Excellence (NICE) [1]. In rheumatology, we have had a similar experience with NICE guidance being at variance with patient and carer opinions [2]. This has been reflected to be due to rationing [3]. In 2005, NICE published the guidelines for the secondary prevention of osteoporosis in post-menopausal women [4]. These have included the advice that women over the age of 75 yrs who have sustained a fracture should be put on bisphosphonates without the need for a dual X-ray absorptiometry (DEXA) scan. We assume that this might have been due to the unequal provision of DEXA scanning nationally. We decided to audit the referrals to a district general DEXA service to ascertain whether osteoporosis (as defined by a T-score of less than −2.5 in the spine or hip) was as prevalent in this age group as NICE have implied.

Morecambe Bay has had a DEXA service since 1992 and since June 2004 has been recording fracture data. Since then, 1551 patients above the age of 75 yrs have been scanned, of whom 711 patients above the age of 75 yrs have been scanned, of whom 711 had experienced an osteoporotic fracture (hip, wrist, spine, pelvis, fibula/tibia, rib). Five hundred and seventy-seven of these patients were women with a mean age of 79.8 yrs (S.D. 3.86). The mean T-score was −2.4 (S.D. 1.29). The distribution of the T-score is shown in the given histogram (Fig. 1) and this indicates that although this population’s scores are shifted to the left, their mean scores would preclude them from automatically being prescribed bisphosphonates. Two hundred and eighty-six (49.6%) of these patients had a T-score that put them outside the osteoporotic range and 63 (10.9%) were not even osteopenic. Although this might be an indicator that bone density is not the perfect surrogate for bone fracture, there is still significant room for misclassification and excessive treatment. This data agrees with recently published studies [5].

This provides some evidence that NICE puts other considerations before issuing evidence-based guidelines. Perhaps commissioning research and surveys like our simple one would have prevented many unnecessary prescriptions and allowed resources to be used more appropriately; this would result in a better developed service to reduce the burden of osteoporotic fracture.

Rheumatology key message

- NICE guidance is potentially flawed as not all patients over 75 yrs with fragility fractures have osteoporosis.
conflicting results in open-label studies of the efficacy of anakinra in AS [1, 2], it is clear from both the studies that certain individuals respond to the drug for up to 24 weeks. In our three cases, the response was sustained for up to 30 months. It may be that patients with sustained response have a more IL-1-mediated disease than non-responders. Our cohort also had a 40% greater baseline CRP compared with the German cohort, indicating that higher serum markers of inflammation may be a predictor of response as has been suggested for anti-TNF response in AS [7]. Haibel et al. [2] reported that 21% of their cohort achieved ASAS40 response that is substantially greater than the reported placebo response of 5.1% in a previous AS anti-TNF study [8].

To summarize, we feel that the available data do not completely exclude a role for anakinra therapy in some cases of AS. It remains a strong possibility that the inflammation associated with AS may be amenable to IL-1 pathway modulation and it is possible that IL-1 blockade, either partially with anakinra or more completely with monoclonal antibodies, may have a role. Future trials will address these issues.

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Academic Unit of Musculoskeletal Disease, Chapel Allerton Hospital, Leeds, UK

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Correspondence to: D. McGonagle.

E-mail: d.g.mcgonagle@leeds.ac.uk


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**Progressive multifocal leucoencephalopathy in a patient with systemic lupus erythematosus treated with rituximab**

Sir, Progressive multifocal leucoencephalopathy (PML) is a rare but usually fatal demyelinating disease of the brain caused by JC papovavirus (JCV). At least 50–75% of the adult population are seropositive for JCV. When JCV reactivation occurs, focal plaques develop in central nervous system white matter. Viral proliferation causes lysis of oligodendrocytes and therefore rapid demyelination [1]. Rituximab is a relatively novel therapy for systemic lupus erythematosus (SLE) and PML has not previously been reported in a patient with SLE treated with rituximab.

The patient was a 45-yr-old woman with SLE, who presented with blurred vision, loss of balance, difficulty in speaking and swallowing and weight loss increasing over 3 weeks. She had a 23-yr history of SLE characterized by photosensitivity, facial rash, alopecia, Raynauld’s syndrome, neutropenia, thrombocytopenia and raised titres of anti-nuclear, anti-Ro, anti-Sm and anti-ribonucleoprotein (RNP) antibodies. Anti-double-stranded deoxy-ribonucleic acid (dsDNA), C3, C4 and complement levels were normal.

In the past, she had been treated with corticosteroids, azathioprine and oral cyclophosphamide, which had failed to control her neutropenia. Three months after discontinuation of cyclophosphamide, because of bone marrow toxicity, she developed severe haemorrhagic cystitis, and a cystectomy and urostomy were performed. There was a 3-month delay between stopping cyclophosphamide and the development of haemorrhagic cystitis that was not compatible with drug-induced bladder toxicity. It is possible that primary infection or reactivation of JCV caused haemorrhagic cystitis, as has been reported with other papovaviruses [2]. Unfortunately, the diagnosis of JCV haemorrhagic cystitis was not suspected at the time of cystectomy in 1999 and immunohistochemical staining for BK virus (BKV) was not carried out.

When neutrophil counts were <0.5 × 10⁹/l, the patient had developed pneumonia, and when prednisolone doses were ≥20 mg or higher, Herpes zoster reactivation occurred on three occasions. Between 2002 and 2005, she received two courses of rituximab, 375 mg/m² × 4 weeks, and a single treatment of rituximab, 375 mg/m². SLE went into remission within 1 month of rituximab treatment and prednisolone was discontinued. At the time of presentation with neurological symptoms, she had been off prednisolone for 1 year and SLE had been in remission.

Examination revealed dysarthria, nystagmus in all directions, pass-pointing, dysdiadochokinesis and an ataxic gait. Muscle power and tendon reflexes were normal, plantar responses down-going and sensation intact. Laboratory investigations revealed a low white cell count of 2.1 × 10⁹/l, neutrophils 1.02 × 10⁹/l and lymphocytes 0.71 × 10⁹/l. Anti-CD19-positive B-cell counts were zero and CD3-positive lymphocytes were 10% (10% CD4 and 8% CD8). Magnetic resonance imaging (MRI) of the brain demonstrated three white matter lesions, one in each cerebellar peduncle and one in the brainstem (Fig. 1). Cerebrospinal fluid analysis revealed increased protein (51 mg/l)...

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Academic Unit of Musculoskeletal Disease, Chapel Allerton Hospital, Leeds, UK

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Correspondence to: D. McGonagle.

E-mail: d.g.mcgonagle@leeds.ac.uk


