Successful Aspiration Thrombectomy for Cocaine-Induced Right Coronary Artery Embolism Secondary to Left Ventricular Thrombus: A Case Report and Literature Review

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Abstract:

Key Clinical Message:

Coronary embolism (CE), though rare, is an essential yet often overlooked cause of acute myocardial infarction. When CE arises from left ventricular thrombus (LVT), as demonstrated in this case, rapid diagnosis and targeted intervention are crucial. Aspiration thrombectomy is an effective, minimally invasive strategy to swiftly restore coronary flow.

CE is a rare yet significant contributor to acute myocardial infarction (AMI), particularly in younger patients. We report a 42-year-old male who experienced an AMI following recent cocaine use. Investigations revealed a left ventricular apical thrombus, identified as the source of a distal embolus occluding the right coronary artery (RCA). Cocaine is known for its thrombogenic effects, promoting thrombus formation through mechanisms such as vasoconstriction and increased platelet aggregation. This unique situation highlights the critical interplay between cocaine use, left ventricular thrombus formation, and the resultant coronary artery embolism, contributing to myocardial ischemia. The patient underwent successful aspiration thrombectomy, restoring coronary blood flow without the need for stenting. This case underscores the importance of recognizing CE as a potential etiology of AMI, particularly in the context of substance abuse, advocating for heightened awareness among clinicians regarding the complexities of managing thromboembolic events.

Keywords: Coronary Embolism, Left Ventricular Thrombus, Cocaine, Aspiration Thrombectomy

Introduction:

Coronary embolism (CE) is a significant yet underrecognized contributor to acute myocardial infarction (AMI), with prevalence estimates between 4% and 13% in angiographic and autopsy studies [1]. Current literature indicates that intracoronary thrombus formation following plaque rupture due to atherosclerosis is the most common mechanism of myocardial infarction; however, non-atherosclerotic causes, such as CE, must also be considered [2,3]. The management for CE differs from atherosclerotic causes, despite similar clinical presentations. In cases of CE, aspiration thrombectomy and balloon angioplasty are preferred, unlike AMI due to atherosclerosis, where coronary stenting is often indicated [4].

CE involves embolic material, such as thrombi, infectious elements, or neoplastic fragments, circulating to and obstructing the coronary arteries, leading to infarction of the myocardium supplied by the affected arteries. Multiple etiologies can give rise to CE, including paradoxical emboli from atrial septal defects, iatrogenic emboli from cardiothoracic procedures, and direct embolization from thrombi originating in the cardiac ventricles [5]. To our knowledge, no cases of the latter have been reported in the literature, particularly

in the context of cocaine abuse, highlighting the significance of this case report as a valuable contribution to to the literature.

Cocaine is recognized for its thrombogenic and angiospastic effects, promoting thromboembolism through several mechanisms[6]. Thrombi originating in the left ventricle or atrium are generally more likely to travel to the carotids or distal systemic circulation, given the linear course from the left ventricular outflow tract. However, coronary vessels are anatomically more protected from embolic events [7], making CE originating from a cardiac thrombus exceedingly rare, a factor that further underscores the uniqueness of our case.

Here, we present a rare case of a 42-year-old male who presented with chest pain following cocaine use. Investigations revealed an embolus obstructing the right coronary artery and a left ventricular apical thrombus on transthoracic echocardiography, which was proposed to be the embolic source, for which he underwent a successful aspiration thrombectomy.

Case History/examination:

A 42-year-old male presented to the emergency department with the acute onset of stabbing chest pain radiating to his back and left arm, accompanied by diaphoresis and nausea. He reported recent cocaine use earlier that morning. Upon arrival, his vital signs were a blood pressure of 126/91 mmHg, heart rate of 109 bpm, and oxygen saturation of 99%. The patient had a history of smoking approximately half a pack of tobacco per day since he was 19 years old, and reported weekend alcohol consumption of about six packs of beer and a pint of vodka per session. Notably, he had consumed four shots of rum the day before admission.

Methods:

An electrocardiogram (ECG) demonstrated ST-segment depressions in leads V5-6 and T-wave inversions in leads II and III. Initial troponin levels were elevated at 684 pg/mL, and he was also noted to have acute kidney injury. A computed tomography angiogram ruled out aortic dissection but revealed a 2.3 cm filling defect in the left ventricle, consistent with a thrombus (Figure 1). After administration of morphine and diazepam, the patient's chest pain improved. He was started on a heparin infusion, aspirin, clopidogrel, and atorvastatin.

