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## Plaque herniation after stenting the culprit lesion with myocardial bridging in ST elevation myocardial infarction: A case report

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**Author contributions:** Ma J participated in the care of the patient throughout the hospitalization, collected clinical information, obtained informed consent for the publication of the manuscript and information, reviewed, edited and approved the manuscript; Gustafson GM performed the coronary angiography and primary percutaneous coronary intervention, initiated the discussion about myocardial bridging in STEMI care and plaque herniation; and reviewed, edited and approved the manuscript; Dai X initiated and proposed manuscript concept, participated literature search and review, wrote and revised manuscript as a senior author.

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### Abstract

#### BACKGROUND

Myocardial bridging (MB) is increasingly recognized to stimulate atherogenesis, which may contribute to an acute coronary syndrome. Stenting the coronary segment with MB has been recognized to have an increased risk of in-stent restenosis, stent fracture and coronary perforation. The safety and efficacy of stenting the culprit lesion with overlaying MB in ST elevation myocardial infarction (STEMI) as primary reperfusion therapy has not been established.

#### CASE SUMMARY

We reported a patient who presented with inferior STEMI with a culprit lesion of an acute thrombotic occlusion in the right coronary artery and thrombolysis and thrombin inhibition in myocardial infarction 0 flow. After the stent placement during primary percutaneous coronary intervention, intravascular ultrasound revealed MB overlying the stented segment where heavy atherosclerotic plaque were present. Likely due to the combination of plaque herniation or prolapse caused by MB, as well as local increased inflammation and thrombogenicity, acute stent thrombosis occurred at this region, which led to acute stent failure. The patient required an emergent repeated cardiac catheterization and placing a second layer of stent to enhance the radial strength and reduce the inter-strut space.

#### CONCLUSION

Plaque herniation or prolapse after stenting a MB segment in STEMI is a potential etiology for acute stent failure.

**Key words:** Case report; ST elevation myocardial infarction; Myocardial bridging; Plaque herniation; Plaque prolapse; Intravascular ultrasound; Acute stent thrombosis

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**Core tip:** Stenting the coronary segment with myocardial bridging is known to have increased risks of in-stent restenosis, stent fracture and coronary perforation. Myocardial bridging is also increasingly recognized to be pro-atherosclerotic and potentially involved in acute coronary syndrome, including ST elevation myocardial infarction (STEMI). The safety and efficacy of stenting the culprit lesion with overlying myocardial bridging in STEMI as primary reperfusion therapy has not been established. Here we present a case where plaque herniation or prolapse occurred after stenting a culprit lesion in STEMI, where overlying myocardial bridging was recognized by post-stenting intravascular ultrasound. The plaque herniation at the stented segment with myocardial bridging contributed to acute stent thrombosis which required a second layer of stent deployment. This case highlighted that plaque herniation or plaque prolapse after stenting a segment with myocardial bridging in STEMI is a potential etiology for acute stent failure, and emphasized the important role of intravascular ultrasound in primary percutaneous coronary intervention.

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## INTRODUCTION

Acute stent thrombosis after coronary artery stent placement is a rare but serious complication in percutaneous coronary intervention (PCI). Stenting culprit lesions in acute myocardial infarction has higher risk of acute stent thrombosis than stable coronary artery disease<sup>[1]</sup>. Coronary dissection, stent mal-apposition, or inadequate antiplatelet/anticoagulation therapy have been considered as the most common causes of acute stent thrombosis, in addition to the local inflammation and thrombogenic environment. We report a patient who experienced acute stent closure after stenting the culprit lesion in the mid right coronary artery (RCA) of an inferior ST elevation myocardial infarction (STEMI). Intravascular ultrasound (IVUS) revealed myocardial bridging (MB) phenomenon overlying the stented segment of the RCA, which likely promoted atherosclerotic plaque herniation (AKA, plaque prolapse) through the stent struts. We are suggesting that the herniated or prolapsed atherosclerotic plaque materials combined with the acute tissue inflammation contributed to the acute stent thrombosis.

## CASE PRESENTATION

### Chief complaint

Recurrence of chest pain 30 min after stent placement.

