Acute renal and hepatic failure: do not miss pericardial tamponade!

Sir,

Pre-renal forms account for 40% of all cases of acute renal failure [1,2]; pericardial tamponade is in this context an unusual aetiology [2].

A 69-year-old man was referred to our hospital because of acute renal failure and cytolytic hepatitis. He had a previous history of long-standing hypertension, and lumbar pain which led to self-medication with non-steroidal anti-inflammatory drugs (NSAIDs). Prior to admission, he was treated with losartan, hydrochlorothiazide and diclofenac. He had noted a flu-like syndrome 10 days before admission, but was asymptomatic when first seen.

Physical examination revealed a man in a relatively good state. His weight was 82.5 kg and had not changed. Blood pressure was 100/70 mmHg, pulse rate 91/min, respiratory rate 20/min, and temperature 37°C. There was no rash, no organomegaly, no lymphadenopathy, no oedema. Lungs were clear to auscultation. No cardiac murmur, no pericardial rub was noted. His jugular veins were slightly distended. Neurologic examination was normal. Urine volume was 500 ml/24 h.

Pertinent laboratory data included normal haemoglobin level, platelets and white blood cell count. Serum sodium was 131 mmol/l, potassium 5.3 mmol/l, chloride 84 mmol/l, bicarbonate 18 mmol/l, creatinine 6.4 mg/dl, SGOT 785 UI/l, SGPT 562 UI/l. There was no proteinuria or haematuria; urinary sodium was 5 mmol/24 h on a normal salt diet. Urinary potassium was 87 mmol/24 h.

Chest radiogram was normal without enlarged cardiac silhouette. Electrocardiogram showed non-specific flat T-waves. Renal sonography showed normal-sized kidneys and no evidence of hydronephrosis.

Common aetiologies of combined hepatic and renal disease such as leptospirosis, Hantavirus disease, angiocholitis were excluded, and the hypothesis of an immunoallergic aetiology was entertained.

The patient received 2 l/day isotonic saline. Two days later, he complained of dyspnea with orthopnea and pain in the right upper abdomen. He gained 2 kg. His lungs remained clear but jugular veins were more distended. Blood pressure decreased to 90/60 mmHg without pulsus paradoxus. The patient was oliguric. The concentrations of liver enzymes increased i.e. SGPT 1600 UI/l, and there was evidence of liver insufficiency (TP 30%). Renal function is reported in Figure 1. The chest radiograph disclosed an enlarged heart silhouette. The echocardiogram revealed massive pericardial effusion with right atrial and ventricular diastolic collapse, inspiratory decrease of transmitral flow velocity and swinging heart. Surgical pericardial drainage was immediately performed and 800 cc of bloody fluid were retrieved. Diuresis immediately increased with prompt recovery of renal function, and normalization of liver tests within 1 week.

We cannot absolutely exclude the possibility of drug-induced haemodynamic acute renal failure, but we consider it unlikely because renal function did not recover after withdrawal of the AT1 receptor antagonist, the NSAIDs, the diuretic and after administration of intravenous NaCl. The response to pericardial drainage, i.e. the immediate dramatic increase of diuresis and improvement in renal function and the normalization of liver tests strongly suggests that pericardial tamponade was responsible.

Finally, although proteinuria is commonly considered a feature of cardiogenic acute renal failure, proteinuria may be absent, as in our case.

The paucity of clinical and radiological signs of pericardial tamponade is also striking in this case. It should be kept in mind that pericardial rub, pulsus paradoxus and enlarged cardiac silhouette may be initially absent [3] as in our case. This case should serve as a reminder not to forget the heart when one is dealing with simultaneous acute renal and hepatic failure.

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