Transthoracic echocardiography revealed a significantly reduced left ventricular ejection fraction of 10-15%, grade III diastolic dysfunction, and the presence of a left ventricular apical thrombus. A repeat ECG showed no new ST elevations, but persistent ST depressions in the high lateral leads and T-wave inversions in the inferior leads were noted. The repeat troponin level increased to 10,970 pg/mL. Given these findings, the decision was made to transfer the patient to the cardiac catheterization laboratory for further evaluation and admit him to the coronary care unit (CCU) for close monitoring, with a diagnosis of non-ST elevation myocardial infarction (NSTEMI).

Coronary angiography revealed a normal right coronary artery (RCA) proximally and mid-segment, but extensive clot burden and total occlusion distally (Figure 2). The left main artery, left anterior descending (LAD) artery, and left circumflex artery were free of significant disease. A left ventriculogram showed no visible ventricular thrombus, but left ventricular end-diastolic pressure (LVEDP) was elevated at 33 mmHg.

A decision was made to proceed with mechanical thrombectomy. Heparin was administered to maintain an activated clotting time of 300-350 seconds, and the RCA was engaged with a Judkins Right 4 (JR4) 6-French guide catheter. A balanced middleweight (BMW) coronary guidewire was used to cross the thrombus and was placed in the distal posterior descending artery. Multiple passes of mechanical thrombectomy were performed using the Penumbra CAT RX device. The wire was then repositioned into the right posterolateral ventricular branch, and another thrombectomy run was completed.

Conclusion and Results:

Post-procedure angiography demonstrated excellent results (Figure 3), with restoration of TIMI grade 3 flow, successfully revascularizing the right coronary artery without the need for balloon angioplasty or stenting. Due to the ongoing thrombus risk, the patient was started on an eptifibatide drip and remained in the CCU

on aspirin, clopidogrel, a statin, and a heparin infusion. He was monitored with serial ECGs and troponin levels.

Discussion:

CE is a non-atherosclerotic cause of AMI that can present as either STEMI or NSTEMI. CE accounts for 4% to 13% of acute MI cases, as observed in angiographic and autopsy studies. The first description of CE-related MI dates back to 1856 by Rudolf Virchow. The primary causes of CE include atrial fibrillation (AF), cardiomyopathy, and malignancy; however, 26.3% of cases have no discernible source[8,9].

CE can theoretically arise from three primary sources. The first is direct embolism, occurring from thrombi originating in the left atrial appendage, left ventricle, or heart valves, often due to conditions such as infective endocarditis or cardiac tumors. The second source is paradoxical embolism, which occurs when venous thrombi travel into systemic circulation through openings like a patent foramen ovale or atrial septal defects. The third source is iatrogenic embolism, which can result from medical procedures such as aortic valve replacements or percutaneous coronary interventions (PCI). Although coronary arteries are generally protected from systemic embolisms, risks like coronary air embolism can arise during PCI, potentially leading to vessel occlusion. Furthermore, complications such as device embolization and microthrombi during STEMI interventions can complicate diagnosis, especially when thrombi coexist with underlying atherosclerosis [8].

Diagnosing CE can be challenging due to its clinical presentation, which resembles atherosclerotic acute coronary syndromes (ACS). To help differentiate CE from atherosclerotic coronary artery disease (CAD), Shibata et al. introduced diagnostic criteria based on clinical and angiographic findings, classifying CE as either "definite" or "probable" (Table 1). In CE cases, coronary artery stenosis is typically minimal, aiding in distinguishing it from atherosclerotic causes. Imaging techniques such as angiography, transthoracic echocardiography (TTE), transesophageal echocardiography (TEE), and magnetic resonance imaging (MRI) are essential for identifying embolic sources, with AF being the most commonly implicated risk factor[4,9].

