Correspondence

Retinoids and hyperostosis

Sir, The article by van Dooren-Greebe et al. requires clarification. The authors indicate that 77 of their subjects were treated with synthetic retinoids for psoriasis but give no indication how many of these subjects also had evidence of psoriatic arthritis or psoriatic spondylitis. Nor is it clear how many of the patients having baseline X-rays had psoriasis.

This is important for two reasons:

1. If none of the 77 patients with psoriasis had psoriatic arthropathy or spondylitis, it is possible the survey has excluded a population of patients susceptible to retinoid-induced spinal abnormalities.

2. The radiographic changes of psoriatic spondylitis may be indistinguishable from DISH and retinoid-induced hyperostosis. As the radiologist was "unaware of details from the patients records", there is the possibility of misinterpretation of spinal findings.

Perhaps the authors could look again at their data with particular reference to psoriatic arthritis and spondylitis.

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References


Reply

Sir, We would like to reply to the letter of Drs Cheesbrough, concerning our recent article.1

In the present retrospective study we focused on the relationship of the duration of oral retinoid treatment and the prevalence of spinal abnormalities. We could not demonstrate such a relationship. Not a single patient was excluded from retinoid treatment because of pre-existing skeletal abnormalities. Patients with psoriatic arthritis or psoriatic spondylitis were not excluded from retinoid treatment.

As the present study had a retrospective design, and because of the fact that baseline X-rays were not available in all patients, we were not able to make a subdivision in patients with and without (pre-existing) psoriasis-induced spinal abnormalities.

As we stated in the discussion part of this communication, we agree with Drs Cheesbrough and Hellwell that it might be possible that a certain group of patients, for example those patients with psoriatic arthritis or psoriatic spondylitis, might be at high-risk for the development of retinoid-induced skeletal abnormalities. Further (prospective) investigations should indeed be focused on defining possible high-risk patient groups in this respect.

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Reference


Is methotrexate liver toxicity modest worldwide?

Sir, I read with interest the article of Dr M.J.Boffa et al. on methotrexate (MTX) liver toxicity in psoriasis.1 MTX has been neglected for years in Italy because of an overemphasized fear of liver toxicity. The reappraisal is therefore welcome. However, how can a study on liver toxicity be performed without any evaluation of previous hepatitis C virus (HCV) infection? As far as I am aware, no studies on MTX liver toxicity have investigated the possible interaction HCV infection. HCV infected patients may have neither symptoms nor elevation of liver enzymes yet still have chronic active hepatitis. Transaminases often show transitory elevations which may pass unnoticed. In such patients, introduction of a toxic and immunosuppressive drug like MTX may reactivate the virus and increase liver toxicity.

Actually, as HCV infection in Dr Boffa’s geographical area affects only 0.04% of blood donors,2 none of his patients was likely to be HCV positive. However, lack of information about HCV status, does not permit optimistic conclusions about MTX safety that may not be shared in countries with higher HCV prevalence than the U.K.1

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References