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Correspondence

Tendon rupture in systemic lupus erythematosus

SIR, We note with interest the report on spontaneous rupture of weight bearing tendons in systemic lupus erythematosus (SLE).[1] We report a case of rupture of a non-weight bearing tendon, namely the long head of biceps.

A 35 year old woman had a six year history of SLE, which has been clinically quiescent since 1982. In January 1985 she sustained a fracture of the surgical neck of the left humerus with minimal displacement. This was treated conservatively, and she made an uneventful recovery. In August 1985 she presented with a one month history of spontaneous swelling in the left arm. Examination showed a non-tender mass in the middle third of the arm, which became more marked with resisted elbow flexion. Overall function was well preserved and surgical repair was not undertaken.

This is the first report of spontaneous rupture of a non-weight bearing tendon in SLE. At presentation, SLE was quiescent and there was no history of left shoulder arthritis or bicipital tenosynovitis. In keeping with previous reports[1] our patient was on long term (four and a half years) corticosteroid therapy, possibly resulting in attenuation of the biceps tendon. It is unlikely that the humeral fracture caused the tendon rupture in January 1985 and remained undetected for seven months. We conclude that spontaneous rupture may occur in both non-weight bearing and weight bearing tendons in SLE patients.

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Reference

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Amyloid arthropathy in patients with chronic renal failure

SIR, I was interested in the recent paper by Muñoz-Gómez *et al* on amyloid arthropathy[1] and Dr Rowe's very readable editorial review[2] also entitled 'Amyloid arthropathy' which preceded it. I am, however, concerned by the name given to this disorder which implies a causative relation between amyloid in the joint and the patients' symptoms. Although it is undeniably possible that this could be the case, the link does not appear to have been established. It is important to know whether Muñoz-Gómez and colleagues had failed to show the presence of amyloid in the synovial fluid or synovium of asymptomatic patients on long term haemodialysis, in patients with primary hyperparathyroidism or, indeed, in age and sex matched controls. In this laboratory we have also been engaged in looking for amyloid in material removed at carpal tunnel decompression. While in our study biopsy specimens from patients receiving haemodialysis have shown amyloid deposits in perineural connective tissue, so too have specimens from other members of the 'control' group, all of whom have normal renal function.

Even if it could be shown that the amyloid deposition described by Muñoz-Gómez *et al* is unique to those patients on chronic haemodialysis who present with joint symptoms it still does not establish a causal relationship. It would be just as valid to claim that because most patients with osteoarthritis have amyloid within their joints, then amyloid is the cause of osteoarthritis.

The clinical and pathological findings of Muñoz-Gómez *et al* are interesting, but I can see nothing in the present paper which excludes their being epiphenomena. Until such time as properly controlled studies are performed and a mechanism linking the clinical syndrome with the microscopic findings can be identified there appears to be little justification for using the term 'amyloid arthropathy', with all its attendant pathogenetic implications, for the disorder which they describe.

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2 Rowe I F. Editorial: Amyloid arthropathy. *Ann Rheum Dis* 1985; **44**: 727–8.

Antikeratin antibody in rheumatoid and psoriatic arthritis

SIR, We were interested to read the recently published studies[1 2] investigating the disease specificity of antikeratin antibodies. The knowledge that psoriasis is a disorder characterised by an abnormality of keratinisation prompted us to carry out a preliminary study into the prevalence of antikeratin antibodies in patients with

psoriatic arthritis, and compare this with the observed frequency in patients with rheumatoid arthritis.

Sera from 14 patients with psoriatic arthritis and 13 patients with rheumatoid factor positive arthritis, fulfilling American Rheumatism Association criteria for definite or classical rheumatoid arthritis, were examined for the presence of antikeratin antibodies using the method described by Young *et al.*[3] Estimation of rheumatoid factor titre was carried out by an enzyme linked immunosorbent assay.