Coronary arteries are generally more protected from embolic events than other vascular systems due to several factors, including their smaller diameter, higher vascular resistance, and the anatomical positioning of the aortic valve and sinus of Valsalva, which reduce the likelihood of embolization during systole [4]. However, in patients with AF, dilated cardiomyopathy, or prosthetic heart valves, emboli can lodge in the coronary arteries, causing CE [10]. Notably, CE preferentially affects the left coronary system, occurring three to four times more frequently than the right, particularly in the left anterior descending artery, due to its anatomical orientation, which makes it more susceptible to embolization. In contrast, coronary vasospasm is more frequently reported in the proximal right coronary artery [1].

It is crucial to emphasize the relationship between MI and the presence of a left intraventricular thrombus. In these instances, CE can either serve as a cause, where MI leads to the formation of the ventricular thrombus, or as a consequence, where CE arises from a pre-existing ventricular thrombus [8].

Cocaine use stimulates the formation of intraventricular thrombus through sympathetic activation, resulting in prolonged coronary vasospasm and direct myocardial injury. Additionally, its prothrombotic effects further elevate the risk of intracardiac thrombus formation[6,11].

Left ventricular (LV) thrombus is a significant complication of LV dysfunction, closely associated with systemic embolism, morbidity, and mortality. Heart failure and acute MI, particularly STEMI, are common causes of LV thrombus. Reduced ejection fraction (EF) is prevalent in these cases, with 36% of patients having an EF <20%, 38.7% with an EF between 20% and 30%, and 25.3% with an EF between 30% and 40%. Cases with an EF between 41% and 49% are less common (8%), as are those with an EF [?]50% (5%) [12]. The LAD artery is the most frequently involved culprit artery, accounting for 77.8% of cases. In contrast, RCA occlusion, as seen in our patient, is rare. Thrombus formation is significantly related to the region with the most severe functional impairment, often reflecting infarction or scarring. Global LV dysfunction is present in 64.8% of cases, with regional dysfunction commonly affecting the apex (92.7%), anterior (26.8%), septal (26.8%), inferior (14.6%), and lateral (9.8%) regions[12,13].

The combination of blood stasis, endothelial injury, and hypercoagulability, collectively known as Virchow's triad, is essential for thrombus formation. In the context of acute MI, particularly associated with cocaine use, all three components of this triad are evident. Regional wall akinesia and dyskinesia contribute to blood stasis; prolonged ischemia leads to subendocardial tissue injury and inflammation; and cocaine use promotes a hypercoagulable state[6,13].

Cocaine is emerging as a significant cause of MI through both coronary vasospasm and thrombosis. It increases platelet aggregation and induces vasoconstriction, leading to thrombus formation within the coronary arteries. The drug's ability to enhance norepinephrine activity heightens sympathetic activation, resulting in coronary vasospasm that can lead to ischemia and MI. Cocaine not only enhances thromboxane A2 production, a promoter of platelet aggregation and vasoconstriction, but also reduces the release of prostacyclin, which normally inhibits platelet aggregation. These imbalances promote a prothrombotic state, while cocaine also increases platelet sensitivity to aggregating agents such as adenosine diphosphate (ADP) and epinephrine, further heightening platelet activity. The combination of endothelial dysfunction and increased platelet activation raises the risk of coronary thrombosis and MI, even in the absence of atherosclerotic disease. Additionally, chronic cocaine use can lead to premature atherosclerosis, cardiomyopathy, and conduction abnormalities, further elevating the risk of MI [6(p1),14].

The combination of smoking and cocaine use, as observed in our patient, amplifies the risk of cardiovascular events due to their combined impact on endothelial function and thrombotic pathways. Both substances cause endothelial injury and impair prostacyclin production, exacerbating platelet aggregation. Smoking also increases oxidative stress and inflammatory markers, further contributing to endothelial dysfunction[6(p1),14]. Additionally, chronic smokers have elevated fibrinogen levels, which, when combined with cocaine's effects, enhance platelet aggregation and increase the risk of thrombus formation. Moreover, the effects of cocaine can be exacerbated by concomitant alcohol consumption, which may also be relevant to our patient[6].

Aortic dissection is one of the cardiac manifestations that cocaine can induce, and it should be highly suspected in patients presenting with chest pain radiating to the back following cocaine use. In such cases, a CT angiogram can be utilized to rule out the condition. Cocaine has several pathophysiological effects that may increase the risk of aortic dissection. By inhibiting catecholamine reuptake, cocaine stimulates the autonomic nervous system, resulting in elevated heart rate and blood pressure. Additionally, cocaine acts synergistically with alpha agonists to induce vasoconstriction. Chronic cocaine consumption can alter the elastic properties of the aorta, leading to decreased strain, reduced distensibility, and increased aortic stiffness. Furthermore, the sudden and severe elevation of blood pressure induced by cocaine produces increased shear stress in the aorta, contributing to intimal disruption that allows the blood column to enter the media and leads to subsequent antegrade or retrograde propagation of the medial hematoma[15].

