PSORIASIS

THE EFFECT OF GUSELKUMAB ON PSORIASIS IN PATIENTS WITH ACTIVE PSORIATIC ARTHRITIS: RESULTS FROM A PHASE 2 STUDY

Ab Gottlieb [1] - W-h Boehncke [2] - P Helliwell [3] - A Deodhar [4] - Xl Xu [5] - S Xu [6] - Y Wang [7] - Y Zhuang [8] - Ec Hsia [9]

New York Medical College, Metropolitan Hospital, Dermatology, New York, United States [1] - University Of Geneva, Immunology, Geneva, Switzerland [2] - Leeds Institute Of Rheumatic And Musculoskeletal Medicine, University Of Leeds, Rheumatology, Leeds, United Kingdom [3] - Oregon Health & Science U, Rheumatology, Portland, United States [4] - Janssen Research & Development, Llc, Immunology, Spring House, United States [5] - Janssen Research & Development, Llc, Immunology, Raritan, United States [6] - Janssen Research & Development, Llc, Clinical Biostats, Spring House, United States [7] - Janssen Research & Development, Llc, Clin Pharm, Spring House, United States [8] - Janssen Research & Development, Llc/university Of Pennsylvania, Immunology, Spring House/philadelphia, United States [9]

Introduction/Objective: The effect of guselkumab on psoriasis was evaluated in a phase 2 study in patients with active psoriatic arthritis (PsA).

Materials/Methods: Patients with ≥3 tender and ≥3 swollen joints, C-reactive protein ≥3 mg/L, and ≥3% plaque-psoriasis body-surface area (BSA), despite treatment, were randomized 2:1 to receive subcutaneous guselkumab 100mg (n=100) or placebo (n=49) at wk0, 4, then q8wks through wk44. At wk16, patients with <5% improvement in swollen and tender joint counts were eligible for early escape (EE) to open-label ustekinumab. Remaining placebo patients crossed over to receive guselkumab 100mg (PBO→GUS) at wks24, 28, 36, and 44. Psoriatic Area and Severity Index (PASI) responses through wk24 were analyzed using last-observation-carried-forward for missing data and data post EE. After wk24, observed data were analyzed

Results: At baseline, BSA and PASI scores were consistent with moderate-to-severe psoriasis (mean (SD): 16.0 [14.70], 11.32 [9.78], respectively) in the overall population, and 80.5%, 43.0%, and 41.6% of patients had scalp, nail, and hand/foot psoriasis, respectively. Higher baseline BSA and PASI scores were associated with greater ACR 20 response at wk24 in GUS patients (BSA≤10%, 55.8%; BSA>10, 59.6% and PASI<12, 50.0%; PASI≥12, 71.1%).
At wk24, significantly more GUS vs PBO patients achieved PASI 75 (78.6% vs 12.5%, p<0.001), PASI 90 (66.3% vs 6.3%), and PASI 100 (39.8% vs 6.3%, p<0.001).

Improvement in PASI responses was first observed at wk4 (PASI 75/90/100: 16.3%/9.2%/3.1% for GUS vs 4.2%/0/0 for PBO). PASI 75/90 response at wk16 correlated with trough GUS serum concentration at wk12. PASI 75/90/100 responses were maintained through wk56 in GUS patients (85.4%/78.0%/57.3%). After wk24, rapid improvements in PASI 75/90/100 responses were also observed in PBO→GUS patients (81.5%/74.1%/55.6% at wk56).

Conclusions: GUS demonstrated robust efficacy for treatment of cutaneous manifestations of psoriasis in PsA pts with ≥3% BSA, and responses were maintained through wk56.