Th1/Th17/Th22 immune response and their association with joint pain, imagenological bone loss, RANKL expression and osteoclast activity in temporomandibular joint osteoarthritis: A preliminary report

Monasterio G.

Castillo F.

Rojas L.

Cafferata E.A.

Alvarez C.

Carvajal P.

Núñez C.

Flores G.

Díaz W.

Vernal R.

It is well accepted that the presence of cytokines belonging to the Th1/Th17/Th22 axis of immuno-inflammatory response in the joint environment, such as IL-1?, IL-17 and IL-22, respectively, are associated with pathogenesis of several synovial joint degenerative disorders. During temporomandibular joint osteoarthritis (TMJ-OA), IL-1? and IL-17 have been implicated in the inflammation and resorption of sub-chondral bone; however, the role of Th22 response in the TMJ-OA pathophysiology has not been established. This study aimed to compare the expression of Th1/Th17/Th22-type cytokines, chemokines and chemokine receptors in synovial fluid samples obtained from TMJ-OA or disk displacement with reduction (DDWR) patients. In addition, it aimed to associate these levels with joint pain, imagenological signs of bone degeneration, RANKL production, osteoclastogenesis and osteoclast-induced bone resorption. Higher levels of IL-1?, IL-17 and IL-22 were expressed in TMJ-OA compared with DDWR subjects, and these increased levels significantly correlated with RANKL expression, joint pain and articular bone degeneration. Higher levels of CCR5, CCR6 and CCR7, as well as their respective ligands CCL5 and CCL20, responsible

for recruitment of IL-1?, IL-17 and IL-22-producing cells, were over-expressed in TMJ-OA compared with DDWR subjects. Osteoclastogenesis and osteoclast-induced bone resorption were significantly greater in presence of synovial fluid from TMJ-OA compared with DDWR subjects. These data demonstrate that cytokines, CCLs and CCRs associated with the Th1/Th17/Th22 axis of immuno-inflammatory response are involved in TMJ-OA pathogenesis. These findings suggest that IL-22 is involved in the RANKL expression in TMJ-OA, which in turn induces differentiation of osteoclasts and subsequent resorption of sub-chondral bone.

bone resorption

chemokines

cytokines

interleukin-22

RANKL

temporomandibular osteoarthritis

osteoclast differentiation factor

adult

aged

cell culture

cell differentiation

cytology

female

helper cell

human

immunology

male

metabolism

middle aged

osteoarthritis

osteoclast

osteolysis

pathology

pathophysiology

synovial fluid

T lymphocyte subpopulation

temporomandibular joint

temporomandibular joint disorder

young adult

Adult

Aged

Bone Resorption

Cell Differentiation

Cells, Cultured

Female

Humans

Male

Middle Aged

Osteoarthritis

Osteoclasts

RANK Ligand

Synovial Fluid

T-Lymphocyte Subsets

T-Lymphocytes, Helper-Inducer

Temporomandibular Joint

Temporomandibular Joint Disorders

Young Adult