



CASE REPORT

Splenic Subcapsular Hematoma Presenting as Acute Abdomen After Brachial Access Coronary Angiography: A Rare Complication

✉ Melik Demir¹, ✉ Ömer Ferudun Akkuş¹, ✉ Gökay Taylan²

¹Clinic of Cardiology, Sultan 1. Murat State Hospital, Edirne, Türkiye

²Department of Cardiology, Trakya University Faculty of Medicine, Edirne, Türkiye

ABSTRACT

Percutaneous coronary intervention performed via upper-extremity access is a highly favorable clinical strategy because it is associated with significantly lower access-site bleeding rates and improved patient comfort. However, mechanical catheter manipulation and hydrophilic guidewire advancement into the descending aorta may occasionally result in rare but life-threatening visceral organ injuries. A 62-year-old woman presenting with non-ST-elevation myocardial infarction underwent coronary angiography via the right brachial artery using a 6-Fr sheath and a hydrophilic guidewire after unsuccessful bilateral femoral access attempts. Because the occlusion in the right coronary artery could not be crossed, aggressive intervention was deferred in favor of conservative medical management. Within 24 hours after the procedure, the patient developed sudden hemodynamic instability and severe anemia (hemoglobin level of 6 g/dL). Contrast-enhanced abdominal computed tomography revealed a 12×35 mm subcapsular splenic hematoma with associated hemoperitoneum. Following emergency splenectomy, the patient was successfully extubated on postoperative day 3 and discharged in stable condition while receiving dual antithrombotic therapy. In upper-extremity catheterization, altered entry angles through the complex aortic arch, combined with the low-friction properties of hydrophilic guidewires, may increase the risk of subclinical visceral branch microtrauma or focal ischemic infarction, which can subsequently progress to massive intra-abdominal hemorrhage in patients receiving potent antithrombotic therapy. Interventional cardiologists should maintain a high index of suspicion for occult intra-abdominal injury in patients with unexplained postprocedural hypotension and a marked decline in hemoglobin, facilitating prompt cross-sectional imaging and immediate surgical consultation.

Keywords: Acute abdomen, brachial access, percutaneous coronary intervention, splenic rupture, subcapsular hematoma

INTRODUCTION

Percutaneous coronary intervention (PCI) is widely and safely performed for the diagnosis and treatment of ischemic heart disease.¹ However, because it is an invasive procedure, coronary angiography and PCI may be associated with various cardiac and systemic complications.² Although the radial artery is the preferred vascular access site for coronary angiography because of its lower risk of access-site bleeding, the brachial artery remains an important alternative in patients with unsuitable vascular anatomy. Upper-extremity approaches, including radial and brachial access, are generally favored because they are associated with lower bleeding rates and greater patient comfort. Nevertheless, these approaches remain susceptible to unique procedural complications. Catheter manipulation through upper-extremity access may increase the risk of subclinical microtrauma to the side branches of the descending aorta. This access route may create a superior entry angle into the descending aorta, directly

opposing the cranially oriented origins of visceral branches, such as the celiac trunk and superior mesenteric artery. In contrast to the relatively linear trajectory of the femoral approach, upper-extremity catheterization requires navigation through the complex anatomy of the aortic arch, which may reduce coaxial support and mechanical leverage. During aggressive attempts to cross technically challenging or totally occluded lesions, this altered entry angle may impair torque transmission and increase catheter tip displacement caused by respiratory motion. In addition, hydrophilic-coated guidewires, which are commonly selected for upper-extremity procedures to minimize vascular spasm and facilitate passage through tortuous vessels, may be inadvertently directed into the descending rather than the ascending aorta because of respiratory movement or anatomical variation. Their low-friction surface and high trackability allow them to enter the origins of descending aortic side branches, including the celiac trunk and intercostal or lumbar arteries, with minimal tactile resistance, thereby increasing potentially the risk of vascular injury,

Address for Correspondence: Melik Demir MD, Department of Cardiology, Kırklareli University Faculty of Medicine, Kırklareli, Türkiye

