Late Spontaneous Internal Thoracic Artery Graft Dissection after Coronary Bypass Grafting: a case report

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Abstract

Background: Internal thoracic artery grafts are commonly used for coronary artery bypass grafting, with dissection to the graft being a rare occurrence. Herein, we describe a case of spontaneous internal thoracic artery graft dissection occurring 11 years after grafting, with no
clear precipitating incidence.

**Case summary:** The patient was a 61-year-old man who presented with a 3-month history of chest pain and dyspnea. Dissection of the left internal thoracic artery graft was observed on angiography, with a thrombolysis in myocardial infarction (TIMI) grade 2 blood flow. Intravascular ultrasound confirmed an intimal tear in the proximal graft, with an intramural hematoma. In the absence of atherosclerotic changes, the dissection was treated directly using multiple drug-eluting stents to prevent further extension of the intramural hematoma proximally into the subclavian artery and distally to the anastomosis site. Post-procedural angiography revealed an enlarged true lumen of the left internal thoracic artery, shrinking of the intramural hematoma, and improvement in blood flow to a TIMI grade 3. Chest symptoms resolved immediately after the procedure, with the patient remaining asymptomatic over the 6-month period following the procedure.

**Discussion:** Dissection of the internal thoracic artery graft can occur spontaneously long after the initial grafting. Intravascular ultrasound is useful for diagnosis. Ensuring adequate coverage of the edges of the dissection with stenting could prevent further extension of the intramural hematoma.
Learning Points

1. Chronic spontaneous dissection of an internal thoracic artery is a rare occurrence.

2. Intravascular ultrasound is useful to differentiate the dissection, hematoma, and true lumen of the vessel and to guide treatment.

3. Adequate coverage of the edges of the dissection during stenting was effective to prevent progression of the dissection and resolution of symptoms.
<table>
<thead>
<tr>
<th>Time</th>
<th>Events</th>
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<tbody>
<tr>
<td>28 September 2010</td>
<td>Coronary artery bypass grafting consisted of anastomosis of the left internal thoracic</td>
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<td>artery (LITA) to the left anterior descending artery and an I-shaped graft, composed of</td>
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<td>the right internal thoracic artery and the radial artery, anastomosed to the diagonal</td>
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<td>branch, posterolateral, and posterior descending arteries, sequentially.</td>
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<td>16 September 2017</td>
<td>Good patency of both bypass grafts in computed tomographic angiography (CTA).</td>
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<td>16 March 2021</td>
<td>Chest pain and repeat CTA revealed diffuse stenosis in the proximal half of the LITA.</td>
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<td>Angiography revealed an extensive dissection of the LITA, with impaired blood flow,</td>
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<td>assessed as a thrombolysis in the myocardial infarction (TIMI) grade 2.</td>
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<td>2 April 2021</td>
<td>Percutaneous coronary intervention of the LITA with multiple drug-eluting stents were</td>
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<td>performed.</td>
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<td>7 September 2021</td>
<td>No recurrence of chest pain.</td>
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Introduction

The internal thoracic artery (ITA) is resistant to accelerated atherosclerotic changes due to endothelial vasodilators like nitrous oxide. However, the ITA can be ruptured by physical force due to the scant presence of smooth muscle cells in the thin-walled media. Although dissection of an ITA graft used for cardiac bypass grafting (CABG) is rare, it may occur due to iatrogenic injury or spontaneously. Spontaneous dissection can occur with a sudden increase in blood pressure during intense exercise, as well as a complication of collagen diseases, such as Marfan’s or Ehlers Danlos syndrome, or increased secretions of stress catecholamines. Herein, we present a 61-year-old man who developed a spontaneous ITA dissection 11 years after CABG, with successful treatment using percutaneous intervention with multiple drug-eluting stents (DES).

