Patients with anti-SRP antibodies have a clinical pattern typical of PM (typically developing rapidly progressive, severe, proximal muscle weakness with frequent occurrence of muscle atrophy) [2–4]. In general, patients have extremely high CK levels at presentation. The histological appearance of muscle biopsy specimens in anti-SRP myopathy is of sparse inflammatory infiltrate but significant muscle necrosis [2, 4].

In general, patients with anti-SRP antibodies have a good initial response to high-dose steroid therapy but the myopathy relapses with dose reduction leading to the requirement for very high cumulative doses of steroids [4]. Both of our patients failed AZA, MTX and mycophenolate mofetil prior to B-cell depletion therapy as is typical in these patients [3].

Anti-SRP myopathy is felt to be the disease primarily of the humoral immune system [5] and anti-SRP antibodies have been shown to inhibit the function of signal recognition proteins by inhibiting the binding of SRP to the SRP receptor [6], supporting a role for B-cell depletion in the treatment of the disease.

Rituximab has been used on a number of occasions for the treatment of refractory myositis [7–9] and good results have been reported in the treatment of anti-SRP myopathy with the use of rituximab following plasma exchange [5]. In our experience, whereas both patients had a rapid biochemical response the clinical response was not as impressive as previously reported. This may be due to the fact that some SRP-positive patients have been noted to have a paucity of hypertrophic fibres on muscle biopsy suggesting that the persistent weakness may be due to a lack of compensatory muscle hypertrophy [3].

In summary, we have reported our experience of treating two patients with anti-SRP myopathy, with aggressive B-cell depletion. Both patients had a poor clinical response, which is in contrast to the only other report of the use of rituximab in this patient population.

Disclosure statement: The authors have declared no conflicts of interest.
SIR, We thank Dr Kittisupamongkol for raising the important question of analgesic drug use [1] in our cohort study of the mortality experience of individuals with musculoskeletal pain [2]. To analyse the effect of these drugs it would have required the collection of continuous drug information for the whole period of follow-up which was out-with the design of the study. The study was of a population cohort and all types of musculoskeletal pain were recorded. Over half (52.1%) of all subjects reported experiencing pain in the past month that had lasted for ≥1 week. Only a minority of those reporting pain would have arthritis and it is unlikely that a significant proportion of subjects with pain would be taking COX-2-specific drugs, although ibuprofen and diclofenac use would be more widespread. Although data showing an increased cardiovascular risk for these drugs do exist, the size of the risk is moderate [3]. The use of NSAIDs for pain should have resulted in a reduction of colorectal cancer risk [4]. Although we were unable to directly assess that relationship in the current study, this was one of the cancers researched in excess in our previous study [5]. The increased risk of cardiovascular deaths attributable to analgesic drugs in our study population is likely to be minimal and would be unlikely to explain the magnitude of the effect observed.

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

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Accepted 2 February 2009

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Rheumatology 2009;48:595
doi:10.1093/rheumatology/kep041
Advance Access publication 18 March 2009

Comment on: Musculoskeletal pain is associated with a long-term increased risk of cancer and cardiovascular-related mortality: reply

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