"help-rejecting complainer," to the extremely straightforward. As an example of the latter, I offer the lady who came to me some years ago saying, "Well, doctor, you know I'm a picker. My mother was a picker, I'm a picker, and my daughter's a picker." She announced it in the same tone of voice and with the same demeanor and conviction as if she had just told me they were all Republicans, and proud of it.

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Treatment of psoriasis with cyclosporine in patients with hepatitis C infection: Risk or opportunity?

To the Editor: We appreciated the article by Frankel et al[1] in the December 2009 issue of the *Journal* regarding the treatment of psoriasis in patients with hepatitis C virus (HCV) infection and the considerable efforts of the Frankel et al to develop practical therapeutic guidelines and arrive at a consensus on treating psoriasis in this particular cohort of patients. Indeed, as a consequence of the worldwide distribution of HCV, it is not infrequent to encounter in daily clinical practice patients with psoriasis also carrying HCV. In such cases, the treatment of psoriasis often raises a special challenge for physicians.

However, the clinical dermatologist could be misguided about the concern regarding the use of cyclosporine (CyA) that arises from this paper. As a matter of fact, the rank of this drug in the treatment of psoriasis in patients affected by HCV has been penalized as compared to the other systemic therapies. In particular, Frankel et al put CyA as a third-line therapy along with azathioprine, while acitretin, etanercept, and possibly other tumor necrosis factor inhibitors, along with psoralen plus ultraviolet A light phototherapy, are regarded as second-line therapies.[1] The authors affirm that CyA, according to the current literature, is relatively contraindicated in patients with HCV because of its immunosuppressive effect, although it may actually have an antiviral effect and therefore be considered cautiously for treatment of psoriasis in patients with HCV.[1]

From a "historical" point of view, CyA is undoubtedly regarded as an immunosuppressive drug; however, the recent discoveries of its anti-HCV effects disclose innovative perspectives for considering this drug in the treatment of psoriasis in HCV patients.

Since 1997, HCV reactivation has been reported after the tapering of CyA dosage in posttransplant patients, consistent with a flare from immune reconstitution.[2] In 2003, Watashi et al[3] showed that CyA exerts an inhibitory effect on HCV replication in vitro. From then on, many clinical observations have confirmed a beneficial impact of CyA on HCV. Inoue et al[4] showed that CyA combined with interferon (IFN) produced a greater sustained biochemical and virology response than IFN monotherapy among HCV patients. A combination of IFN beta with CyA proved be effective for HCV patients who failed previous pegylated-IFN or classical IFN combined ribavirin treatment.[5] Among a number of immunosuppressants used in clinical renal transplantation alone or in combination with IFN in 153 HCV-positive recipients of renal transplantation, only CyA, when used alone, strongly inhibited the growth of HCV-RNA, effectively preventing the progression of chronic hepatitis.[6]

The inhibitory effect on HCV replication exerted by blocking cellular cyclophilin B is independent of the immunosuppressant function of CyA, which is exerted through inhibition on calcineurin.[7] Cyclophilin inhibitors, a novel class of drugs including CyA, are among the most promising of the new anti-HCV agents under development, and their efficacy profiles make them attractive candidates for combination with current and future HCV treatments.

Although literature data concerning safety and efficacy of CyA in the treatment of HCV-infected psoriatic patients are limited to few case reports, additional clinical evidences can be found in studies on patients affected by other autoimmune disorders carrying HCV.

Miura et al[8] described four patients affected by different dermatologic disorders, including psoriasis and HCV infection, who were treated with long-term administration (24-36 months) of CyA with reduced

HCV load and aminotransferase levels in three out of four patients. The safety of CyA in the treatment of autoimmune disorders with concomitant HCV infection, even in patients with chronic active hepatitis, was confirmed in two different studies on 26 individuals, seven of whom had psoriatic arthritis.[9-10]

We can conclude that, according to Frankel et al,[1] further investigations on the safety of CyA in the treatment of psoriasis in HCV-infected patients are advisable, but until this time, the available data indicate that CyA can contribute to a good outcome in patients affected by psoriasis and concomitant HCV infection in terms of both safety and efficacy.

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Scalp necrosis in giant cell arteritis after initiation of therapeutic corticosteroids

To the Editor: We read with interest the article by Tsianakas et al[1] in the October 2009 issue of the *Journal* entitled "Scalp necrosis in giant cell arteritis: case report and review of the relevance of this cutaneous sign of large-vessel vasculitis," in which the authors analyzed 78 case reports published between 1946 and 2007. Important findings of this article included an increased incidence of vision loss, an increased rate of mortality, and a delay of about 1 month in diagnosis of giant cell arteritis (GCA) with associated scalp necrosis compared with those of patients with GCA alone.[2] The authors emphasized the need for early treatment with glucocorticoids to help prevent serious complications, and noted that scalp necrosis has never been reported after initiation of adequate treatment. Three case reports in the medical literature have detailed patients in whom scalp necrosis developed with low dose (range, 5-35 mg daily) prednisolone,[3] whereas therapeutic doses are generally thought to be 40 mg to 60 mg or more.[4]

We recently evaluated an 80-year-old woman who presented with biopsy-confirmed bilateral GCA and subsequent onset of scalp necrosis 1 week after initiation of prednisone 60 mg daily. The patient had a 14-month history of polymyalgia rheumatica (with a moderately elevated erythrocyte sedimentation rate of 31 [reference range, 0-29 mm/h]) and more recently (1 month before presentation to our dermatology clinic) had experienced headaches, diplopia, jaw claudication, sore throat, and scalp tenderness. The physical evaluation revealed bilateral frontotemporal scalp erosions and necrosis with hemorrhagic and fibrinous crusting (Fig 1). She also had a slight loss of peripheral vision in the left eye, which was confirmed by ophthalmologic evaluation. Biopsy specimens of the temporal arteries taken at another institution were reviewed, and the diagnosis of GCA was reconfirmed. Findings of polymerase chain reaction