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Osteoporosis and Related Bone Metabolic Disease

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Many heterogeneous causes (e.g., metabolic, inflammatory, autoimmune, vascular, and renal diseases, and even drugs), collectively grouped as secondary causes of osteoporosis, may lead to bone loss or damage to architecture through a number of mechanisms. Although these secondary causes of osteoporosis are the most frequently observed causes of unexpected bone loss, they can only be diagnosed via a high degree of suspicion and clinical experience and by performing the appropriate investigations. In inflammatory disorders such as rheumatoid arthritis or chronic inflammatory bowel diseases, as well as vascular diseases, T-cell activation, and consequently pro-inflammatory cascades, trigger the increased expression of T-cell-derived RANKL. In addition, a new biomarker signature of bone-related miRNAs is promising in certain clinical features. Glucocorticoids, often used to control disease activity, decrease the number and function of osteoblasts and inhibit OPG expression. The ubiquitous occurrence of disease-related secondary changes in bone metabolism implies that numerous medical disciplines need to interact. Screening for secondary causes of osteoporosis and the search for new modes of action should present a substantial aspect of osteoporosis management. In the book, the current management of osteoporosis and related metabolic bone diseases is discussed.

