The serum MMP3 measurement was performed by ELISA assay.

Results: Serum MMP3 was significantly higher in RA patients vs. healthy controls (p < 0.0001). Inflammatory rheumatism (p < 0.0001) and CID (p < 0.05) and is particularly high in women with high Disease Activity Score 28 joints (DAS28) vs. Moderate DAS28 (p < 0.05). In addition, it is higher if patients have: positive CRP: p < 0.05; positive RF: p < 0.05; positive ACPA: p < 0.05 and Bone erosion: p < 0.01 compared to other patients. Finally, there is a positive correlation between MMP3 production and RF (p < 0.0001, Spearman r = 0.35) and ACPA (p < 0.01, Spearman r = 0.26) but not with DAS28 joining the results published by Mahmood et al. (2013) and Fadda et al. (2018). Furthermore, in RA patients, there is no significant difference in MMP3 levels according to whether the erythrocyte sedimentation rate (ESR) is accelerated or not, unlike CRP which represents the best marker of inflammation in this disease, according to the literature. Finally, the production of MMP3 is greater in case of erosion and is associated to bone and joint destruction.

Conclusion: MMP3 serum measurement is a particularly useful marker of inflammatory activity in RA and may have potential predictive value in the development of bone and joint destruction.

REFERENCES

Disclosure of Interests: None declared

AB0058

SERUM OXYTOCIN LEVELS IN PATIENTS WITH ANKYLOSING SPONDYLITIS AND NONRADIOGRAPHIC AXIAL SPONDYLARTHРИTHIS

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Background: Spondyloarthritis refers to a group of chronic inflammatory diseases with unknown etiologies that involve particularly sacroiliac joints and spine but may also affect the remaining joints and entheses and exhibit extra-articular involvement in some patients. Oxytocin is a peptide hormone released from hypothalamus and stored in pituitary gland. It has been known for a while that oxytocin has anti-inflammatory effects and can cause a reduction in TNF-alpha levels.

Objectives: The aim of this study was to investigate the serum levels of oxytocin and its potential association with disease activity, spinal mobility and some other laboratory parameters such as erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) in patients with ankylosing spondylitis (AS) and non-radiographic axial spondylarthritis (nrAxSpA).

Methods: Seventy-one patients with nrAxSpA and 38 patients with AS who presented to the Outpatient Clinic of Physical Medicine and Rehabilitation of Dicle University Hospital between March 2017- October 2017 and 67 healthy control subjects were included in this study. All the subjects underwent a thorough physical examination. Disease activity was assessed by Bath Ankylosing Spondylitis Disease Activity Index, and spinal mobility by Bath Ankylosing Spondylitis Metrologic Index. Laboratory examinations included complete blood count, ESR, CRP, and oxytocin tests.

Results: There was no significant difference in serum levels of oxytocin among the three groups (p = 0.973). However, serum levels of oxytocin correlated negatively with both ESR (r = -0.359, p = 0.027) and BASDAI (r = -0.448, p = 0.005) in patients with AS. On the other hand, serum levels of oxytocin had a negative correlation only with ESR in the patients with nrAxSpA (r = -0.321 p = 0.009).

Conclusion: ESR is one of the parameters associated with disease activity in patients with inflammatory rheumatoid diseases. Serum levels of oxytocin correlate negatively with both ESR and BASDAI scores in patients with AS and only with ESR in patients with nrAxSpA suggests that low levels of oxytocin may be associated with increased disease activity. This study lays the foundation for further studies that may aim to investigate how addition of oxytocin to the treatment regimen impact on the disease activity in patients with AS who exhibit particularly low levels of oxytocin during active disease period.

REFERENCES

Disclosure of Interests: None declared

AB0059

通过MAPK和NF-KB信号通路自细胞抑制药物抑制细胞介素-37炎方

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Background: Autophagy has been shown as an intrinsic cellular defense mechanism in innate and adaptive immune responses and is reported to regulate inflammation during infection [1]. Accumulating evidence indicates that autophagy suppresses inflammasome activation [2-5]. IL-37 has been identified as an anti-inflammatory factor that translocates into the nucleus and downregulates other pro-inflammatory cytokines [6]. Nevertheless, whether autophagy regulates the expression of IL-37 has not been elucidated.

Objectives: The purpose of this study was to investigate the role of autophagy in regulating IL-37, an anti-inflammatory cytokine highly expressed in tissues of patients with inflammatory and autoimmune diseases.

Methods: PMA-transformed and LPS-stimulated U937 macrophages were treated with three autophagy-modifying reagents—3MA, chloroquine, and rapamycin. The expression of three IL-1 family cytokines (IL-37, IL-18, and IL-1β) and two receptors (IL-1R8 and IL-18RA) was determined by quantitative PCR. Intra-cellular IL-37 expression was detected by flow-cytometry. Western blot analysis and immunofluorescence assay were used to determine the levels of P62/SQSTM1 and LC3, and autophagic U937 cells were isolated by sorting for quantifying IL-37 expression. We also used agonists and antagonists of the MAPK and NF-κB pathways to investigate the possible pathway involved. Peripheral blood mononuclear cells from one healthy donor were also used for autophagy modification and intracellular IL-37 quantitation.

Results: IL-37 was upregulated by rapamycin and chloroquine in U937 cells stimulated by LPS. IL-37 was preferentially expressed in autophagic cells accompanied with LC3 conversion. Higher IL-37 expression was found in “Cyto-ID” positive cells than in negative cells. Inductive IL-37 expression, but not constitutive IL-37 expression, could be abolished by inhibitors of the MAPK and NF-κB pathways, whereas it was augmented by MAPK agonists.

Conclusion: IL-37 levels were enhanced by rapamycin and chloroquine dependent on MAPK and NF-κB pathway activation, probably through LC3 accumulation. This study suggests a possible novel mechanism of chloroquine and rapamycin action in autoimmune inflammatory diseases.

REFERENCES

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