168 BODY MASS INDEX DOES NOT INFLUENCE THE EFFICACY OF ABATACEPT IN PATIENTS WITH PSORIATIC ARTHRITIS: RESULTS FROM THE ASTRAEA TRIAL

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Background: Obesity is a risk factor for the development and severity of psoriatic arthritis (PsA). Patients with increased BMI are less likely to achieve sustained minimal disease activity (MDA) compared with those with normal BMI, independent of treatment. Moreover, obese patients with PsA respond less favourably to TNFα inhibitors than those with normal BMI. In the Phase III ASTRAEA trial NCT01860976, abatacept significantly improved disease activity and was well tolerated; the primary endpoint (ACR20 at 24 weeks [W]) was met. Here, we evaluated posteriori the relationship between BMI and abatacept response in ASTRAEA.

Methods: Patients were randomised (1:1) to weekly SC abatacept 125mg or placebo for 24 weeks. Patients without ≥20% improvement in joint counts at W16 were switched to open-label abatacept (early escape). Early escape patients or those with missing data were imputed as non-responders. ACR20/50/70 responses and percentages of patients with DAS28 (CRP) <3.2 or <2.6, MDA, HAQ-DI response (change from baseline [CFB] ≥0.35) and radiographic non-progression (PsA-modified total Sharp/van der Heijde score, CFb:0.3) at W24 were compared for abatacept vs placebo between three BMI subgroups (underweight/normal: <25kg/m²; overweight: 25-30kg/m²; obese: >30kg/m²) using univariate and multivariate analyses. BMI-29kg/m² subgroup was the reference and key potential confounding factors for treatment efficacy were included in the multivariate model. Odds ratios (ORs), 95% CIs and p values were calculated for each BMI subgroup comparison.

Results: Overall, 212 abatacept- and 210 placebo-treated patients had available baseline BMI status. For abatacept vs placebo, 31 (14.6%) vs 39 (18.6%) were underweight/normal, 77 (36.3%) vs 57 (27.1%) were overweight and 104 (49.1%) vs 114 (54.3%) were obese. In the abatacept and placebo groups, neither overweight nor obese patients had a significantly lower ACR20 response compared with underweight normal patients in the univariate model. These results were confirmed in multivariate models in overweight and obese patients vs underweight/normal patients: abatacept (OR (95% CI): 1.215 (0.437, 3.378); p = 0.7087 and 0.446 (0.162, 1.228); p = 0.1181; placebo group: OR (95% CI) 0.554 (0.189, 1.621); p = 0.2811 and 0.460 (0.166, 1.271); p = 0.1343. Similar results were observed for the other outcomes: MDA response: abatacept: p = 0.5392 and p = 0.7896; placebo: p = 0.3292 and p = 0.8996; DAS28 (CRP): 2.6: abatacept: p = 0.7189 and p = 0.5486; placebo: p = 0.0876 and p = 0.0281; HAQ-DI response: abatacept: p = 0.729 and p = 0.5957; placebo: p = 0.8749 and p = 0.6993; radiographic non-progression: abatacept: p = 0.4983 and p = 0.9601; placebo: p = 0.907 and p = 0.8509.

Conclusion: As in RA, BMI does not appear to affect the efficacy of abatacept in PsA across objective and patient-reported outcome measures. As one in three patients with PsA are overweight/obese, these data strongly suggest an advantage of abatacept in this disease. First presented at DGRh 6-9 September, 2017 (abstract Sp.06; doi: 10.3205/17S04620).

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