A 50-year-old man presented to his local emergency department with acute-onset retrosternal chest pain associated with nausea. Five years previously, he had undergone primary percutaneous coronary intervention for the management of ST-segment myocardial infarction and received a 3.0×18 MultiLink Vision bare metal stent to an occluded proximal circumflex. On arrival in the emergency department, his pain was relieved with sublingual nitrate and did not recur. His ECG was normal, but his high-sensitivity troponin was elevated at 12 hours (237 ng/L), in keeping with an acute coronary syndrome. Routine hematologic testing also revealed a previously undiagnosed profound microcytic, hypochromic anemia (hemoglobin, 88 g/L; mean cell volume, 57 fl; mean cell hemoglobin, 16 pg), and on further questioning, the patient admitted to a recent change in bowel habits.

The patient underwent diagnostic angiography via the right radial artery that demonstrated a filling defect within the stent at the bifurcation between the proximal circumflex and a small obtuse marginal branch. There was an associated 50% to 60% stenosis with Thrombolysis in Myocardial Infarction grade 3 distal flow (Figures 1 and 2). The other coronary arteries had minor nonobstructive disease. Because of the profound and unexplained anemia, percutaneous coronary intervention, or intravascular imaging was not performed.

The patient’s anemia was subsequently found to be due to severe iron deficiency, and a colonoscopy revealed a fungating cecal adenocarcinoma. Further investigations revealed no evidence of local or distal metastases. The patient underwent a right hemicolectomy and made an uneventful recovery.

Six weeks after surgery, the patient returned for repeat coronary angiography. The filling defect within the proximal circumflex persisted, but now had a new double lumen appearance (Figure 3). Optical coherence tomography was undertaken and demonstrated extensive intrastent dissection with clear separation of the neointima away from the endothelial aspect of the stent (Figures 4 and 5). The entry and exit points of the dissection flap were demonstrable (Figure 5), and organized thrombus was visible immediately distal to the distal end of the dissection (Figure 6). A diagnosis of acute coronary syndrome secondary to intrastent dissection was made. Because the patient had been stable since suffering...
acute coronary syndrome, there was no flow-limiting stenosis, and the optical coherence tomography findings were consistent with a healing dissection, it was decided not to perform percutaneous coronary intervention. The patient was managed medically with dual antiplatelet therapy, statin therapy, and an angiotensin-converting enzyme inhibitor. He has remained well with no unplanned hospital admissions.

Spontaneous coronary dissection is a rare but well-described phenomenon that can be attributed to different etiologic factors such as atherosclerosis, collagen disorders, and pregnancy.\(^1\) Spontaneous dissection occurring within a previously stented segment of coronary disease, however, is exceptionally rare. The phenomenon has been documented during autopsy studies\(^2\) but has been reported to have occurred in a survivor on only a single occasion, with the diagnosis suggested on coronary arteriography.\(^3\) Our case, however, involving optical coherence tomography, clearly demonstrates a dissection plane within the stented segment, with clear entry and exit points and organized thrombus in the false lumen.

The pathophysiology of intrastent dissection is speculative but likely involves rupture of new plaque within the stent, with propagation of the dissection plane along the interface between the stent and neointima. Given the length of time since the stent was inserted and the fact that the dissection

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**Figure 3.** Repeat angiography at 6 weeks shows persistence of the filling defect with new double-lumen appearance (arrow).

**Figure 4.** Optical coherence tomography demonstrating clear separation of the neointima from the stent struts within the proximal segment of the stent (solid arrow). Long-axis view demonstrates the dissection plane (dotted arrow). Asterisk indicates the branch vessel.

**Figure 5.** Within the midportion of the stent, the dissection plane persists (solid arrow). On the long axis, the entry and exit points of the dissection are visible (dotted arrows).

**Figure 6.** Organized thrombus within the distal portion of the dissection plane separating the neointima from the stent struts (solid arrow). The dissection starts and ends within the stented segment of artery (dotted arrow).
plane originated and resolved within the stent, propagation of an original stent edge dissection during initial percutaneous coronary intervention is highly unlikely.

Late stent thrombosis is often attributed to contact between blood and thrombogenic stent struts that have not developed neointima after cessation or interruption of antiplatelet therapy. Our case highlights another possible cause of late stent thrombosis and the role of optical coherence tomography in such cases. It can be argued that all thrombotic complications involving implanted coronary stents should undergo mandatory intravascular imaging as intrastent dissection may be an underrecognized phenomenon.

Disclosures
None.

References
Spontaneous Intrastent Dissection: Late Neointimal Separation Within a Bare Metal Stent Causing Acute Coronary Syndrome
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