

The “Chimney Sweep Technique” and Recurrent Coronary Artery Dissections

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ABSTRACT: We are reporting a case of a 65-year-old female who experienced three ST elevation myocardial infarctions (STEMI) during the course of 3 weeks. The first infarct was related to intramural coronary hematoma that propagated proximally after stenting, the second involved progression of this hematoma with obstruction of the vessel lumen, and the third caused subacute stent thrombosis that required “stent cleaning” using the inflated balloon technique.

INTRODUCTION

An intramural hematoma is a manifestation of a spontaneous coronary artery dissection. In the setting of an acute coronary syndrome (ACS), it may be difficult to distinguish an intramural hematoma from an acute plaque rupture using coronary angiography. Although there is no definitive data to help manage these lesions, conservative treatment has become the suggested practice in the absence of ongoing ischemia or an ACS. Even when invasive strategies are implemented, angioplasty lends itself to poor outcomes due to hematoma propagation, poor stent approximation, and subsequent dissection. This case highlights the difficulties encountered with both invasive and conservative management pathways.

CASE DESCRIPTION

A 65-year-old female originally presented to a regional hospital with new-onset angina at rest lasting 1 hour. She had bradycardia and profound hypotension consistent with cardiogenic shock. The electrocardiogram (EKG) revealed an inferior ST elevation myocardial infarction (STEMI), and the patient was transferred for primary percutaneous coronary intervention (PCI) within 120 minutes. On arrival, she underwent emergent coronary angiogram that revealed ulcerative plaque in the mid right coronary artery (RCA) with distal thrombosis (Figure 1 A); this was successfully treated with balloon angioplasty and placement of a drug-eluting stent. The post-PCI angiogram demonstrated an eccentric protuberance adjacent to the proximal stent edge (Figure 1 B). Since there were no observed lesions in this region prior to stent placement, her physicians assumed it was an intramural hematoma and left it to medical management. Both her junctional bradycardia and hypotension persisted;

thus, the patient required an intra-aortic balloon pump and temporary pacing wire. Her troponin peaked at 44.58 ng/mL and an echocardiogram revealed a preserved ejection fraction. Since the cardiogenic shock and rhythm disturbances resolved by the second day, both the pacemaker and balloon pump were successfully removed. She was noted to have episodes of atrial fibrillation associated with sinus conversion pauses up to 4.5 seconds, so a beta blocker was not administered. She was discharged on apixaban, aspirin, clopidogrel, lisinopril, and atorvastatin.

Seven days following discharge, the patient presented to the regional hospital with rest angina and cardiogenic shock. An EKG demonstrated inferior STEMI. After her transfer was delayed due to weather, the patient received

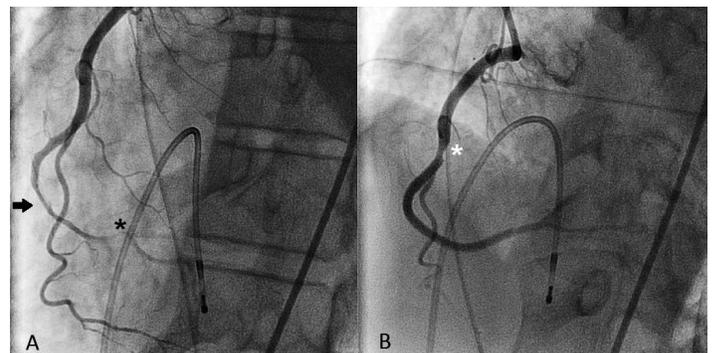


Figure 1.

(A) Angiogram in left anterior oblique (LAO) view demonstrating ulcerative plaque in the mid right coronary artery with 70% stenosis (black arrow) and distal embolism (black asterisk). (B) Angiogram in LAO view demonstrating a de novo lesion with features suggestive of intramural hematoma (white asterisk).

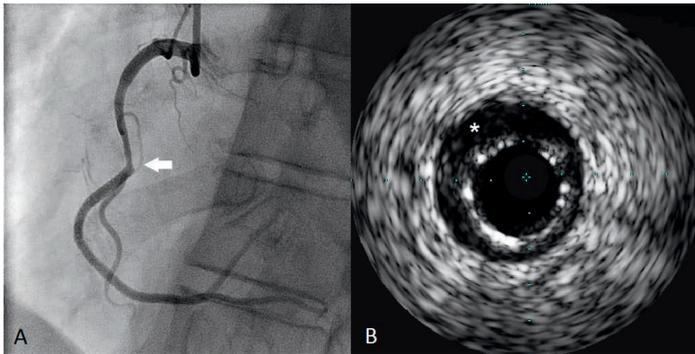


Figure 2.