Case report

The patient provided informed consent for publication of this case. The patient was a 61-year-old man who had undergone CABG 11 years prior, in 2010. CABG consisted of anastomosis of the left internal thoracic artery (LITA) to the left anterior descending artery (LAD) and an I-shaped graft, composed of the right internal thoracic artery and the radial artery, anastomosed to the diagonal branch, posterolateral, and posterior descending arteries, sequentially. Health comorbidities included diabetes mellitus (DM), hypertension, and ischemic cardiomyopathy, with a left ventricular ejection fraction of
In 2017, he underwent computed tomographic angiography (CTA) to verify graft patency before his referral to our hospital upon his relocation (Figure 1A). In 2021, the patient presented with a 3-month history of chest pain and dyspnea, suggesting chronic coronary syndrome because his laboratory tests showed no elevation of myocardial enzyme. An electrocardiogram identified new ischemic changes with T wave inversion in V3-V6. On admission, physical examination revealed no abnormalities. As repeat CTA revealed diffuse stenosis in the proximal half of the LITA (Figure 1B), we performed coronary angiography. His native coronary angiogram showed severe narrowing in the left main artery, severe proximal stenosis in both LAD and a dominant circumflex, and a non-dominant right coronary artery with a proximal chronic total occlusion. Angiography also revealed an extensive dissection of the LITA with impaired blood flow, assessed as a thrombolysis in the myocardial infarction (TIMI) grade 2 (Figure 2). On intravascular ultrasound (IVUS, OptiCross™ HD, Boston Scientific, Natick, MA, USA), an intimal tear was observed in the proximal LITA, with an associated intramural hematoma extending proximally to just before the origin of the LITA and distally to the mid-portion of the LITA. The LITA origin and bypass anastomosis site were intact (see supplementary video). Although the minimum lumen area was 9.3 mm² at the distal end of the LITA, the area of the lumen at the site of dissection was reduced to 3.9 mm². As there was no evidence of atherosclerotic changes, the dissection was directly stented using multiple DES (Synergy-DES of 4.0 × 24 mm, 3.5 × 48 mm and 3.0 × 48 mm; Boston Scientific,
Natick, MA, USA) from distal to proximal to prevent extension of the intramural hematoma proximally into the subclavian artery and distally into the site of anastomosis. On IVUS analysis, there was no malapposition of the implanted DES. Post-procedural angiography revealed an enlarged true lumen of the LITA, shrinkage of the intramural hematoma, and improved blood flow distally (TIMI grade 3 flow; Figure 4). The patient’s symptoms resolved and the patient was discharged on postoperative day 2 with prescriptions for 75 mg/day clopidogrel and 100 mg/day aspirin. There was no symptom recurrence over the following six months.

