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AB0826

PROBABILITY OF SURVIVAL OF USTEKINUMAB IN PSORIATIC ARTHRITIS: A REAL CLINICAL PRACTICE COHORT COMPOSED OF 64 PATIENTS

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Background: Psoriatic arthritis (PsA) is an inflammatory disorder of unknown etiology. Several domains are affected as peripheral or axial joints, enthesitis, dactylitis, nails as well as skin. Diverse cytokines have been described in the pathology of PsA as TNF α , IL-17 and IL-23. Ustekinumab (UST) is a fully human IgG1 κ monoclonal antibody to interleukin 12/23. Its efficacy and safety have been tested in several clinical trials and registries. Nevertheless data from real word evidence studies is needed to understand the effectiveness, safety and behavior of UST in a different population of patients from randomized controlled trials

Objectives: Analyze the persistence of UST 45 and 90mg along 52 weeks of treatment

Methods: Drug survival, effectiveness and security of UST were studied in a population of 64 PsA patients treated in the period between August 2014 to October 2019. Drug survival was defined as the time from initiation to discontinuation (stop/switch) of bDMARDs. For the determination of drug survival, Kaplan-Meier survival curves and Cox-regression analyses were used. Effectiveness was described as a reduction in the use of corticosteroids and in the levels of CRP along the study. All adverse events were recorded during the study

Results: 64 patients were included with a mean follow-up of 57,2 weeks. At baseline the mean age was 47,8 years (8,9). 54,7% of patients were women and 45,3% were male. 31,3% were obese. Mean disease duration was 7,9 (5,0) years. 45,3% presented peripheric arthritis; 32,8% axial involvement; 31,3% enthesitis; 80% psoriasis. Patients were 45% bDMARDs-naïve; had a previous bDMARDs in 20,3% and $\geq$ than two bDMARDs in 34,4%. 30% of the patients had co-therapy with methotrexate and 29,7% of patients received corticosteroid therapy. Mean CRP was 7,9 (12,7) mg/L. The global probability of survival for UST was 96%, 83,9% and 60% at week 12, 24 and 52 respectively. High UST dosage was associated with favorable drug survival (at 52W: UST 45 mg=40,1%; UST 90 mg=75,8%; UST 45 to 90 mg*=88,9%) ($p=0,008$). The bDMARDs-naïve population also correlated with favorable UST survival (at 52W: bDMARDs-naïve=66,1% vs bDMARDs-experienced=56,7%), however no statistical significance was found ($p=0,196$). No difference in survival was observed among patients with or without axial involvement (W52: axial=58,2% vs non-axial=61,6; $p=0,869$). UST produced a reduction in the use of corticosteroids (30% vs 16%) and CRP levels (8,7 vs 7,7). Differences were greater in patients treated more than 28 weeks (maximum efficiency described for UST) (corticosteroids: 26% vs 16%; CRP levels: 8,5 vs 4,4). 4,9% of the patients suffered an AE. Most of them were non-serious AE: infections (3,3%) or headache (1,6%). The main cause of treatment discontinuation was lack of efficacy (30%), followed by primary failure (9,4%) and just a 3% due to AE

Conclusion: The persistence of UST was dose-dependent and greater for the UST 90mg dosage and for the population of bDMARD-naïve patients. Drug survival of UST in the population of patients with axial involvement seems similar to the population of patients without axial affection which provide evidence of the efficacy of the IL23 inhibition in the axial domain of PsA. UST decreased the use of corticosteroids and CRP levels along treatment. The security profile of UST was to the drug. Only few non-serious AE reported during this study

*UST 45 to 90mg, patients who change from UST 45 to UST 90mg dosage

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AB0827

IMPACT OF BASELINE BODY MASS INDEX ON THE EFFICACY AND SAFETY OF TOFACITINIB IN PATIENTS WITH PSORIATIC ARTHRITIS

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Background: Obesity is highly prevalent in PsA (~45%)¹ and is associated with a reduced response to TNF inhibitors.² Tofacitinib is an oral Janus kinase inhibitor for the treatment of PsA.

Objectives: This post hoc analysis assessed tofacitinib efficacy and safety in patients (pts) with PsA by baseline (BL) body mass index (BMI) category.

Methods: Data were pooled from two placebo (PBO)-controlled, double-blind, Phase 3 studies in pts with active PsA and an inadequate response to ≥ 1 conventional synthetic DMARD (OPAL Broaden [12 months; NCT01877668]) or to ≥ 1 TNF inhibitor (OPAL Beyond [6 months; NCT01882439]).^{3,4} This analysis included pts randomised to tofacitinib 5 mg twice daily (BID), tofacitinib 10 mg BID or PBO, stratified by BL BMI: <25 kg/m², ≥ 25 – <30 kg/m², ≥ 30 – <35 kg/m², or ≥ 35 kg/m². Efficacy and safety were reported to Month (M)3. M3 efficacy outcomes included ACR20/50/70 and HAQ-DI responses, dactylitis and enthesitis resolution rates and changes from BL in HAQ-DI, Short Form-36 Version 2 (SF-36v2) Physical (PCS) and Mental Component Summary (MCS) scores, and Functional Assessment of Chronic Illness Therapy-Fatigue (FACIT-F) scores. Safety outcomes included adverse events (AEs), such as cardiovascular (CV) events and changes in lipid levels and liver function tests (LFTs).

Results: This analysis included 710 pts; 43.8% were obese (BMI ≥ 30). At BL, 161 (22.7%) pts had a BMI <25 , 238 (33.5%) had a BMI ≥ 25 – <30 , 186 (26.2%) had a BMI ≥ 30 – <35 and 125 (17.6%) had a BMI ≥ 35 . Most pts were white (92.5–96.8%), middle-aged (mean: 44.5–51.2 yrs) and female (49.5–65.6%). Greater proportions of obese pts were from Russia/Eastern Europe (35.0%) and USA/Canada (31.8%), vs the rest of world. At BL, higher BMI correlated with an increased prevalence of metabolic syndrome (4.3% in BMI <25 to 76.0% in BMI ≥ 35) and CRP levels >2.87 mg/L (49.1% in BMI <25 to 84.0% in BMI ≥ 35). Higher proportions of pts (42.5–47.9%) in BL BMI categories <35 reported no prior biologic DMARD use, vs pts with a BL BMI ≥ 35 (33.6%). At M3, efficacy improvements were greater in tofacitinib-treated pts vs PBO-treated pts (Figure 1). In pts with a BL BMI ≥ 35 , a trend towards fewer pts responding was observed (Figure 1) and mean changes from baseline in SF-36v2 PCS and MCS and FACIT-F generally appeared lower (Figure 2) vs pts in lower BL BMI categories. Up to M3, the proportions of pts with AEs, and percentage change from BL in lipid levels and LFTs, were generally similar across all BL BMI categories. Three CV events were reported: non-fatal cerebrovascular accident, transient ischemic attack (both tofacitinib 5 mg BID, BMI ≥ 30 – <35) and coronary artery revascularisation (PBO; BMI ≥ 35). Limitations include the 3-month observation time, particularly for safety findings, thus longer observation times are warranted.

Conclusion: Regardless of BL BMI, tofacitinib demonstrated greater efficacy than PBO at M3 in pts with PsA. Similar to other advanced therapies,² reduced efficacy was generally observed in tofacitinib and PBO pts with a BL BMI ≥ 35 . Tofacitinib safety appeared consistent across all BL BMI categories.

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