though the different ways in which one condition causes the other are not known. Some studies have suggested that in patients with chronic HCV+ hepatitis, the whole panel of autoantibodies (AMA, ANA, anti-SMA, anti-LKM) could affect the response to interferon and clinical profile of liver disease.[6] However, when ANA was analysed separately, it did not appear to confirm these assumptions.[5] Fortunately, it seems that the presence of these ANAs in HCV+ patients with or without IFN is not related to IFN efficacy.[3]

Our patient became positive only for ANA, remaining negative for the rest of the antibodies related to both lupus and AIH. She has not developed either of these clinical syndromes or classical viral relapse (occult HCV not analysed). Although she has not had a liver biopsy at any time, currently, we see no justification for monitoring these antibodies unless she shows clinical or biochemical abnormalities of some form.

In patients with hepatitis C, with or without interferon treatment, ANA not only bears the possibility of developing SLE, but could also be related with the hepatic infection itself.

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Letter to the Editor – Comments on published articles

Reply to the comment "Infection with hepatitis C virus, interferon α and lupus: An odd association"☆

Respuesta al comentario de «Infección por virus de la hepatitis C, interferón α y lupus, una curiosa asociación»

Dear Editor:

We hereby thank Dr Martín-Gómez for her interest in our article "Infection with hepatitis C virus, interferon α and lupus: An odd association"[1] and her comment on it.

As she clearly explains in her remark, the presence of antinuclear antibodies (ANA) among patients with chronic HCV infection has been extensively described in the literature as an immune epiphenomenon lacking clinical significance in most cases.[2]

Additional supporting tests, including extending the autoantibody profile, should only be performed in patients whose clinical or analytical findings are unrelated to hepatitis C. This rules out associated diseases, such as autoimmune hepatitis or drug-induced lupus.

Following treatment, our patient had fever, asthenia, and arthralgia, as well as positive anti-histone antibodies. The temporal relationship between concomitant interferon treatment and negative HCV tests resulted in the diagnosis and subsequent therapy of the patient.

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By contrast, the advent of new drugs for the treatment of HCV, such as the nucleotide NS5B polymerase inhibitor, sofosbuvir, and the NS5A inhibitor, daclatasvir, paves the way for interferon-free treatments. These new drugs will avoid the interferon-associated adverse effects, while achieving highly sustained virologic response rates. However, further studies should be conducted in special populations,[3] including patients with end-stage renal disease undergoing haemodialysis.

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