E-mail: melikdemir34@gmail.com **ORCID ID:** orcid.org/0000-0003-1711-6707

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dissection, and subsequent visceral rupture.³ In patients receiving intensive anticoagulant therapy, intra-abdominal solid organ injury following PCI is an extremely rare, unpredictable, but potentially fatal complication.⁴

CASE REPORT

A 62-year-old woman with a history of hypertension presented to the emergency department with persistent chest pain and dyspnea that had begun 24-48 hours earlier. She had no history of abdominal trauma, preexisting splenic disease, hematologic disorders, or chronic liver disease. The initial electrocardiogram showed normal sinus rhythm. On admission, her blood pressure was 110/70 mmHg, her heart rate was 95 bpm, her high-sensitivity cardiac troponin level was 681 ng/L, and her hemoglobin level was 13 g/dL.

With a preliminary diagnosis of non-ST-elevation myocardial infarction, the patient received loading doses of 300 mg aspirin, 600 mg clopidogrel, and 5,000 U of unfractionated heparin and was immediately transferred to the catheterization laboratory for coronary angiography. During the procedure, bilateral femoral sheath insertion was unsuccessful. Because the radial pulses were non-palpable, coronary angiography was performed via the right brachial artery using a 6-Fr sheath. Coronary angiography was performed with 6-Fr JR4 and JL4 diagnostic catheters advanced over a hydrophilic guidewire. The culprit lesion was identified distal to the right ventricular branch of the right coronary artery (RCA) (Figure 1).

The RCA was engaged with a 6-Fr JR4 guiding catheter to initiate the intervention. Angiography revealed a totally occluded culprit lesion in the RCA with faint distal filling through acute collateral branches. However, the lesion could not be crossed despite the use of a floppy

guidewire, the parallel-wire technique, and stiffer coronary guidewires. Because the patient remained hemodynamically stable and free of active chest pain during the procedure, further aggressive intervention was deferred, and conservative medical management was selected (Figure 2). The total procedure time was 45 minutes, with a fluoroscopy time of 18 minutes.

The patient was transferred to the coronary care unit for postprocedural monitoring. Within the first 24 hours, she developed sudden hypotension accompanied by a marked decrease in hemoglobin levels. Because her hemoglobin level fell to 6 g/dL, an emergency red blood cell transfusion was initiated. Owing to persistent hemodynamic instability and signs of an acute abdomen, contrast-enhanced computed tomography of the abdomen and pelvis was performed promptly, revealing splenic rupture with a subcapsular hematoma (Figure 3). The patient underwent emergency splenectomy by the general surgery team. She was successfully extubated on postoperative day 3, and hemodynamic stability was achieved. Following postoperative treatment with low-molecular-weight heparin and stabilization of her hemoglobin level, dual antiplatelet therapy was safely resumed, and she was discharged in stable condition.

DISCUSSION

Intra-abdominal visceral hemorrhage, particularly splenic subcapsular hematoma or rupture, following coronary angiography or PCI is an exceptionally rare but life-threatening complication reported in the interventional cardiology literature. Although procedural visceral organ injuries have traditionally been associated with transfemoral access or intra-aortic balloon pump use, only a limited number of cases of splenic injury have been reported following diagnostic catheterization or PCI performed via either femoral or upper-extremity access. In

