Case Report

Massive bowel infarction after percutaneous transluminal renal angioplasty

Daniela Ricciardi, Davide Rossi and Riccardo Maria Fagugli

Nephrology and Dialysis Department, Silvestrini Hospital, Perugia, Italy

Keywords: atherosclerotic renal artery stenosis; cholesterol crystal embolization; percutaneous transluminal renal angioplasty

Introduction

We report the case of a patient with atherosclerotic renal artery stenosis (ARAS) who developed massive bowel infarction caused by cholesterol crystal embolization (CCE) after percutaneous transluminal renal angioplasty (PTRA).

CCE is a complication of diffuse atherosclerosis, and it is responsible for a variety of complex clinical findings due to various degrees of multi-organ damage; it can range from being clinically silent to resembling other systemic diseases, such as vasculitis [1]. CCE in the vessels of the digestive tract suggests a very advanced and diffuse atherosclerosis with a serious outcome. Although CCE is a recognized complication of intra-arterial catheter manipulation, bowel infarction due to CCE after percutaneous treatment of ARAS is rare. Considering that the best treatment of atheroembolic disease is prevention, clinicians should be aware of this condition, especially in view of the increasing size of the population with atherosclerosis that undergoes invasive vascular procedures. This case may suggest the utility of an accurate evaluation of the atherosclerotic condition prior to PTRA, especially in those patients with a higher risk of CCE (elderly patients with diffuse and severe atherosclerosis or previous cardiovascular complications).

Case

A 75-year-old Caucasian male was admitted to hospital because of rapidly worsening renal function (serum creatinine rising from 2.1 to 5 mg/dl within 2 months). He had previously been monitored in the out-patient nephrology service of the hospital, and non-invasive diagnostic tests performed prior to his admission had shown severe nephro-angiosclerosis on the right kidney and ostial renal artery stenosis on the left side. The patient had a history of smoking, arterial hypertension, chronic renal failure and clinical manifestations of diffuse atherosclerosis (myocardial ischaemia, transient ischaemic attack, abdominal aortic aneurysm treated with aorto-aortic bypass graft, and occasional abdominal angina). He was under aggressive pharmacological treatment with statins, antiplatelet agents and angiotensin-converting enzyme (ACE) inhibitors. Signs of a deterioration of the left renal artery stenosis were present on admission, and an aortic angiography was performed, on which the aorto-aortic bypass graft appeared in good condition and significant ostial stenosis of the left renal artery was seen, which was treated with balloon angioplasty and stent placement. An angiogram obtained after the procedure showed good final angiographic appearance of the renal artery (Figure 1, arrow).

Intra-arterial heparin was given during the procedure and, afterwards, the antiplatelet drug was restarted. After that, the patient had epigastric pain, vomiting, elevated blood pressure (160/70 mmHg) and tachycardia (heart rate 130 b.p.m.). Physical examination of the abdomen was normal. Laboratory tests, performed on the same day as the angiography, showed an immediate increase of lactate dehydrogenase (676 U/l) and of the white blood cell count (from 9800 to 14 400/mm³). By the next day, renal function had improved (creatinine 1.55 mg/dl), the patient was asymptomatic, and his examination failed to reveal pathological conditions. On the second day, he had increasing abdominal pain and vomiting for a second time. The physical examination of his abdomen was normal. Laboratory tests, performed on the same day as the angiography, showed an immediate increase of lactate dehydrogenase (676 U/l) and of the white blood cell count (from 9800 to 14 400/mm³). By the next day, renal function had improved (creatinine 1.55 mg/dl), the patient was asymptomatic, and his examination failed to reveal pathological conditions. On the second day, he had increasing abdominal pain and vomiting for a second time. The physical examination of his abdomen revealed a diffuse tenderness, guarding to palpation, and the total absence of bowel sounds. A nasogastric tube was inserted, resulting in drainage of a large volume of fluid. Laboratory examinations showed a further rise in lactate dehydrogenase (777 U/l) and an
increase of aspartate transaminase (66 U/l) and creatine kinase (358 U/l). The white blood cell count was 16,910/mm³. An X-ray of the abdomen showed the presence of multiple air-fluid levels. A diagnosis of abdominal infarction was made, and the patient underwent a resection of the small bowel and ascending and transverse colons for massive infarction. In the post-operative period, because of the extent of the bowel resection, the patient needed parenteral nutritional support to maintain good nutritional status and a positive volume balance. He was discharged in improved condition on the 20th hospital day, on total parenteral nutrition. His management at home was difficult, and a negative fluid balance was progressively evident. Twenty days after discharge, he was admitted to hospital because of deterioration of renal function (creatinine 4.9 mg/dl) and hypotension. The patient was rehydrated, restoring a normal blood pressure, but his kidney function did not recover. Haemodialysis was begun, but after few days he died from an ischaemic stroke.

