Case Report

Acute renal failure associated with carcinoid crisis

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Introduction

The carcinoid syndrome is caused by a malignant tumour that characteristically secretes 5-hydroxytryptamine (5-HT). The syndrome is characterized by flushing of the head and upper body, diarrhoea, and cardiac valve abnormalities [1]. Renal failure is rare in this condition [2]. We report a case of reversible and recurrent acute renal failure associated with a carcinoid crisis.

Case report

A 54-year-old man was admitted acutely to another hospital with a 1-week history of fever, sore throat, headache, loss of appetite, and a 2-day history of rash and watery diarrhoea. His medication was codeine, ibuprofen, and ofloxacin which had been started 2 days previously by his general practitioner. Over the previous year he had been seen by a rheumatologist and a general physician with recurrent episodes of back, shoulder, and limb pains. A diagnosis of polymyalgia rheumatica had been proposed but a trial of steroids had been unsuccessful in relieving the symptoms.

On admission he was unwell with an erythematous rash over his face and upper body (figure 1), heart rate 100 regular, blood pressure 140/80 mmHg. He had a soft systolic murmur, bibasal inspiratory crepitations, and 4 cm tender hepatomegaly. Neurological examination was normal and rectal exam revealed a moderately enlarged smooth prostate.

Initial investigations showed: urea 22 mmol/l, creatinine 416 μmol/l, aspartate aminotransferase 236 IU/l (normal 1–65), alkaline phosphatase 496 IU/l (30–110), albumen 32 g/l, bilirubin 41 μmol/l, creatinine kinase 1751 IU/l (24–196), total white cell count 31.2 (92% neutrophils), haemoglobin 16.1 g/l, platelets 707, ESR 46 mm/h. Abdominal ultrasound showed an enlarged liver with multiple opacities consistent with metastases, kidneys were normal in size and non-obstructed. X-rays of chest and spine showed multiple sclerotic lesions in keeping with metastases. He was oliguric and transferred to our unit for further management.

In view of the possibility of an allergic nephritis, prednisolone 60 mg/day was started and his other medication stopped. Liver and renal biopsies were performed. Haemodialysis was instituted. Renal biopsy showed changes compatible with acute tubular necrosis. Cystoscopy and bilateral ureterograms were normal. Liver biopsy revealed carcinoid tumour and oncology review was arranged. A diagnosis of carcinoid crisis with liver metastases was made.

On day 2 of admission his skin was much worse and he was given an intravenous pulse of methylprednisolone (500 mg) daily for 2 days. His condition improved dramatically over the next few days and he became dialysis independent (figure 2). Twenty-four hour urinary 5-hydroxy indole acetic acid (5-HIAA) was elevated at 462 μmol/l (normal <4). He was discharged home on a reducing dose of prednisolone with a serum creatinine of 248 μmol/l.

Six weeks later he was readmitted with a further carcinoid crisis with severe diarrhoea and rash on
40 mg prednisolone. He was again oliguric with increasing creatinine (figure 2) and a high creatinine kinase. Further intravenous steroids were given, haemodialysis instituted, octreotide and cryptohexidine started. His condition once again improved and he became dialysis independent. He was allowed home on 600 μg of octreotide daily with a serum creatinine of 529 μmol/l (figure 2).

Two weeks later he was readmitted with a further crisis. In view of his failure to respond to octreotide and his poor prognosis, dialysis was not commenced and he died 48 h after admission. Post mortem revealed a 2 cm tumour in the left lower lobe and confirmed liver and bone metastases.

Discussion

Our patient had two episodes of reversible acute renal failure due to biopsy proven acute tubular necrosis (ATN). Because of the striking absence of hypotension at any point during his admission, we believe that ATN arose as a consequence of arteriolar vasoconstriction mediated by vasoactive substances released by the carcinoid tumour or as a direct toxic effect of these substances on tubular cells. Couttenye and colleagues [1] describe a case of carcinoid crisis with significant hypotension during episodes of acute renal failure. They demonstrated high serum levels of 6-ketoPGF1α, a metabolite of PGI2. They postulated that this potent vasodilator was responsible for loss of glomerular filtration pressure due to efferent renal artery vasodilatation during a crisis. The rise in creatine kinase is unexplained, though it is plausible he had muscle inflammation or necrosis associated with his crisis.

It is not clear what caused improvement of the crises. We postulate that the initial dramatic improvement following haemodialysis may have been due to the partial clearance of these substances from the circulation. Steroids may also have a role in improving the flush from carcinoid tumours of bronchial origin [3].

References


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Fig. 2. Changes in serum creatinine with time.