

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

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.⁴ If a patient denies any 'promiscuous activities' or appears to be in a stable relationship should they still be screened? 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).⁴ 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.⁵ 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 guidance.

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.

Bibliografía

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<http://dx.doi.org/10.1016/j.reuma.2013.03.001>

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

Lupus eritematoso cutáneo inducido por la terapia biológica con antagonistas del factor de necrosis tumoral**Cutaneous lupus erythematosus induced by the treatment with tumor necrosis factor antagonists**

Sr. Editor:

Hemos leído atentamente la excelente revisión de Hernández et al.¹ respecto a las lesiones cutáneas que ocurren durante el tratamiento con fármacos antagonistas del factor de necrosis tumoral (anti-TNF), y quisiéramos hacer algunas precisiones adicionales respecto al lupus eritematoso (LE) cutáneo inducido por estos fármacos.

Como los autores refieren, el desarrollo de autoanticuerpos es un evento frecuente en pacientes que reciben fármacos anti-TNF¹, estimándose una prevalencia de positividad de ANA del 25 al 80% y de anti-ADN del 5 al 15%.² Sin embargo, como también ellos establecen, la aparición de LE es bastante infrecuente¹. Los estudios poscomercialización estiman una incidencia de LE inducido del 0,19-0,22% para infliximab, el 0,18% para etanercept y el 0,10% para adalimumab². La ligera mayor frecuencia de LE inducido con infliximab y etanercept puede simplemente reflejar más años de exposición de pacientes en comparación con adalimumab. En relación con los anti-TNF introducidos más recientemente, certolizumab y golimumab, se ha descrito un caso de lupus inducido con el primero³ y otro de exacerbación de LE cutáneo subagudo previo con el segundo⁴. Considerando la alta prevalencia de autoanticuerpos y el gran número de pacientes tratados, se esperaría una mayor frecuencia de LE inducido. Una de las probables explicaciones para esta discrepancia es que el tipo de respuesta autoinmunitaria inducida por los anti-TNF está restringida principalmente a isotipos IgM o IgA

Véase contenido relacionado en DOI:
<http://dx.doi.org/10.1016/j.reuma.2012.04.007>