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Acute kidney injury in a 'well-filled patient': an unusual cause of elevated jugular venous pressure

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Case

A 55-year-old woman was referred to a tertiary referral renal unit with acute kidney injury. Her past medical history included long-term rheumatoid arthritis and osteoporosis. She had recently been diagnosed with heart failure and was commenced on therapy with oral bumetanide (1 mg OD), ramipril (2.5 mg OD) and spironalactone (50 mg OD). Other medications included methotrexate 10 mg once a week, sulphasalazine 500 mg BD, folic acid 5 mg 6 days a week, risedronate 35 mg once a week, ranitidine 150 mg BD and co-codamol 8/500 mg as required.

On examination she was cachectic. Pulse rate was 72 per min, in sinus rhythm and blood pressue (BP) was 60/40 mm Hg. Jugular venous pressure (JVP) was raised at 6 cm and heart sounds were normal with no murmur or pericardial rub. Chest auscultation revealed bibasal crackles. The abdomen was soft and non-tender with no palpable mass. She had pitting oedema of her legs to mid-thigh.

Blood tests revealed the following: Na 137 mmol/L, K 7.5 mmol/L, urea 46.8 mmol/L and creatinine 470 µmol/L (baseline creatinine was 111 µmol/L) and HCO_3 12 mmol/L, Hb 9.3 g/L, WBC 10.8×10^9/L, PLT 82×10^9/L.

The initial working diagnosis was AKI secondary to the combination of angiotensin-converting enzyme inhibitor, diuretics and severe right-side heart failure with a low cardiac output. A differential diagnosis of constrictive pericarditis, pulmonary embolism, pulmonary hypertension and cardiac amyloid was considered for the severe heart failure and a very low cardiac output.

A right internal jugular dialysis catheter was placed and she was commenced on haemodialysis for her acidosis and hyperkalaemia after confirming the line position by performing a chest X ray.

The chest radiograph shows a normal sized heart and a left retrocardiac mass (Figure 1).

An urgent bedside transthoracic echocardiogram was performed which did not show any evidence of pericardial effusion.

A computed tomography pulmonary angiogram (Figure 2a, b and c) was requested to rule out pulmonary embolism, which showed a pulmonary mass and the right atrium almost entirely replaced by a mass that was suspected to be a tumour with no evidence of pulmonary embolism. Her inferior vena cava (IVC) was occluded by thrombus.

The axial CT image shows a soft tissue mass invading the right atrium, a left retrocardiac lung mass and small bilateral pleural effusions (Figure 2a).

The axial CT image shows the extent of the right cardiophrenic mass which invades the right atrium, liver and hemidiaphragm. The IVC is obstructed by this mass (Figure 2b).

The coronal CT image shows a large filling defect within the IVC consistent with a thrombus (Figure 2c).

Despite correction of electrolytes, a low cardiac output persisted. In view of the presence of extensive malignant disease and its associated poor prognosis a decision was made to discontinue dialysis. The patient was transferred to a palliative care pathway.

A subsequent post-mortem examination confirmed that the inferior vena cava was completely occluded by thrombus. There was widespread malignant disease with disease in the left lung, throughout the right atrium and metastases in the right ventricle, liver, kidneys and both adrenal glands. Histopathology showed a poorly differentiated adenocarcinoma.

Question

What is the cause of acute kidney injury in this patient with an elevated jugular venous pressure?

Acute kidney injury in this patient is due to a combination of poor venous return secondary to an occluded inferior vena cava, tumour mass causing right ventricular outflow obstruction, diuretics causing volume depletion and the ACE-inhibitor's haemodynamic effect.

Discussion

Pre-renal failure secondary to decreased renal perfusion is probably the commonest cause of acute kidney injury. The diagnosis is usually based on a combination of patient history and clinical examination findings, typically low blood pressure and tachycardia. While examination of the jugular venous pressure is of limited reliability even amongst experienced clinicians [1], most patients will have a reduced JVP due to hypovolaemia. The finding of an elevated JVP in the presence of hypotension should immediately alert the clinician to a more unusual cause of

shock, typically either cardiogenic or obstructive (Table 1). This would then lead to a detailed assessment to ascertain the underlying pathology. Intracardiac tumour is low down on the list for differential diagnosis.

Metastatic cardiac tumours are more frequent than primary cardiac tumours [2, 3] and are associated with a poor prognosis. Tumours that most often metastasize to the heart include lung, breast, renal cell, melanoma, hepatocellular carcinoma and lymphoma [4]. Cardiac involvement can occur by haematogenous spread, direct invasion from the mediastinum, tumour growth into the vena cava and extension into the right atrium or retrograde lymphatic spread.

The clinical presentation of the cardiac tumour depends on the location of the tumour [4]. A tumour in the right atrium can present with signs and symptoms of right heart failure and haemodynamic compromise [5].

