A rare case of acute renal failure—acute bilateral renal artery embolism

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Introduction

Acute renal failure is an emergency. Its incidence in hospitalized patients varies between 2 and 5% [1]. Rapid diagnosis followed by immediate therapy can frequently restore and preserve renal function, as acute renal failure is frequently a reversible condition. The definition proposed by the acute kidney injury network (AKIN) is often used for diagnosis of acute renal failure [2]. The causes of acute renal failure can be divided into three categories: prerenal, intrinsic and postrenal. Diagnostic procedures should be focused on early detection of the underlying mechanism. Bilateral acute renal thromboembolism is a rather rare cause of acute renal failure.

We present here a patient with bilateral renal artery embolism. He was diagnosed at least 2 days after admission and onset of symptoms. However, endovascular treatment, including angioplasty with stenting and local thrombolysis, was still effective and kidney function improved substantially. This case report should encourage clinicians to enforce immediate radiological intervention, especially in the patient at risk. Even if diagnostic procedures are delayed, radiological intervention should be taken into account.

Case report

A 68-year-old male patient presented at a community hospital with moderate pain in the left lower back and dyspnoea. Onset of symptoms was acute and present for 12 h before admission. The breathing rate was increased (20 min⁻¹) and atrial fibrillation (AF, heart rate 100 min⁻¹) terminating spontaneously within 2 h was detected. Laboratory findings revealed serum creatinine 1.34 mg dl⁻¹, blood urea nitrogen 14.7, lactate dehydrogenase 358 U l⁻¹ (normal: −225), leucocytes 8.3 g dl⁻¹, thrombocytes 140 000 μl⁻¹, haemoglobin 16.4 g dl⁻¹ and CRP 1.7 mg dl⁻¹. D-Dimers were 3.11 μg ml⁻¹ (normal: 0–0.5) and cardiac troponine was negative. Urinalysis showed 10–20 erythrocytes, 10–20 leukocytes, no casts, no dysmorphic erythrocytes but traces of protein by urine dip-stick analysis. Diuresis was 1600 ml/24 h and dyspnea was ameliorated by nasal oxygen insufflation. Pulmonary embolism was excluded by CT angiography; pulmonary infiltrates and congestion were absent. B-mode sonography showed kidneys of normal size (length: left 9.7 cm; right 10.3 cm) without postrenal obstruction. Within 2 days, creatinine rose gradually to 4.4 mg dl⁻¹ and diuresis declined progressively. Medical history revealed arterial hypertension since 1994, atrial fibrillation, chronic obstructive lung disease due to a history of smoking with 46 pack years; he had stopped smoking in 1997. The stable peripheral artery occlusive disease was treated by an aorto-bifemoral allograft in 1998. Medication was by sotalol 80 mg bid and furosemide 40 mg once daily. Due to recurrent mild nocturnal epistaxis the patient had stopped phenprocoumon treatment 3 weeks prior to admission. On Day 3 the patient was transferred to our hospital with oliguric acute renal failure. Duplex-Doppler sonography (DDS) revealed a ‘no-flow signal’ in both kidneys. MR angiography was performed immediately and showed bilateral occlusion of the renal arteries with protrusion of thrombotic material into the lumen of the abdominal aorta (Figure 1). The clinical diagnosis was bilateral renal artery embolism. Due to the acute onset of symptoms, the modest increase in serum creatinine on admission and kidneys of normal size, we believed that radiological intervention with recanalization of renal arteries might be of value. Angiography confirmed occlusion of both renal arteries (Figure 2). After balloon dilatation a stent was inserted into the right renal artery. After aspiration of thrombotic material segmental arteries of the left kidney were subjected to thrombolysis by local administration of r-TPA for 50 min followed by stenting of the left renal artery. Blood pressure was 140/90 mmHg...
before and 120/80 mmHg after intervention. Angiography performed 24 h after the intervention showed restored perfusion of both kidneys (Figure 3). The patient had to be dialyzed once after the procedure due to hyperkalaemia and hypervolaemia. Within the following days urine production increased and the patient became polyuric. Starting at Day 5 after the intervention, serum creatinine began to fall and reached 1.68 mg dl$^{-1}$ on dismissal (Figure 4).

Transesophageal echocardiography showed a left atrial thrombus ($1.5 \times 3.5$ cm). Anticoagulation was initiated with heparin and switched to phenprocoumon after 5 days. Echocardiography after 14 days showed a complete resolution of the left atrial thrombus, probably due to spontaneous lysis as neurological disorders were absent [3].

Discussion

We present here a rare case of bilateral acute renal thromboembolism. As the period from the onset of symptoms to the intervention was longer than 72 h it is remarkable that kidney function recovered substantially [4,5]. To our knowledge there are no studies in humans that investigated the role of the duration of ischaemia for the recovery of acute renal failure in renal vascular disease in detail and prospectively. Han and co-workers report a case of
bilateral renal artery occlusion and full recovery after a long period of ischaemia, although due to the clinical conditions they were not able to elaborate the exact time of ischaemia (possibly days). However, in their case aortography revealed collateral arterial blood flow to the kidneys whereas in our case there was no collateral blood flow detectable [6]. A recently published report by Sezer et al. depicts two cases of bilateral renal artery occlusion with improvement of renal function after revascularization of only one of the occluded renal arteries. Again the authors were unable to define duration of ischaemia prior to revascularization [7]. We believe that in our patient total occlusion of both renal arteries for 72 h would have resulted in irreversible renal failure as we know that renal infarction can occur within 60 min [8,9]. However successful revascularization has been reported after extended periods of anuria (up to 42 days). However, kidneys that can be salvaged after prolonged periods of ischaemia generally have some collateral arteries or a nonocclusive embolus. As we know from the MR angiography and the arterial angiography, the patient presented here did not have collateral arteries and he had an occlusive embolus in both renal arteries. Considering the clinical symptoms, the patient presented here only suffered ischaemic pain on the left side. On this side thrombotic occlusion of even the segmental renal arteries was revealed. It is therefore conceivable that occlusion of the right kidney was due to prolonged embolization occurring within days and thereby limiting ischaemic injury. Indeed, scintigrafic detection of kidney function 17 days after the intervention showed that the right kidney contributed to total tubular excretion rate with 80%. Together with the acute onset of symptoms the normal configuration and size of both kidneys made us believe that embolism of both renal arteries was acute. This was supported by the serum creatinine that was only slightly increased on primary admission.

Discontinuation of phenprocoumon therapy in a patient with intermittent AF could be causative in this setting. Cardiac origin of the thrombotic material is most likely.

Our diagnostic algorithm starting with DDS followed by MR angiography and finally arterial angiography with intervention was effective in quickly rendering the diagnosis and initiating treatment. We reviewed the literature concerning available data on this issue. However randomized, controlled trials are missing concerning the role of the different diagnostic procedures in acute bilateral renal artery occlusion. Concerning treatment, non-ostial stenoses should be treated by angioplasty and stenting whereas percutaneous treatment of ostial stenoses is more complicated and sometimes requires surgical procedures [10–13]. However, to our minds the experienced physician should use percutaneous angioplasty in the first line as complications are far less than by surgery.

This case demonstrates that the diagnostic algorithm in acute renal failure should comprise early detection of renal perfusion. Acute onset of the disease should encourage clinicians to consider immediate radiological intervention, especially in the appropriate clinical setting [5].

Conflict of interest statement: None declared.

References

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