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Steroid treatment should be started without delay to avoid the high risk of blindness.

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1. Goldberg JW, Lee ML, Sajjad SM. Giant cell arteritis of the skin simulating erythema nodosum. *Ann Rheum Dis* 1987;46:706-8.
2. Baum EW, Sams WM, Payne RR. Giant cell arteritis: a systemic disease with rare cutaneous manifestations. *J Am Acad Dermatol* 1982;6:1081-8.
3. Soderstrom SW, Seehafer JR. Bilateral scalp necrosis in temporal arteritis: a rare complication of Horton's disease. *Am J Med* 1976;61:541-6.
4. Cohen DN. Facial swelling in giant cell arteritis. *J Rheumatol* 1982;9:325-8.
5. Jeanmaire H *et al.* Lesion ulceronecrotique de la face relevant une maladie de Horton. *Presse Med* 1992;21:1820.
6. Wong RL, Korn JH. Temporal arteritis without an elevated erythrocyte sedimentation rate. *Am J Med* 1986;80:959-63.
7. Huston KA, Hunder GG, Lie JT, Kennedy RH, Elveback LR. Temporal arteritis: a 25 year epidemiologic clinical and pathologic study. *Ann Intern Med* 1978;88:162-7.
8. Allsop CJ, Gallagher PF. Temporal artery biopsy in giant cell arteritis: a reappraisal. *Am J Surg Pathol* 1981;5:317-23.

Arthritis and Spondylodiscitis Caused by *Mycobacterium xenopi* in a Patient with Systemic Lupus Erythematosus

SIR—We read with interest the article by Coombes *et al.* [1], on a case of tenosynovitis in an immunocompetent patient involving *Mycobacterium xenopi*, and would like to report a patient with arthritis of the left shoulder and spondylodiscitis due to *M. xenopi*.

A 56-yr-old woman presented to the out-patient clinic with a painful left shoulder. This pain started 2 months before, and gradually increased. The patient had been suffering from systemic lupus erythematosus (SLE) for >20 yr, which was treated with a combination of low-dose corticosteroids and azathioprine. She had never before experienced painful joints. Apart from the painful left shoulder, she had some dyspnoea on exertion. Physical examination was unremarkable except for a swollen left shoulder with a solid, 2.5 cm tumour on top of the acromioclavicular joint. Movements of the shoulder were tender and moderately limited in all directions. Laboratory examinations revealed a slightly elevated ESR (28 mm/h); renal and liver function tests, and blood counts were in the normal range. Aspiration yielded 2.5 ml of clear synovial fluid, without crystals on microscopic examination. Gram and Ziehl-Neelsen stains were negative, as were routine bacterial and fungal cultures. Aspirate was inoculated onto Lowen-

stein-Jensen and Middlebrook K7 H10 culture medium, and incubated at 30 and 37°C. A Mantoux reaction remained negative. A chest X-ray showed no abnormalities, an X-ray of the left shoulder revealed some decalcification of the distal part of the clavicle, and an enlarged joint space of the acromioclavicular joint. Ultrasound examination showed an increased mass of the synovial tissue of the acromioclavicular and glenohumeral joints, and of the subdeltoid and subacromial bursa. Magnetic resonance imaging (MRI) confirmed these findings. A proposed arthroscopy was cancelled because of spontaneous reduction of the pain and swelling of the left shoulder.

Two months later, she presented with back pain. The VIIIth and IXth thoracic vertebrae were painful on palpation. The left shoulder was still slightly limited in motion. Routine laboratory examinations were normal except for an elevated ESR (31 mm/h). X-ray examination of the thoracic spine was suggestive for spondylodiscitis of these vertebrae. MRI examination showed a decreased T1 and increased T2 signal intensity of the VIIIth and IXth thoracic vertebrae, and the intervertebral disc, compatible with spondylodiscitis. Histological examination of several biopsies, taken from the inflammatory lesion of the VIIIth and IXth thoracic vertebrae, revealed low-grade chronic osteomyelitis, without granulomata. Gram and Ziehl-Neelsen stains of the biopsy specimen were negative. At this time, cultures of the synovial fluid of the left shoulder, taken 2 months previously, demonstrated growth of a *Mycobacterium* species, later identified as *M. xenopi*. Cultures from the biopsy specimen became positive for *M. xenopi* after 7 weeks of incubation. Sensitivity testing showed that *M. xenopi* was sensitive to isoniazid, clarithromycin, ciprofloxacin, amikacin, ansamycin, lampren, protionamide, cycloserine and streptomycin, and resistant to rifampicin and ethambutol. Triple anti-tuberculous therapy, consisting of isoniazid, clarithromycin and ciprofloxacin, was initiated. After 2 months of treatment, the pain in her spine and left shoulder abated, and the ESR had fallen to 17 mm/h. Anti-tuberculous treatment was discontinued after 9 months. Since then, with a follow-up of 30 months, the patient feels well and no recurrence of the infection was noted. Recently performed ultrasound examination of the left shoulder showed calcifications of the tendons of the biceps and supraspinatus muscles, and an MRI of the thoracic vertebrae showed destruction of the VIIIth vertebra, with normal T1 and T2 signal intensities.

