Comment on:
Reclassification into very-high cardiovascular risk in psoriatic arthritis as well as axial spondyloarthritis

Sirs,
We read with great interest the recent paper by Rueda-Gotor et al. (1) in which they studied whether patients with axial spondyloarthritis (axSpA) were more commonly reclassified into the very high cardiovascular risk category than controls after performing carotid ultrasound (US). Similarly to spondyloarthritis, a concern exists that the CV risk in psoriatic arthritis (PsA) may be underestimated (2,3). That is why we aimed to evaluate the presence of subclinical CV disease in patients with PsA. We performed a transversal multicentre study collecting demographical data, the presence of classical CV risk factors and events, and different PsA characteristics. The probability of fatal CV events was calculated by SCORE adapted for Spain. Then all patients underwent bilateral standardised common carotid US. Plaques were defined according to the Mannheim consensus (4), and the patients’ risk was reassessed upon US. Descriptive, univariate and multivariate analyses (ANOVA) were performed to study the presence of plaque and its association with the dependent variables. 399 patients were included (men 51.8%; age 54.8 - SD 12.5), with an average duration of the disease 10.4 years. At least one classic CV risk factor was present in 35% of the patients, and 30.4% had 2 or more. Clinical stratification by SCORE showed 13.3% patients with low risk, 58.9% with intermediate, 24.3% with high risk and 3.6% patients with very high risk. In Rueda-Gotor et al., carotid plaques were higher in the patients (36% vs. 25% controls), and 34% were reclassified into high-risk. In our patients with PsA, similarly to previous results of our group (5), plaques were present in 30.7%, and as much as 30.1% were reclassified into the very high-risk group (Fig. 1).

In the association study, univariate analyses found that plaques were significantly associated with age, diabetes, hypertension, hyperuricaemia, and dyslipidaemia. In the multivariate analyses, the presence of plaque was only associated with age >55yo (OR 2.84; CI 1.54–5.55) and the number of classical CV risk factors (OR 1.49; CI 1.13–1.97). No associations with any PsA characteristics or treatments were found. Similarly in Rueda-Gotor et al., the association of plaques with axSpA characteristics was lost after adjusting for cardiovascular risk. The risk for CV disease and death in PsA is increased comparing to the general population (6), although very few studies have evaluated the presence of subclinical CV disease in PsA. In a case-control study the prevalence of carotid plaques among 59 patients with PsA was 15% (7). In other case-control studies, patients with PsA exhibited higher carotid plaque index (8). Other case-control study found a prevalence of 11.7% of carotid plaques among 77 patients with PsA, significantly higher than the controls and only slightly higher than psoriasis (9). In these studies the presence of plaques was associated to the age and to cholesterol levels, but all failed to find an association between plaques and the activity or duration of the disease. This difficulty may be related to the fact that we lack a standardised activity index for PsA. The main difference with those previous studies is that we included further more patients and the prevalence of carotid plaque was considerably high. Although this is a retrospective study, and we cannot define the predictive value of carotid US in the prevention of CV, our results show considerable subclinical CV disease in patients with PsA, in which classical CV risk factors are inherently frequent. The clinical risk stratification charts potentially underestimate the real CV risk in PsA as it seems to do in axSpA, and patients with clinical intermediate or high risk, who additionally have at least one classical CV risk factor, would benefit the most from a carotid US scan.

M.P. MARTÍNEZ-VIDAL, MD, PhD
J.A. LORENZO-MARTÍN, MD
M. ANDRÉS, MD, PhD
V. JOVANJ, MD, PhD
C. SANTOS-RAMÍREZ, MD
C. ROMERA-LOPEZ, MD, PhD
C. FERNÁNDEZ-CARBALLIDO, MD, PhD
R. QUEIRO-SILVA, MD, PhD
Hospital Universitario San Juan, Alicante;
Hospital Central Universitario de Asturias, Oviedo;
Hospital General Universitario de Alicante, Alicante;
Hospital Virgen de los Lirios, Alcoy;
Hospital General Universitario de Elda, Spain;
Please address correspondence to:
María Paz Martínez-Vidal,
Hospital Universitario de San Juan de Alicante,
Ctra. Naln. 32, s/n,
03550 Sant Joan d’Alacant, Alicante, Spain.
E-mail: mpamvidal@yahoo.es
ORCID: 0000-0002-4837-5237
Competing interests: none declared.
© Copyright CLINICAL AND EXPERIMENTAL RHEUMATOLOGY 2020.

Clinical and experimental Rheumatology 22

References

Fig. 1. Cardiovascu-lar risk distribution before and after carotid ultrasonound. Final classification showed a decrease in all the groups at the expense of an increase in the patients classified as very high risk.