Overexpression of MMPs, cytokines, and RANKL/OPG in temporomandibular joint osteoarthritis and their association with joint pain, mouth opening, and bone degeneration: A preliminary report

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Abstract

Objective This study aimed to determine the expression of distinct matrix metalloproteinases, cytokines, and bone resorptive factors in temporomandibular joint osteoarthritis (TMJ-OA) patients and their association with joint pain, mouth opening, and subchondral bone degeneration. Materials and methods Twelve patients affected with TMJ-OA (n = 5), disk displacement without reduction (DDWoR) (n = 3), or disk displacement with reduction (DDWR) (n = 4) were selected. Joint pain was quantified by using visual analog scale, mouth opening was quantified at the maximum pain-free aperture, and bone degeneration was quantified using joint imaging. Synovial fluid samples were collected and immediately processed for cell and synovial fluid recovering. From cells, the MMP-1, MMP-2, MMP-8, MMP-13, IL-6, IL-23, and TNF-alpha expression was quantified by qPCR. From synovial fluid, the RANKL and OPG levels were quantified by ELISA. Results Higher levels of MMP-1, MMP-8, MMP-13, IL-6, IL-23, TNF-alpha, and RANKL/OPG ratio were detected in TMJ-OA compared with DDWoR and DDWR patients (p < .05). Joint pain significantly correlated with TNF-alpha levels (r = .975, p = .029). Besides, imaging signs of bone degeneration significantly correlated with RANKL/OPG ratio (r = .949, p = .042). Conversely, mouth opening did not correlate with any of the analyzed mediators. Conclusion During TMJ-OA, a pathological response characterized by the overexpression of TNF-alpha and RANKL/OPG could be involved in joint pain and subchondral bone degeneration.

Palabras clave

Palabras clave de autor: cytokines; MMPs; OPG; RANKL; temporomandibular osteoarthritis

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