Catheter-induced Spasm of the Left Main Coronary Artery Due to Anatomic "Kinking" in Its Course

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Catheter-induced spasm of the left main coronary artery is rare but has been previously reported. The accurate diagnosis and differentiation of left main coronary spasm from fixed left main obstructive disease has obvious significant clinical implications.[1] Although spasm of the left main coronary artery has been attributed to either catheter-induced mechanical irritation or the presence of Prinzmetal's variant angina,[2,3] the factors predisposing to catheter-induced spasm are poorly defined. Upon review of literature, we did not find any reports of anatomical variations of the left main coronary artery that may predispose to either spontaneous or catheter-induced spasm.

We report here a case of catheter-induced left main spasm in a patient with a significant bend in the course of the left main coronary artery.

Case Report. *A 62-year-old woman with a history of hypertension and prior stent placement in the proximal LAD was admitted to the hospital after an episode of chest pain. The previous stent placement took place two years before the current hospital admission. At that time, the left main coronary artery was reported to have a 30–40% stenosis. The current ECG obtained in the ER showed normal sinus rhythm with non-specific ST-T wave changes. Myocardial infarction was ruled out, but because of her history of prior stent placement, she was referred for cardiac catheterization and coronary angiography.*

A 6 Fr JL-4 catheter was used to engage the left main coronary artery. Initial contrast injections demonstrated what appeared to be a 70–80% obstruction in the proximal portion of the left main coronary trunk. The left main was long and had a sharp bend in its course. The mid-LAD stent was widely patent and all of the other coronary arteries were free of disease. Intracoronary nitroglycerin and subsequent angiography (including non-selective injection of the left main coronary) demonstrated that the obstruction was in the range of about 30% rather than 70–80%. The patient remained hemodynamically stable and chest pain-free throughout the procedure. The previous angiogram at the time of coronary intervention to the left anterior descending artery was reviewed. It showed no disease in the left main coronary artery and confirmed the presence of unusual tortuosity and a sharp bend in the course of the left main coronary artery. Subsequent dobutamine echocardiography revealed no evidence of stress-induced wall motion abnormalities. The patient was discharged home and has continued to do well with medical therapy.

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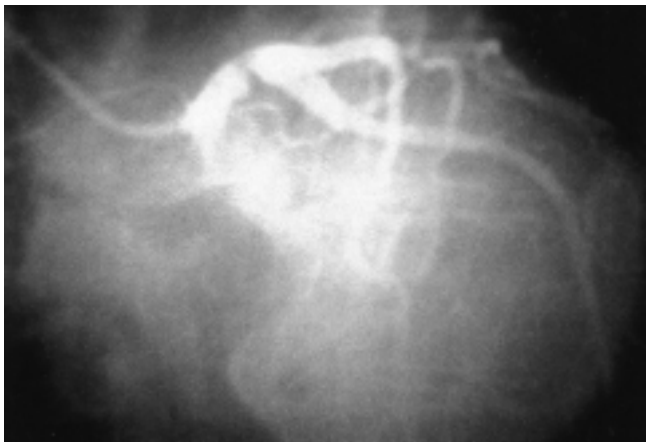
Discussion. Catheter-induced spasm of the right coronary artery is a common phenomenon;[1] however, left main spasm during routine diagnostic catheterization is a relatively rare occurrence.[1,4] A review of the literature that exists on left main spasm narrows the etiology to either spasm resulting from mechanical irritation following catheter engagement, or to spontaneous spasm in patients with Prinzmetal angina.[2,3] Although there is general consensus that catheter-induced spasm of the left main coronary artery can and does occur, the



Figures 1. *Selective angiography of the left main coronary artery (LMCA) in a LAO projection reveals an apparent significant stenotic lesion in its mid-portion.*

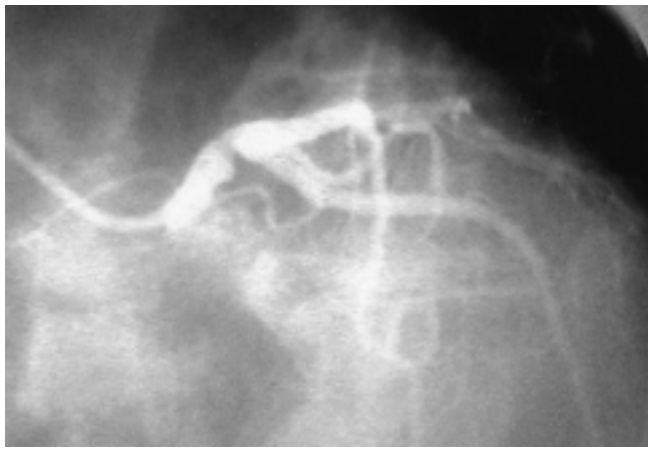


Figure 2. *Selective angiography of the left main coronary artery (LMCA) in a LAO projection reveals an apparent significant stenotic lesion in its mid-portion.*

