Comment

One in two postmenopausal women and one in five older men are at risk for a low-energy fracture. Bone fractures exacerbate the course of underlying diseases and significantly increase mortality, especially in men.

The risk of bone fractures is influenced by a number of factors, including bone mineral density (BMD), bone quality and non-skeletal agents affecting the incidence of falls. Each of these factors is under, at least partial, genetic control. The role of genetic determinants of skeletal fractures is discussed in the article "Genetic factors, osteoporosis and bone fractures". The authors indicate that important determinants of BMD, such as peak bone mass and the rate of age-related bone loss are genetically determined. They present current methods of genetic studies and discuss the importance of candidate genes for osteoporosis classified according to metabolic or hormonal pathways.

Most fractures, especially hip fractures, are the results of falls. The role of reduced mass and strength of skeletal muscles, particularly of lower extremities, in the rate of age-related fragility fractures is presented in the paper entitled "Sarcopenia and osteoporosis – cofactors of increased risk of falls and bone fractures". The authors emphasize that sarcopenia, which decrease bone strength and mobility in elderly people, is the result of reduced physical activity and age-related metabolic abnormalities leading to e.g. accumulation of fat between and within muscle fibers.

Numerous bone fractures are the result of secondary osteoporosis. The authors of the article entitled "Bone fractures in cancer patients" present the mechanisms responsible for bone loss and increased incidence of skeletal fractures among patients with neoplastic diseases. They indicate that hormonal therapies used in patients with hormonally-responsive cancers as well as chemotherapy result in hypogonadism and progressive loss of bone mass. The authors remind that patients with high risk of fragility fractures should be given preventive treatment as soon as possible.

The authors of the paper entitled "Bone fractures after stroke" discuss the main causes and mechanisms leading to accelerated bone loss and increased rate of fragility fractures in patients following stroke. The authors describe that main factors influencing bone mass loss are duration of hemiplegia-induced immobilization, time and degree of functional recovery, and severity of functional deficits. They emphasize that effective management for prevention of post-stroke fragility fractures should include physical exercise regimens, the use of hip protectors, correction of vitamin D deficiency, and – in patients with high risk of fractures – antiresorptive treatment.

Patients with type-1 and type-2 diabetes mellitus (DM) were found to have significantly increased risk of low-energy fractures. The authors of the paper entitled "Diabetes mellitus, osteoporosis and bone fractures" describe the mechanisms of fragility fractures, partly independent on BMD, and emphasize that conventional diagnostic methods of fracture risk assessment are not sufficient in patients with DM. The authors also describe anti-diabetic therapies which can additionally enhance, have not affect, or significantly reduce the incidence of bone fractures.

To reduce the rate of skeletal fractures effective therapy should be implemented as soon as possible. The authors of the article entitled "Vitamins D and K and bone fractures" describe an important role of both vitamins in bone tissue metabolism and fracture prevention. They emphasize that significant vitamin D deficiency results in muscle weakness and increased risk of falling, while vitamin K deficiency decreases bone quality diminishing the γ-carboxylation of the osteocalcin. The authors indicate that proper supplementation of vitamins D, K and calcium is able to reduce the incidence of bone fractures.

In patients with high risk of fractures additional, effective therapy has to be employed. In the article entitled "Bisphosphonates and denosumab – the efficacy in the fracture prevention" the results of randomized, controlled clinical trials on efficiency of these anti-osteoporotic drugs are discussed. The authors indicate that bisphosphonates and denosumab significantly reduce the risk of vertebral and nonvertebral fractures in patients with primary and secondary osteoporosis. Long-term treatment with these drugs may result, however, in rare but serious side effects. The authors of the paper entitled "Osteonecrosis of the jaw and atypical femoral fractures as complications of antiresorptive therapy" describe the causes, pathophysiology and risk factors of both complications.

The report entitled "A case of an (a)typical course of thyrotoxic storm" has been included in this issue of "Progress in Medicine" to illustrate the difficulties in proper diagnosis of the disease in everyday clinical practice and to remember that hyperthyroid state is related to significantly accelerated bone turnover and increased risk of bone fractures.

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