Discussion

This report presents several unique features. First, the spontaneous dissection of the LITA graft occurred 11 years after CABG. ITA dissections that occur at a short latency after CABG or selective ITA angiography are suggestive of an iatrogenic injury, particularly when involving the proximal ITA. Only four previous cases of chronic phase ITA dissection (occurring 18 months and 2, 3, and 8 years after CABG) have been reported. In a case reported by Suresh et al., ITA dissection occurred eight years after CABG as the patient lifted a heavy bookcase; in the other three reports, dissection cause was not specified. Here, the reason for ITA dissection was unclear; he had no history of connective tissue diseases or fibromuscular dysplasia, and iatrogenic insult was unlikely as there was no intervention since the CABG. The chronicity of the dissection of the ITA, 11 years after CABG, is the
longest duration reported to date. Second, our case demonstrates the usefulness of IVUS for diagnosis and treatment. Although optical coherence tomography may be superior to IVUS for identification of disrupted flaps, it might not be effective to visualize the full extent of the damage to the vessel wall in the presence of a large intimal hematoma. Use of contrast media or low-molecular-weight dextran to remove residual blood could increase the risk of dissection due to the pressure load on the vessel wall. By comparison, IVUS clearly shows the whole vessel wall, differentiating the dissection, hematoma, and true lumen. IVUS could also help with decisions on stent positioning, early after assessment of the site of lesion. As such, IVUS is useful for both assessment and treatment. Third, the optimal treatment for ITA graft dissection remains unclear. Akita et al. indicated that observation is an option for asymptomatic patients with good distal blood flow. Absorbable stents or bioresorbable vascular scaffold implantation may be options for temporary scaffolds, because most patients with spontaneous coronary artery dissection have little or no atherosclerosis and may heal spontaneously. In our case, there were several reasons for performing revascularization for the dissected ITA rather than the native coronary artery. Revascularization for the native coronary artery was challenging because he had triple-vessel coronary disease including the left main artery, one vessel occlusion with DM, and severely impaired left ventricular function. In contrast, revascularization for LITA was easier than for the native coronary artery because the dissected ITA had no atherosclerosis. Moreover a report by
Michael et al. demonstrated coronary revascularization with CABG leads to lower all-cause mortality than other interventions in patients with multivessel disease and DM. Gruberg et al. reported that revascularization of the ITA graft can be performed safely with low target lesion revascularization rates in long-term follow-up. These reports indicate that treatment strategies should be decided on a case-by-case basis, depending on symptoms, location and length of the hematoma, degree of compromise of the vessel lumen, and extent of blood flow restriction distal to the dissection. As shown here, stenting multiple DES from distal to proximal was our strategy to avoiding the so-called “tooth-pasting” effect and propagation of the dissection. The risk for acute and late stent thrombosis or in-stent restenosis with the use of longer stents needs to be considered. An individualized approach based on ischemic vs. bleeding risk assessment is warranted. Particularly in patients with longer or complex treated segments, ischemic risk prevails over bleeding risk. Late stent malposition is also concerning for an increased very late stent thrombosis risk, following the resorption and healing of intramural hematoma. In this case, a follow-up CTA before cessation of dual antiplatelet treatment may be useful, and after dual antiplatelet treatment for 12 months, termination of this case’s second antiplatelet will be based on a follow-up CTA of stent patency.

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.

**Availability of data**

The authors confirm that the data supporting the findings of this case report are available within the article and its supplementary materials.

**References**


Figure Legends

Figure 1. (A) CTA assessed at 7 years after CABG, showing good graft patency without any abnormal findings. (B) CTA assessed at 11 years after CABG, showing diffuse stenosis in the proximal half of the LITA (yellow line).

CABG: coronary artery bypass grafting, CTA; Computed tomographic angiography, LITA; left
Figure 2. (A) Angiography, showing an extensive dissection of the LITA and associated impairment in blood flow through the graft. (B) Tip injection by microcatheter from the middle portion of the LITA confirmed a comprised blood flow in the LAD. The white dotted line identifies the native LAD and the yellow arrow shows the anastomosis site of the LITA with the LAD. (a) origin, (b) long smooth narrowing of the vessel leading to the dissection, (c) ulcer-like appearance, and (d) healthy area in the middle portion of the LITA.

LAD; left anterior descending artery, LITA; left internal thoracic artery

Figure 3. Longitudinal IVUS images showing (a) absence of dissection at the origin of the LITA and (b) intramural hematoma (*), confirmed by the white-black-white appearance of the intimal-medial membrane (yellow arrow). (c) The intramural hematoma (*), extending proximally and distally from the site of dissection (white arrows). (d) Intact middle portion of the LITA.

LAD, left anterior descending artery; LITA, left internal thoracic artery; IVUS, intravascular ultrasonography.
Figure 4. Angiography images after implantation of multiple stents, showing (A) the area of the repaired dissection with no evidence of blood flow restriction in the LITA and (B) recovery of the LAD patency. The white dotted line shows the native LAD and the yellow arrows shows the anastomosis site of the LITA with the LAD.

LAD; the left anterior descending artery. LITA: left internal thoracic artery

Video. IVUS imaging of the LITA from its origin to the bypass the anastomosis site.

LITA, left internal thoracic artery; IVUS, intravascular ultrasonography.