As made clear in the article of Coombes *et al.* [1], *M. xenopi* rarely causes non-pulmonary disease. Only a few reports mention musculoskeletal disease due to *M. xenopi* [2]. Spinal infections due to *M. xenopi* have been reported before in four patients [3-6]. Like our patient, two of these patients were suffering from SLE, treated with low doses of corticosteroids. In our patient, *M. xenopi* was the cause of both the arthritis of the left shoulder and the spondylodiscitis. To the best of our knowledge, this combination of infections

caused by *M. xenopi* has not been reported before. It illustrates that in patients with rheumatic diseases with recent onset inflammation of any part of the musculoskeletal system, mycobacteria should be considered as a possible causative agent, especially when immunosuppressive drugs are used.

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1. Coombes GM, Teh LS, Denton J, Johnson AS, Jones AKP. *Mycobacterium xenopi*—an unusual presentation as tenosynovitis of the wrist in an immunocompetent patient. *Br J Rheumatol* 1996;35:1008–10.
2. Simor AE, Salit IE, Vellend H. The role of *Mycobacterium xenopi* in human disease. *Am Rev Respir Dis* 1984;129:435–8.
3. Prosser AJ. Spinal infection with *Mycobacterium xenopi*. *Tubercle* 1986;67:229–32.
4. Rahman MAA, Phongsathorn V, Hughes T, Bielawska C. Spinal infection by *Mycobacterium xenopi* in a non-immunosuppressed patient. *Tubercle Lung Dis* 1992;73:92–5.
5. Miller WC, Perkins MD, Richardson WJ, Sexton DJ. Pott's disease caused by *Mycobacterium xenopi*: Case report and review. *Clin Infect Dis* 1994;19:1024–8.
6. Jones PG, Schragar MA, Zabransky RJ. Pott's disease caused by *Mycobacterium xenopi*. *Clin Infect Dis* 1995;21:1352.

Informing the Public About Treatment Advances

SIR—In their viewpoint article, Deighton and Doherty [1] propose guidelines for the publicity of therapeutic research advances. Unfortunately, their proposals serve no useful purpose and do not relate in any way to the process by which the media report and comment upon scientific developments. Your journal would do better to have commissioned an article from those who have dealt more fully with this area and have consulted more widely.

In the case of the developments with which I was involved [2] and which Deighton and Doherty quote, the initial publicity was generated by a national charity at the time of the award of the research grant which made the study possible. It was reinforced by the learned society which decided to issue a press release at the time of presentation of the earliest abstract, and magnified by press coverage related to the annual Arthritis Education Week (which was not directly related to the report). In practice, however, the Press Association and other journalists were continually pressing for further information about the study from its earliest days. It was seen by scientific reporters as an important story, and did, after all, report a new and important therapeutic advance which was immediately available to thousands of people all over the world.

Publicity cannot be controlled. Deighton and Doherty's suggestions are uninformed, naive and impossible to implement. Openness with information and honest debate can at least make the processes of science accessible to the public.

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1. Deighton C, Doherty M. Treatment advances—when and how to inform the public? *Br Rheumatol* 1996;35:1175–7.
2. Kirwan JR, Arthritis and Rheumatism Council Low-Dose Glucocorticoid Study Group. The effect of glucocorticoids on joint destruction in rheumatoid arthritis. *N Engl J Med* 1995;333: 142–6.

Reply

The response by Dr Kirwan unfortunately reinforces the concerns expressed in our viewpoint article [1]. His perspective as the principal investigator, echoed widely by the media, was that low-dose corticosteroid 'is a new and important therapeutic advance'. This treatment was not new, was not a major advance, did not have its potential caveats discussed, and was not placed in the context of other treatments. Subsequent to its publication [2], the study has not been widely perceived as the seminal work Dr Kirwan believes it to be [3]. Public 'education' at the time of his grant award, when the abstract was first presented, and then on the day the paper appeared, was hardly likely to encourage balanced appraisal of the merits of the work. Because corticosteroids were 'immediately available', considerable re-education of disappointed patients was required in the wake of the publicity. Dr Kirwan does not concede this problem and may have missed it whilst engaged with the media.

We may be naive and uninformed in many matters, but between us have preceded and succeeded Dr Kirwan on the ARC Education Committee, have been involved in *British Medical Journal* policy on national media coverage, and have been directly involved in newspaper, radio and television 'news items'. Before writing the viewpoint, we canvassed opinion from GPs, rheumatologists and uARC officers, many of whom shared our concerns. Dr Kirwan accepts the immediacy of the press and believes it leads to honest debate. His own example, however, does not inspire confidence. We do not accept the inevitability of media domination, but favour continuing debate on how to improve mechanisms that impart accurate information with minimal bias and sensationalism.

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1. Deighton C, Doherty M. Treatment advances—when and how to inform the public? *Br J Rheumatol* 1996;35:1175–7.
2. Kirwan JR. The effect of glucocorticoids on joint destruction in rheumatoid arthritis. *N Engl J Med* 1995;333:142–6.
3. Morrison E, Capell H. Corticosteroids in the management of rheumatoid arthritis. *Br J Rheumatol* 1996;35:2–4.