(A) Angiogram in left anterior oblique view demonstrates haziness within a 70% stenosis proximal to stent edge (white arrow). This was the region of previous suspected hematoma; however, stenosis progressed. (B) Intravascular ultrasound reveals an intramural hematoma (white asterisk) proximal to the stent.

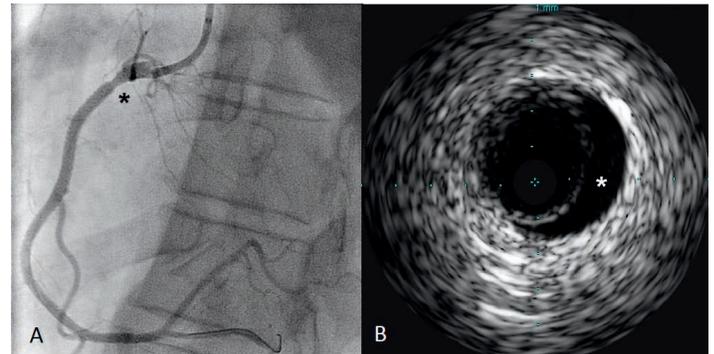


Figure 3.

(A) Angiogram in left anterior oblique view following proximal stent deployment shows a de novo stenosis of 50% to 60% (black asterisk). (B) Intravascular ultrasound of proximal right coronary artery lesion confirms the intramural hematoma (white asterisk).

thrombolysis therapy with tenecteplase. She was then transferred for rescue PCI when her condition did not significantly improve with thrombolysis. An emergent coronary angiogram revealed hazy 70% stenosis in her mid RCA proximal to the stent (Figure 2 A). Intravascular ultrasound (IVUS) confirmed the presence of significant stenosis associated with an intramural hematoma (Figure 2 B). Subsequent angioplasty and stenting was performed, and a de novo mild stenosis developed in the proximal RCA near the new stent edge (Figure 3 A). It was confirmed to be an intramural hematoma but was not stented since it was nonobstructive (Figure 3 B). Oral anticoagulation and dual antiplatelet therapies were resumed. Shortly after the procedure, the patient developed dysphasia and altered mentation. A brain computed tomography (CT) scan revealed multiple large intracranial hemorrhages involving the left frontal lobe, occipital lobe, and left and right cerebellum. She immediately received platelets, fresh frozen plasma, and prothrombin complex concentrate to reverse her anticoagulated state. Over the course of several days, she had minimal neurological improvement, and the decision was made to commence hospice care. Her cognition and physical impairments improved while in hospice, so she was transferred to an inpatient rehabilitation program. Due to the severity of the intracerebral hemorrhage, all antiplatelet and oral anticoagulation medications were withheld.

Her neurological deficits resolved during her rehabilitation period, and aspirin was commenced with planned reintroduction of clopidogrel at a later date. On day 14 following the second STEMI, the patient developed angina at rest and bradycardia. Another EKG revealed ST elevation

in the inferior leads with anterior ST depression consistent with STEMI. An emergent angiogram revealed acute in-stent thrombosis with TIMI 0 flow in the RCA (Figure 4). The “dottering” technique and balloon angioplasty were performed with anticoagulation using heparin and clopidogrel. Of note, the proximal prestented segment hematoma completely resolved (Figure 4 B). However, thrombus continued to develop immediately following balloon angioplasty. It was thought that the residual thrombus following angioplasty was a likely nidus for thrombogenesis, so an ad hoc technique was employed—referred to here as the “chimney sweep technique.”

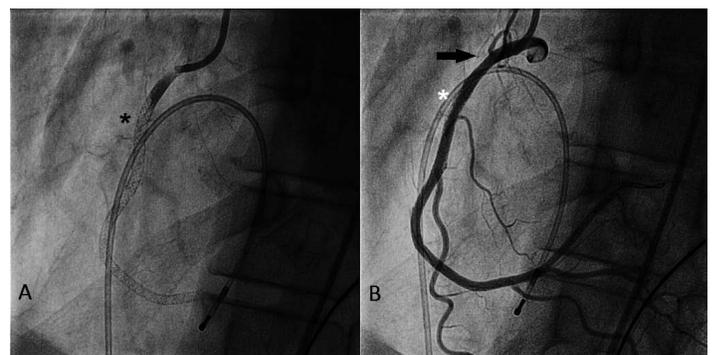


Figure 4.

