Acute bacterial endocarditis and renal microaneurysms

Georges Mourad and Valérie Garrigue

Department of Nephrology, Lapeyronie Hospital, Montpellier Medical School, Montpellier, France

Introduction

Renal aneurysms are common in polyarteritis nodosa, either idiopathic or associated with HBV or HCV infection [1]. They have been rarely reported in fibromuscular hyperplasia, systemic lupus erythematosus, Wegener granulomatosis, and Henoch-Schönlein purpura. During infective endocarditis, mycotic aneurysms may involve renal arteries but, to the best of our knowledge, the occurrence of bilateral, diffuse intraparenchymatous renal artery aneurysms has not been reported.

Case

A 34-year-old woman, i.v. heroin abuser, was admitted with acute nephritic syndrome and severe renal failure. One year before, she presented with an acute HCV hepatitis. On admission, she complained of fatigue, fever, oliguria and oedema of the legs. Blood pressure was 110/70 mmHg, 24-h urine volume 1100 ml, serum creatinine 800 μmol/l, blood urea 41 mmol/l. Urine was dark and contained proteins (1.8 g/l), numerous red blood cells and few RBC casts. WBC count was 8800/mm³ (68% of neutrophils), CRP 54 mg/l and fibrinogen 4.5 g/l. Total complement and the C4 fraction were decreased; no cryoglobulins were detected. Echocardiography showed three vegetations on the tricuspid valve. Multiple blood cultures grew Staphylococcus aureus. Percutaneous renal biopsy showed proliferative endocapillary glomerulonephritis with crescents in 20% of glomeruli. As the biopsy was complicated by gross haematuria, a renal angiography was performed; it showed an arteriovenous fistula in the left kidney and, unexpectedly, bilateral diffuse renal aneurysms (Figure 1a). There were no signs nor symptoms of polyarteritis nodosa nor microscopic polyarteritis. The arterio-venous fistula was successfully treated by embolization. The patient received antibiotics for 10 weeks (teicoplanin, ofloxacin and meticillin). Due to gross haematuria and radiocontrast media injection, renal function initially worsened and the patient was treated by haemodialysis for 2 weeks; subsequently, renal function gradually improved and serum creatinine reached 120 μmol/l and proteinuria 0.6 g/l at discharge (2 months). A second angiography, performed 6 months later, showed the total disappearance of renal aneurysms; in addition, there were no aneurysms in the coeliac or mesenteric arteries (Figure 1b and c).

Discussion

Despite HCV infection, our patient had no vasculitis in renal biopsy nor microaneurysms in other splanchnic arteries; in addition, there were no clinical symptoms or signs of polyarteritis nodosa. Acute bacterial endocarditis may result in emboli in the renal arterial system, originating from the infected heart valves, or in the development of immune-mediated glomerular disease [2]. Mycotic aneurysms develop in 3–15% of patients; the sites most often involved are the proximal aorta and arteries to the viscera, to the extremities and to the brain [3]. We suggest that renal artery aneurysms are a very unusual and reversible complication of acute bacterial endocarditis.

References

Fig 1. (a) Small diffuse intraparenchymatous aneurysms (renal angiography performed during the acute phase of the disease, in September 1998). (b) Disappearance of renal aneurysms after the patient was treated with antibiotics (control angiography performed in March 1999). (c) No aneurysms are seen in coeliac or mesenteric arteries, as is often the case in polyarteritis nodosa; the embolization device is seen in the left kidney (control angiography of March 1999).