

vs. 25% placebo) and incidence of acute liver failure (20% vs. 42.5%). The study also showed that the sooner the treatment is initiated, the better the results obtained. In two additional studies,^{6, 7} patients with severe acute or fulminant hepatitis B were treated with lamivudine, demonstrating the safety and efficacy of this antiviral drug, with a capacity for improving the prognosis of these patients.

On the basis of these studies, it appears reasonable to recommend antiviral treatment for patients with severe AHB, as it improves survival rates and reduces the incidence of acute liver failure. Despite the absence of studies with tenofovir or entecavir, these are the drugs that could be recommended by major clinical practice guidelines.^{8, 9}

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Letter: pathogenesis of tumour necrosis factor-alpha antagonists-induced psoriasiform lesions

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SIRS, We read with interest the article by Buisson *et al.*¹ As described by the authors' the pathogenesis of the tumour necrosis factor-alpha antagonists (anti-TNF- α)-induced psoriasiform lesions has not yet been fully clarified.² The most widely accepted hypothesis is based on an interaction between the reduction in TNF- α and the increase in interferon-alpha (IFN- α),^{2, 3} instead of the increase of interferon- γ described in the study.¹ It is believed that plasmacytoid dendritic cells (natural IFN- α producers) are capable of inducing psoriasis through IFN- α production.^{4, 5} Since such plasmacytoid cells are usually down-regulated by TNF- α , its inhibition by the biological may determine increased and uncontrolled

IFN- α production and consequently induce or exacerbate psoriasis.⁶

In the literature, in addition to the *in vitro* studies described by the authors,¹ there are other reports^{3, 7} that support such a relationship between IFN- α and anti-TNF- α -induced psoriasiform lesions. A study³ detected strong production of protein MxA (a specific marker for IFN signalling) in the inflammatory cells of skin samples of anti-TNF- α -induced psoriasis as compared with controls. Another study⁷ found increased IFN- α expression in the psoriatic lesions of patients receiving anti-TNF- α therapy, as compared with spontaneous psoriasis.

Furthermore, since the time between anti-TNF- α administration and the development of psoriatic lesions may be extremely variable,² an environmental trigger could be involved in this pathophysiological mechanism.⁸ In addition, since TNF- α antagonists have been administered to more than two million patients worldwide⁹ and cases of this adverse cutaneous event remains in the hundreds,² this paradoxical phenomenon may be related to a genetic predisposition.^{2, 10} Consequently, future genetic studies may be able to help with the identification of predisposed patients as well as with the elucidation of the specific immunopathogenic mechanism.

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