In terms of management, coronary interventions for CE differ from those for atherosclerotic MI. Thrombus aspiration and balloon angioplasty are preferred approaches, often eliminating the need for stent implantation[4]. Aoun et al. proposed a comprehensive algorithm for managing CE [16]. In cases of small, distal CE without hemodynamic instability, the recommended approach is anticoagulation therapy alone. However, in the presence of high intracoronary thrombus burden accompanied by instability, thrombus aspiration should be prioritized. If angiography indicates CE and the coronary anatomy is favorable, thrombectomy may be attempted. While the efficacy of intracoronary thrombolysis or glycoprotein IIb/IIIa inhibitors has not been established through randomized clinical trials, their use is frequently reported in clinical cases. Notably, Popovic et al. found that interventional procedures for CE involved significantly higher utilization of glycoprotein IIb/IIIa inhibitors compared to non-CE cases (73.9% vs. 37.7%), alongside a markedly lower rate of angioplasty (45.3% vs. 95.5%) [10]. In instances where thrombus aspiration results in a TIMI flow grade of less than 2, primary stenting may be considered. Conversely, if TIMI flow is 2 or greater following thrombus aspiration, intravascular ultrasound or optical coherence tomography may be employed to investigate potential plaque erosion. A minimalistic revascularization strategy aimed at achieving TIMI flow grade 3 is preferred, ideally without the need for stenting[8].

The Penumbra CAT RX has demonstrated significant efficacy and safety in thrombus removal in acute

settings, without the heightened risk of systemic emboli associated with manual aspiration thrombectomy. Approved for coronary use in 2017, this device features an atraumatic catheter combined with a sustained vacuum source, providing consistent power aspiration throughout the procedure. Clinical outcomes reveal that after the use of the CAT RX, 88% of patients achieved a post-procedure TIMI flow of 3, 9% attained TIMI 2 flow, and 3% experienced TIMI 0 flow[17].

In conclusion, this case underscores the critical need for heightened awareness of coronary embolism (CE) as a potential cause of acute myocardial infarction (AMI), particularly in younger patients with a history of substance abuse, such as cocaine use. The interplay between cocaine's thrombogenic effects, the formation of a left ventricular thrombus, and subsequent coronary artery embolism highlights the complexities of diagnosing and managing such cases. Successful aspiration thrombectomy in this patient not only restored coronary blood flow but also emphasizes the importance of tailored therapeutic approaches in managing CE, differing from conventional treatment strategies for atherosclerotic causes. Clinicians should consider CE in the differential diagnosis of AMI, especially in patients with risk factors associated with thrombus formation, and remain vigilant for potential complications arising from both substance use and cardiovascular pathology.

Declarations:

Ethical approval: Our institution does not require ethical approval for reporting individual cases or case series.

Informed consent to participate: Written informed consent was obtained from the patient himself. The participant has consented to the submission of the case report to the journal.

Competing interests: On behalf of all authors, the corresponding author states that there is no conflict of interest.

Data availability statement: Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

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Authorship Contributions

Abraam Rezkalla and Ahmad Nouri : contributed equally to the article. They were both involved in case conceptualization, literature review, and manuscript drafting.

Fadu Hadya: contributed to drafting the discussion, conducting the literature search, and editing the manuscript.

Islam Rajab: contributed to data acquisition, drafting the discussion, and manuscript editing.

Emile Doss: prepared the figures, managed clinical aspects of the case, and oversaw the final revisions.

All authors reviewed and revised the manuscript and approved the final version for submission.

