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Multiple Culprit Lesions in STEMI - Invitation to Collaborate, A Case Series and Literature Review

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Introduction

Acute ST-segment elevation myocardial infarction (STEMI) is usually attributed to an occlusion in one single coronary artery. The etiologies are numerous, ranging from thrombotic, embolic, vasospastic, and dissection among others. In rare instances, multiple thrombotic occlusions can occur in more than one "culprit" artery. The incidence of simultaneous multi-vessel coronary thrombosis was 4.8% at the time of primary percutaneous coronary intervention (PCI)in one single study center [1]. However, autopsy series report the incidence of multiple thrombotic coronary occlusions to be 50% in patients who died from sudden cardiac death [1]. This suggests that the incidence may be higher but is underreported as many patients die before medical intervention.

Prior cases with identified etiology were attributed to coronary vasospasm [2,3], cocaine abuse [4], and thrombophilic states (heparin induced thrombocytopenia [5], antithrombin III deficiency [6], idiopathic thrombocytopenic purpura [7], tamoxifen use [8], essential thrombocytosis [9-12], and hyperhomocysteinemia [13]). However, other cases with unidentified etiology were speculated to be caused by simultaneous plaque rupture in each culprit vessel. This could be caused by diffuse inflammation in each coronary artery, or platelet activation from catecholamine surge [14]. Others have postulated that the initial STEMI in the territory of the culprit artery causes cardiogenic shock, resulting in decreased blood flow to other stenotic coronary arteries [15].

We describe three rare cases of simultaneous thrombotic occlusions in multiple coronary arteries where each was treated successfully with percutaneous coronary intervention at the time of the initial procedure.

Case 1

A 68 year-old male with stable angina, hypertension, and hyperlipidemia presented with acute onset, substernal chest pain associated with diaphoresis, nausea, and lethargy. Initial electrocardiogram showed atrial fibrillation with ST elevations in leads II, III, aVF, and ST depressions in leads V1 through V3. Initial troponin was 0.0117 ng/mL consistent with an acute myocardial infarction. His vital signs remained stable otherwise. He was given aspirin 162 mg, ticagrelor 180 mg, atorvastatin 80 mg, intravenous metoprolol 5 mg, sublingual nitroglycerin 0.4 mg, and a heparin bolus.

Emergent cardiac catheterization revealed a thrombotic occlusion of the distal RCA feeding a right posterolateral branch (rPL). He also had a thrombotic occlusion of a large caliber 2nd Obtuse marginal (OM2). There was an 80% focal lesion in the Left Anterior Descending artery (LAD) as well. A ventriculogram revealed a hypokinetic inferior wall with estimated left ventricular ejection fraction of 45%. He underwent successful PCI with a drug-eluting stent (DES) to the distal RCA/rPL which restored TIMI 3 flow. Subsequently, he also had a successful PCI to OM2 with a DES, which restored TIMI 3 flow.

Post-catheterization electrocardiogram revealed return to normal sinus rhythm and near-resolution of ST elevations in the inferior leads. The rest of his hospital course was complicated by a 4cm x 8cm hematoma in the radial arteriotomy site. This was managed with compression and resolved. His Troponin peaked at 111.182 ng/mL.

The patient then underwent staged PCI of the LAD with successful placement of 1 DES. He was continued on goal directed medical therapy for CAD and was started on Ranolazine for persistent chest pain. This improved his symptoms and the patient was discharged.

Case 2

A 55 year-old male with a previous history of coronary vasospasm thought secondary to cocaine use presented with acute onset substernal chest pressure while walking. Medical history notable for hypertension, diabetes mellitus, hyperlipidemia, and substance abuse. On examination he was diaphoretic, tachycardia, and appeared in acute distress. Electrocardiogram showed antero-septal ST elevations in leads V1-V4 with otherwise stable vitals. Troponin was elevated to 0.336 ng/mL. Urine toxicology was positive for benzodiazepines, opioids, and negative for cocaine. He was loaded with aspirin (325mg) and ticagrelor (180mg). A heparin drip was initiated, and atorvastatin was given.

He underwent emergent coronary catheterization that revealed total thrombotic occlusion of distal RCA and total occlusion of mid LAD. Ventriculogram revealed severely hypokinetic apical and inferior regional wall motion abnormalities with estimated left ventricular ejection fraction of 30-40%. He underwent percutaneous coronary intervention of the mid LAD with (1) drug eluting stent with successful restoration of TIMI 3 Flow afterwards. He also underwent percutaneous transluminal coronary angioplasty (PTCA) of the mid and distal right coronary artery (RCA) which improved flow, but due to significant tortuosity, a stent was not placed. Flow was re-established with plain old balloon angioplasty (POBA). The patient was transferred to the CCU where he was started on goal directed medical therapy and heparin infusion for left ventricular thrombus prophylaxis given the apical hypokinesis on the ventriculogram.

