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Polymyalgia rheumatica

Editor – Dasgupta, writing on behalf of the polymyalgia rheumatica (PMR) guideline development group, presents a welcome and thorough overview of this common condition (*Clin Med* June 2010 pp 270–4). I have concerns with the recommended three-monthly 'lab monitoring' of full blood count, erythrocyte sedimentation rate/C-reactive protein (ESR/CRP), urea and electrolytes and glucose. The management of straightforward PMR is to relieve symptoms (and not to treat inflammation) until the condition runs its natural course. Steroid withdrawal should be based on the clinical picture and not on the level of ESR and this is alluded to in the article 'raised ESR/CRP without clinical symptoms is not an indication to continue corticosteroids'.

It is my belief, based on reviewing many patients with PMR and iatrogenic Cushing's/osteoporosis, that the main reason for the continuation of higher dose steroids is the regular checking of an ESR to follow disease activity. The secret to the successful management of straightforward PMR is, once the diagnosis has been made, never to check an ESR/CRP unless there is a clinical indication.

GRANVILLE MORRIS
Consultant geriatrician
Prince Philip Hospital, Llanelli

In response

Editor – We thank Dr Morris for highlighting an important issue – the objectives of steroid treatment for polymyalgia rheumatica (PMR). Steroids are prescribed for their important effect on pain, disability and stiffness and the quality of life in untreated PMR is lower than in most other comparable conditions. On the other hand steroids also have many side effects and over-treatment based on raised inflammatory markers alone may prolong duration of treatment and induce treatment co-morbidities such as fractures, diabetes, hypertension, weight gain and cataracts.

However, we now know that the PMR constitutes only one of many conditions that can present with bilateral shoulder pain and stiffness. Such conditions include late onset rheumatoid arthritis, other arthropathies, spondyloarthropathies and connective tissue diseases. Large vessel vasculitis may also present with polymyalgia, constitutional symptoms and raised inflammatory markers. Other serious pathology, such as systemic infection, disseminated cancer and so on, may also be mistaken as PMR and may have an initial response to high dose steroids.

We therefore stand by our recommendation of inflammatory marker testing in the context of a clinical review – especially in the first year of disease. Transient elevations of CRP/ESR may be due to common causes such as urinary or chest infections and urinalysis and chest radiographs may be considered. Persistent elevation in the presence of definite symptoms suggests partial or non-response to steroids, search for alternative pathology or adjuvant immunosuppresives and a specialist referral. Persistent symptoms in the absence of elevated markers suggests evaluation of co-existing non-inflammatory conditions such as osteoarthritis, rotator cuff and other local shoulder conditions, fibromyalgia, etc. These conditions should be dealt with by explanation, reassurance and local treatments such physiotherapy, injections and exercises; while the steroid dose is tapered.

BHASKAR DASGUPTA
Consultant in rheumatology and honorary professor
Essex University Southend University Hospital
Westcliff-on-Sea, Essex
On behalf of the PMR guideline development group

A complicated hyperglycaemic emergency

Editor – I read with interest the article by Vidyarthi and Chowdhury describing a hyperosmolar non-ketotic diabetic emergency complicated by diabetes insipidus (*Clin Med June* 2010 pp 264–5).

I agree that these complex cases are best managed in a critical care environment where point of care testing is available to guide therapy. I feel a number of other features merit clarification, however. Firstly, the authors fail to emphasise that hyperglycaemia causes water shift from intracellular fluid (ICF) to extracellular fluid (ECF). Correction of hyperglycaemia thus causes in a influx of water back into the ICF causing a rise in serum sodium despite reduced free water losses. As this rise is accompanied by an influx of water into the brain, osmotic demyelination syndrome (central pontine myeliniolysis) should not arise, as long as serum osmolarity is falling. Conversely cerebral oedema can be a risk if serum osmolarity falls very rapidly with volume expansion. However, this danger may have been overemphasised in this case where serum osmolarity paradoxically rose with therapy, attributed by the authors to diabetes inspidus of uncertain aetiology. I feel administration of large volumes of 0.9% saline may have contributed to this outcome. The patient described was in early shock with a mean