Oliguric acute renal failure as a complication of treatment of an infrarenal aortic aneurysm by implantation of an aortic stent

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To the editor: Endovascular repair of abdominal aortic aneurysm (AAA) is a therapeutic alternative to open surgery in selected patients.[1,2] Occurrence of acute renal failure (ARF) following implantation of an aortic stent in a patient with AAA is reported here.

CASE REPORT

A 58-year old male with a history of NSAID intolerance, a former smoker of 60 cigarettes daily recently diagnosed locally advanced non-small cell lung carcinoma. The patient had started biweekly chemotherapy with cisplatin-gemcitabine, of which two cycles had been administered. Baseline kidney function was normal (serum creatinine levels, 0.8 mg/dL).

The patient experienced acute ischaemia in the right lower limb. An angio-CT revealed a 4.5-cm infrarenal aortic aneurysm extending to common iliac arteries and thrombosis of the right external and internal common iliac arteries. A right femoral thromboembolectomy was performed. One day later, aneurysm exclusion was performed using a left monoiliac aortic stent and a left-to-right femorofemoral bypass. After this procedure, the patient experienced oliguric ARF that required haemodialysis replacement therapy. Despite the finding of vascular patency in the arterial Doppler and control arteriography, kidney function was not recovered, and continued haemodialysis was required. A subsequent scan showed absence of renal uptake. Because of the underlying disease (lung cancer), vascular conservative treatment was decided, and the patient continued on haemodialysis.

DISCUSSION

AAA is a serious vascular condition characterised by a permanent focal dilation in the aorta.[3] More than 90% of AAAs are secondary to arteriosclerosis, and most of them are infrarenal in location.[4] This condition is more common in males, and its incidence substantially increases from 55 years of age.[5] Endovascular repair of AAAs is an alternative to elective open surgery, particularly in selected patients, with low mortality and acute complication rates.[6] In the case reported, and because of the history of lung neoplasm, endovascular repair was decided, and oliguric renal failure occurred as a complication of such procedure. Since the subsequent arterial Doppler and control arteriography showed patent renal and femoral flow, and in the CT scan the right kidney appeared normal and the left kidney showed hypodense areas suggesting infarction, the possibility that the ARF was related to the renal ischaemia-acute tubular necrosis caused by the procedure and was potentially reversible was considered. Failure of kidney function to recover over time may perhaps be related to late migration of the stent to renal arteries, which was subsequently confirmed by the absence of renal uptake in a scan.

In conclusion, migration of the aortic stent is a potential cause of ARF in patients with AAA undergoing this endovascular procedure.

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Acute renal failure after intake of mushrooms: the orellanus syndrome

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To the editor: Harvesting and intake of wild mushrooms causes a significant number of poisonings, particularly in autumn. A patient with a mixed syndrome of hepatic and renal failure following intake of mushrooms from the species *Amanita phalloides* and *Cortinarius orellanus* is reported. No description of any poisoning showing such an association has been found in the literature. A 74-year old male patients with an unremarkable history attended the emergency room for intractable vomiting and diarrhoea. The patient reported to have taken mushrooms 12-15 hours before. Physical examination showed an acceptable general condition and haemodynamic stability. Laboratory test results included: urea 89 mg/dL, creatinine 3.4 mg/dL, Na 137 mmol/L, K 4 mmol/L, GOT 1406 IU/L, GPT 1170 IU/L, LDH 1319 IU/L. Coagulation: PAI 71%, APTT 43.4 sec, INR 1.24. Complete blood count: Hb 18 mg/dL, haematocrit 53.3%, WBCs 11,200/mm^3 (N 78%). Urine: Na 30 mmol/L, K 66 mmol/L, urea 16.3 g/L, creatinine 155.4 mg/dL. Acute renal failure due to volume depletion and hepatic failure secondary to mushroom intake were diagnosed, and the patients was admitted to the intensive care unit. Treatment was started with penicillin G so-

