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CORRESPONDENCE

Migratory pulmonary infiltrates in a patient treated with sotalol

To the Editors:

We read with interest the case study by FALLER *et al.* [1] concerning migratory pulmonary infiltrates in a patient treated with sotalol. The authors concluded that β-blockers produce an adverse effect, but we have good reason to think that the diagnosis can be refined. In particular, the patient could have been suffering from an underlying disease, such as a drug-induced lupus, and this point did not appear to have been discussed.

First, the patient was female, and this is an epidemiological argument for exposure to systemic lupus erythematosus (SLE), even though this predominance is less marked in the case of induced lupus. The clinical presentation included nonspecific signs of SLE: slight fever, systemic hypertension and, in particular, a more specific sign, Raynaud's phenomenon, that should not be neglected. The patient was treated with β-blockers, which are known to provoke induced lupus [2, 3]. These are often pauci-symptomatic and benign forms of SLE and do not fit the American Rheumatism Association (ARA) criteria of SLE.

Secondly, immunological investigations did not contain a dosage of soluble antinuclear factors and, in particular, antihistone antibodies. The latter have a good sensitivity in these cases of induced lupus, and their absence may have diverted the suspicion of an induced lupus. Furthermore, even if seric antinuclear antibodies were absent here, as is commonly observed, one could perform antibody immunodetection on the lung biopsy with the same aim.

Thirdly, although specific pulmonary involvements of SLE are rare compared to infectious manifestations, there have been several observations of bronchiolitis obliterans organizing pneumonia (BOOP) accompanying a lupus [4–7] since the first description by MATTAY *et al.* [8] in 1974, and this would not be an isolated report.

Finally, the authors did not mention the evolution of the Raynaud's syndrome after the discontinuation of treatment; it would be interesting to know the course of all of the clinical symptoms, particularly this uncommon symptom, after the resolution of BOOP.

For all of these reasons, we believe that this BOOP was not an adverse effect of the β-blocker treatment, but appeared as a manifestation of an induced lupus. This case report may represent more accurately the first case of a bronchiolitis obliterans organizing pneumonia in the course of a drug-induced lupus in a patient treated with sotalol.

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REPLY

From the authors:

We thank L. Portel for his remarks regarding our paper "Migratory infiltrates in a patient treated with sotalol", published recently in the Journal [1].

β-blockers are well known to be responsible for the development of a lupus [2]. In the patient studied, the slight fever, systemic hypertension and Raynaud's syndrome could be part of a drug-induced lupus. However, Raynaud's syndrome and systemic hypertension were present for many years before introducing sotalol. After resolution of the bronchiolitis obliterans organizing pneumonia (BOOP), these two clinical manifestations persisted. It is therefore difficult to connect these two symptoms with the pulmonary manifestations, even if it is known that Raynaud's phenomenon may persist in spite of the treatment and may, by itself, be a predictive factor for the development of a drug-induced lupus [3].

With reference to the antinuclear antibodies, we read in the recent "Cecil Textbook of Medicine" that the seric antinuclear antibodies are positive in 95% of spontaneous lupus and 95% of drug-induced lupus [4]. Antihistone antibodies are present in 25% of spontaneous lupus and

90% of drug-induced lupus, and may also be positive in other diseases as well as rheumatoid arthritis [3, 4]. Antinuclear antibodies are frequent with β-blocker treatment and, a small number of these cases develops a drug-induced lupus [3].

Accordingly, since the seric antinuclear antibodies were absent, we therefore did not look for the antihistone antibodies. We can find neither clinical nor biological support for the hypothesis of a drug-induced lupus.

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