

this case, the simultaneous occurrence of anti-Jo-1 and anti-U1RNP autoantibodies is a difficult diagnostic problem. Autoantibodies against Jo-1 are usually highly specific for the anti-synthetase syndrome.¹ However, in this case, the manifestation was incomplete, lacking pulmonary and peripheral vascular involvement. On the other hand, anti-U1RNP antibodies are a serological marker for MCTD, in which myositis is not an essential part but a typical symptom.² However, the titre of the anti-U1RNP antibodies was not as high as it is typically for MCTD. The finding of low complement levels is not typical for myositis and might have been due to overlap of myositis with MCTD as complement factors can be decreased, especially in MCTD. Although an association between anti-Jo-1 and anti-Ro/SSA 52 kDa responses is relatively common, as reported by former studies,^{4,6} the coincidence of anti-Jo-1 and anti-U1RNP antibodies appears to be less common.³ Autoantibodies against proteasome were recently found in association with autoimmune myositis and other connective tissue diseases.^{7,8} In this case, the unique autoantibody pattern detected impressively reflects the variability of these myositis related markers and emphasises that they belong to the common immune repertoire seen in autoimmune myositis.

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Degenerative disc disease and pre-existing spinal pain

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Appportioning pain and disability after a car accident or work related injury can be difficult. Many doctors who undertake this task often state that because an *x* ray examination or magnetic resonance imaging (MRI) scan soon after the injury shows degenerative disc disease (DDD), some or all of the patient's spinal pain and disability must be pre-existing. This interpretation of imaging is not consistent with the peer reviewed medical published reports.

For this statement to be true, there would need to be a strong connection between MRI or *x* ray evidence of DDD and pain/disability. If we look at this concept and compare it with the published reports, we see that DDD, as seen on imaging, is not a painful condition.

Several studies have been performed in this area. The oldest was published in the *Journal of Neuroimaging* in 1991. In this study patients without low back pain underwent an MRI scan; 39% of this normal group had evidence of DDD.¹ A *New England Journal of Medicine* article in 1994 found similar results. It showed that of 98 subjects without low back pain, 52% had DDD on MRI.² Similar findings were discovered in the thoracic spine (upper back) by Wood *et al* in the *Journal of Bone and Joint Surgery* in 1995. Thoracic MRI scans were performed in 90 asymptomatic adults; 73% of these patients had DDD at least one level.³ Similar findings have been found in the radiographic analysis of asymptomatic cervical spines, with the prevalence of DDD increasing with age. In addition, MRI has been found to have high false negative and positive rates for predicting painful discs in this area.⁴

If DDD is not painful, then why do MRI scans and *x* ray examinations of people with spinal pain often show DDD? The reason is probably that DDD can predispose a patient to a painful spinal condition. Important clues can be gleaned from recent research showing that painful discs have nerve in-growth.⁵ Additional research has shown that degenerated discs move abnormally and this property may predispose them to injury in a traumatic event.⁶ Finally, we have much to learn about the cause of axial spinal pain, but it seems clear that MRI scans and *x* ray examinations are often not sufficiently sensitive to show us the cause.

Attribution is yet another problem. For instance, if a patient has evidence on examination of a right sided L5 radiculopathy, then looking for right sided L5-S1 disease may be fruitful. However, the converse is problematic. If the patient has DDD of the right L5-S1 area on an old low back *x* ray but clearly has no symptoms or signs of this disorder on examination, then we must assume the problem had not yet reached the point of being symptomatic.

In summary, DDD as seen on *x* ray examination and MRI scans is not a painful condition, therefore evidence of this "disorder" before an accident or injury does not mean that the patient had a painful pre-existing condition. Although it is true that some patients with DDD do have pain, it is also true that many patients without DDD have pain. Furthermore, high percentages of the normal, pain-free population have DDD. From the peer reviewed research in this area, DDD seems to be a normal part of the aging process and not "smoking gun" evidence of a pre-existing problem.

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Tuberculous tonsillitis in a patient receiving etanercept treatment

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Since the approval by the Food and Drug Administration (FDA) of tumour necrosis factor α antagonists, infections have accounted for 21% of the adverse events reported to the FDA for etanercept and 20% of those reported for infliximab.^{1,2}

As of May 2001, from approximately 147 000 subjects receiving infliximab treatment, 70 patients have been reported to have developed active tuberculosis. Of these, 52% presented with extrapulmonary tuberculosis while 24% presented with disseminated disease.³ As of April 2001, from approximately 102 000 subjects receiving etanercept treatment, nine patients have been reported to have developed active tuberculosis.⁴ With the case report presented herein we want to add evidence to suggest that atypical presentation of active tuberculosis may also be seen in patients receiving etanercept treatment.

CASE REPORT

The patient, a 56 year old Filipino man with no significant past medical history other than receiving BCG immunisation at age 23 before he emigrated to the United States, presented to our clinic for a third opinion for his polyarthritis symptoms. He had initially been seen two years previously by a rheumatologist for left knee swelling and diffuse asymmetric arthralgias. A raised erythrocyte sedimentation rate and a raised rheumatoid factor prompted a diagnosis of rheumatoid arthritis. Treatment was started with a tapering dose of prednisone and weekly oral methotrexate. The patient discontinued both drugs about two months later and sought the advice of another rheumatologist about a year later. At that time it was noted that the patient had swelling of his wrists, ankles, and knees, and etanercept was started for a presumptive diagnosis of rheumatoid arthritis. No radiological studies were performed.

About a week before our evaluation the patient had developed a swollen left tonsil and was evaluated by an otolaryngologist, who performed a resection of his left tonsil to rule out a possible malignancy or infection.

On physical examination there was evidence of small bilateral knee effusions as well as an erythematous rash over his upper arms. There were no palpable nodules and he complained of morning stiffness that lasted for about 10 minutes. He had no respiratory complaints and his lung fields

were clear to auscultation. Radiographic studies of his hands, wrists, and knees showed osteoarthritic changes without erosions or periarticular osteopenia. A diagnosis of inflammatory osteoarthritis was made, and the patient was treated with hydroxychloroquine 200 mg twice daily, while etanercept was discontinued because of the new diagnosis, and also because stains of the left tonsil had shown acid fast bacilli. Antituberculous treatment was initiated by a consulting infectious disease specialist once disseminated disease was ruled out.

DISCUSSION

Two issues come to the forefront with this case. Even though biological agents are new treatments for rheumatoid arthritis, the increased incidence of infectious adverse events, should make us reserve these treatments for patients who meet the clinical criteria for a diagnosis of rheumatoid arthritis. Also of importance, and as described in this case report, is the fact that not only patients receiving infliximab but also patients receiving etanercept can have atypical presentations of *Mycobacterium tuberculosis* infections.

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