Case Report

Mesangiocapillary glomerulonephritis in a patient with Nocardia pneumonia

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Introduction

Nocardiosis is an opportunistic infection that requires aggressive and prolonged antibiotic treatment. The genus Nocardia was named after Edmond Nocard who isolated the organism from cattle on the island of Guadeloupe in 1888 [1]. They are Gram-positive variably acid-fast aerobic bacteria that form filamentous branched cells. These cells are found within the environment in soil, water and vegetable matter. Infections in humans may result from inhalation of organisms into the lungs, through trauma directly into the tissues, or ingested through contaminated food sources. The various clinical presentations include pulmonary, extrapulmonary, CNS, cutaneous or systemic infections by different species including N. asteroides, N. brasiliensis, N. farcinica, N. caviae and N. transvalensis. Nocardia infection is well recognized in immunosuppressed individuals [2], and is usually pulmonary or systemic, but to our knowledge has not been described in association with glomerulonephritis or rapidly progressive renal failure. We report a 40-year-old man who presented with atypical pneumonia and developed nephrotic-range proteinuria due to an immune complex glomerulonephritis, followed by progressive deterioration in renal function to dialysis-dependant renal failure within 3 months.

Case presentation

The patient was married with one son, worked as a taxi driver in a small town, and drank 10 g of alcohol and smoked 15 cigarettes per day. He had a 15-year history of cluster headaches that only responded to sumatriptan or oral corticosteroids. From 1990 to 1996 he was on oral prednisolone, self-administering doses of up to 100 mg daily.

He presented to a country hospital with a cough, arthralgia and fever of 2 weeks duration, and was initially treated with erythromycin for a left lower lobe pneumonia. He deteriorated rapidly and required ventilation and transfer to our hospital. Medication on admission included prednisolone 100 mg daily, verapamil, sodium valproate and diazepam for treatment of his headaches. Clinical and radiological examination was consistent with left lower lobe consolidation. His initial urinalysis was negative for protein, his serum albumin was 15 g/l (reference range 34–48 g/l) and creatinine 0.04 mmol/l (range 0.05–0.12 mmol/l). Nocardia asteroides and N. brasiliensis were grown from a tracheal aspirate and IV bactrim (trimethoprim–sulfamethoxazole) was commenced. Progressive improvement in respiratory function allowed extubation and discharge 2 weeks later on oral bactrim. His urinalysis remained negative for protein. Prednisolone was withdrawn slowly.

He presented again 3 weeks later to his local hospital with increasing anorexia, dyspnoea and abdominal pain. Bactrim was ceased and minocycline was commenced. On transfer to our hospital, urine examination showed heavy proteinuria and granular casts. Biopsy of a skin rash was consistent with leucocytoclastic vasculitis. There was no arthralgia, Raynaud’s phenomenon, alopecia or haemoptysis. Twenty-four hour protein excretion was quantitated at 3.6 g with a serum albumin of 15 g/l and serum creatinine 0.06 mmol/l. Serum antinuclear factor, antineutrophil cytoplasmic antibody, protein electrophoresis, rheumatoid factor, alpha-1-antitrypsin level, C₃, C₄, cryoglobulins, hepatitis B,C and HIV serology, extractable nuclear antigens and mycoplasma serology were all normal or negative.

A renal biopsy was performed. On light-microscopy there was a diffuse proliferative crescentic glomerulonephritis with glomerular endocapillary proliferation and an accentuated lobular architecture (Figure 1).
Mesangiocapillary glomerulonephritis

There was no significant interstitial scarring. Double contouring of the glomerular capillary walls was focally evident in the silver stains (not shown). Immunofluorescent staining revealed generalized global granular capillary loop staining for IgA (2+), IgG (2+), C3 (1+) and fibrinogen, and electron-microscopy showed small mesangial, subendothelial and subepithelial deposits and focal mesangial interposition (Figure 2).

The patient developed progressive renal dysfunction, oliguria and respiratory failure (requiring ventilatory support), and dialysis was commenced on day 40 of the second admission. Bactrim and cefotaxime were administered intravenously. Following improvement in respiratory function and extubation, a second renal biopsy was performed to determine renal prognosis. This again showed a diffuse endocapillary proliferative glomerulonephritis, with nearly 100% of the glomeruli distorted by cellular or fibrocellular crescents. A moderate interstitial infiltrate with some eosinophils raised the possibility of an interstitial nephritis. Immunofluorescent staining revealed global diffuse granular-capsillary-loop staining for IgA (+/−), but stains for IgG, IgM, Clq, and fibrinogen were negative. Electron-microscopy showed distortion of the glomerular tuft by ischaemic changes with expansion of some mesangial areas and interposition, but no electron-dense deposits.

He was discharged from hospital on oral bactrim and sodium valproate, and received haemodialysis three times a week. Following 8 months of haemodialysis his urine output increased progressively, renal function improved, and dialysis was successfully withdrawn. His serum creatinine concentration remains stable at ~0.25 mmol/l, and he is well with no further episodes of cluster headache (on bactrim and sodium valproate) 12 months after his initial illness. Repeat chest X-rays show residual scarring but otherwise resolution of the nocardial pneumonia.

Discussion

The diffuse proliferative glomerulonephritis suffered by this individual fits best into the category known as mesangiocapillary glomerulonephritis (MCGN). To our knowledge this is the first description of an association between MCGN and nocardiosis. A similar immune complex glomerulonephritis has been described in association with numerous infections, and differs from post-infectious glomerulonephritis in both its histopathological appearance and clinical course [3]. This patient’s presentation followed self-medication with corticosteroids, which is likely to be the major predisposing factor for the infection. No primary immune deficiency disorder was found. The normal serum creatinine and urinalysis at first presentation argues against significant pre-existing renal disease. The appearance of proteinuria 8 weeks after the illness began occurred when the lung disease was deteriorating, but also corresponded to withdrawal of corticosteroids, suggesting that the immunosuppression may have had a protective effect against the glomerulonephritis. Bactrim is unlikely to be the cause of the MCGN, despite the timing of its introduction, because the patient remained on it while the renal disease improved. An association between the nocardiosis and the MCGN is further supported by the observation that resolution of the pneumonia was followed, with a lag period, by clinical improvement in the renal disease.

In summary, we report a 40-year-old man with nocardiosis who developed a mesangiocapillary-type glomerulonephritis leading to prolonged but reversible dialysis-dependant renal failure.

References


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