Osteoporosis International (2022) 32 (Suppl 1):S91-S92
https://doi.org/10.1007/s00198-021-06122-y

ABSTRACT

World Congress on Osteoporosis, Osteoarthritis and Musculoskeletal Diseases (WCO-IOF-ESCEO 2021): Committee of Scientific Advisory Abstracts

CSA-OC1
BIOMARKERS OF BONE FRAGILITY IN PATIENTS WITH DIABETES

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In recent years, fragility fractures have been recognized as an important complication of patients with type 1 and type 2 diabetes. The exact mechanisms accounting for bone fragility in diabetes are poorly understood. Determinants of reduced bone strength include micro- and macroarchitectural changes, cellular and molecular mechanisms, poor glycaemic control, and presence of diabetes-related complications.
Cellular and molecular changes in diabetic bone disease include a state of low bone turnover, altered calcium and PTH metabolism with relative hypoparathyroidism, a decrease in enzymatic crosslinking and hyperglycaemia-induced accumulation of AGEs, alterations in osteocyte function with changes in protein levels (sclerostin, periostin), increase in pro-inflammatory cytokines (TNF, IL-6, and IL-1) and markers of inflammation (CRP), dysregulation of adipokines (adiponectin, leptin) and altered hormone levels (amylin, insulin, IGF-1, and gonadal hormones).
Some of these biochemical markers are commercially available to be measured in serum or urine. They may be used to reflect diabetes-specific structural and/or material changes in bone properties and may be used for fracture risk assessment in patients with type 1 and type 2 diabetes.
This lecture will characterize cellular and molecular markers reflecting diabetic bone disease, review the interaction of these molecular markers in the pathogenesis of diabetic bone disease, discuss their potential use in clinical practice with specific focus on their analytical performance and evaluate whether these markers may be used as clinical tools to predict bone loss and fracture risk in patients with diabetes.

CSA-OC2
PATHOPHYSIOLOGY OF VASCULAR CALCIFICATION AND BONE LOSS: LINKED DISORDERS OF AGING?

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Vascular calcification, bone loss, and increased fracture risk are current age-associated disorders frequently considered as "physiological" aging. Clinical and experimental data suggest that vascula calcification and bone loss, beyond aging, could be influenced by multiple factor that can promote, at the same time, vascular calcification and bone loss. Vascular calcification is an active process of calcium and phosphate precipitation that involves the transition of the vascular smooth muscle cells (VSMCs) to osteoblast-like cells. If mineralization takes place, the clinical consequence in the large and medium-caliber arteries is an increased stiffness with negative impact on cardiovascular outcomes. The molecules involved in the change of the VSMC phenotype have been extensively studied, the evidence suggests there are driven factors that promote and/or inhibits vascular calcification.
Parathyroid hormone (PTH) plays a key role in bone metabolism and vascular calcification acting through several mechanisms which includes the regulation of the RANK/RANKL/OPG system, the Wnt/ß-catenin pathway and the modulation of several factors, such as calcium, phosphorus, and vitamin D. The micro RNAs has been also implicated as they are regulators not only of skeletal related genes, but also of genes involved in cardiovascular complications, such as vascular calcification, left ventricle hypertrophy, and myocardial fibrosis.
Important progress has been made in this field; however, the complete understanding of interactions between aging, vascular calcification and bone loss still remains uncomplete.

CSA-OC3
SCREENING FOR HIGH FRACTURE RISK IN PRIMARY CARE

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The burden of long-term osteoporosis management falls on primary care in most healthcare systems. However, past and recent studies demonstrate a wide and stable treatment gap, most of which appears to be secondary to a lack of awareness of fracture risk. In most countries, screening is regarded as a public health measure for the purpose of identifying individuals who are likely to benefit from further investigations and/or treatment to reduce the risk of a disease or its complications. Well-established criteria for the development of a screening program have existed for over 50 years [1] and osteoporosis and its ensuing fractures fulfill many of these. For example, the condition should be a significant health burden, with a sufficiently large, identifiable target population to enable safe, clinically, and cost-effective screening. Likewise, there is an obvious need for an established testing procedure and effective interventions to prevent the outcome of interest.
The effectiveness of screening programs incorporating the FRAX fracture risk assessment tool has recently been evaluated in three large randomized, controlled studies [2,3,4]. Despite important differences in study design and

approaches to intervention thresholds, two of the studies showed significant reductions in hip fractures [2,3]. While the third study failed to show such an effect, a meta-analysis of all three studies showed a 20% reduction in hip fractures with smaller but significant reductions in major osteoporotic fractures, and all osteoporotic fractures [4,5]. The approaches, particularly that utilized in the SCOOP study in the UK is highly cost-effective or cost-saving [6,7]. These studies support the proposal that screening for high fracture risk in primary care should strongly be considered for incorporation into many health care systems to reduce the burden of fractures, particularly hip fractures. The key remaining hurdles to overcome are engagement with primary care healthcare professionals, and the implementation of systems that facilitate and maintain the screening program.

References

1. Wilson JMG, Jungner G (1968) Principles and practice of screening for disease. World Health Organization https://apps.who.int/iris/handle/10665/37650

2. Shepstone L, Lenaghan E, Cooper C et al (2018) Screening in the community to reduce fractures in older women (SCOOP): a randomized controlled trial. Lancet 391:741–747

3. Rubin KH, Rothmann MJ, Holmberg T et al (2018) Effectiveness of a two-step population-based osteoporosis screening program using FRAX: the randomized Risk-stratified Osteoporosis Strategy Evaluation (ROSE) study. Osteoporos Int 29:567–578

4. Merlijn T, Swart KM, van Schoor NM et al (2019) The Effect of a Screening and Treatment Program for the Prevention of Fractures in Older Women: a Randomized Pragmatic Trial. J Bone Miner Res 34:1993–2000

5. Merlijn T, Swart KMA, van der Horst HE, Netelenbos JC, Elders PJM (2020) Fracture prevention by screening for high fracture risk: a systematic review and meta-analysis. Osteoporos Int 31:251–257

6. Soreskog E, Borgstrom F, Shepstone L et al (2020) Long-term cost-effectiveness of screening for fracture risk in a UK primary care setting: the SCOOP study. Osteoporos Int 31:1499–1506

7. Turner DA, Khioe RFS, Shepstone L et al (2018) The cost-effectiveness of screening in the community to reduce osteoporotic fractures in older women in the UK: economic evaluation of the SCOOP study. J Bone Miner Res 33:845–851