

CONSACRAT ANIVERSĂRII A 75-A DE LA FONDAREA USMF "NICOLAE TESTEMIȚANU"

Introduction

Osteoporosis (OP) is a common complication observed in rheumatoid arthritis (RA) patients. Two types of bone loss localized and generalized are documented in RA patients. It is considered that pathogenesis of RA plays an important role in the development of these bone changes.

Keywords: Rheumatoid arthritis, osteoporosis, bone pathology

Purpose

To identify and analyze the most relevant articles on the topic of bone pathology in rheumatoid arthritis published in Pubmed resource during 2000-2020

Synovial cytokines, especially MCS-F and RANKL, promote the differentiation of osteoclasts and their invasion in the periosteal area adjacent to the articular cartilage. TNF- α , IL-1, II-6 and IL-17 intensify differentiation and OK activation.

Conclusions: Bone pathology in RA is one of the key mechanisms in arthritis progression along with synovia pathology. Generalized OP in RA is associated with increased disability and mortality. Patients with RA should be screened for OP as early as possible and treated , including profilaxy.

Osteoporosis in Rheumatoid Arthritis

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Material and methods

Pubmed database with key words rheumatoid arthritis, osteoporosis, bone pathology



Localised osteoporosis

Both types have the same pathogenetic

Assessment of fracture risk in patients with RA Most paradoxical fractures occur in patients without OP, in those with osteopenia. Other factors such as sarcopenia, vitamin D deficiency must be considered in assessing the total risk of fracture. A good method for assessing fracture risk

can serve **FRAX**.



Positive Anti CCP patients, especially if at high levels, should be investigated and treated with bone protection agents. DMARD anti-rheumatic drugs, which lower the antiCCP titer, can have positive effects on systemic bone mass.



After exclusion of less relevant articles, 105 sources were analyzed

RANKL in synovia



Non active RA

Healthy contro

Crotti et al, Annals of Rheumatic Diseases 200

In vitro RANKL expression is stimulated by cytokines: TNF α , IL-1. In patients with RA, the RANKL level is increased both in the serum and in synovia