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Anti-CD20 monoclonal antibody (rituximab) as an adjunct in the treatment of giant cell arteritis

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We describe a patient with polymyalgia rheumatica/giant cell arteritis (PMR/GCA) whose disease was refractory to a reduction in the dose of her glucocorticoid to an acceptable level. Our patient improved after B lymphocyte depletion but developed respiratory problems. To our knowledge this is the first description of such a case.

CASE REPORT

An 82 year old woman presented with a 4 week history of symptoms consistent with GCA of the temporal arteries and PMR. Of significance in her past medical history she had significant chronic airflow limitation with an FEV_1/FVC (forced expiratory volume in 1 second/forced vital capacity) of 0.7/1.2. Computed tomography of her chest identified a small area of bronchiectasis in the left lower lobe of her chest.

Her erythrocyte sedimentation rate (ESR) and C reactive protein (CRP) were, 109 mm/1st h (normal 1–20) and 230 mg/l (normal 0–5.0), respectively. Treatment was started with 50 mg of prednisolone. Three weeks later the patient developed a nasal left upper visual field defect. The dose of prednisolone was gradually reduced over the next 5 months.

She was, however, experiencing side effects of her prednisolone treatment, including weight gain, proximal muscle weakness, and the development of a cushingoid face. However, at a dose of 15 mg of prednisolone a day, her symptoms of GCA returned with an increase of the ESR to 53 mm/1st h. Azathioprine 100 mg/day was not tolerated. Four months later while receiving 15 mg of prednisolone her GCA again became symptomatic with transient visual changes including flashes of light. Her CRP rose to 92 mg/l.

At this stage her GCA was not adequately treated and a decision to use B lymphocyte depletion therapy was made. Intravenous cyclophosphamide (500 mg) with mesna followed by a single infusion of 1 g rituximab preceded by 100 mg of methylprednisolone intravenously was given. An absolute CD19 count 1 month after treatment was 0.00/l, confirming that B lymphocyte depletion had been successful.

Fluorodeoxyglucose positron emission tomography (FDG-PET) undertaken before B lymphocyte depletion and 4½ months after treatment provides objective confirmation of her arteritis and demonstrates her response to B lymphocyte depletion (fig 1). After B lymphocyte depletion the patient's symptoms also resolved and her CRP returned to normal. Six months after B cell depletion our patient has no symptoms suggestive of GCA. Her ESR is currently 13 mm/1st h and her CRP 6.0 mg/l.

Four days after B lymphocyte depletion our patient developed respiratory failure and was transferred to the intensive therapy unit. A chest radiograph showed lobar consolidation bilaterally, the cause of which remains unclear but may be a result of treatment with rituximab. Her chronic airflow limitation remains a major constraint on her mobility and she has until now required 24 hour domiciliary oxygen (2 l/min).

DISCUSSION

Our patient's GCA was inadequately controlled with a dose of prednisolone of 20–30 mg and treatment was associated with significant side effects. An alternative treatment is needed in such cases.

The aetiology of PMR/GCA is unclear, but muscle biopsy specimens from patients with PMR show immunoglobulin deposits around fascicles,[1] and serum samples from patients with PMR have been found to contain immune complexes.[2] These features suggest that PMR/GCA may be mediated by a particular type of small immune complex. We have previously suggested that binding of such complexes to matrix bound FcγRIIIa on fibrillin based microfibrils in muscles and in the internal elastic lamina of arteries may initiate local disease which will lead to symptoms associated with PMR and GCA.[3]

Taken together with overlapping clinical and immunogenetic features (HLA-DR4) this suggests that PMR/GCA might respond to new treatments being developed for RA.

B lymphocyte depletion treatment has recently proved successful in rheumatoid arthritis.[4] On the basis of inadequate control of the GCA and the above theoretical considerations, a decision was made to treat this patient . Our patient's favourable response suggests that this approach to the management of patients with GCA may be worthy of further investigation.

A significant number of lower respiratory tract infections have occurred in our cohort of patients with rheumatoid arthritis treated with B cell depletion (125 cycles) at University College London.[5]

This case illustrates the possible risk that B cell depletion may pose in patients with marginal respiratory reserve.

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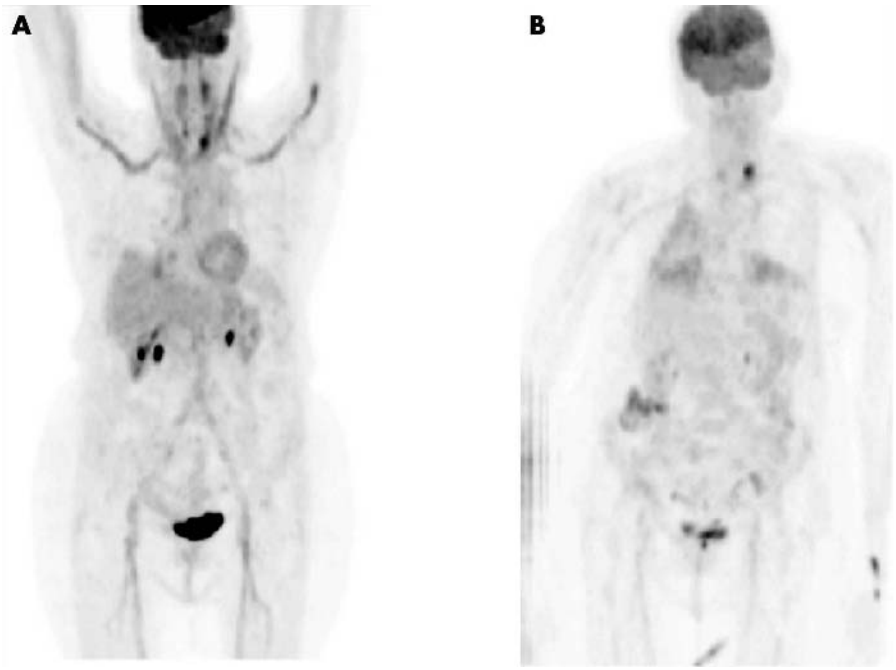

Figure 1 (A) FDG-PET before B lymphocyte depletion shows significant vascular uptake of FDG in both external carotid and subclavian arteries. Part of the abdominal aorta and both iliac arteries also show increased vascular FDG uptake. (B) FDG-PET 4½ months after treatment shows a reduction of vascular uptake with no uptake in the iliac vessels or ascending aorta.

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