

observed 1 severe adverse reaction alone, although a 0.9% nonfatal shock rate in 352 consecutive patients during 4 years has been reported.²

Severe myocardial ischemia secondary to an anaphylactic reaction has been observed in patients with coronary arteries without lesions, and has been induced by circumstances as varied as eating shellfish, insect bites or the use of different drugs.³ Electrocardiographic findings (ST-segment depression in lead I together with ST-segment elevation in II, III, VF and ST-segment depression in precordial leads, but with mild elevation in V₆) and the absence of significant lesions in the coronary angiography suggest right coronary spasm before the AV node artery, but after the RV artery as the most probable mechanism of action. However, it cannot be ruled out that critical hypoperfusion due to the severe hypotension that always accompanies an anaphylactic reaction may have also contributed to the event. The main pathogenic mechanism by which anaphylactic reactions are linked to coronary ischemia is via the release and activation of vasoactive substances produced by mast cells in the human heart. Antispasmodic agents were not considered given that contrast agents will never again be used in this patient. The composition of the microbubble shell triggers the anaphylactic reaction rather than the gas core, and this may be the reason why some contrast agents cause more adverse effects than others.

Contrast echocardiography rather than conventional echocardiography is indispensable when quantifying volumes, since it provides the accuracy and reproducibility of cardiac magnetic resonance imaging. Safety studies support their general use, but

only providing staffing and technical requirements are met such that any adverse effect can be dealt with immediately and effectively.⁴

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Percutaneous Coronary Intervention Through an Axillo-Bifemoral Bypass

Intervención coronaria percutánea a través de puente axilobifemoral

To the Editor,

Use of radial access has increased spectacularly. At the time of writing, it is found in 45% of all percutaneous coronary interventions in Spain.¹ However, despite this, femoral access remains the most frequently used site in many catheterization laboratories. This is especially the case in North America and in patients in whom radial access cannot be canalized or when large caliber catheters are needed for complex procedures. We present the case of a patient with peripheral vascular disease and totally unsuitable for radial access who underwent coronary angiography and percutaneous intervention via a right axillo-femoral bypass.

A 61-year-old man with a history of smoking, high blood pressure, and hypercholesterolemia was admitted to a regional hospital with acute non-ST segment elevation coronary syndrome. In 2002, he had an aorto-bifemoral bypass for Leriche syndrome with juxta-renal obstruction of the aorta. Six years later he presented symptoms compatible with graft thrombosis. He was indicated for urgent arteriography and right branch thrombosis was confirmed. The patient received fibrinolysis, which was initially effective. However, when treatment ended he had another thrombotic episode leading to the decision for an urgent axillo-bifemoral bypass intervention that placed an 8 mm polytetrafluoroethylene (PTFE) prosthesis from the right axillary artery to the deep femoral artery. At the time, coronary angiography was

indicated at a regional hospital and the patient's clinical history showed the aorto-bifemoral bypass with no mention of the urgent axillo-femoral bypass. Echocardiography indicated normal ejection fraction. Radial access was attempted on both sides but the radial delivery catheter's hydrophilic guidewire could not advance.

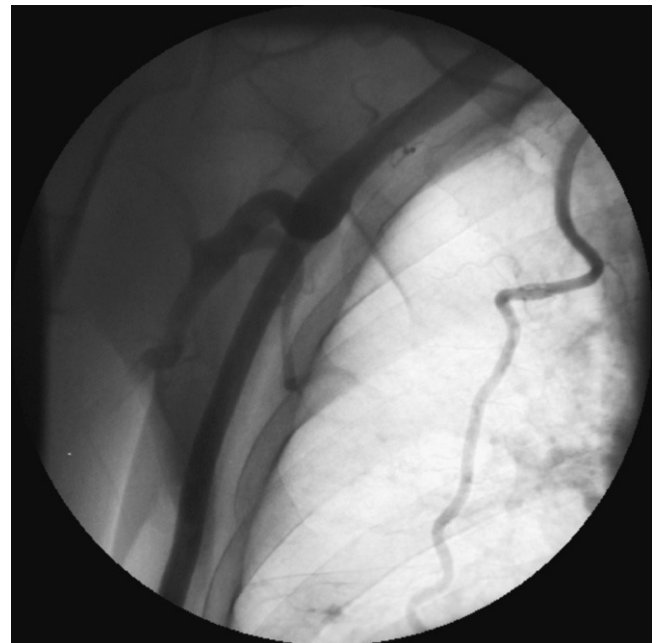


Figure 1. Contrast injection in right axillary artery. The brachial artery can be seen on the left. The axillo-bifemoral graft is in the center of the photograph and the right mammary artery is to the right.

