Letters to the Editor

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Fatigue in rheumatoid arthritis

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Sir, We write in response to an editorial in *Rheumatology* defining what is known and what remains to be learned about fatigue in RA [1]. Repping-Wuts *et al.* [1] recognize fatigue as a common and severe complaint, having a major impact on the quality of life in patients with RA. They emphasize that doctors often underestimate this effect and also that the issue is usually raised by patients rather than by health care professionals. We agree with this and welcome the suggestion that a more proactive approach to the identification and management of fatigue in RA is necessary. However, it is important that fatigue is not assumed to be secondary to RA in isolation. Other potentially treatable contributory conditions should be actively sought. Anaemia is frequently associated with RA and often presents with fatigue. Hypothyroidism is also common in patients with RA, usually as a result of autoimmune disease, and symptoms may include fatigue. Most rheumatologists would recognize these associations and check a full blood count and thyroid stimulating hormone in an RA patient with such symptoms.

We recently investigated fatigue in our RA population by checking random cortisol levels in 50 patients who complained of excessive fatigue, and in whom anaemia and hypothyroidism had already been excluded. We performed a Synacthen test in the eight patients whose results were low (<200 nmol). This was normal, excluding hypoadrenalism, in four. Two further patients had a blunted response as a result of previous long-term oral steroid therapy producing inhibition of adrenal response. The two remaining patients had little response to ACTH, with high circulating ACTH levels and anti-adrenal antibodies. Baseline cortisol levels were 26 and 51 nmol, rising 30 min after ACTH to just 112 and 120 nmol, respectively. These two patients had primary adrenal failure (Addison's disease) of autoimmune aetiology. They subsequently responded well to physiological doses of hydrocortisone. Ethical approval for the study was provided by the Northern Regional Ethical Committee.

Clearly our data do not allow any firm conclusions to be drawn about the prevalence of Addisons disease in RA. However, it is feasible that autoimmune adrenalitis may be more common in RA than in the population at large [2]. Certainly an association between Addisons disease and other chronic disorders has been demonstrated, with the authors postulating links through autoimmunity [3]. We suggest that fatigue in patients with RA, in addition to being a feature of that disease, might represent coexisting physical disorders relating to other autoimmune processes. Before assigning such symptoms to RA itself, clinicians may be well advised to check thyroid function and cortisol levels. Such a strategy should sensibly preface the introduction of oral steroids.

Rheumatology key message

- Fatigue in RA may be due to other autoimmune disorders.

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

1 Repping-Wuts H, van Riel P, van Achterberg T. Fatigue in patients with rheumatoid arthritis: what is known and what is needed. Rheumatology 2009;48:207–9.
2 Oelkers W. Adrenal insufficiency. N Engl J Med 1996;335:1206–12.
3 Biagi F, Campanella J, Soriani A, Vailati A, Carazza GR. Prevalence of coeliac disease in Italian patients affected by Addisons disease. Scand J Gastroenterol 2006;41:302–5.

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Efficacy of rituximab in refractory and relapsing myositis with anti-JO1 antibodies: a report of two cases

Sir, The anti-Jo1 (histidyl tRNA synthetase) antibodies are often associated with myositis, arthritis, RP, mechanic's hand and interstitial lung disease (ILD), the latter governing the prognosis of this anti-synthetase syndrome. A 49-year-old woman presented marked muscular weakness (psoas and deltoids MRC 3/5, legs held outstretched at 45° supine: 22 s, normal >75 s) with anti-Jo1 antibodies. Creatine kinase (CK) level was 1114 U/l ($N < 170$ U/l). Muscle biopsy confirmed myositis. CT scan was normal. The patient was treated with prednisone 1 mg/kg/day (60 mg/day) associated with AZA (150 mg/day). Three months later, she improved (psoas and deltoids MRC 4/5, legs outstretched at 45° supine: >75 s) with normalization of CK levels. At 12 mg/day of prednisone (after 20 months), she relapsed with muscle weakness (psoas and deltoids MRC 3/5, legs outstretched at 45° supine: 30 s), polyarthralgia and increased CK levels (1018 U/l) leading to introduce monthly high-dose (2 g/kg) intravenous immunoglobulins (IVIgs) and daily oral mycophenolate mofetil (MMF, 2 g/day). However, after nine courses of IVIg, weakness progressed (psoas and deltoids MRC 3/5, legs outstretched at 45° supine: 10 s) and CK continued to rise (3296 U/l). We decided to then use rituximab (2 × 1 g, 2 weeks apart), while increasing prednisone to 1 mg/kg/day and maintaining MMF. A third injection of rituximab (1 g) was performed 6 months later. The patient showed then total recovery with normal muscle strength, normal CK level, allowing to taper corticosteroid doses at 10 mg/day and to stop MMF. Ten months after the third injection of rituximab, she exhibited a further relapse with polyarthralgia, proximal muscle weakness (psoas MRC 3/5 and deltoid MRC 4/5, legs outstretched at 45° supine: 26 s) and CK levels at 4863 U/l. She then received again rituximab (1 g 2 weeks apart). Six months later, symptoms disappeared and CK decreased to 150 U/l.

A 19-year-old woman presented with proximal muscle weakness (psoas and deltoids MRC 4/5, legs outstretched at 45° supine: 70 s) with anti-Jo1 antibodies. CK level was 11 990 U/l. Muscle biopsy confirmed myositis. CT scan revealed ILD. Prednisone 1 mg/kg/day associated with MMF (2 g/day) were started with a