Coronary artery bypass graft • Gastroepiploic artery • Intravascular ultrasound • Case report

Learning points

- Even if a bypass graft is a patent, the acute coronary syndrome can occur if a thrombus from a culprit lesion extends beyond the anastomosis, causing total occlusion of both the native coronary artery and graft.
- A multimodal approach including intravascular ultrasound can help to understand the complex coronary anatomy in patients with a history of coronary bypass surgery and determine the strategy in the setting of urgent percutaneous coronary intervention.

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Handling Editor: Francesco Giannini

Peer-reviewers: Ying Xuan Gue and Sameh Shaheen

Compliance Editor: Abdelrahman Ibrahim Abushouk

Supplementary Material Editor: Ayse Djahit

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Introduction

Patients with a history of coronary artery bypass grafting (CABG) pose a clinical challenge when performing primary percutaneous coronary intervention (PCI) for the acute coronary syndrome (ACS). This patient cohort represents a high-risk group with advanced comorbidities resulting in poor outcomes.^{1,2} In addition, limited information regarding patients' history of bypass surgery and preceding native coronary and bypass graft patency often hampers a swift treatment response. Some authors have considered that ACS in the territory of a bypass graft is caused by either abrupt occlusion of a bypass or disease progression distal to the graft.³ We herein describe a patient with a history of CABG who developed ST-elevation myocardial infarction (STEMI) and subsequently underwent successful PCI for the proximal native coronary artery. Intravascular ultrasound (IVUS) was useful to understand the complex coronary anatomy and determine the treatment strategy.

Timeline

22 years prior to admission	Patient undergoes coronary artery bypass grafting
Emergency hospital admission	Patient presents with inferior ST-elevation myocardial infarction (STEMI). Primary percutaneous coronary intervention for the occluded proximal right coronary artery is performed. Contrary to angiographic failure for recanalization after balloon inflation, intravascular ultrasound reveals recanalized distal flow and a patent bypass graft. The findings suggest that the native coronary thrombus has also occluded the previously patent bypass graft, resulting in STEMI. A stent is implanted, covering only the culprit lesion.
5 months after admission	The patient is discharged 2 weeks later. Coronary angiography shows a patent stent and bypass graft.
3 years after admission	Both the native coronary artery and the bypass graft remain patent as confirmed by coronary computed tomography angiography.

Case presentation

An 83-year-old Japanese man with a history of CABG, hypertension, and dyslipidaemia presented to our hospital because of sudden-onset chest pain. The chest pain was vague but accompanied by shortness of breath, which prompted him to visit the emergency department. Twenty-two years prior to presentation, he had undergone CABG involving a saphenous vein graft for the left circumflex artery and a right gastroepiploic artery (RGEA) graft for the right coronary artery

(RCA). Coronary angiography had been performed 10 years before presentation, but the result was unavailable. His medical conditions had been well controlled with aspirin, an angiotensin-converting enzyme inhibitor, and a statin. The physical examination findings were unremarkable with the exception of an irregular cardiac rhythm. Auscultation revealed no additional heart sounds and no pulmonary rales. He was haemodynamically stable with a blood pressure of 164/66 mmHg and a heart rate of 60 beats/min. His qualitative rapid troponin T assay (Roche TROP T Sensitive; Roche, Basel, Switzerland) was positive, and an electrocardiogram showed ST-segment elevation in the inferior leads with reciprocal changes (Figure 1).

Urgent coronary angiography was performed and revealed a total occlusion of the proximal RCA (Figure 2A, Video 1). The left main coronary artery exhibited moderate stenosis, and the mid-portion of the left circumflex artery was chronically occluded with distal reconstitution from collaterals; neither of these conditions seemed related to the presentation. Given the patient's clinical situation and our lack of expertise in probing RGEA grafts, we did not check the graft patency. We assumed instead that the RGEA had been occluded, and we thus proceeded to perform a native RCA intervention. A guidewire was navigated to the distal RCA through the occlusion without any difficulty; however, even after several thrombus aspirations and repeated inflations with a 2.0-mm balloon, the antegrade flow was not attained angiographically (Figure 2B, Video 2). Intravascular ultrasound was performed to elucidate this unexplained no-reflow state without attempting to administer intracoronary agents. Intravascular ultrasound showed that the distal RCA had a well-delineated vessel architecture with dark-coloured lumen, which was suggestive of sufficient blood flow (Figure 3C, Video 3). Intravascular ultrasound also showed that the apparently patent bypass graft was anastomosed to the mid-portion of the RCA and that a massive plaque burden with a ruptured plaque was present proximal to the anastomosis (Figure 3A and B). More forceful dye injection with a guide extension support demonstrated recanalization of the RCA and backward flow to the RGEA (Figure 3, Supplementary material online, Video S1). We concluded that the first assumption had been incorrect and that the plaque rupture on the proximal native RCA had caused the acute occlusion and thrombus formation extending to and overlying the patent RGEA, resulting in transmural STEMI. A 2.5-/38-mm everolimus-eluting stent (Synergy; Boston Scientific, Marlborough, MA, USA) covering the culprit lesion was implanted, and antegrade flow was restored with minimal distal embolization in the posterolateral branch (Figure 4, Supplementary material online, Video S2). On the final angiogram, the RGEA had a large calibre (Figure 4, arrowheads), indicating that the bypass had been supplying dominant blood flow and that the native proximal RCA appeared to have been severely stenosed. The final IVUS confirmed that the stent was fully expanded and that an intended residual stenosis was present immediately proximal to the anastomosis (Figure 4A and B, Supplementary material online, Video S3).