Discussion

CCE is defined as the migration of cholesterol crystals from the ulcerated atherosclerotic plaques of large or medium sized arteries into small arteries, arterioles and capillaries, where they elicit an inflammatory response with macrophage, giant cell and eosinophilic infiltration. The final result is multifocal ischaemic lesions and progressive tissue loss [1,2]. This process often leads to ischaemic injury to the skin, kidney, brain, myocardium and intestine, but any organ distal to the lesion may be affected [1]. Although spontaneous atheroma rupture is possible, CCE is more often observed after invasive vascular procedures (angiography, balloon angioplasty, stent deployment or vascular surgery) or as a complication of anticoagulant/thrombolytic treatment [1,3,4]. The precise overall incidence of CCE is poorly defined, ranging from 0.15–3.4% in autopsies of unselected populations (5) to 25–77% in patients who died after angiography or vascular surgery (6). With regard to gastrointestinal involvement, autopsies have shown it to occur in one-third of CCE cases [1,2].

Percutaneous renal artery stent placement, which is one of the therapeutic options in ARAS, is not without risk of significant morbidity. A variety of complications have been reported related to this procedure, such as haematoma at the puncture site, azotaemia from the dye load, cholesterol emboli, dissection or occlusion of the renal artery, retroperitoneal bleeding and occasionally death. CCE complicating renal artery stenting can occur in 10% of patients treated with PTRA for ARAS [7]. We have reported here a case of superior mesenteric artery embolism after PTRA due to dislodgement of atheroembolic material from the coeliac trunk. There are no published data on the incidence of bowel ischaemia due to CCE after PTRA, only very few anecdotal reports [8,9].

If we consider the PTRA technique, the preliminary step consists of a midstream angiogram obtained with a pig-tail catheter located just above the expected origin of the renal arteries. Then a guidewire is introduced and its tip is directed toward the target renal artery. The examination of the angiogram of the abdominal aorta of our patient showed the presence of atherosclerotic plaques throughout the coeliac trunk (Figure 2, arrow 1).
We hypothesized that atheromatous fragments might have been dislodged from the atherosclerotic plaques of the coeliac trunk into the superior mesenteric artery as a result of direct mechanical trauma or sudden arterial distension during the aortic angiography, leading to bowel ischaemia. Although the image is non-selective (Figure 2, arrow 2), the arteriogram obtained before PTRA shows a narrowed superior mesenteric artery, in agreement with symptoms described by the patient.

We can expect that the frequency of CCE will increase in the future, because of the rise in indications for vascular surgery, angiography and angioplasty in a population that is becoming older and often is obese and diabetic. These invasive vascular procedures may induce rupture of atherosclerotic plaques with subsequent dislodgement of cholesterol crystals into the bloodstream. Considering the deleterious consequences that CCE might have, the best policy is to try to prevent the atheroembolic disease. In the specific case of ARAS, nephrologists should be particularly aware of the risk of CCE and should accurately weigh up the possible benefits and risks of a PTRA. In fact, the current published data strongly support the use of renal revascularization in only a few cases, such as in the case of patients with severe stenoses in their sole functioning kidneys. The effect of PTRA on renal function in unilateral or mild bilateral disease has not yet been elucidated through large, controlled randomized trials. For this reason, nephrologists could be allowed to treat conservatively those patients who have higher risks of CCE (during PTRA) and who do not have very strong indications for revascularization. In elderly patients with cardiovascular risk factors, diffuse atherosclerosis or vascular diseases, it is mandatory to assess the condition of the aorta and to clarify CCE risk prior to PTRA. Magnetic resonance angiography could be a powerful diagnostic tool for studying the aorta, assessing the presence of atherosclerotic plaque and grading the more advanced lesions [10]. This approach can help in making the decision to perform angiography and the stenting procedure, so that the less risky intervention and therapy will be chosen for patients.

Acknowledgements. The authors would like to thank Mrs Gina Danon for language revision.

Conflict of interest statement. None declared.

References


Received for publication: 20.4.05
Accepted in revised form: 25.5.05