### History of present illness

A 72-year-old woman with a history of hypertension, dyslipidemia, diabetes mellitus, and remote history of thyroid cancer status post thyroidectomy, who presented with acute onset substernal chest pain (CCS class IV angina pectoris) associated with nausea and diaphoresis. The 12-lead electrocardiogram (ECG) showed ST elevations in II, III and aVF with reciprocal ST depressions met the clinical diagnostic criteria of inferior STEMI (Figure 1A). The patient was pre-loaded with aspirin (325 mg) and P2Y12 receptor antagonist (clopidogrel 600 mg) in the emergency department, and was brought to the cardiac catheterization laboratory for emergent coronary angiography followed by reperfusion therapy by primary PCI. An acute thrombotic occlusion in the mid RCA with thrombin inhibition in myocardial infarction 0 flow was identified as the culprit lesion (Figure 1B, white arrow). Diffuse atherosclerotic disease was also seen in the proximal RCA (Figure 1B, orange arrows). Weight-based direct thrombin inhibitor (bivalirudin) infusion (0.75 mg/kg IV bolus immediately followed by 1.75 mg/kg/h IV infusion) was used for anticoagulation during the primary PCI. Primary PCI was performed with initial aspiration thrombectomy and followed by the placement of two over-lapping drug eluting stents (3.0 × 28 mm drug

eluting stents deployed at 12 ATM, and 3.25 mm × 12 mm NC balloon for 18 ATM post-dilatation), which achieved 0% residual stenosis and thrombin inhibition in myocardial infarction 3 flow angiographically (Figure 1C). The patient's symptoms and ST elevations on ECG completely resolved after this primary PCI. Pre-stenting IVUS was not performed. Serial post stenting IVUS studies were performed which revealed heavy atherosclerotic plaque burden at the stented area (Figure 1E, blue arrows). The IVUS images demonstrated that there was overlying MB with the typical IVUS appearance of echo-lucent area overlying the stented segment (the "half-moon phenomenon") (Supplement Video 1; Figure 1E, orange arrows; Figure 1C, boxed area, arrows). IVUS also confirmed fully expanded and well apposed stent. With the recognition of the stent in a segment with overlying MB, there was some concern and discussion, but no further prophylactic action was taken. At the conclusion of the primary PCI, bivalirudin infusion was discontinued. The patient was subsequently transferred to the coronary care unit (CCU) for further post-STEMI monitoring.

Shortly after her arrival to the coronary care unit (30 min after the discontinuation of bivalirudin infusion), the patient experienced recurrent chest pain. Again, the pain was substernal, 9-10 out of 10 in pain scale. It was associated with nausea and diaphoresis. The patient appeared to be in significant distress although, vital signs were stable. She had mild shortness of breath associated with the chest pain, denied of palpitations, and any neurological symptoms. She did not have hypotension, nor evidence of tamponade on bedside transthoracic echocardiography. Other review of systems were negative except detailed above.

Immediate bedside 12-lead ECG revealed the re-appearance of the inferior leads ST elevations, which was previously confirmed to have resolved after stenting the mid RCA. An acute stent failure/thrombosis leading to acute occlusion of the stented RCA was suspected. The patient returned to the CCL immediately for repeat coronary angiography and primary PCI.

### **History of past medical and surgical history**

Hypertension, hyperlipidemia, diabetes mellitus, thyroid cancer status post thyroidectomy on thyroid hormone replacement therapy.

### **Physical examination**

Vital signs: Temperature 36.9 °C, heart rate 58 bpm, respiratory rate 15 per minute, BP 105/68 mmHg, SpO<sub>2</sub>: 93% on room air. The patient appeared in mild distress, skin was mildly moisture suggestive for mild diaphoresis. Alert awake oriented × 3. There was no significant evidence of jugular vein distention, mild crackles in the bilateral lung bases without wheezing. There was no significant murmur audible. The right femoral access site for the previous emergent catheterization was clear without evidence of hematoma. Peripheral pulses were intact bilaterally. Other physical examinations were unremarkable.

### **Laboratory examinations**

Labs were drawn at her initial emergency department presentation with returned results by the time when she returned to the CCL with recurrent chest pain. Her complete blood count results were white blood cell, 9.16 K/ $\mu$ L, Hb 11.2 g/dL; Hct 34.1%, platelet 160 K/ $\mu$ L; her basic chemistry panel showed: Na<sup>+</sup> 131 mEq/L, K<sup>+</sup> 4.1 mEq/L, Cl<sup>-</sup> 95 mEq/L, HCO<sub>3</sub><sup>-</sup> 24 mmol/L, blood urea nitrogen 18.5 mg/dL, Mg<sup>2+</sup> 2.3 mEq/L, Troponin 3.48 ng/mL. Her liver function panel were within normal range.