Given the existence of a very weak brachial pulse and good femoral artery pulse, coronary angiography was performed via the right femoral artery. The catheter was inserted with no complications and the PTFE prosthesis puncture dilated with a 6F introducer dilatation catheter (Cordis, Johnson & Johnson). The artery was canalized with the 6F delivery catheter and the guidewire and dilatation catheter extracted. When the JL4 6F catheter was advanced over the metal guidewire we discovered the patient had an axillo-bifemoral bypass, not just the aorto-bifemoral bypass described (Fig. 1). We photographed the left coronary artery and a 285 cm exchange guidewire was used to complete coronary angiography via the JR4 6F catheter. This showed no significant lesions in the left coronary artery and a long, severe lesion in the proximal and mid right descending coronary artery. Successful percutaneous placement of two drug-eluting stents was via a JR4 6F catheter guidewire similar to that used in femoral and radial access interventions. The procedure was completed with deferred manual extraction of the femoral introducer at 6 h post-procedure. The patient was discharged next day with no complications.

Although percutaneous procedures on PTFE prostheses entailing puncture with the Seldinger technique have been described previously,^{2,3} to our knowledge this is the first time this percutaneous coronary intervention has been performed via an axillo-bifemoral bypass. As radial and brachial access are possible on both sides, the need to resort to this access site can only arise in exceptional circumstances but may sometimes be of use. The procedure was conducted with standard percutaneous intervention materials with no difficulty. Hence, questions such as a

theoretical limitation due to catheter length, difficulty in advancing through the graft, or lack of anatomy support, seem to be answered by the successful outcome of this case.

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Elderly Patient With Platypnea-Orthodeoxia: Check the Ascending Aorta

Paciente anciana con platipnea-ortodesoxia: hay que verificar la aorta ascendente

To the Editor,

A previously asymptomatic 78-year-old woman was admitted because of dyspnea and a sense of fainting when moving from the recumbent to the erect position. When supine the patient was not dyspneic and on clinical examination a soft basal diastolic murmur was heard. Arterial blood oxygenation on room air was 95%. On standing up, she became tachypneic and cyanotic, with a blood desaturation to 84%, not responding to supplemental oxygen. The presence of a platypnea-orthodeoxia syndrome (POS) was evident, while signs of orthostatic hypotension were absent. The electrocardiogram was normal. Chest radiography showed an enlarged and tortuous thoracic aorta.

A transthoracic echocardiogram revealed normal ventricles and left atrium. A dilated ascending aorta of 6.0 cm was distorting and severely reducing the right atrium (Fig. 1A). Slight aortic and tricuspid valve regurgitations were noticed. Systolic pulmonary artery pressure was estimated at 35 mmHg. When agitated saline was injected intravenously in a seated position, microbubbles appeared immediately in the left atrium, confirming the existence of a right to left intracardiac shunt (Fig. 1B). With the patient in the recumbent position, the quantity of bubbles passing to the left cardiac chambers was dramatically reduced. The location of the shunt was not apparent transthoracically, but transesophageally a patent foramen ovale (PFO) of 5 mm was found and the amount of

shunt was calculated at 25% (Fig. 1C). Although the early appearance of contrast in the left atrium made the existence of an intrapulmonary fistula unlikely, a chest computerized tomography (CT) and magnetic resonance imaging were deemed necessary. In particular, chest CT showed the aortic aneurysm severely deforming the right atrium to a vermiform cavity (Fig. 1D). No signs of intrapulmonary arteriovenous malformations were found. The patient was referred for resection and grafting of the ascending aortic aneurysm, with closure of the PFO. Unfortunately, the planned interventions were cancelled 10 days later, when the patient sustained a hemorrhagic stroke.

A POS consists of dyspnea and arterial oxygen desaturation when assuming the sitting or erect position from the recumbent one.¹ A right-to-left interatrial shunt through a PFO is frequently the anatomical substrate that is responsible.^{2,3} Persistence of a PFO in adulthood reaches a prevalence of almost 25%, usually remaining clinically silent. If right atrial pressure exceeds the left one, a right-to-left interatrial shunt can manifest paradoxical embolisms or POS, sometimes provoking cryptogenic strokes. Alternatively it can present even in the absence of elevated right atrial pressure. This is a case of a position- and not a pressure-mediated shunt. An enlarged ascending aorta can distort the right atrium, giving a horizontal direction to the atrial septum and a counterclockwise rotation of the heart.^{4,5} This distortion favors a caval inflow against a PFO, especially in the sitting and erect position, when the postural changes may exacerbate a right-to-left shunt.⁶

As life expectancy is increasing, aortic dilatation will become more common. When elderly patients present with otherwise unexplained dyspnea, positional manipulations should be performed. If the suspicion of POS is raised, the possibility of an aneurysmatic ascending aorta distorting a previously silent PFO should not be overlooked.