Myocardial bridging (MB) is a term that describes an anatomic variant where the coronary artery runs underneath the myocardium for varying lengths before returning to the epicardium. MB could be found in any of the epicardial arteries but predominantly (over three-fourths) involves the left anterior descending (LAD) artery. From an anatomic perspective, MB has traditionally been considered a benign clinical condition. However, recent findings suggesting a relationship between MB and adverse cardiovascular outcomes have led to a reappraisal of the clinical relevance of MB. Considering that MB may physically compress the coronary artery during systole, treating a lesion in the MB-affected location during percutaneous coronary intervention remains clinically challenging. With the use of intravascular imaging, cardiologists can now detect the severity of MB and evaluate the spatial relationship between lesions and MB. Although atherosclerosis is usually present in an LAD segment proximal to the MB, intimal thickening occurs in tunneled segment in a few patients. Here we report a case of MB-associated acute coronary syndrome (ACS) induced by plaque disruption at an LAD-MB segment.

Case
A 35-year-old man who was a smoker presented with paroxysmal retrosternal chest pain that had persisted for 2 weeks. This patient was diagnosed with ACS based on the abnormal findings of an electrocardiogram (Q waves in leads I and aVL and T-wave inversion in leads V1-V5, Supplemental Fig. S1) and elevated troponin I level (0.25 ng/mL). Trans-thoracic echocardiogram showed decreased contraction activity in septal and apical segments of the left ventricular anterior wall with a floating mural thrombus (10 × 4 mm) in the apical segment of the anterior wall of the left ventricle. Coronary angiography was performed, revealing a total occlusion at the middle segment of the LAD (Fig. 1A). We selected a 7F EBU 3.5 guiding catheter for the left coronary artery and a Sion Blue as the working wire. After passing the culprit lesion, we used a 2.0 × 15 mm Tazuna balloon for predilation at 8 atm. A repeated angiogram showed restored blood flow in the coronary artery.

ABSTRACT
This report describes a case of a 35-year-old man who presented with acute coronary syndrome. An angiogram and intravascular ultrasound revealed atherosclerotic stenosis in the myocardial bridging segment of the mid-left anterior descending artery. The culprit lesion was treated using a drug-coated balloon, and no residual stenosis was observed, which was later confirmed by intravascular ultrasound and optical coherence tomography at a 1-year coronary angiographic follow-up. This case provides evidence that drug-coated balloon could be a potential treatment strategy for atherosclerosis located in the myocardial bridging segment and suggests advantages of the “leave nothing behind” strategy in such clinical scenarios.

Case Report
Drug-Coated Balloon Treatment for ACS Induced by Myocardial Bridging: An Intravascular Ultrasound-Guided PCI
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Ethics Statement: The present research has adhered to the relevant ethical guidelines.

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See page 375 for disclosure information.
LAD with severe systolic compression (suggesting the presence of MB) at the middle segment (Fig. 1B). Intravascular ultrasound (IVUS) identiﬁed an MB with a length of 36.12 mm (Fig. 1G), maximal systolic arterial compression as 31.5% (Fig. 1F), and maximal halo thickness as 1.87 mm (Fig. 1E) in the middle segment of the LAD. Atherosclerosis in the MB segment was detected with a maximal plaque burden of 60% (Fig. 1D). Considering that the vessel diameter at the culprit lesion located in the MB segment was approximately 1.5-2.0 mm, we treated the culprit lesion with the use of a 2.0 × 20 mm Sequent Please drug-coated balloon (DCB) for dilation for 60 seconds at lesion at 10 atm. Thrombolysis in myocardial infarction flow grade 3 was observed after dilation. The patient was discharged with dual antiplatelet therapy (ie, 

**Novel Teaching Points**

- Given the physical compression of the coronary artery during systole in patients with myocardial bridging (MB), interventional treatment of the MB-associated lesion remains clinically challenging.
- This case report presented the usefulness of intravascular imaging on detecting and assessing the severity of MB and the spatial relationship between the lesion and MB during percutaneous coronary intervention.
- Drug-coated balloon angioplasty may avoid coronary stent–associated complications, especially when the lesion is located at the tunneled segment of MB.

Figure 1. Myocardial bridging (MB) and culprit lesions in acute coronary syndrome. (A) Angiographic appearance of culprit lesions in the mid-left anterior descending (LAD) artery (white arrowhead). (B) Angiographic appearance of opened coronary artery and MB in mid-LAD (white dashed line). (C) Intravascular ultrasound (IVUS) image of proximal MB. (D) Maximum plaque burden in the MB segment (Max PBmb) was 60%. (E) Site with maximal halo thickness. (F) Site with maximal arterial compression. (G) Longitudinal IVUS view. MB was identified by IVUS as an echolucent-band “halo” (red asterisk; E, F). S1, septal 1; S2, septal 2.
aspirin plus clopidogrel) for 1 year and was instructed to take warfarin (INR range as 2-3) for 3 months along with atorvastatin, isosorbide mononitrate, and metoprolol. At the 1-year coronary angiographic follow-up, no residual stenosis was observed at the original location of the culprit lesion, which was confirmed by IVUS and optical coherence tomography (Fig. 2).