Post-cath course was largely unremarkable. His Troponin peaked to 65.7. A transthoracic echocardiogram was obtained that ruled out left ventricular thrombus and revealed moderately to severely reduced left ventricular systolic function with an ejection fraction of 30%. He was brought back to the catheterization lab for another attempt at PCI to the RCA, which was successful. The LAD stent was also found to be patent. Given his history of cocaine use, metoprolol was switched to carvedilol. The following day, the patient symptoms had resolved, and he was discharged.

Case 3

A 49-year-old incarcerated male with history of hypertension and asthma presented to the detention center clinic with chest and abdominal pain. Shortly after, he became unresponsive, pulseless, and went into circulatory arrest. Subsequently he was given (1) round of cardiopulmonary resuscitation and (1) defibrillation, advised by the machine. On transfer to the hospital, aspirin was given. In the ED he developed v-fib arrest and return of spontaneous circulation was achieved after (1) shock. He was then loaded with Ticagrelor but needed to be intubated for airway protection and hypoxemia. His initial 12 lead electrocardiogram revealed ST elevations in leads II, III, and aVF with reciprocal ST depressions in leads I and aVL. Initial troponin was 0.626 ng/mL. He underwent emergent left heart cardiac catheterization, with coronary angiography that revealed thrombotic occlusion of the proximal RCA with TIMI 0 flow. It also showed a complete thrombotic occlusion in the prox-mid LAD. Ventriculogram revealed akinetic inferior wall with an estimated left ventricular ejection fraction of 45%. Decision was made to intervene on both vessels. The RCA was sought after first, but the patient had 3 episodes of Polymorphic VT that all responded to (1) defibrillation each. He required ino-pressor support with three agents. Subsequently a right heart catheterization was performed that showed cardiac output of 2.96 L/min and a cardiac index of 1.47 L/min/m2, indicating refractory cardiogenic shock. Intra-aortic balloon pump was inserted. He then underwent aspiration thrombectomy and PCI with DES to the RCA. After revascularization, there was poor re-flow and intravenous verapamil and eptifibatide was initiated. There was evidence of a ruptured plaque in mid left anterior descending artery that required thrombectomy and PCI with DES as well.

The patient was transferred to the cardiac intensive care unit for further management. Hypothermia protocol and heparin infusion was initiated. He was started on daily aspirin 81 mg, atorvastatin 80 mg, and ticagrelor 90 mg. He was successfully weaned off of vasopressor support after several days. Post op course complicated by aspiration pneumonia and hyperreflexia of lower extremities. A subsequent MRI brain showed left thalamic and cerebellar infarcts. He was treated medically. Prior to discharge the patient returned for surveillance angiography, which showed full opacification of the RCA, which was not seen before due to reflow phenomenon. His stents were patent. No intervention needed. His mental status improved and he was discharged on goal directed medical therapy.

Discussion

These cases represent patients who presented with STEMI due to simultaneous thrombus formation in multiple coronary arteries. Thrombus burden was evaluated with fluoroscopy guidance and decision was made not use intracoronary imaging given acuity of presentation. In all three cases, the RCA was acutely occluded. A recent case series also found the RCA as the most common vessel involved in patients who presented with multiple occluded vessels [1]. In two of our three patients with RCA and LAD occlusion, the EKG was indicative of infero-posterior myocardial infarction and did not show any signs of LAD occlusion. Only one of the patients developed cardiogenic shock requiring mechanical support. A systematic review of prior case reports and prospective trials showed that cardiogenic shock was present in 41% of the 56 patients who presented with multi-vessel coronary thrombosis in the setting of a STEMI. Ventricular arrhythmias were the presenting diagnosis in 25% of cases. Like our patients, the majority of those patients underwent multiple PCI on each culprit vessel in the same procedure setting. In the same systematic review, the in-hospital mortality rate for multi-vessel coronary thrombosis was 5% [16]. All three of our patients survived.

In patients with STEMI in which multiple coronary vessels are affected and without hemodynamic compromise, the guidelines recommend primary PCI on the culprit lesion and subsequent staged PCI on the non-culprit vessels [17]. However, no randomized clinical trials exist evaluating the efficacy and adverse outcomes for patients with STEMI from multiple culprit lesions due to the rarity of the condition. When presented with this clinical scenario,

cardiologists have relied on clinical judgment and expertise. When extrapolating from our patients, and the previous case series: Re-establishing distal flow in both lesions during the initial procedure appears to be beneficial in patients who present with acute total thrombotic occlusion in multiple vessels.

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