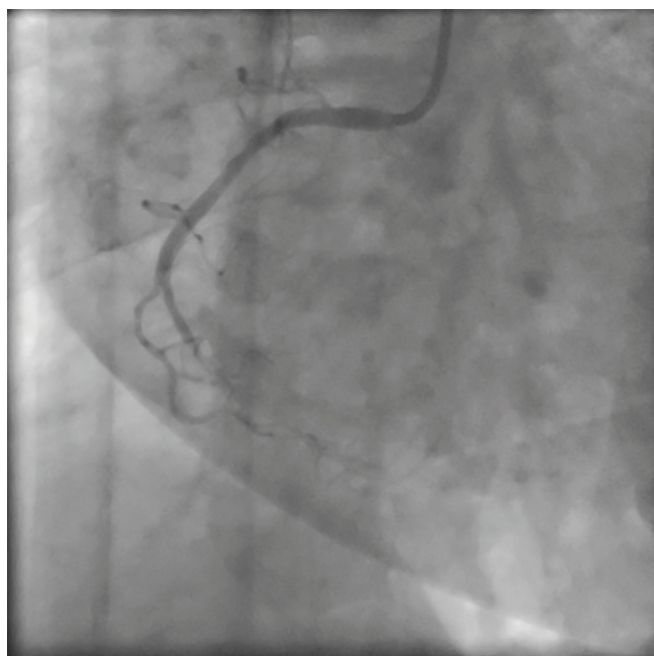


Figure 1. Selective right coronary angiography performed via right brachial access using a 6-Fr JR4 diagnostic catheter, demonstrating an acutely occluded culprit lesion in the distal segment of the right coronary artery (arrow)



Figure 2. Final post-procedural right coronary angiography image following unsuccessful crossing attempts with coronary wires, confirming persistent complete occlusion of the distal right coronary artery (arrow)

these cases, the proposed mechanisms primarily involved procedural microembolization leading to splenic infarction with secondary hemorrhagic transformation during intensive antithrombotic therapy or subtle guidewire-induced vascular microtrauma.⁵

In the present case, the development of a massive subcapsular splenic hematoma following an unsuccessful coronary intervention via brachial access underscores the importance of maintaining a high level of clinical vigilance and multidisciplinary management. From a pathophysiological perspective, establishing a direct causal relationship between upper-extremity access and intra-abdominal visceral injury based on a single case remains speculative. However, we hypothesize that a combination of biomechanical, anatomical, and pharmacological factors contributed to this complication. Unlike the relatively linear trajectory of transfemoral access, upper-extremity catheterization (via the brachial or radial artery) creates a superior entry angle into the descending aorta, directly opposing the cranially oriented origins of visceral branches, such as the celiac trunk. The use of hydrophilic-coated guidewires to overcome arterial spasm or vessel tortuosity, together with respiratory motion or anatomical variation, may inadvertently direct the guidewire into the descending aorta rather than the ascending aorta.⁶ Because of their low-friction surface and high trackability, these guidewires may inadvertently engage or microscopically penetrate the origins of descending aortic side branches without appreciable tactile resistance, particularly during aggressive catheter manipulation and torque transmission in technically challenging coronary lesions. Alternative causes, including spontaneous splenic rupture, preexisting splenic disease, or blunt abdominal trauma, were considered unlikely based on the patient's clinical history and the post-splenectomy histopathological findings, which demonstrated benign splenic parenchyma. Nevertheless, procedural microembolization leading to focal splenic ischemia cannot be completely excluded.³ Regardless of whether the initial

event was subtle mechanical injury to a visceral branch or subclinical embolic infarction, the intensive antithrombotic regimen, consisting of dual antiplatelet therapy and systemic unfractionated heparin administered for acute coronary syndrome, likely contributed to the progression of a minor parenchymal injury into a massive subcapsular hematoma and subsequent splenic rupture.⁴

CONCLUSION

Although upper-extremity interventional approaches remain highly favorable in contemporary practice because of their lower access-site bleeding rates, clinicians should recognize that catheter manipulation through the complex anatomy of the aortic arch may, under certain circumstances, contribute to visceral vascular injury. Given the inherent limitations of a single case report, any direct causal relationship between catheter manipulation and intra-abdominal organ injury should be interpreted with caution. Nevertheless, interventional cardiologists should not attribute unexplained postprocedural hypotension, flank pain, or transfusion-refractory anemia solely to cardiac or access-site causes. Prompt multidisciplinary collaboration with general surgery and timely abdominal imaging are essential to identify or exclude occult intra-abdominal pathology in this vulnerable patient population.

Informed Consent: Written informed consent was obtained from the patient for publication of this case report.

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Figure 3. Axial contrast-enhanced abdominal computed tomography scan demonstrating a prominent subcapsular splenic hematoma measuring approximately 12×35 mm in maximum dimensions (arrow), accompanied by perisplenic free intra-abdominal fluid (hemoperitoneum)

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