### **Imaging examinations**

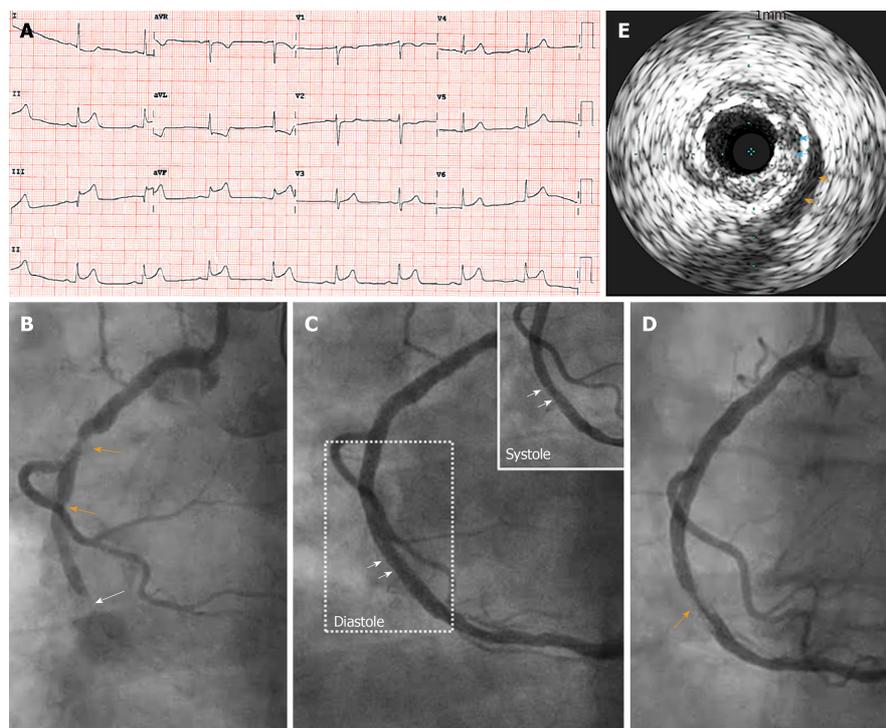
**12-leads ECG:** Sinus bradycardia at 53 beat per minutes, with ST segment elevations > 0.1 mV in II, III and aVF and reciprocal ST depressions in leads I and aVL.

**Transthoracic echocardiography (post-PCI):** Mild segmental left ventricular systolic dysfunction, estimated EF 50%. The basal to mid inferior wall is hypokinetic. The inferolateral wall appears mildly hypokinetic. Normal right ventricle size and function. No significant valvular abnormalities. No evidence of pericardial effusion. No evidence of pulmonary hypertension.

**Chest X-ray (post-PCI):** Increased interstitial markings throughout both lungs, which may represent pulmonary edema. No pleural effusions. No pneumothorax.

## **FINAL DIAGNOSIS**

Acute stent thrombosis due to plaque herniation after stenting a STEMI culprit lesion with overlying MB.



**Figure 1** Images of the recurrence of inferior ST segment elevation myocardial infarction and intra-procedural findings during reperfusion therapy. A: Presenting 12-lead electrocardiogram showed ST elevation in inferior leads; B: Culprit lesion in the mid right coronary artery (RCA) (white arrow) and diffuse atherosclerotic lesions in the proximal RCA (orange arrows); C: Initially stented RCA with systolic narrowing (insert, systolic phase of the dashed area in C) showed mild compressions of the stented lumen (white arrows); D: Circumferential filling defect in the stented segment (orange arrow); E: Intravascular ultrasound evidence of myocardial bridge with echo-lucent half-moon sign (orange arrows) and plaque between stent and myocardial bridge (blue arrows).

## TREATMENT

With the presumed diagnosis of acute stent thrombosis which led to the recurrent presentation of inferior STEMI, the patient was brought back to the CCL emergently for a repeat reperfusion therapy. Repeat angiography confirmed a filling defect with subtotal occlusion in the mid portion of the stented segment (Figure 1D, orange arrow), where overlying MB had been recognized on IVUS in the earlier study. MB induced plaque herniation was thought to be the likely contributing factor for the acute thrombotic closure, where the stent had been well expanded and apposed on IVUS at the end of previous procedure. In order to enhance radial strength and reduce the inter-strut spaces to minimize plaque herniation through the stented MB segment, a second layer of DES was deployed with additional post dilations. Repeat IVUS study confirmed an improved stent expansion and luminal gain without evidence of acute plaque herniation. Since the acute stent thrombosis occurred only 30 min from the discontinuation of the bivalirudin (less than one half-life), and the P2Y12 inhibitor (clopidogrel) was loaded approximately 2 h earlier, we considered that an ineffective dual antiplatelet therapy was less likely to be the major contributor to the event. Nevertheless, the patient's P2Y12 antagonist was changed to ticagrelor to enhance antiplatelet therapy.

