Trauma and seronegative spondyloarthropathy

Sir, Olivieri and his colleagues report the apparent precipitation of seronegative spondyloarthropathy by physical injury. The first case can easily be explained as the onset of a reactive arthritis following gastrointestinal infection while in hospital. It is extremely interesting that the mother of that individual had what sounds very much like ankylosing spondylitis. The second patient may well be a patient with intermittent hydroarthrosis of the knees related to the B27 arthropathy. Trauma being a common event in life, it may simply be related stoichiometrically to the effusion and not causally.

The coincidence of two events does not imply causality and great caution should be exercised in attributing such a causality as there are major legal implications in cases of compensation due to injury at work or elsewhere.

Rheumatology Unit Shepherds House, Guy's Hospital, London SE1 9RT

Reference


Synovial fluid and amyloidosis

Sir, We read with interest the article on dialysis related arthropathy, published in the Annals, by Hurst et al., with whom we agree in general.

With reference to the method used for the diagnosis of synovial fluid amyloidosis, and especially to their conclusion that ‘this technique requires further evaluation before it can be accepted as diagnostically useful’ we would like to point out the following. In our work we did not use the immunoperoxidase technique to diagnose the presence of amyloid in the synovial fluid sediment, but to confirm that the amyloid fragments that we found with the Congo red staining and polarising microscopy contained \( \beta_2 \) microglobulin.

We think that the authors’ explanation for their false positive results with the immunochemical technique — ‘soluble \( \beta_2 \) microglobulin precipitates as an amorphous deposit’ — is very interesting and indeed could be the answer.

Therefore we and others consider that the diagnosis of synovial amyloidosis of any origin can be achieved by studying the synovial fluid sediment and that the immunochemical techniques must be used to show the type of amyloid deposits previously detected with Congo red and polarising microscopy.

Departments of Rheumatology and Pathology, Hospital Clinico, 08036 Barcelona, Spain

References


Sir, We thank Drs Muñoz-Gómez and Solé for their interest in our article and for drawing attention to our comments about the problems associated with the use of immunoperoxidase staining of synovial sediments to identify \( \beta_2 \) microglobulin related amyloidosis.

Our intention was simply to emphasise that immunoperoxidase staining of synovial fluid sediment could not be used alone as a diagnostic test and could give rise to false positive results. The implication in our text that this approach had been suggested by Muñoz-Gómez et al who, as they point out, used Congo red as their primary diagnostic test, was quite unintentional.

Sentimos mucho el error!

Rheumatology Unit, The Queen Elizabeth Hospital, Woodville, South Australia 5011

References