(A) Angiogram in left anterior oblique (LAO) view confirming the presence of acute in-stent thrombosis (black asterisk) within the proximal right coronary artery. (B) Angiogram in LAO view showing resolution of thrombus (white asterisk) and proximal prestented hematoma (black arrow) following angioplasty and completion of the “chimney sweep technique.”

Intracoronary heparin was administered, and the balloon was inflated and pulled in the stent backwards to remove residual thrombus and clean the stent surface (Video 1). The patient did well in the recovery period, although she required a pacemaker to manage her tachycardia-bradycardia syndrome. She was discharged home in good condition and was doing well at her 6-month follow-up.

DISCUSSION

An intramural hematoma is a manifestation of a spontaneous coronary artery dissection (SCAD) in which separation of the vascular wall layers creates a potential space. This space within the arterial wall fills with blood from either the endoluminal space or vasa vasorum, thereby creating a mass capable of compressing the arterial lumen.^{1,2} Compression of the lumen not only may reduce antegrade flow but also may precipitate ischemia or infarction. Although uncommon, coronary dissections are more prevalent in women, especially in the setting of an ACS. In case series, coronary dissections account for 10.8% to 24% of all cases of ST elevation myocardial infarctions in younger women (< 50 years of age).³ In addition to gender and age, other clinical features suspicious for spontaneous coronary dissection include absence of typical atherosclerotic lesions, peripartum state, fibromuscular dysplasia, absence of traditional risk factors, and precipitating stressful events.¹ Currently, there are no prospective randomized data to direct the management of spontaneous coronary dissections, and it is unclear if goal-directed therapies for ACS are beneficial. There are reports of clinical deterioration with the use of thrombolytic therapy and a potential for increased bleeding and lesion extension with glycoprotein inhibitors; therefore, these agents should be avoided if a SCAD is suspected. Fortunately, beta blockers and nitroglycerin have beneficial effects as they reduce wall shear stress and alleviate vasospasm.⁴

The long-term outcome following a dissection is favorable as the majority of lesions heal, conferring a 1-year mortality rate of 1% to 4% and a reinfarction rate of 4.8 percent.^{1,3,5,6} Conservative treatment has been advocated in stable patients with no ongoing ischemia or with lesions not involving the proximal regions of coronary vessels.⁵ When required, PCI is notoriously challenging and lends itself to suboptimal results. The reasons for poor outcomes include propagation of the dissection plane with further luminal compromise, an extensive region of dissection requiring longer stents, small vessel caliber, and strut malposition following hematoma resorption predisposing to very late stent thrombosis.¹ Even when implementing modern therapies, the observed rate of subsequent dissection remains high at 17%.⁴

CONCLUSION

Our patient demonstrates the failure of current treatment strategies to prevent dissection recurrence and propagation. The intramural hematoma that was treated conservatively following stent placement likely progressed and was the nidus for both thrombus formation and the development of recurrent STEMIs. The use of oral anticoagulation to manage atrial fibrillation likely promoted hematoma extension within the vessel wall. The third STEMI undoubtedly resulted from the discontinuation of antiplatelet therapy in the setting of an extensive intracranial hemorrhage. The technical difficulties associated with stent malposition were unlikely contributing factors as IVUS demonstrated good luminal gain and stent apposition. Interestingly, the final angiographic picture obtained during STEMI management demonstrated resolution of the stent-induced hematoma in the proximal RCA by day 14.

Due to the patient's history of recent intracerebral hemorrhages, no further stents were deployed while managing her third coronary syndrome; instead, the focus was on the dissolution of thrombus. The "dottering" technique and balloon angioplasty were ineffective at maintaining luminal patency. This was due to rapid reaccumulation of thrombus within the stent that was observed on fluoroscopic images. It appears that use of the "chimney sweep technique" completely disintegrated the thrombus and achieved successful long-term outcomes. We suggest that this technique may be utilized in similar patients who present technical and clinical challenges.

Conflict of Interest Disclosure

The authors have completed and submitted the *Methodist DeBakey Cardiovascular Journal* Conflict of Interest Statement and none were reported.

Keywords

spontaneous coronary artery dissection, recurrent STEMI, in-stent thrombosis, chimney sweep, intramural hematoma

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