Acute renal failure due to gold

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Summary

A patient with rheumatoid arthritis is described who developed acute renal failure whilst receiving gold. This occurred despite the normal precautions of patient monitoring before each dose was given. The clinical picture suggests this was a hypersensitivity reaction to chrysotherapy.

Introduction

Transient proteinuria is an occasional complication of chrysotherapy and usually disappears when the drug is withdrawn, although progression to the nephrotic syndrome has been reported (Vaamonde and Hunt, 1970). Acute renal failure is rare and only 3 patients with this complication have been described previously, 2 of whom died (Olmer and Sarradon, 1934; Mathers, 1945; Derot *et al.*, 1954). A patient with rheumatoid arthritis is reported, who developed acute renal failure whilst receiving gold but which had a successful outcome.

Case report

A 68-year-old woman with a 15-year history of seropositive rheumatoid arthritis had been treated with small daily doses of prednisolone for 5 years until 1975. Latterly, her therapy had included naproxen 750 mg daily and dihydrocodeine tablets. Articular symptoms persisted and gold treatment was started. Her blood urea, creatinine and urinalysis were normal at that time. The Hb concentration was 10.4 g/dl. The first injection (5 mg of sodium aurothiomalate) was given in April, 1978. A further 4 injections were given at weekly intervals when, after a total of 60 mg of the drug, she complained of itching and sore throat. Proteinuria was detected and chrysotherapy was discontinued. Over the next 8 days nausea, diarrhoea, confusion and oliguria developed, necessitating her admission to hospital. Examination revealed an anaemic and drowsy, but afebrile patient with the typical articular stigmata of rheumatoid arthritis. A faint

rash was present on the face and arms. The BP was 95/65 mmHg with no postural fall. Peripheral oedema was absent. Catheterization of the bladder produced 10 ml of urine which contained protein (3+) and copious red and white cells but no casts. The urinary sodium concentration was 65 mmol/l (mEq/l). Blood analysis showed a normochromic normocytic anaemia with an Hb concentration of 7.7 g/dl, a normal differential WCC and a platelet count of $485 \times 10^9/l$. The blood urea was 26 mmol/l, serum creatinine 875 $\mu\text{mol/l}$, sodium 122 mmol/l, potassium 4.5 mmol/l and bicarbonate 15 mmol/l. Tests for rheumatoid and antinuclear factor were negative, complement levels were normal, immunoglobulin levels were within normal limits. Her HLA status was not determined. High dose intravenous pyelography showed poor concentration of dye, but normal sized kidneys with no evidence of obstruction.

The patient's condition deteriorated with increasing drowsiness, irregular respiration, and hyponatraemia. Peritoneal dialysis reversed the biochemical abnormalities and full consciousness was restored. There was a diuresis on the third day followed by a return of normal renal function. During the recovery period she developed a severe but transient oropharyngitis. Throat swab culture grew no bacterial or fungal pathogens; the anti-streptolysin-o titre was normal. Her Hb had fallen to 5.5 g/dl, but there was no clinical evidence of bleeding or haemolysis, indeed, the sternal marrow showed reduced erythropoiesis and normal iron stores. The differential WCC remained normal with a platelet count of $410 \times 10^9/l$. However, the direct antiglobulin test was positive, the patient's erythrocytes being coated with complement ($C_4 < C_3$) and the serum contained a cold pan-autoantibody showing anti-I specificity. This made crossmatching difficult although blood was transfused uneventfully. The direct antiglobulin test has remained positive. Six weeks later she was well with quiescent joint symptoms, a normal haemoglobin concentration and normal renal function.

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Discussion

The history and evolution of this patient's renal failure suggests a causal relationship between this and her treatment with gold. This is supported by the development of pruritus, a skin rash and oropharyngitis—well recognized complications of chrysotherapy. These features and the development of renal failure after such a small quantity of the drug suggest this was a hypersensitivity reaction and not a dose-related effect.

A review of the literature showed only one case of gold-induced acute renal failure in a patient with rheumatoid arthritis (Derot, 1954), the earlier reports referring to patients with tuberculosis (Olmer and Sarradon, 1934; Mathers, 1945). That patient died, the renal failure having developed after 350 mg of sodium aurothiomalate, and tubular necrosis was found at post-mortem—an appearance more in favour of a toxic effect than a hypersensitivity reaction.

Acute renal failure during chrysotherapy is rare, but may develop with small doses of gold salts and despite the normal precaution of monitoring the patient before each injection is given.

Acknowledgments

We are grateful to Dr Douglas Lee of the National Blood Transfusion Service, Lancaster District, for the immunological studies.

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