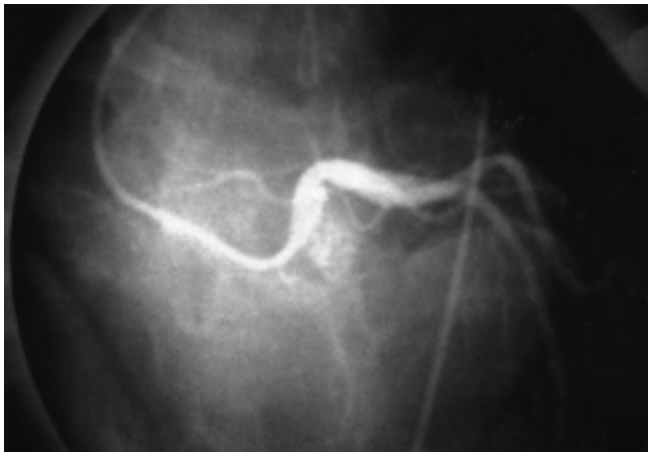


Figure 3. *Selective angiography of the LMCA after administration of intra-coronary nitroglycerin shows some improvement of the apparent stenosis, suggesting a component of spasm.*

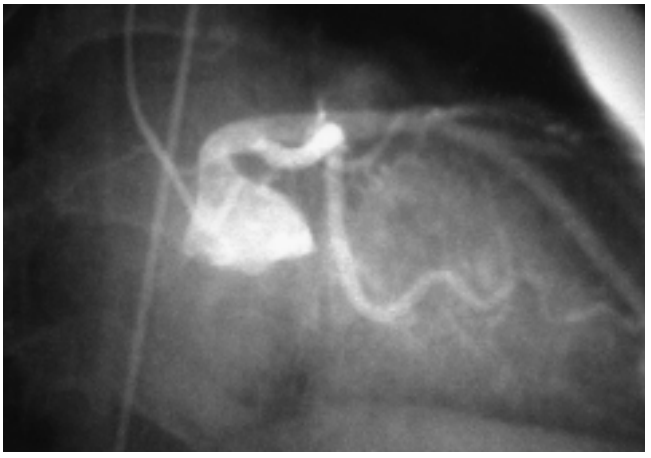


Figure 4. *Non-selective angiography of the LMCA, coupled with administration of intra-coronary nitroglycerin, demonstrates complete resolution of the spasm and reveals only an underlying 90° turn in its course.*

factors predisposing patients to this phenomenon are poorly defined. We did not find any reports in the literature on any anatomical variations of the left main coronary artery suspected in playing a role in its susceptibility to catheter-induced spasm. It is conceivable that certain anatomic variations in the length, caliber, course, or even origin of the left main coronary artery may predispose it to spasm. In the case of the right coronary artery, it is not unusual for a small and/or non-dominant vessel to develop spasm with catheter engagement of the ostium. However, this is not as common with the left main coronary artery, where it is rare for a truly non-diseased LMCA to be of such small caliber that spasm will occur with catheter engagement. However, rare, unusual bends in its course may theoretically predispose the artery to spasm, or lead to trauma and dissection by virtue of asymmetric engagement of a coronary catheter. In the patient presented here, this clearly appears to be the case. Abrupt engagement of the catheter into the transition zone of the vessel (i.e., at the bend in its course) led to spasm of the artery, with the subsequent appearance of an angiographically significant lesion. Non-selective injections of the LMCA, coupled with intracoronary nitroglycerin administration, demonstrated no evidence of LMCA disease, although there was an approximately 90º bend in the mid-portion of the vessel. Of note, during the catheterization performed two years earlier, there were no non-selective injections of the LMCA. This may explain why the vessel was presumed to have a fixed obstruction.

In this high-volume cardiac catheterization era, it is of utmost clinical importance to correctly identify the phenomenon of catheter-induced spasm of the left main coronary artery in order to make appropriate management decisions and to avoid unnecessary bypass surgery.[5] Although surgery has been the mainstay of therapy for fixed, significant left main coronary obstructions, the treatment of left main spasm is not well established. There is at least one reported case of left main spasm refractory to medical therapy and requiring coronary bypass surgery.[6] However, most patients with documented spasm of the left main coronary artery are treated medically with the combination of nitrates and calcium channel blocking agents. Due to the wide disparity between treatment strategies for fixed obstruction versus spasm of the left main coronary artery, it becomes imperative that the operators differentiate spasm from fixed obstruction during coronary angiography. The possibility of spasm should be especially entertained in the setting of anatomical variations in the course of the left main coronary artery, as occurred in this case.

