Dear Editor,

We read in interest Espinoza and García-Valladares's article entitled ‘Of Bugs and Joints’.1 We agree that the epidemiology of reactive arthritis (ReA) is difficult to determine, especially in the absence of any internationally validated diagnostic criteria or guidelines. Whilst the clinical features of a ReA secondary to a sexually transmitted infection (STI) are indistinguishable from those caused by an enteric organism, the management could potentially be different. As was discussed, there is evidence that chlamydia induced ReA may benefit from a prolonged course of combination antibiotics.1,3

We wondered how good clinicians were at identifying the responsible organism? Is sexually acquired ReA (SARA), an under-recognised diagnosis, perhaps due to a reluctance from the rheumatologist to discuss and investigate such matters?

We conducted an audit to establish whether patients with suspected ReA were screened for STIs. The first clinic letter of all new referrals <30 years of age to both the general rheumatology and the early arthritis clinics in the preceding 6 months was reviewed. Out of 244 referrals, 42 patients were considered to potentially have ReA and of these only 24% (10/42) were screened for an STI (all negative).

It is not reassuring that no STIs were detected because over three quarters of patients were not tested. STIs are common in the young sexually active population, with chlamydia affecting 5–10% of those under 24 years, and in females especially it can be completely asymptomatic.5 If a patient denies any ‘promiscuous activities’ or appears to be in a stable relationship should they still be screened?

References


Emily Pease ∗ Benedict Pease Colin Pease
Leeds Teaching Hospitals NHS Trust, Yorkshire, United Kingdom

Corresponding author.
E-mail address: emilypease@doctors.org.uk (E. Pease).

DOI of refers to article: http://dx.doi.org/10.1016/j.reuma.2012.06.008

Dear Editor,

We read in interest Espinoza and García-Valladares’s article entitled ‘Of Bugs and Joints’.1 We agree that the epidemiology of reactive arthritis (ReA) is difficult to determine, especially in the absence of any internationally validated diagnostic criteria or guidelines. Whilst the clinical features of a ReA secondary to a sexually transmitted infection (STI) are indistinguishable from those caused by an enteric organism, the management could potentially be different. As was discussed, there is evidence that chlamydia induced ReA may benefit from a prolonged course of combination antibiotics.1,3

We wondered how good clinicians were at identifying the responsible organism? Is sexually acquired ReA (SARA), an under-recognised diagnosis, perhaps due to a reluctance from the rheumatologist to discuss and investigate such matters?

We conducted an audit to establish whether patients with suspected ReA were screened for STIs. The first clinic letter of all new referrals <30 years of age to both the general rheumatology and the early arthritis clinics in the preceding 6 months was reviewed. Out of 244 referrals, 42 patients were considered to potentially have ReA and of these only 24% (10/42) were screened for an STI (all negative).

It is not reassuring that no STIs were detected because over three quarters of patients were not tested. STIs are common in the young sexually active population, with chlamydia affecting 5–10% of those under 24 years, and in females especially it can be completely asymptomatic.5 If a patient denies any ‘promiscuous activities’ or appears to be in a stable relationship should they still be screened?

References


Emily Pease ∗ Benedict Pease Colin Pease
Leeds Teaching Hospitals NHS Trust, Yorkshire, United Kingdom

Corresponding author.
E-mail address: emilypease@doctors.org.uk (E. Pease).

DOI of refers to article: http://dx.doi.org/10.1016/j.reuma.2012.06.008

Dear Editor,

We have carefully read the excellent review by Hernandez et al.,1 with regard to skin lesions that occur during treatment with antagonist of tumor necrosis factor (anti-TNF), and we would like to make some additional comments with respect to cutaneous lupus erythematosus (LE) induced by such drugs.

As the authors report, the development of autoantibodies is a frequent event in patients receiving anti-TNF drugs,2 with an estimated prevalence of ANA positivity ranging from 25% to 80% and anti-DNA ranging from 5% to 15%.2 However, as they state, the appearance of LE is quite rare.3 Postmarketing studies estimate the incidence of induced LE at 0.19%–0.22% for infliximab, 0.18% for etanercept and 0.17% for adalimumab.2 The slightly higher frequency of LE induced with infliximab however, the mechanism remains unclear.

The frequency with which these cases appear in the literature contrasts to those described in RA clinical trials with long-term follow-up.4,5 As was discussed, there is evidence that chlamydia induced ReA may benefit from a prolonged course of combination antibiotics.3,4

Another factor that could influence this is the underlying disease. Although the proportion of autoantibodies is similar among the different diseases treated with these agents, most cases have been described in RA patients, as evidenced by a review of Costa et al.,6 who found that of 33 published cases of induced LE due to anti-TNF drugs, 76% of patients had RA. The frequency with which these cases appear in the literature contrasts to those described in RA clinical trials with long-term follow-up, so it should be noted that these cases are generally based on retrospective observations that often lack serological data prior to starting anti-TNF therapy and there may be some overlap of RA and LE before treatment.3

LE cases induced by anti-TNF comply with 4 or more ACR classification criteria in 40%, 3 criteria in 21%, and 2 or less in 39%.3 Up to 67% of cases have cutaneous manifestations,2 corresponding generally to maculopapular, pruritic erythematous rash affecting photosensitive areas, as mentioned by the authors,1 however, the spectrum is much broader. Both LE-specific lesions (cutaneous acute, subacute and discoid), and other non-specific findings including urticarial lesions, scarring, alopecia and purpura may occur.2 Within difficult to classify cutaneous LE lesions, there has also been published cases of LE tumidus and lupus pernio (LP) induced by anti-TNF. LE tumidus is characterized by the appearance of papules on exposed areas, erythematous plaques or nodules without other associated epidermal changes; one of the cases found in the literature occurred with infliximab and adalimumab in another,9 both in RA patients. Our group conducted a review of 5 cases of LP associated with anti-TNF,10 a rare form of cutaneous LE characterized by papules or plaques with erythematous violaceous acral distribution that simulate ischemic injury. Four of these cases occurred in patients with RA and one in ankylosing spondylitis.