The patient's clinical course after the PCI was uneventful, and he was discharged in 2 weeks. Follow-up angiography 5 months later showed no stent restenosis with competitive flow from the RGEA and dissipation of the distal embolization (Figure 5A and B, Supplementary material online, Videos S4 and S5). Coronary

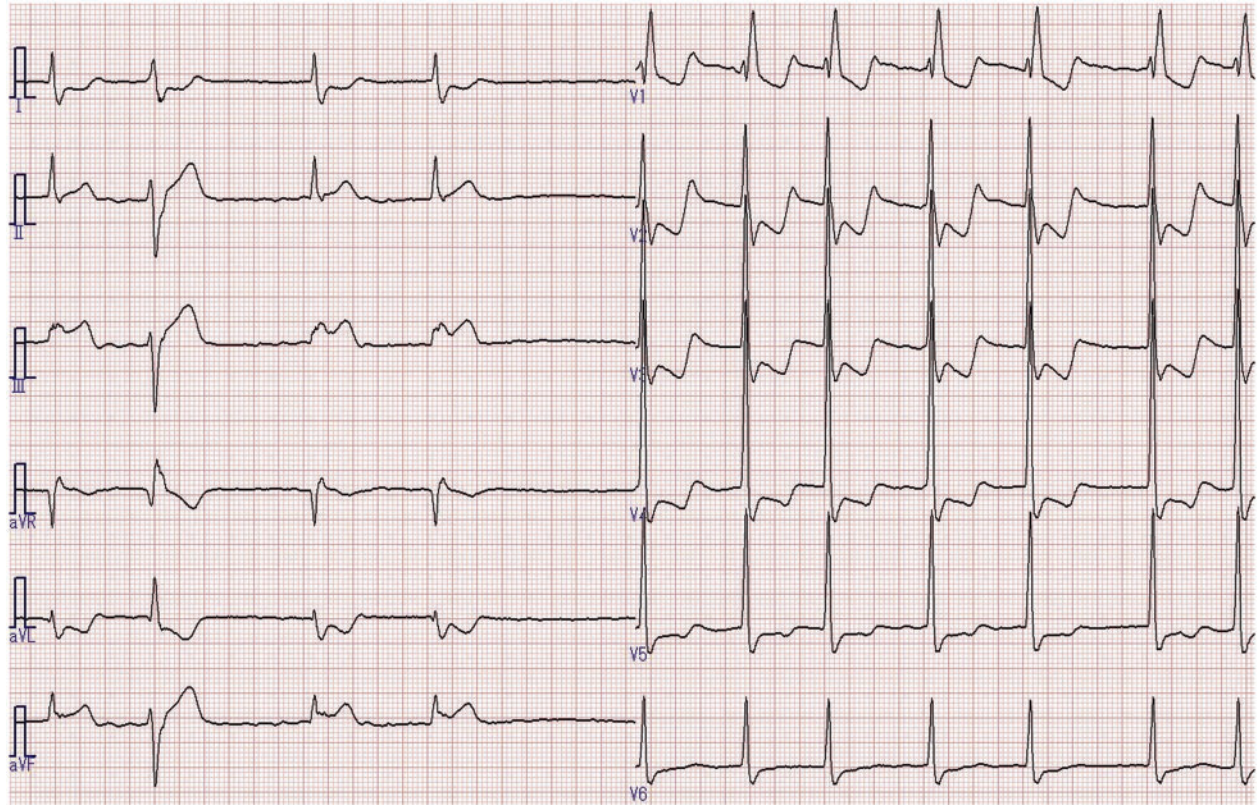


Figure 1 Electrocardiogram at the initial presentation shows ST-segment elevation in the inferior leads. Additionally, the presence of atrial fibrillation and right bundle branch block are suggestive of the severe nature of the disease.

