may pioneer further studies investigating the usage of adipoins such as vaspin and lipocalin2 levels as biomarkers in the diagnose and disease course of PsA.

Disclosure of Interest: None declared.


AB0902

EFFICACY OF TOFACITINIB BY BACKGROUND METHOTREXATE DOSE IN PATIENTS WITH PSORIATIC ARTHRITIS: A POST-HOC ANALYSIS OF POOLED DATA FROM 2 PHASE 3 TRIALS

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Background: Tofacitinib is an oral Janus kinase inhibitor for the treatment of psoriatic arthritis (PsA). The efficacy of tofacitinib has been evaluated in 2 Phase 3 studies in patients (pts) with PsA. Objectives: To describe the efficacy of tofacitinib by background methotrexate (MTX) dose in pts with PsA.

Methods: This post-hoc analysis utilised efficacy data pooled from 2 Phase 3, randomised, double-blind, placebo-controlled studies (OPAL Broaden: NCT01877668 and OPAL Beyond: NCT01882439) in pts with a diagnosis (≥6 months) of active PsA (≥3 swollen and ≥3 tender joints). Pts in OPAL Broaden were tumour necrosis factor inhibitor (TNFi)-naive and had an inadequate response to csDMARDs (≥1 conventional synthetic disease-modifying anti-rheumatic drug [csDMARD]). Pts in OPAL Beyond had an IR to ≥1 TNFi. Pts were randomised to tofacitinib 5 or 10 mg twice daily (BID), placebo or adalimumab 40 mg subcutaneous every 2 weeks (OPAL Broaden; adalimumab data not shown). All pts received a stable dose of 1 csDMARD (eg MTX, leflunomide or sulfasalazine) as background therapy. The maximum dose of MTX allowed per protocol was 20 mg/week. Efficacy outcomes for tofacitinib at Month 3 were broadly similar between background MTX dose groups (table 1). Baseline demographics and disease characteristics were generally similar across all groups. No statistical testing was performed.

Results: In total, data from 556 pts who received tofacitinib plus MTX only or placebo were included. Most pts were treated with background MTX dose at weeks 15 vs 15 mg/week (n=371, 66.7%; mean [SD] dose, 12.6 [3.1] mg/week) or >15 mg/week (n=185, 33.3%; mean [SD] dose, 19.8 [0.8] mg/week). Baseline demographics and disease characteristics were generally similar across subgroups. ACR20/50/70 response rates were >20/50/70% improvement from baseline, respectively. Health Assessment Questionnaire-Disability Index (HAQ-DI) response rates (reduction from baseline ≥0.35 points) and mean change from baseline in HAQ-DI score. Analyses were based on the full analysis set for pts receiving MTX on Day 1; pts with missing data were considered as having a non-response for binary endpoints. No statistical testing was performed.

Table 1: Baseline demographics and disease characteristics at baseline, and efficacy outcomes at Month 3 by MTX dose

Disclosure of Interest: None declared.

DOI: 10.1136/annrheumdis-2018-eular.1278

AB0903

EFFICACY OF TILDRAKIZUMAB IN ETANERCEPT PARTIAL OR NONRESPONDERS


Background: Etanercept (ETN) is an anti-tumour necrosis factor (TNF) medication that was among the first biologics approved for psoriasis. Additional medications have been developed or are in development for psoriasis, and patients who do not adequately respond to ETN may benefit from these more recent biologics.

Objectives: Here we report the efficacy of tildrakizumab (TIL), a humanised anti-IL-23p19 monoclonal antibody, as evaluated in patients with moderate to severe chronic plaque psoriasis who were partial (Psoriasis Area and Severity Index [PASI]: 50–75) or nonresponders (PASI <50) to ETN and subsequently randomized to TIL in the phase 3 reSURFACE 2 trial (NCT01729754).

Methods: Patients with psoriasis (≥10 body surface area, Physician’s Global Assessment [PGA], and PASI ≥12) participated in reSURFACE 2, a 3-part, 52 week, randomised controlled trial. In Part 1 (Weeks 0–12), patients were randomised to subcutaneous TIL 200 mg, TIL 100 mg, or placebo (PBO) administered at Weeks 0 and 4, or ETN 50 mg administered twice weekly. In Part 2 (Weeks 12–28), TIL and ETN patients remained on the same treatment (TIL administered at Week 16; ETN once weekly), whereas PBO patients were rerandomized to TIL 200 mg, TIL 100 mg, or placebo. In Part 3 (Weeks 28–52), ETN responders (PASI ≥75) were discontinued, and partial and nonresponders were switched to TIL 200 mg (administered at Weeks 32, 36, and 48). For this post hoc analysis, the proportion of patients with PASI 75% improvement from baseline (PASI 75%) was compared in PASI responders (score of 0 [clear] or [minimal] with at least a 2-grade score reduction from baseline) were determined at Week 52. Primary results from the trial have been previously reported.1

Results: In total, 1090 patients were randomised. Of the 313 patients randomised to ETN, by Week 28 there were 83 partial responders and 39 nonresponders. At Week 52 (after 20 weeks of TIL treatment) for ETN partial responders, 75%±5%, 54%±5%, 15%±4%, and 58%±5% had achieved PASI 75, 90, and 100, and PGA response of 0/1, respectively, with TIL 200 mg treatment. At Week 52 for ETN nonresponders, 54%±6%, 31%±5%, 10%±3%, and 56%±5% had achieved PASI 75, 90, 100, and PGA response of 0/1, respectively, with TIL 200 mg treatment. Adverse events were similar in patients switched from ETN to TIL at Week 28, compared with the patients who were maintained on TIL through Week 52.