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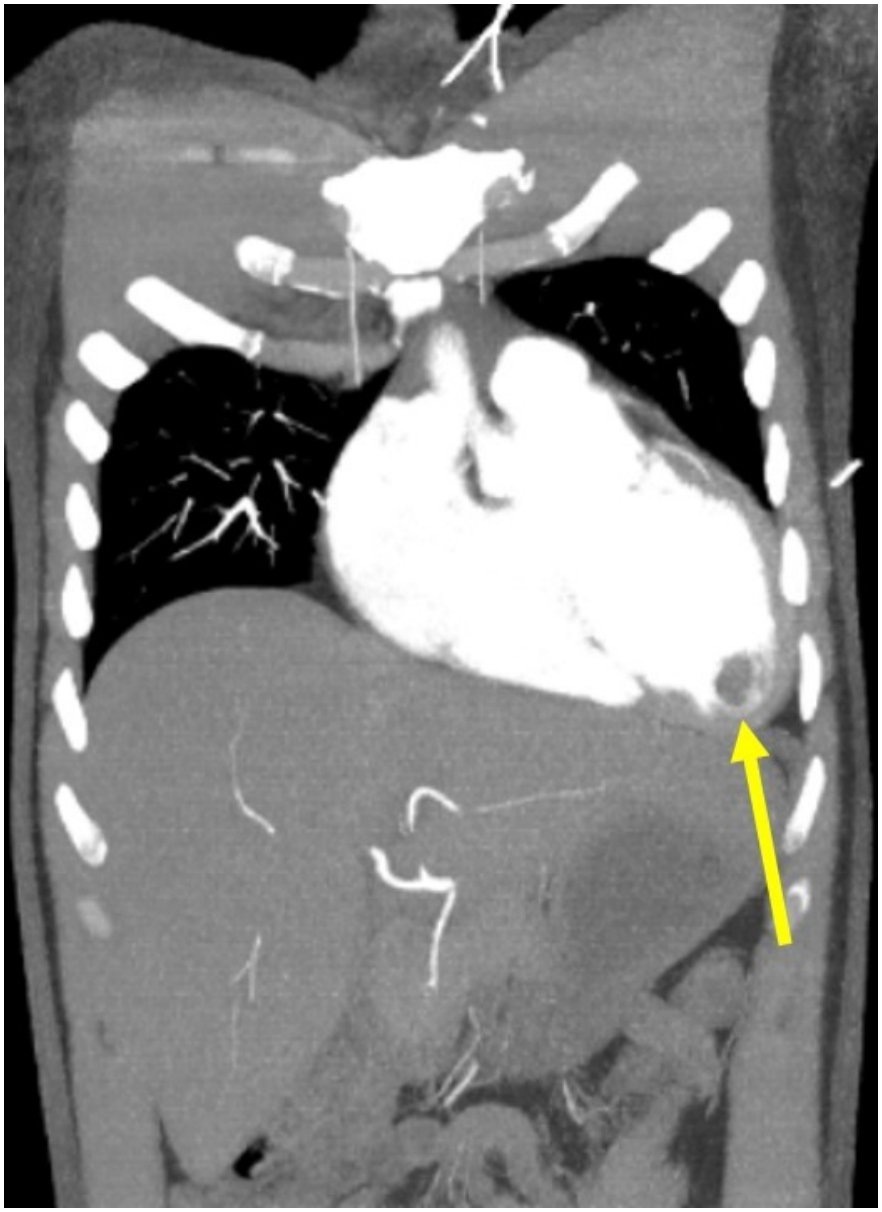

