Reply to: Calcium pyrophosphate disease and polymyalgia rheumatica: association or coincidence?

Sirs,

We have read with interest the comments from Milchert *et al*. regarding our recent study about the ultrasound (US) shoulder assessment of calcium pyrophosphate disease (CPPD) among patients with suspected polymyalgia rheumatica (PMR) (1). In their correspondence, the authors highlighted two important points that need to be discussed. First, the authors discussed the fact that CPPD can coexist with PMR and that finding CPPD in PMR-suspected patients could be a coincidence. Additionally, they suggested that CPPD is an infrequent PMR-mimicking condition (2, 3). Indeed, regarding the studies by Manzo *et al*. and Ceccato *et al*., the proportion of CPPD patients was low (2, 3). However, in those studies, screening of CPPD was not systematically performed without US assessment limiting the ability to diagnose CPPD. Moreover, patients were followed in primary care whereas our patients were referred to a tertiary care hospital. In contrast, in the study by Pego-Reigosa *et al*., one third of their PMR-suspected patients were classified PMR/CPPD (4). We fully agree that it is difficult to conclude that CPPD should be a diagnosis of exclusion among PMR-suspected patients. The targeted population (elderly people) is similar in both diseases. In absence of specific markers for PMR diagnosis and due to the fact that CPP crystals do not disappear, a coexistence of the two diseases remains possible. We discussed this point in the manuscript. We think that presence of CPPD do not allow for excluding PMR. Our results suggested that, in presence of CPPD, the physician needs to search for atypical clinical presentation of PMR such as asymmetry of joint pain or involvement of joint sites evocating CPPD (4). In those patients, which could be named PMR/CPPD, it could be proposed a short-term steroids therapy.

Another point was discussed by Milchert *et al*. They re-evaluated retrospectively an US database of confirmed PMR and analysed US CPPD of acromioclavicular (AC) joint. They observed only one CPPD patient of the 198 available pictures. One of the explanations to this contradictory result could be the possibly distinct population. We do not have the details of the population but some patients seemed to be managed in outpatient clinic. Our patients were all referred to a tertiary care university that could represent a bias of recruitment. Moreover, US analysis of Milchert *et al*. was retrospective and used static pictures that could limit the ability to observe CPPD. Our study was designed to screen systematically CPPD by US leading to a better determination of CPPD. Despite not performing AC joint evaluation, Falsetti *et al*. observed that 78% of CPPD patients had menisci calcifications (5). According to the OMERACT, AC joints are, with knees and wrists, the most relevant joint site for US screening of CPPD. The interobserver and intraobserver kappa values of AC joint for CPPD are moderate to excellent according to the OMERACT (6). Thus, adding US assessment of AC joints might be useful in clinical practice.

In conclusion, we fully agree that CPPD and PMR can coexist. Adding AC joint analysis to clinical and US evaluation appear to be relevant and finding CPPD might suggest to the physician that short-term steroids therapy could represent an alternative to usual treatment of PMR.

S. Ottaviani, *MD*

Université Paris Diderot, Sorbonne Paris Cité, UFR de Médecine, Paris, and AP-HP, Service de Rhumatologie, Hôpital Bichat, Paris, France.

Please address correspondence to: Sébastien Ottaviani, Service de Rhumatologie, Hôpital Bichat, 46 rue Henri Huchard, 75018 Paris, France. E-mail: sebastien.ottaviani@aphp.fr

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