Case Report

Rosiglitazone as a cause of acute interstitial nephritis

Clare Castledine, David Wright and Edward Kingdon

Royal Sussex County Hospital, Brighton, Renal Department, Brighton, E. Sussex, UK

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Introduction

Acute interstitial nephritis (AIN) is characterized histologically by the infiltration of the non-vascular regions of the kidney with polymorphonuclear cells, eosinophils and plasma cells [1]. The main causes are either infectious or drug related. Infectious causes are Legionella, Streptococcus, Staphylococcus, EBV, CMV, HIV as well as Rickettsia, leptospirosis and toxoplasmosis. Drug causes include antibiotics [2], NSAIDS [3,4] and diuretics as well as proton pump inhibitors [5], captopril, phenytoin, allopurinol and interferon.

Rarely, AIN can be caused by anti-tubule basement membrane disease, sarcoidosis and tubulo-interstitial and uveitis syndrome [1].

Case report

A 55-year-old gentleman was admitted via his GP with a two week history of nausea, anorexia, weight loss and a dull ache in both the loins. He was found to have acute renal dysfunction with a creatinine of 458 μmol/l. His creatinine had been measured one month previously at 97 μmol/l.

He denied any dysuria, frequency, haematuria, rashes, joint, respiratory or ENT symptoms.

Fig. 1. Serum creatinine over time.
Previous history was of type II diabetes and hypercholesterolaemia only.

Medications were metformin 500 mg bd, atorvastatin 10 mg od and rosiglitazone 4 mg od. He had been taking the atorvastatin and metformin for several years. The rosiglitazone, however, had been commenced three weeks previously due to poor glycaemic control.

He had not taken any other medicines during this period, including any NSAIDS, complementary medicines or antibiotics.

On arrival, BP was 130/60, P72 Temp 37.9 and Sats 96% air. He was euvoalaemic and physical examination revealed tenderness of the left loin. Urine dipstick revealed glucose ++ protein ++ blood ++++. A renal ultrasound was performed, which did not reveal any evidence of urinary tract obstruction or calculi and 16.6 cm kidney on the right and 15.8 cm kidney on the left. Metformin and rosiglitazone were ceased and a renal biopsy was performed which revealed an AIN as demonstrated by lymphocytic tubulitis and eosinophilic infiltrates.

He was commenced on an intermediate dose reducing course of prednisolone, commencing at (0.5 mg/kg/day) and the renal function quickly improved over the next two weeks without the need for dialysis (Figure 1).

Conclusion

The chronology of events in this case led us to believe that rosiglitazone was the cause for the interstitial nephritis. Ceasing the offending drug and a reducing course of steroids has led to an improvement in the renal function in this case. Follow-up at 6 months revealed a creatinine 139 µmol/l. This is the first time that rosiglitazone has been implicated as a cause for interstitial nephritis in the literature.

Conflict of interest statement. None declared.

References


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