

Resource

Osteoporosis in RA



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Osteoporosis in Rheumatoid Arthritis

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Introduction

Osteoporosis is a common feature in adults with rheumatoid arthritis (RA) and may lead to an increased risk of fracture. Patients who fracture are often immobilised for a significant period, and this may have a further adverse effect on bone. In general, several studies have shown a two-fold increase in osteoporosis in patients with RA compared to individuals of the same age and sex who do not have RA. Several factors may contribute to the increased risk, including difficulty with taking exercise and long-term use of corticosteroids (often referred to as 'steroids'). Osteoporosis can, of course, occur for reasons other than having RA, so in any patient diagnosed with osteoporosis, the appropriate (and usual) tests should be performed to exclude other explanations. This review highlights the steps that can be taken to prevent this important complication in RA.



What is osteoporosis?

Osteoporosis means porous bone, and it is a condition of the skeleton characterised by reduced bone quantity and quality. Bone mass peaks by about the age of thirty and slowly decreases thereafter. Bone undergoes a continuous process of break down and formation so that every year approximately 10% of the skeleton in adults is remodelled. An imbalance between the rate of breakdown and formation leads to bone loss. This results in fragile bones and an increased risk of fractures. The most common sites for fracture are the hip, spine and wrist. Osteoporosis is common; it is estimated to affect over 200 million people worldwide. One in three women and one in five men, older than 50 years may eventually experience osteoporotic fractures.

Why are people with RA at increased risk?

In RA, bone may be affected by structural joint damage (erosions) and osteoporosis. The causes of osteoporosis associated with RA are numerous and include the effects of chronic inflammation, the effects of medication and lifestyle factors.

Osteoporosis in RA can present in two ways: generalised bone loss or periarticular (around joint space) osteoporosis. The latter is probably due to local release of inflammatory agents. Inflammation leads to more severe bone loss in the hand as compared with at the hip or spine and has been shown to be reduced in patients whose inflammatory disease is treated more aggressively. The remainder of this article focuses on generalised osteoporosis.

In RA risk factors of generalised osteoporosis and fractures could be divided into two groups: 1) disease related risk factors and 2) traditional risk factors. The most frequently reported RA related risk factors are particularly inflammation, disease duration, but also immobility, disability and high dose corticosteroid use). In addition to the factors described above, there are a number of traditional risk factors that are not specific to RA. These include being female, increasing age, a postmenopausal state, family history of osteoporosis, being underweight, inadequate physical activity, cigarette smoking, high alcohol intake and increased fall risk.

How is osteoporosis diagnosed?

Bone density is measured by a type of scan called 'dual-energy x-ray absorptiometry' (DEXA). DEXA is the standard method used to establish or confirm a diagnosis of osteoporosis. This technique uses low doses of radiation, is quick and requires no undressing. It is suitable for individuals who suffer from claustrophobia as the patient is not enclosed during the scan. Results from the scan may be incorporated into an online web-based tool called FRAX to calculate an individual's risk of breaking a bone over the next 10 years. Patients who feel that they might be at risk of osteoporosis can discuss this with their GP or hospital consultant who can advise further. In some cases, patients may be started on treatment without needing to have a DEXA scan performed if their risk of an osteoporotic fracture is high. In general, while an initial scan is often helpful and commonly performed, follow up scans are less commonly used now. In cases where they are indicated, these would typically be every 3-5 years. Your hospital consultant can advise on the need for this.

What are the treatment options?

An important part of the management of osteoporosis is education, as lifestyle changes can reduce

the chance of developing osteoporosis. A healthy diet (rich in calcium and vitamin D), weight-bearing exercise and sensible exposure to sunlight (the main source of vitamin D) can all help to maintain bone mass. Smoking and excessive alcohol intake have a harmful effect and therefore, should be avoided. Calcium and vitamin D supplements may be prescribed if the dietary intake and sunlight exposure are inadequate.

There are also a number of drugs available to reduce the risk of fracture—these act either by reducing bone breakdown or stimulating bone formation. The usual first-line therapy is a group of drugs called bisphosphonates, that include the agents alendronate and risedronate, and act to reduce bone breakdown. These drugs can be given orally, or intravenously, so if tablets are not suitable (for example if you suffer from gastric problems) an infusion (such as zoledronate) may be more suitable. Another group of drugs that could be used to target cellular pathways that are important in controlling the cells responsible for bone breakdown. This may be important for the development of both regional and generalised osteoporosis, and in the prevention of the development of erosions. One such drug, denosumab (administered as a subcutaneous injection), has been shown to reduce bone turnover and increase bone mineral density in postmenopausal women with low bone mineral density, reduce fracture risk in women with postmenopausal osteoporosis, and reduce structural damage in patients with rheumatoid arthritis when added to ongoing methotrexate treatment. However, it may not be suitable for all patients. For some patients at highest fracture risk and in whom other therapies may have failed, teriparatide (given by daily injections for limited periods of time) may be used. It is a parathyroid hormone treatment and works by increasing the activity of bone-building cells. Novel therapies such as monoclonal antibody against sclerostin are being developed and present promise for future use.

In all cases, it is recommended that a clinician reassesses the need for treatment after three years of intravenous bisphosphonate/subcutaneous denosumab and five years of oral bisphosphonate. For high-risk patients, continuation of treatment is usually warranted, but where there have been no new fractures, and bone density has improved, a period without treatment may be recommended. Importantly, denosumab should not be stopped without considering an intravenous bisphosphonate injection or other treatment, as discontinuation has been associated with spinal fractures. Needless to say, the lifestyle measures considered in the previous section are also very important factors to consider alongside drug therapy, and the good control of joint inflammation is critical.

Conclusion

Osteoporotic fractures are common, and patients with RA may be at an increased risk. However, we have excellent methods for detection and therapy, with lifestyle measures being an important part of prevention and treatment of this condition.

Useful links

[Royal Osteoporosis Society](#)

[Strong Bones and Me](#)

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