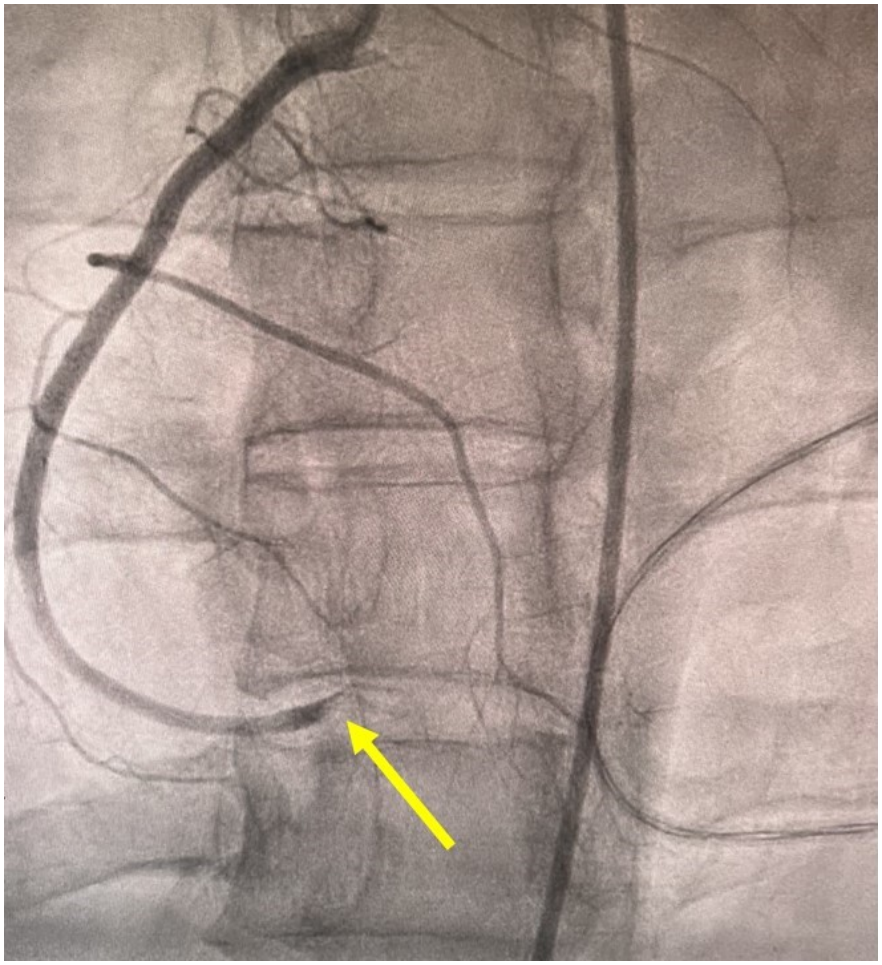

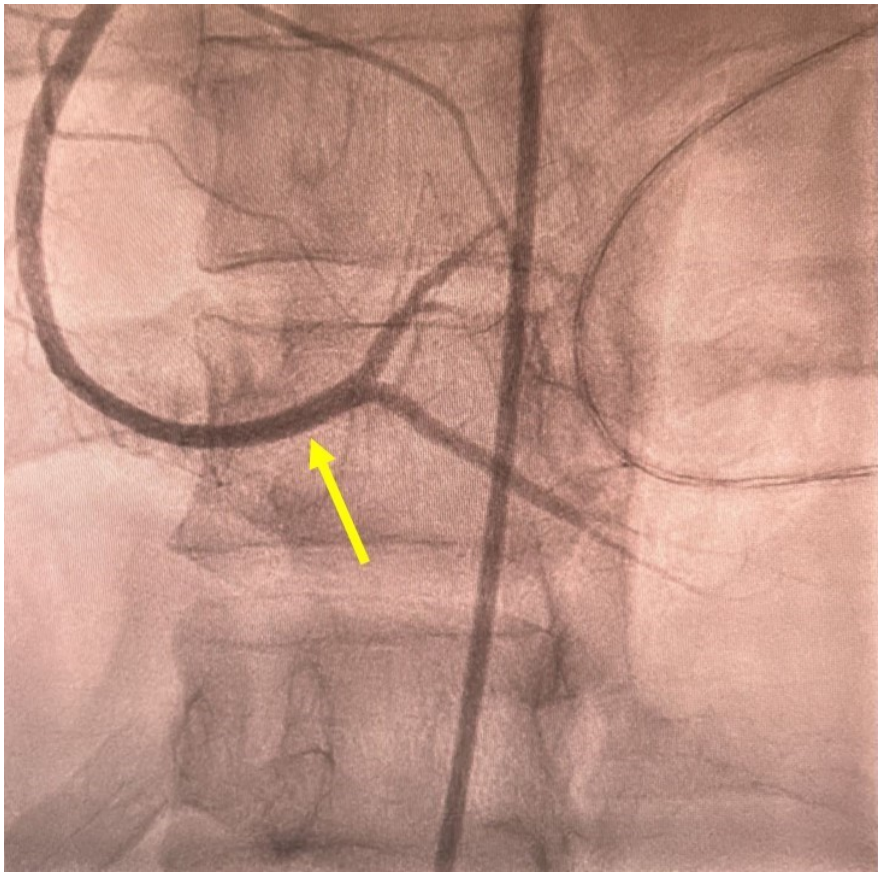

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