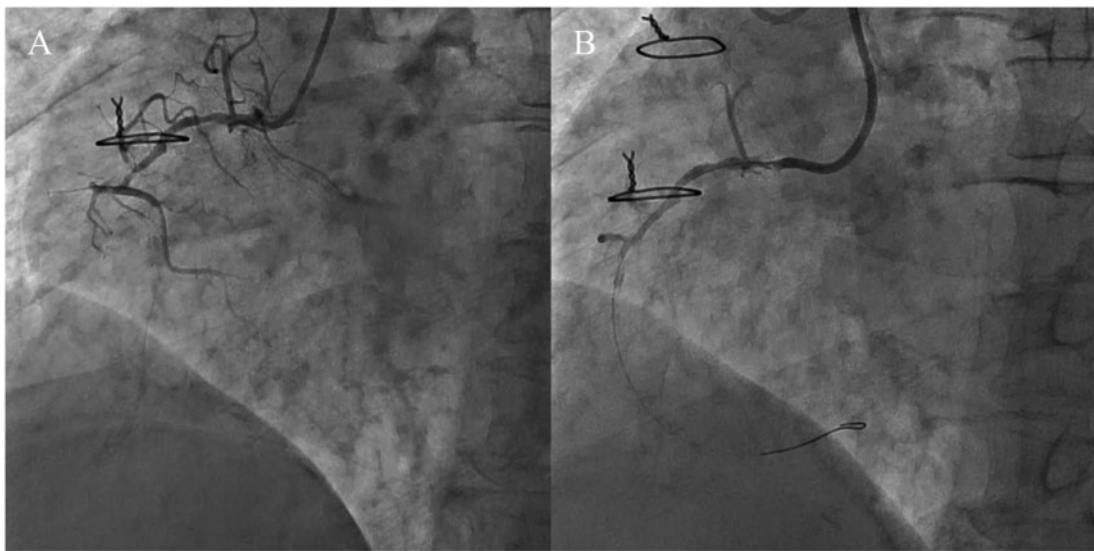


Figure 2 (A) The initial angiogram reveals proximal right coronary artery occlusion. (B) Angiographically non-recanalized right coronary artery after several thrombus aspirations and repeated balloon inflations.

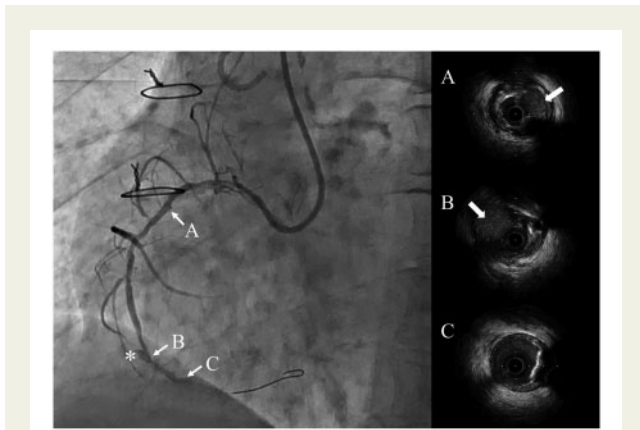


Figure 3 Angiography with more forceful dye injection and a guide extension support shows restored antegrade native right coronary artery flow and a patent bypass graft (asterisk). Corresponding intravascular ultrasound images demonstrate (A) a positively remodelled vessel with a high plaque burden and probable ruptured plaque (arrow) suggestive of the culprit lesion. (B) Patent anastomosis (arrow) corresponding to angiography. (C) Evidence of distal blood flow with intravascular ultrasound showing a dark-coloured lumen and distinctive vessel architecture.

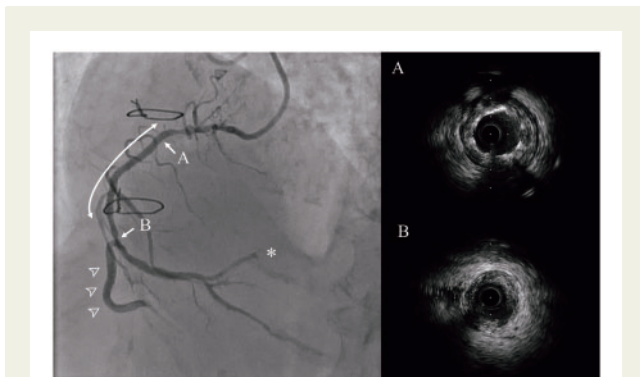


Figure 4 Final angiogram after stent implantation (double-sided curved arrow) and corresponding intravascular ultrasound images. (A) Fully expanded stent covering the culprit lesion. Competitive flow from the right gastroepiploic artery is visible (arrowheads). Distal embolization (asterisk) was treated conservatively. (B) Intravascular ultrasound shows residual stenosis with mild dissection immediately proximal to the bypass graft.

computed tomography angiography 3 years later confirmed long-term patency in both the native coronary stent and the bypass graft (Figure 5C). The patient remained angina-free after discharge, and he was well managed on an outpatient basis without rehospitalization at the time of this writing.

