Do rheumatologists think about sex? ¿Piensan los reumatólogos en el sexo?

Dear Editor:

We read in interest Espinoza and Garcia-Valladare'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.4 If a patient denies any ‘promiscuous activities’ or appears to be in a stable relationship should they still be screened?

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Cutaneous lupus erythematosus induced by the treatment with tumor necrosis factor antagonists

Lupus eritematoso cutáneo inducido por la terapia biológica con antagonistas del factor de necrosis tumoral

Dear Editor,

We have carefully read the excellent review by Hernandez et al., 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, with an estimated prevalence of ANA positivity ranging from 25% to 80% and anti-DNA ranging from 5% to 15%. However, as they state, the appearance of LE is quite rare. Postmarketing studies estimate the incidence of induced LE at 0.19%–0.22% for infliximab, 0.18% for etanercept and 0.10% for adalimumab. The slightly higher frequency of LE induced with infliximab [last accessed 21.11.12]. This is no more onerous than routinely testing the same people for rheumatoid factor, anti-CCP and HLA B27.

Our findings were presented both locally and at the 2012 Rheumatology conference in Glasgow, reiterating the importance of STI screening. A re-audit 1 year later found that STI screening had increased to 50% (13/26) and two chlamydia infections were identified. This increase indicates that with clear guidance, clinicians are more likely to carry out an STI screen, and supports the need for a national ReA guideline.

As the authors described so well, the relationship between ‘bugs and joints’ is clear, however, the underlying STI may not be obvious. SARA may well be an under-recognised diagnosis due to the absence of testing, not the absence of infection. Clinicians must view STI screening as ‘routine’, if only those who are perceived to be high risk for infection are tested, then infection will be missed.

We suggest if a diagnosis of ReA is being considered all patients should be tested, regardless of the social circumstances that they chose to disclose in their rheumatology consultation. The initial screening for an STI need involve only a first pass urine sample in males and in females a self-taken vulvo-vaginal swab sent for nucleic acid amplification testing (NAAT). This is no more onerous than routinely testing the same people for rheumatoid factor, anti-CCP and HLA B27.

In conclusion, we would encourage clinicians to be aware of the frequency of ReA in patients with anti-TNF, and we would like to make some additional comments with respect to cutaneous lupus erythematosus (LE) induced by such drugs.


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References

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In summary, although induced LE is a rare adverse event seen during anti-TNF treatment, it is important to have in mind because of its varied clinical expression, especially on the skin, and to identify those cases that actually are due to this entity, given the trend that may lead to overdiagnosis.

References


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