Pseudoneuropathic CPPD arthropathy: magnesium matters

Sue R. Whelan, F. O’Shea, G. McCarthy

Department of Rheumatology, Mater Hospital, Dublin, Ireland

Accepted 8 January 2008

Rheumatology key message

- Hypomagnesaemia as a cause of CPPD should be considered in the context of thiazide diuretic use.

Disclosure statement: The authors have declared no conflicts of interest.

B. R. Whelan, F. O’Shea, G. McCarthy

Department of Rheumatology, Mater Hospital, Dublin, Ireland

Accepted 8 January 2008
Correspondence to: B. R. Whelan, Department of Rheumatology, Mater Hospital, Eccles St, Dublin 7, Ireland.
E-mail: bryanwhelan@ireland.com


Rheumatology 2008;47:552–553
doi:10.1093/rheumatology/kem357
Advance Access publication 15 February 2008

Successful treatment of resistant scleroderma-associated interstitial lung disease with rituximab

SIR, We report the case of a 57-year-old patient who presented with an 18-month history of scleroderma. This had been diagnosed previously in another institution where she was treated with i.v. infusion therapy of methylprednisolone at 10 mg/kg and cyclophosphamide 15 mg/kg. She failed to respond to the initial six courses of therapy, and had a further five infusions. Unrelated to her medical condition, she moved to our region where she presented to the respiratory service in February 2004 with worsening dyspnoea. She reported hand pain, stiffness and swelling and restriction of shoulder movements, with worsening Raynaud’s phenomenon, and was also referred to rheumatology.

When she attended the rheumatology clinic in August 2004, she was wheelchair bound due to dyspnoea and had an intermittent cough, but not productive of sputum. There was no chest pain or haemoptysis. The patient was in respiratory distress and had difficulty talking, with a resting respiratory rate of 40/min and oxygen saturation of 92% on room air. She was not centrally cyanosed and had bibasal coarse crackles, but her jugular venous pulse was not elevated, heart sounds were normal, no murmurs were detectable and she had no dependent oedema. She had puffy scleroderma of the hands with marked synovitis and bilateral shoulder capsulitis. There was extensive telangiectasia on her hands and face, and facial skin tightening with beaking of the nose. She also had symptoms of heartburn and was taking omeprazole 20 mg daily.

The chest X-ray demonstrated signs of pulmonary fibrosis at both bases. Her blood count was normal, ESR and CRP were 30 mm/h and 28 mg/l (normal <10 mg/l), respectively; rheumatoid factor was negative but ANA was strongly positive at a titre of 1:2560 with a homogeneous pattern; extractable nuclear antigens including Scl-70 were negative. High-resolution CT (HRCT) performed before her rheumatology review showed basal honeycombing and a ground glass appearance compatible with scleroderma-related interstitial lung disease (ILD). Her pulmonary function test showed a forced vital capacity (FVC) of 1.84 l (100% predicted), forced expiratory volume in 1 s (FEV1) to FVC ratio was 88.01, and the diffusing capacity of the lung for carbon monoxide (DLCO) was 1.1 mmol/min/kPa (62.7% predicted).

In September 2004, she was treated with rituximab 1000 mg 2 weeks apart with 100 mg of methylprednisolone premedication prior to the infusions. She improved within 3 weeks and managed to walk up to 500 m. Her FVC improved to 2.01 l (109.5%). Her arthropathy resolved completely. She started prednisolone 5 mg daily and cyclosporin 50 mg twice daily. In late 2005, to walk up to 500 m. Her FVC improved to 2.01 l (109.5%). In February 2006 a repeat HRCT showed ground glass changes compatible with midzone pneumonitis (Fig. 1A). Her FVC had reduced to 1.69 l (93.5% predicted) and her DLCO was 0.62 mmol/min/kPa (34.3% predicted). She was developing cor pulmonale; an echocardiogram performed an enlarged right ventricle with impaired function with right ventricular systolic pressure of 132 mmHg and raised pulmonary artery pressures. In view of the success of the original infusion she was retreated with the same rituximab regimen. Following therapy she resumed her walking distance of 500 m. Her repeat FVC and DLCO improved to 2.02 l (113% predicted) and 0.84 mmol/min/kPa (48% predicted), respectively, and the repeat HRCT (Fig. 1B) showed an improved Air bronchograms evident on HRCT in the upper and mid-zones were less conspicuous following therapy.

Peripheral blood cytometric evaluation using a sensitive assay after the second treatment was determined. Following therapy the B-cells were 0.0001 l x 10^9/l (normal levels 0.06–0.66 l x 10^9/l) and pre-plasma cells 0.0002 l x 10^9/l. The good clinical response noted in the face of peripheral blood B-cell lymphopenia likely reflects the presence of pathogenic B cells in the lymphoid and possibly pulmonary tissues.

The monoclonal antibody rituximab is directed against the CD20 antigen that is expressed on B lymphocytes. Rituximab has been used to treat non-Hodgkin’s lymphoma where it has good

![Fig. 1. HRCT showing (A) pre-retreatment with rituximab and (B) post-treatment with the second course of rituximab, demonstrating a reduction of the ground glass appearance and resolution of the air bronchograms in midzones (arrows). At this stage, the lung bases demonstrated irreversible fibrotic changes with honeycombing.](https://academic.oup.com/rheumatology/article-abstract/47/4/551/1790360)