## OUTCOME AND FOLLOW-UP

The patient did well after the second procedure, and was discharged to home after 3 d of uneventful hospitalization. A pre-discharge echocardiography showed mild segmental left ventricular systolic dysfunction (EF 50%) with hypokinesia of the basal-to-mid inferior and inferolateral walls. She was doing well at 30-d follow up.

## DISCUSSION

MB is a coronary anomaly in which a segment of epicardial coronary artery coursing

under a “bridge” of overlying myocardium<sup>[2]</sup>. This phenomenon was initially discovered in autopsy. Coronary artery angiography, coronary computer tomographic angiography (CCTA) and IVUS are now commonly used imaging modalities to identify MB. In general population, the prevalence of MB reported by autopsy series was roughly 25%, similar by CCTA and lower in coronary angiography series (0.5%-12%)<sup>[2]</sup>. Angiographically, MB is most commonly seen in the mid segment of left anterior descending artery, followed by left circumflex and least common in RCA. However, some CCTA and autopsy series found similar involvement of left circumflex and RCA in MB<sup>[3-6]</sup>. The overlying myocardial fibers often lead to vessel compression during systole in cardiac cycle. Depending on the depth of MB, location of the MB, with or without myocardial hypertrophy, as well as systemic and coronary hemodynamic status, MB may result in clinical symptoms such as angina, myocardial ischemia or even ventricular arrhythmias. The first line of therapy of stable symptomatic MB remains medical treatment with beta-blockers and non-dihydropyridine calcium-channel blockers, while nitrates provides less consistent effects.

Stenting a coronary artery segment with overlying MB is associated with an increased risk of in-stent restenosis, stent fracture and coronary perforation. Therefore, stenting the coronary artery with MB is generally not recommended for management of ischemic symptoms in stable patients. MB is also increasingly recognized to be pro-atherosclerotic, especially at the peri-bridge edges, due to endothelial dysfunction, thickened intima and turbulent flow dynamics<sup>[7-9]</sup>. Rupture of atherosclerotic plaque in the segment of MB could result in acute thrombotic occlusion and STEMI presentation<sup>[10]</sup>. Primary PCI with stent placement remains the treatment of choice in the acute STEMI setting with or without the recognition of MB by angiography or IVUS. How often an overlying MB is involved in STEMI culprit lesion is not known. In our case, the MB overlying the stented segment involved in the culprit lesion of STEMI was recognized by post stenting IVUS during the initial primary PCI. Although concerns were raised during the initial primary PCI after the recognition of MB, and options such as empirically deploying a second layer of stent was discussed, there was no clinical evidence to guide this prophylactic use of second layer of stent against acute stent failure in this setting. In retrospect, potential adverse consequences after stenting coronary segment with overlying MB, such as acute plaque herniation, recoiling, and re-thrombosis had been underestimated. Plaque herniation or prolapse after stenting occurs up to 25% of stent placement in acute MI by IVUS imaging<sup>[11,12]</sup>. It is associated with worse clinical outcome including stent thrombosis<sup>[13]</sup>. Like the case presented here, in the setting of acute STEMI with the culprit lesion in a coronary artery segment with an overlying MB and heavy atherosclerotic plaque burden, stenting of this lesion resulted an increased risk of plaque herniation through the stent struts. With the combination of local inflammation, and the thrombogenic nature of the disrupted atherosclerotic plaque, acute stent thrombosis and closure occurred which required repeat revascularization. It's a perfect storm. In our case, placing a second layer of stent to improve radial strength and also reduce the space between struts especially in open-cell designed stents was performed to minimize plaque herniation.

IVUS is an important imaging modality to identify MB. On IVUS, the tunneled segment of coronary artery underneath the overlying myocardium demonstrates systolic vessel compression and highly specific echo-lucent “half-moon” appearance as shown in **Figure 1E** (orange arrows). The use of IVUS in primary PCI is a valuable technique to provide information to recognize overlying MB and plaque herniation, which can promote the consideration of approaches of prophylaxis of acute stent failure after stenting MB segment<sup>[14,15]</sup>. We now felt strongly that pre-stenting IVUS may provide potential MB information before stent placement, which may change clinical decision making. When MB phenomenon identified by post-stenting IVUS, then empirically deploying an additional layer of stent may be advantageous after the recognition of MB and significant plaque burden and herniation in the stent on IVUS. This case further emphasizes the value of IVUS (pre- and post- stenting) during primary PCI.

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## CONCLUSION

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The risk related to MB in the STEMI culprit lesion was under-recognized. Stenting the culprit lesion with overlying MB may result in plaque herniation, leading to acute stent failure. IVUS study in primary PCI is an useful tool for detecting MB. Empirical treatment with double layered stents may be necessary to prevent acute stent failure.

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