Discussion

MB is a common coronary abnormality present in approximately a quarter of adults. Although cases of MB could be asymptomatic or benign, several reports have shown that it could cause severe mechanical compression of the LAD, and symptomatic patients with MB may present with myocardial ischemia, ACS, coronary spasm, or sudden death, even in those without significant clinical risk factors. The anatomic properties of MB relevant to clinical pathophysiology include depth (superficial: > 1-2 mm vs deep: > 2 mm) and length of the encasement. In this case, the patient was young with a low cardiovascular risk (ie, 10-year risk of fatal cardiovascular disease estimated as < 1% using the systematic coronary risk estimation system) but suffered ACS caused by a thick (1.87 mm) and long (36.12 mm) LAD-MB.

The primary treatment of symptomatic patients with MB is pharmacologic therapy. Intravascular imaging (eg, IVUS) during coronary angiography may provide additional assistance in the recognition of plaque location and its spatial relationship with MB. Stent implantation may rectify the abnormal hemodynamics in patients with flow-limiting plaque but with concerns on perforation during stent deployment, stent fracture, in-stent restenosis, and stent thrombosis. However, it may not be possible to avoid these interventional complications, especially when the target lesion is located at the MB segment. DCB could inhibit smooth muscle cell proliferation and thus eliminate stent thrombosis and reduce restenosis by directly contacting the anti-proliferative drug with the vessel wall by a semicompliant balloon. Thus, this approach could be an alternative treatment option in de novo coronary lesions. With the “leave nothing
behind” strategy afforded by DCB angioplasty, complications associated with stent implantation may be avoided in patients undergoing interventional treatment of the coronary lesion in the MB segment. Accordingly, in this case, we recognized MB using IVUS, treated the lesion with DCB only, and prescribed optimal medical therapy. No recurring angina had presented within 1 year of the procedure, and no residual stenosis was observed at the 1-year coronary angiographic follow-up, indicating that IVUS-guided DCB angioplasty could optimize the outcome in patients with MB-associated ACS. However, because MB exists at the original location, long-term MB-associated changes in wall shear stress and formation and progression of atherosclerosis may occur. A careful long-term management of the patients’ lifestyle and medical treatment should be provided and another interventional procedure or coronary artery bypass grafting may be required if MB patients are resistant to optimal medical treatment.

**Conclusion**

This case demonstrates the utility of an IVUS-guided interventional treatment of MB-induced ACS. DCB angioplasty may avoid coronary stent—associated complications when the lesion is located within the tunneled segment of the MB.

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**Disclosures**

The authors have no conflicts of interest to disclose.

**References**

1. Tarantini G, Migliore F, Cademartiri F, Fraccaro C, Iliceto S. Left anterior descending artery myocardial bridging: a clinical approach. J Am Coll Cardiol 2016;68:2887-99.
2. Ural E, Bildirici U, Celikyurt U, et al. Long-term prognosis of non-interventionally followed patients with isolated myocardial bridge and severe systolic compression of the left anterior descending coronary artery. Clin Cardiol 2009;32:454-7.
3. Piepoli MF, Hoes AW, Agewall S, et al. 2016 European Guidelines on cardiovascular disease prevention in clinical practice: The Sixth Joint Task Force of the European Society of Cardiology and Other Societies on Cardiovascular Disease Prevention in Clinical Practice (constituted by representatives of 10 societies and by invited experts) developed with the special contribution of the European Association for Cardiovascular Prevention & Rehabilitation (EACPR). Eur Heart J 2016;37:2315-81.
4. Lu H, Ge L, Ge J. Coronary aneurysm and stent fracture following stenting of a myocardial bridge. Catheter Cardiovasc Interv 2016;87:E15-8.
5. Kunamneni PB, Rajdev S, Krishnan P, et al. Outcome of intracoronary stenting after failed maximal medical therapy in patients with symptomatic myocardial bridge. Catheter Cardiovasc Interv 2008;71:185-90.
6. Jiang Q, Liang C, Wu Z. Myocardial bridging is a potential risk factor of very late stent thrombosis of drug eluting stent. Med Sci Monit 2012;18: HY9-12.
7. Yerasi C, Case BC, Forrestal BJ, et al. Drug-coated balloon for de novo coronary artery disease: JACC state-of-the-art review. J Am Coll Cardiol 2020;75:1061-73.
8. Ishikawa Y, Akasaka Y, Suzuki K, et al. Anatomic properties of myocardial bridge predisposing to myocardial infarction. Circulation 2009;120:376-83.

**Supplementary Material**

To access the supplementary material accompanying this article, visit CJC Open at https://www.cjcopen.ca/ and at https://doi.org/10.1016/j.cjco.2020.10.013.