SERUM IL-12/23 AND IL-17 LEVELS IN PATIENTS WITH SPONDYLOARTHRITIS WERE NOT INFLUENCED BY TNF-BLOCKADE

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Background: Spondyloarthritis (SpA) refers to a heterogeneous group of disorders with clinical features that can include axial and peripheral arthritis, inflammatory bowel disease, uveitis, and psoriasis. Several cytokines including interleukin (IL)-12/23, IL-17 and tumor necrosis factor (TNF) are involved in pathogenesis of SpA. It is assumed that TNF is the upstream cytokine in the cytokine cascade (Schett, et al., 2013).

Objectives: To investigate whether TNF inhibitors decrease serum IL-12/23 and IL-17 levels in patients with SpA.

Methods: Serum were obtained from 23 SpA patients (AS, 10 patients; PsA, 13 patients) enrolled in this study, baseline, 24 and 48 weeks of TNF inhibitor treatment. Serum IL-12/23 and IL-17 levels were measured using LEGEND MAX Human IL-12/23 (p40) ELISA Kit (BioLegend) and Human IL-17A ELISA kit (Invitrogen), respectively. IL-6 levels, the other downstream cytokine, was measured using Lumipulse G600II (FUJIREBIO) as a control.

Results: Any significant reduction in IL-12/23 levels (143.9±143.6 pg/mL at baseline, 56.3±56.7 pg/mL at 24 weeks and 43.9±43.6 pg/mL at 48 weeks) as well as that in IL-17 levels (13.6±5.9 pg/mL at baseline, 12.3±4.1 pg/mL at 24 weeks and 11.6±3.2 pg/mL at 48 weeks) were not observed in 23 SpA patients. On the other hand, serum IL-6 levels were significantly decreased after treatment (4.0±2.4 pg/mL at baseline, 1.8±1.4 pg/mL, p=0.002, at 24 weeks; 1.6±1.9 pg/mL, p=0.002 at 48 weeks. Pain-VAS was significantly reduced at 24 and 48 weeks compared with that at baseline. No significant differences in serum levels of IL-17 level were significantly decreased after treatment (4.0±1.3 pg/mL at baseline, 1.8±1.3 pg/mL, p=0.002, at 24 weeks; 1.6±1.7 pg/mL, p=0.002 at 48 weeks).

Conclusion: TNF inhibitors did not alter serum IL-12/23 and IL-17 levels but reduced IL-6 levels in patients with SpA. These results imply that IL-12/23 and IL-17 expression might be regulated by alternative pathways.

References:

PATIENT PERCEPTIONS OF FIBROMYALGIA SYMPTOMS AND THE OVERLAP WITH AXIAL SPONDYLOARTHRITIS

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Background: In clinical practice, it is often challenging to distinguish fibromyalgia syndrome (FMS) from axial spondyloarthritis (axSpA), which includes ankylosing spondylitis and non-radiographic axSpA.1,2 Early stages of axSpA may present with an onset similar to FMS,3 and likewise patients with FMS may have symptoms that are similar to axSpA. Differentiating between axSpA and FMS can also be challenging for patients and cause confusion about their diagnosis.

Objectives: To examine the prevalence of axSpA symptoms among patients with FMS and differences in the pathway to diagnosis among patients with and without concomitant axSpA.

Methods: Adult US patients with FMS without concomitant rheumatoid arthritis or psoriatic arthritis in the ArthritisPower registry received email invitations to participate. Participants (pts) were asked whether they had a diagnosis of axSpA or ankylosing spondylitis and completed patient-reported outcome measures including Patient Reported Outcomes Measurement Information System (PROMIS) measures for Pain Interference, Sleep Disturbance and Fatigue, and the Bath Ankylosing Spondylitis Disease Activity Index (BASDAI). Pts then responded to a 57-item customized survey developed by the researchers in collaboration with patient partners. Results are descriptively reported.

Results: As of January 2020, 231 pts completed the survey; 97% female, 89% White, mean (SD) age of 52 (11). Mean (SD) Pain Interference score was 68 (5); Sleep Disturbance 63 (8); Fatigue 66 (7); and BASDAI 46 (9). Of the pts, 40 (17%) reported concomitant axSpA, 64% osteoarthritis, 6% gout, 5% Crohn’s or ulcerative colitis, and 4% lupus. Half of all pts perceived their FMS to be ‘rarely’ or ‘never’ well managed and 80% felt that they had an undiagnosed condition in addition to their FMS and their other current diagnoses. Three-fourths (75%) of pts reported being able to tell the difference between their FMS pain and pain they experience as a part of the concomitant disorder. Back pain lasting >3 months was reported by 95% of axSpA pts and 94% of non-axSpA pts and 12% reported all of the symptoms consistent with patient reported versions of the Assessment of SpondyloArthritis International Society (ASAS) criteria (back/buttock pain >3 months; age of symptom onset <45; sacroiliitis diagnosis; at least on spondyloarthritis feature) (Figure 1), and of these, 39% reported an axSpA diagnosis. More pts with axSpA received their FMS diagnosis by a rheumatologist (45%) than without (41%) (Figure 2), and of the pts without an axSpA diagnosis (n=191), only 6% had recalled their provider ever discussing the possibility of axSpA, including non-radiographic axSpA diagnosis. Half (53%) of pts with axSpA believe that their axSpA should have been diagnosed earlier; with 33% reporting that one reason for the delay was their doctors’ belief that FMS was the cause of any axSpA symptoms they experienced.

Conclusion: Patients with FMS often experience symptoms of axSpA and the two conditions can occur concomitantly. Additional research is needed to improve the triage, diagnosis, and education of patients with FMS and symptoms of axSpA.

References:

USEFULNESS OF THE TRABECULAR BONE SCORE AS A PREDICTOR OF VERTEBRAL FRACTURE IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS

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Disclosure of Interests: Kelly Gavigan: None declared, W. Benjamin Nowell: None declared, Regan Reynolds: None declared, Laura Stratford: None declared, Jeffrey Curtis Grant/research support from: AbbVie, Amgen, Bristol-Myers Squibb, Corrona, Janssen, Lilly, Myriad, Pfizer, Regeneron, Roche, UCB, Consultant of: AbbVie, Amgen, Bristol-Myers Squibb, Corrona, Janssen, Lilly, Myriad, Pfizer, Regeneron, Roche, UCB, Alexis Ogdie Grant/research support from: Pfizer, Novartis, Consultant of: Abbvie, Amgen, BMS, Celgene, Corrona, Janssen, Lilly, Pfizer, Novartis