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Can achieving Minimal Disease Activity (MDA) prevent progression of subclinical atherosclerosis and arterial stiffness? A two-year prospective cohort study in Psoriatic Arthritis

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Introduction

PsA patients have higher cardiovascular disease (CVD) risk due to underlying inflammation. While achieving MDA was associated with articular benefits, its effect on CVD risk remained uncertain.

Objectives

This study aimed to investigate the effect of achieving sustained MDA on subclinical atherosclerosis

Methodology

101 consecutive PsA patients without overt CVD were recruited for this prospective cohort study. All patients received protocolized treatment aiming at MDA for a period of 2 years. High-resolution ultrasound (for subclinical atherosclerosis) and arterial stiffness were assessed annually. The primary objective was to investigate the effect of achieving MDA (MDA group) at 12 months on the progression of subclinical atherosclerosis (carotid intima-media thickness [IMT] & plaque) over a period of 24 months. Secondary objectives were to compare 1) the changes in arterial stiffness (branchial-ankle pulse wave velocity [baPWV] and augmentation index [AIX]) over 24 months between the MDA and non-MDA groups; 2) changes in subclinical atherosclerosis and arterial stiffness markers in patients who achieved sustained MDA (sMDA: defined as achieving MDA from month 12 to 24) compared to those who did not (non-sMDA group). Carotid plaque progression was defined as an increase in number or region harboring plaque compared with baseline.

Result

90 PsA patients [male:52(57.8%); age: 5011] who completed 24-months of follow-up were included in this analysis. At 24 months, 62 (69%) were on csDMARDs, 20 (22%) were on anti-TNF-? and 8 (9%) were on Secukinumab. A significantly increased proportion of patient achieved MDA (baseline: 16.7% ; 12 months: 63.3% ; 24 months: 68.9%) after intensive treatment. Subclinical atherosclerosis outcomes were similar between the MDA and non-MDA groups. 41 (45.6%) patients achieved sMDA. At baseline, a higher prevalence of subjects in the non-sMDA group were smokers, were treated with NSAIDS and csDMARDs; fewer subjects were on bDMARDs, and they were having higher disease activity (higher enthesitis scores, VAS pain, patient and physician global scores, DAPSA and HAQ) compared with the sMDA group. 34/90(37.8%) patient had plaque progression. The prevalence of plaque progression was numerically higher in the non-sMDA group [22(44.9%) vs 12(29.3%), p=0.128]. Using multivariate analysis, achievement of sMDA had protective effect on plaque progression [OR=0.273, 95%CI:0.088 to 0.846, p=0.024] after adjustment of baseline difference (Table 1). Achievement of sMDA was also related to less progression of total plaque area (TPA), mean and maximum IMT, baPWV and AIX. Overall, there was no association between the change in vascular parameters and clinical or laboratory parameters of disease activity.

Conclusion:

Effective suppression of inflammation by achieving sustained MDA may prevent progression of subclinical atherosclerosis and arterial stiffness in PsA patients.

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