Conclusion. We report a case of left main coronary spasm, which was initially diagnosed as a fixed obstructive lesion and subsequently recognized as spasm. Cardiac catheterization was followed by a negative dobutamine echocardiographic study, which supported the diagnosis of catheter-induced spasm rather than a fixed obstructive lesion. We propose that operators be particularly suspicious of catheter-induced spasm of the left main coronary artery in cases where there is significant tortuosity in the course of this artery. Such bends may predispose the artery to asymmetric engagement of the catheter with subsequent spasm or even trauma. In such cases, the operator should be especially diligent in ruling out catheter-induced spasm. This may require numerous non-selective injections, administration of intracoronary nitroglycerin and the avoidance of "abrupt" engagement of the catheter into the transition zone of the bend.

References

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How Would You Treat This Patient?

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Vasospasm of the left main coronary artery is an uncommonly described event for which clinicians have a very limited understanding. Among the few reports of left main coronary spasm, most descriptions are related to catheter-based procedures of diagnostic angiography or percutaneous revascularization. Conversely, the causal relationship of left main vasospasm to the over 335,000 sudden cardiac death events that occur annually in the United States is entirely uncertain.

In this report, *Lingegowda et al.* describe a patient in whom catheter-induced left main spasm was relieved only following administration of intracoronary nitroglycerin. Although removal of the catheter is in many instances the only effective method to relieve vasospasm, the operator must first be certain that antegrade flow is present, and that catheter-induced dissection has not occurred. In this latter instance, immediate revascularization may instead be required.

Among patients with observed vasospasm during catheter engagement, a remaining uncertainty is whether the observation of catheter-induced spasm implies a predisposition to spontaneous vasospasm and chest pain syndromes. In this particular case, considering the clinical implications of spontaneous left main vasospasm, I also would prescribe intensive medical therapy with vasodilators (typically calcium channel blockade with or without nitrates) and antiplatelet therapy with aspirin. Further, given the potential for abnormal endothelial function, I would also treat this patient with statin therapy, and if the patient had hypertension despite calcium channel blocker therapy, I would also include treatment with an ACE inhibitor.

Other secondary considerations for this patient would be the performance of multi-row detector CT angiography and measurement of markers of systemic inflammation. Although diagnostic cardiac catheterization enables assessment of the vessel lumen, CT angiography may help clarify the anatomical course of the left main, in addition to the presence or absence of intraluminal soft plaque, a finding that would only verify the need for intensive medical therapy. As a noninvasive imaging method, CT angiography would also obviate the risk of vasospasm induced by an intravascular ultrasound catheter. Second, assessment of systemic inflammation in a patient otherwise without angiographic evidence of atherosclerotic disease may also be of clinical use. Elevated measure of high-sensitivity C-reactive protein, for example, may also imply the need for medical therapy and risk factor modification. It is noteworthy, however, that elevated markers of inflammation may not be routinely associated with coronary vasospasm and superimposed thrombosis, but instead with the development of more progressive atherosclerosis.

When coronary vasospasm does occur, it is frequently associated with ventricular arrhythmias (specifically, torsade des pointes) and, potentially, sudden cardiac death. As a final recommendation, considering 1) the clinical implications of left main coronary vasospasm, and 2) the benefits of external automated defibrillators (AED), I would advocate this patient invest in an AED in the unlikely (but potential) event of witnessed sudden cardiac death.

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This case is an excellent demonstration of one of the many important challenges that face the invasive cardiologist and for which there are no randomized trials or other evidence-based knowledge sets. However, although we don't have specific evidence-based guidelines to fall back upon, the risk for the patient may be considerable. Indeed, when there is a question about the left main coronary artery, making the wrong decision may be disastrous.

This patient appears to have had catheter-induced spasm of the left main coronary artery triggered by a pre-existing anatomic kink of the normal vessel that responded well to intra-coronary nitroglycerin. In my experience, even after nitroglycerin administration, there often remains some question as to the status of the left main coronary artery. Under these circumstances, intravascular ultrasound can be very effective in clarifying the issue.

Thereafter, however, the question remains about what to do with this patient long-term. It is not always clear what causes coronary spasm, although an inherited natural tendency to have spontaneous spasm, such as in typical Prinzmetal's variant angina, may be an important contributor. Additionally, atherosclerosis-associated endothelial dysfunction may also increase the risk of coronary spasm. This patient had known coronary artery disease, and so aggressive secondary prevention therapy was already indicated. Also, although it is very possible that the spasm only occurred as a direct result of catheter trauma, the fact that left main coronary artery spasm is rare in patients undergoing coronary angiography indicates that she likely has an increased tendency toward coronary spasm. My strategy would be to treat the patient with a vasodilator, particularly when the left main coronary artery is a risk. Generally, I would use amlodipine and increase the dose as tolerated. Long-acting nitroglycerin is another alternative, although many patients cannot tolerate it because of the headaches it induces. Certainly, having the patient carry sublingual nitroglycerin for emergencies may be advisable.

Due to the risk associated with left main coronary artery occlusion, some may consider prophylactic coronary bypass surgery. I do not recommend this, however. Most patients with coronary spasm can expect to have a long and event-free life, though many of them will continue to have intermittent episodes of chest pain.