None of the 14 patients with psoriatic arthritis had identifiable antikeratin antibodies or significant levels of rheumatoid factor. Of the patients with rheumatoid disease, four of 13 were positive for antikeratin antibodies including, most notably, the two patients with highest titres of rheumatoid factor (336 and 304 IU/l respectively). These results are in accord with the finding of both a high specificity of antikeratin antibodies for rheumatoid arthritis, and the observed correlation between antibody level and disease activity;[4] they certainly do not support a role for them in the pathogenesis of psoriatic arthritis.

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2 Youinou P, Goff P Le, Colaco C B, *et al.* Antikeratin antibodies in serum and synovial fluid show specificity for rheumatoid arthritis in a study of connective tissue diseases. *Ann Rheum Dis* 1985; **44:** 450–4.
3 Young B J J, Mallya R K, Leslie R D G, Clark C J M, Hamblin T J. Antikeratin antibodies in rheumatoid arthritis. *Br Med J* 1979; **ii:** 97–9.
4 Mallya R K, Young B J J, Pepys M B, Hamblin T J, Mace B E W, Hamilton E B D. Antikeratin antibodies in rheumatoid arthritis: frequency and correlation with other features of the disease. *Clin Exp Immunol* 1983; **51:** 17–20.

Sweat gland function in Sjögren's syndrome

SIR, Although it is generally agreed that the skin dryness associated with Sjögren's syndrome (SS) is caused by decreased secretion from chronically inflamed sweat glands,[1] sweat output in patients with SS has not been fully investigated. In the only study specifically to address this issue Bloch *et al*[2] reported normal sweat production and a normal sweat sodium content in each of the seven patients they tested with pilocarpine iontophoresis. However, in presenting their findings, these authors neither described their study design nor recorded the ages and symptomatology of their study population.

To investigate further whether diminished sweat output is indeed a feature of SS and to assess whether alterations in sweat sodium concentrations are an additional feature of the condition we studied 12 patients (nine women, three men) with SS of one to 13 years' duration. Their ages ranged from 28 to 65 years. Of the 12, three had the sicca complex alone, while the remaining nine had associated connective tissue disease (rheumatoid arthritis in three and SLE in six). Eight of the 12 reported skin dryness as a specific, troublesome symptom of their disease.

Since sweat output is known to diminish with advancing age and to differ between the sexes,[3] we matched each patient for age and sex with a normal control. Each subject in the study was referred for pilocarpine iontophoresis which was carried out according to a standardised method recommended by Schwartz *et al.*[4] After applying a 3 × 3 cm square of filter paper of known weight to the iontophoresed area for one hour the paper square was reweighed. The difference between these two weights provided a measure of sweat production, expressed in milligrams. Sweat sodium concentrations were then calculated.[4]

By means of a two tailed unpaired *t* test at the 0·05 significance level we were unable to show a statistically significant difference between the mean sweat weights of the group with SS and their matched controls (Table 1). Moreover, the mean sweat sodium concentrations for both patients and controls fell within the normal range (<60 mmol/l) and there was no statistically significant difference between the two groups. When we compared the eight patients who specifically complained of skin dryness with the four who did not there was again no detectable difference in either sweat weights or sodium concentrations.

Although our results suggest that there is neither a measurable diminution in sweat output nor a change in sweat sodium concentration in patients with SS, it is possible that we have missed a statistically significant difference between patients and controls because of our small sample size.[5] It is also possible that our method of pilocarpine iontophoresis, although standardised and universally accepted for use in adults with cystic fibrosis, may be insensitive to small changes in the sweat secretions and sodium concentrations of patients with SS. Nonetheless our findings do seem to cast some doubt on the generally

Table 1 *Sweat weights and sweat sodium concentrations in 12 patients with Sjögren's syndrome and their age and sex matched controls*

	Mean (standard deviation)		*p Value*
	Patients	*Controls*	
Sweat weight (mg)	0·33 (0·12)	0·30 (0·14)	NS*
Sweat sodium concentration (mmol/l)	48·71 (17·10)	61·61 (14·11)	NS*

*Not significant at 0·05 significance level.