Results. In patients with RA four biomarkers are found to predict fracture sites, Tartrate-resistant acid phosphate 5b (TRACP-5b), undercarboxynated osteocalcin (Uc-OC) and bone specific alkaline phosphate (BAP) are able to realize both BMD and bone quality while homocysteine is able to realize only bone quality, In RA patients annual bone mineral density changes are 0.14 + 2.70 in lumbar spine, 0.46 + in proximal hip and 1.14 + 1.85 in forearm. Some studies show that in lumbar spine Homocysteine is the significant predictor for fractures, while in the proximal hip and forearm homocysteine does not have any significance. The most potent predictors for hip and forearm fractures are DAS28-ESR, blood pressure and Vitamin D levels other authors consider a better predictor to be ACPA and Methotrexate dosage use. Another hypothesis suggests that mycobacterium Avium Paratuberculosis (MAP) infection associated with TNF polymorphisms in patients with rheumatoid arthritis might cause secondary osteoporosis and it was found that the association between MAP infection in patients with rheumatoid arthritis and a risk for development of osteoporosis.

Conclusions. Osteoporosis is a common condition diagnosed in patients with RA. Secondary osteoporosis due to RA depends on the disease activity, ACPA level, MTX dosage. Some biochemical markers, as homocysteine, TRACP-5b, Uc-OC and bone specific alkaline phosphate can serve as predictors for osteoporotic fractures at different sites

Key words: osteoporosis, rheumatoid arthritis, biomarkers, fracture risk, hip, forearm, lumbar spine, bone mineral density

178. ASSOCIATION BETWEEN ESSENCIAL HYPERTENSION AND BONE MINERAL DENSITY

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Introduction. With society trending towards aging and unhealthy lifestyle changes the prevalence rate of essential hypertension (EH) and osteoporosis (OP) increases every year, to a point where they have become the two most common diseases in the world.

Aim of the study. To highlight the relationship between essential hypertension (EH) and bone mineral density (BMD).

Materials and methods. A systematic review on the published literature was conducted. 17 articles on the topic of association between EH and BMD were selected after searching PubMed, Medline, Medscape, and Google Scholar. The data were anaysed and statistically compared .

Results. The 17 articles used have a total of 39,491patients. Of these, 13,375 were patients with EH and 26,116 were patients without EH. The most relevant meta-analysis results showed that EH can reduce the BMD of the lumbar spine (95% CI: −0.08∼0.01, P=0.006), femoral neck (95% CI: −0.09∼-0.02, p = 0.001), ward's triangle (95% CI: −0.45∼-0.25, p=0.000), femoral intertrochanteric (95% CI: −0.90∼-0.64, p = 0.000), calcaneus (95% CI: −0.31∼-0.18, p = 0.000) and distal forearm (95% CI: −0.09∼-0.03, p = 0.000), but EH cannot reduce the BMD of the femur rotor (95% CI: −0.07∼0.24, p = 0.273). Another valuable study showed that EH can reduce the BMD of the lumbar spine (95% CI: −0.11∼-0.03, p = 0.000) and femoral neck (95% CI: −0.11∼-0.07, p = 0.000) in Asian populations. In non-Asian populations, EH

can reduce the BMD of the femoral neck (95% CI: 0.04~0.19, p = 0.002), but cannot reduce the BMD of the lumbar spine (95% CI: −0.04~0.11, p = 0.346).
Conclusions. Summarizing the articles and results analysis suggests that EH can have a negative effect on BMD, for different parts of bone, the degree of reduction is different. Furthermore, the reduction level of BMD can vary for different regions and populations.
Key words: association, essential hypertension, bone mineral density, meta-analysis

179. THE ROLE OF TRIGGER INFECTIONS IN REACTIVE ARTHRITIS

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Introduction. Reactive Arthritis(ReA) is an immune-mediated synovitis resulting from slow bacterial infections and showing intra-articular persistence of viable nonculturable bacteria and/or immunogenetic bacterial antigens synthesized by metabolically active bacteria residing in the joint and/or elsewhere in the body.Reactive arthritis is known to be triggered by a bacterial infection, particularly of the genitourinary (Chl. trachomatis, Neisseria gonorrhea, Mycoplasma hominis, and Ureaplasma urealyticum) or GI tract (Salmonella enteritidis, Shigella flexneri, and disenteriae, Yersinia enterocolitica, Campylobacter jejuni, Cl.difficile). The incidence is about 2% to 4% after a urogenital infection mainly with chlamydia trachomatis and varies from 0% to 15% after gastrointestinal infections with Salmonella, Shigella, Campylobacter, or Yersinia.
Aim of the study. To identify the most common infections that lead to the reactive arthritis and to highlight the pathogenetic mechanisms of action, which would help to improve the treatment tactic.
Materials and methods. The relevant articles on the topic were taken from the databases NCBI, PubMed, Medline,and ScienceDirect .
Results. Reactive arthritis is an immune-mediated syndrome triggered by a recent infection. It is hypothesized that when the invasive bacteria reach the systemic circulation, T lymphocytes are induced by bacterial fragments such as lipopolysaccharide and nucleic acids. These activated cytotoxic-T cells then attack the synovium and other self-antigens through molecular mimicry. This is supported by the evidence of Chlamydia trachomatis and C pneumoniae ribosomal RNA transcripts, enteric bacterial DNA and bacterial degradation products in the synovial tissue and fluid. It is believed that anti-bacterial cytokine response is also impaired in reactive arthritis, resulting in the decreased elimination of the bacteria.
Conclusions. Current evidence supports the concept that reactive arthritis (ReA) is an immune-mediated synovitis resulting from slow bacterial infections and showing intra-articular persistence of viable, nonculturable bacteria and/or immunogenetic bacterial antigens synthesized by metabolically active bacteria residing in the joint and/or elsewhere in the body. The mechanisms that lead to the development of ReA are complex and basically involve an interaction between an arthritogenic agent and a predisposed host. The way in which a host accommodates to invasive facultative intracellular bacteria is the key to the development of ReA. The details of the molecular pathways that explain the articular and extra-articular manifestations of the disease are still under investigation.
Key words: bacterial infection,trigger, reactive arthritis