

Overwhelming diffuse psoriasis during chronic HCV infection, after peginterferon-ribavirin treatment, supported by frequent resort to filgrastim rescue

TO THE EDITOR

Associated treatment with pegylated interferon plus specific antivirals significantly improved the prognosis of chronic hepatitis C and B,¹ although these drugs (especially interferon and its derivatives) tend to be myelotoxic. Some rescue treatments, like human recombinant granulocyte colony-stimulating factors (which are extensively administered in order to correct neutropenia induced by antiviral therapy), may also be involved in prompting or exacerbating cutaneous psoriasis and its systemic complications.

A representative case report is the one of a woman with chronic, progressive, hepatitis C, who underwent long-term treatment with combined pegylated interferon plus ribavirin, and resorted to multiple cycles of filgrastim to recover from a severe and recurring granulocytopenia caused by antiviral therapy itself and to maintain an effective dosage of anti-HCV antivirals. The patient developed an extensive and severe cutaneous psoriasis, which improved only after specific cyclosporin treatment.

From a pathogenetic point of view, in our case it remains extremely difficult to distinguish the role of pegylated interferon from that of the accompanying ribavirin and from that of the frequently administered granulocyte growth factor (filgrastim), since all forementioned drugs were administered concurrently over several months. According to evidence from the existing literature, all of these drugs have the potential to induce psoriasis as an untoward effect in subjects suffering from chronic hepatitis. Cyclosporin treatment induced a stable remission of this last severe cutaneous complication, but the efforts to contain the progression of the underlying hepatitis C were blunted by the difficult-to-treat genotype 1 HCV infection, and the frequent need to lower drug dosages and/or to interrupt antiviral therapy, because of myelotoxic and later cutaneous complications prompted by anti-HCV therapy itself. With regard to the management of psoriasis in these special hosts, the dermatologist needs to be involved in order to obtain an early diagnosis, to consider differential diagnoses of HCV-infected patients receiving multiple pharmacological treatments and with non-infrequent co-morbidities (i.e. HIV disease), and to monitor patients over treatment and over time.² Also in HCV-infected patients with a newly recognized psoriasis or with an exacerbating psoriatic disease, cyclosporin seems a suitable treatment, with times and modes of delivery tailored on every single patient and disease presentation and evolution, as recently commented.³ However, novel-generation compounds (like etanercept) are also expected to play a role in the future, since they act on the activated component of TNF-alpha, which may have a remarkable role in the pathogenesis and treatment of HCV disease itself, complicated by psoriasis.⁴

[*Braz J Infect Dis* 2010;14(4):327]©Elsevier Editora Ltda.

REFERENCES

1. Simin M, Brok J, Stimac D, Gluud C, Gluud LL. Cochrane systematic review: pegylated interferon plus ribavirin vs. interferon plus ribavirin for chronic hepatitis C. *Aliment Pharmacol Ther* 2007; 25:1153-62.
2. Yamamoto T, Yokozeki H, Nishioka K. Evaluation of psoriatic patients with HCV infection. *J Dermatol* 2005; 32:769-70.
3. Imafuku S, Tashiro A, Furue M. Cyclosporin treatment of psoriasis in a patient with chronic hepatitis C. *Br J Dermatol* 2007; 156:1367-9.
4. Linardaki G, Katsarou O, Ioannidou P, Rarafoulidou A, Boki K. Effective etanercept treatment for psoriatic arthritis complicating concomitant human immunodeficiency virus and hepatitis C virus infection. *J Rheumatol* 2007; 34:1353-5.

Authors

Sergio Sabbatani, MD¹
Roberto Manfredi, MD¹

¹Department of Clinical and Experimental Medicine, Division of Infectious Diseases, "Alma Mater Studiorum" University of Bologna, S. Orsola-Malpighi Hospital, Bologna, Italy.

Submitted on: 03/04/2010
Approved on: 03/20/2010

Correspondence to:

Roberto Manfredi, MD
Associate Professor of Infectious Diseases, University of Bologna c/o Infectious Diseases, S. Orsola Hospital Via Massarenti 11 – Bologna, Italy I-40138
Phone: +39-051-6363355
Fax: +39-051-343500
E-mail: roberto.manfredi@unibo.it