dium, activated charcoal, water and electrolyte replacement, pyridoxine, vitamin K, traxenamic acid, and fresh plasma. The reference liver transplant unit was contact because of suspected poisoning by *Amanita phalloides*. The cytolysis pattern and coagulation changes started to improve on the third day of stay at the ICU, and the patient was discharged to the gastroenterology ward. On the fourth day of stay at the ward (7 days since mushroom intake), creatinine levels of 4.2 mg/dL (as compared to a previous value of 1.5 mg/dL) were reported to the nephrology department. Urine: Na 95 mmol/L, K 49.08 mmol/L, urea 14.94 g/L, creatinine 100 mg/dL, protein 0.5 g/L, no RBCs. Normal complete blood count without eosinophilia, and normal C3 and C4. A further evaluation ruled out a prerenal cause, nephrotoxic agents, and an obstructive cause (by ultrasonography). Since a relationship with mushroom intake was suspected, mushrooms were analysed by an expert mycologist, who identified several species, including *Amanita phalloides* and *Cortinarius orellanus*. Support measures were started and an adequate water balance was ensured. Patient remained asymptomatic with a preserved urine output and maximum creatinine levels of 7.1 mg/dL with metabolic acidosis. Liver enzymes and coagulation were normal. Renal replacement therapy was not required at any time, and kidney function gradually improved until basal creatinine levels of 2 mg/dL were achieved. These levels have been maintained to date.

Mushroom poisoning is classified into two large groups based on whether the time elapsed from intake to symptom occurrence is shorter or longer than 6 hours. Poisonings caused by the *Amanita* and *Cortinarius* genera belong to the latter group (2-21 days). The potential occurrence of mixed syndromes due to the concomitant intake of several species, as occurred in our case, should also be taken into account.

Species from the genus *Cortinarius* have two types of toxins, cortinarins and orellanines. Orellanines show a high renal tropism, inhibiting protein synthesis in tubular cells. Orellanine degradation produces oxygen free radicals and glutathione depletion. Orellanines remain in renal tissue for up to 6 months after intake.

Renal failure occurs in 30%-75% of all poisonings depending on individual sensitivity and the amount ingested. End-stage chronic renal failure occurs in approximately one third, temporal haemodialysis is required in another third in which total or partial recovery of kidney function is subsequently achieved, and the remaining third experience no renal damage.

Non-specific gastrointestinal symptoms initially occur. These are associated to urinary frequency, that is occasionally followed by an oliguric phase with onset of uremic symptoms. Hepatic damage is rare, and some cases of transient cytolysis have only been reported.

Renal biopsy mainly shows interstitial nephritis with tubular necrosis and infiltration by lymphocytes, plasma cells, and PMNs with no glomerular involvement.

There is no specific antidote. Treatment should be supportive and symptomatic. Haemodialysis and plasmapheresis are not effective for toxin removal because of the long symptom-free period involved in late diagnosis. However, good results have been reported in some cases when performed within 5 days of poisoning. Use of corticoids and N-acetylcysteine for its antioxidant and glutathione-donating effect has been reported, but their efficacy is controversial.

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Acute pancreatitis and polycystic kidney disease

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To the editor: Adults with polycystic liver and kidney disease have cysts in the kidneys and, in many cases, asymptomatic cysts in the liver, pancreas, ovaries, and spermatic duct.[1,2] A patient with polycystic kidney disease and pancreatic cysts who experienced acute pancreatitis is reported.

The patient was a 47-year old male without no toxic habits. He had been on regular haemodialysis since September 2006 due to adult polycystic liver and kidney disease, and had underwent nephrectomy because of multiple complications derived from his renal cysts (infections, ruptures…).

The patient reported nausea, vomiting, severe abdominal pain, and loose stools.

The most common extrarenal complications in polycystic kidney disease include cerebral aneurysms, hepatic cysts, cardiac valve disease, colonic diverticulosis, and abdominal and inguinal hernias.[3]

Physical examination revealed diffuse abdominal pain, liver increased of