Discussion

Atherosclerotic progression of the native coronary artery leading to chronic total occlusion after CABG is frequently

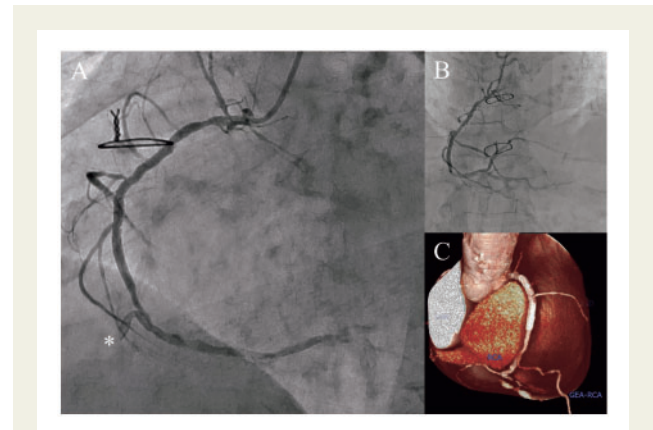


Figure 5 (A) Coronary angiography 5 months after the event shows the patent native right coronary artery and competitive flow from the bypass graft (asterisk). (B) The cranial view proves that the distal embolization observed at the time of the primary percutaneous coronary intervention has resolved. (C) Coronary computed tomography angiography taken 3 years later demonstrates the patent native coronary stent and the bypass graft.

observed with a reported prevalence of >40%.⁴ In contrast, a previous study showed that the rate of occlusion of the RGEA graft was 13% at 3 years.⁵ Acute coronary syndrome in the territory of a bypass graft has been generally attributed to either early graft failure with a subsequent native coronary event or an acute graft occlusion in the setting of chronic total occlusion of the native artery.

Even with a patent bypass graft, ACS can occur if a thrombus that has formed in a culprit lesion distal to the anastomosis extends proximally across the anastomosis, occluding both the native coronary artery and bypass graft. In our case, however, IVUS did not demonstrate such findings in the distal segment. We conclude that the proximal native coronary artery plaque rupture and subsequent thrombus formation had occluded the previously patent bypass graft, leading to STEMI. To the best of our knowledge, this is the first report to describe ACS caused by proximal native coronary occlusion with a thrombus overlying and occluding the previously patent bypass graft.

A possible reason for this rare phenomenon is that the bypass graft was anastomosed onto the mid-portion of the RCA near the culprit. This might have resulted in a silent chronic total occlusion if the bypass had been anastomosed more distally. In such a case, there would have been three options: (i) simply finish the procedure with the restored antegrade flow, (ii) implant a stent covering only the culprit lesion with plaque rupture, or (iii) place a stent covering the entire stenosed lesion and jailing the bypass graft. Given the high plaque burden, we chose the second option because we were concerned about re-occlusion if the first option was selected. We also contemplated that the stent-jailed bypass graft with the completely recanalized antegrade native coronary flow would result in acute graft failure, which could be hazardous if in-stent restenosis occurred at a later time.

Arterial graft conduits have a more dynamic nature than saphenous vein grafts and can restore patency afterward.^{6,7} For our

patient, although the bypass did not seem to be predominantly supplying the distal flow after PCI, we expect that it will recover dominance in the event of restenosis as long as the bypass is patent. Intravascular ultrasound was indispensable not only to comprehend this unusual clinical situation but also to determine the optimal stent size and position to achieve the second of the three options mentioned above. The mid-term patency of both the native and bypass artery indicates that our strategy has worked well so far. As noted above, patients with a history of CABG who develop ACS are a high-risk group with worse clinical outcomes. However, a previous study suggested that outcomes can be improved with contemporary standardized management involving early reperfusion therapy.⁸ The present case provides additional insight into how a culprit lesion should be determined in this specific subset.

In summary, our report suggests that even if a bypass graft is a patent, ACS can occur if a thrombus of a proximal culprit lesion extends beyond the anastomosis, causing total occlusion of both the native coronary artery and the bypass graft. Intravascular ultrasound is useful to understand the complex coronary anatomy when performing primary PCI in patients with a history of CABG.

Lead author biography



Dr Kazunari Kitazono graduated from Kumamoto University School of Medicine in 1998. He completed residency training at Kagoshima Tokushukai Hospital and a cardiology fellowship at Tokyo Women's Medical University Hospital. He has been working as a cardiologist at Tanegashima Medical Center and Tenyokai Chuo Hospital since 2013.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Acknowledgements

We thank Angela Morben, DVM, ELS, from Edanz Group (<https://en-author-services.edanzgroup.com/ac>), for editing a draft of this manuscript.

Slide sets: A fully edited slide set detailing these cases and suitable for local presentation is available online as [Supplementary data](#).

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: None declared.

Funding: None declared.

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