In the case discussed, the initial presentation included significant leg oedema, leading to a diagnosis of heart failure and treatment with diuretics and ACE inhibitor. Acute kidney injury developed due to a combination of poor venous return secondary to an occluded inferior vena cava, obstructed right ventricular outflow secondary to

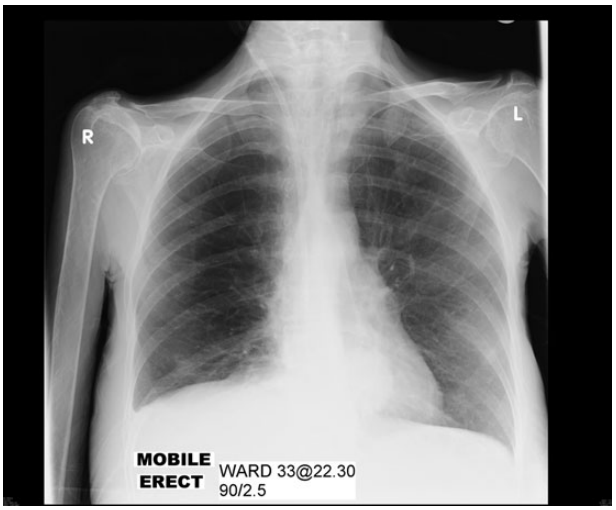

Fig. 1. Chest X-ray demonstrating dialysis catheter in place, a normal sized heart and a left retrocardiac mass.

Table 1. Hypotension and elevated JVP

Cardiogenic causes	Obstructive causes
Pump failure (heart failure)	Pulmonary embolus
Right ventricular infarction	Cardiac tamponade
Restrictive cardiomyopathy	Constrictive pericarditis
	Pulmonary hypertension
	Tension pneumothorax
	Intracardiac thrombus
	Intracardiac tumour

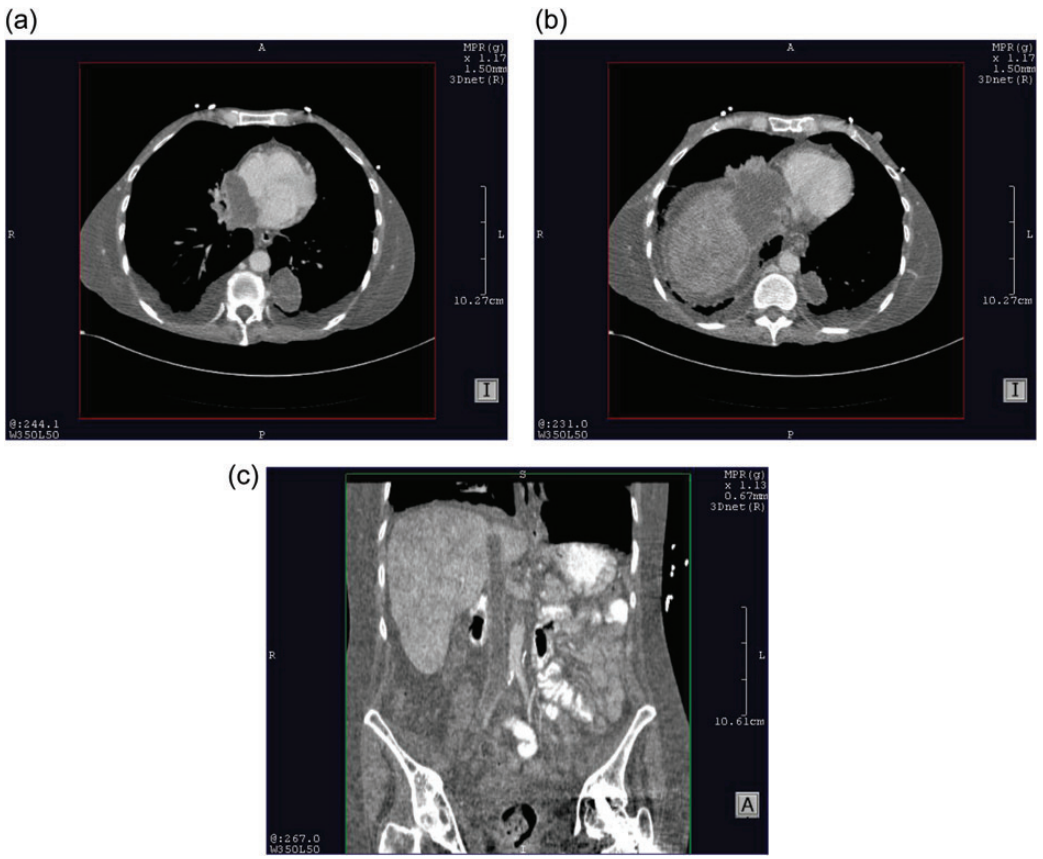

Fig. 2. (**a**) Axial CT image, which shows a soft tissue mass invading the right atrium, a left retrocardiac lung mass and small bilateral pleural effusions. (**b**) An axial CT image that shows the extent of the right cardiophrenic mass, which invades the right atrium, liver and hemidiaphragm. The IVC is obstructed by this mass. (**c**) Coronal CT image, which shows a large filling defect within the IVC consistent with a thrombus.

tumour mass, volume depletion secondary to diuretics and the haemodynamic effects of ACE inhibition.

Recognition of the underlying diagnosis was important as it allowed the treating clinicians to explain the inconsistencies in the clinical picture, but more importantly it allows an appropriate discussion of diagnosis and prognosis with the patient. This avoided futile attempts at prolonged dialysis support and allowed appropriate implementation of supportive palliative care.

Conflict of Interest Statement. None declared.

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