Osteomalacia

Osteomalacia is the softening of the bones caused by impaired bone metabolism primarily due to inadequate levels of available phosphate, calcium, and vitamin D, or because of resorption of calcium. The impairment of bone metabolism causes inadequate bone mineralization. Osteomalacia in children is known as rickets, and because of this, use of the term "osteomalacia" is often restricted to the milder, adult form of the disease. Signs and symptoms can include diffuse body pains, muscle weakness, and fragility of the bones. In addition to low systemic levels of circulating mineral ions necessary for bone and tooth mineralization, accumulation of mineralization-inhibiting proteins and peptides (such as osteopontin and ASARM peptides) occurs in the extracellular matrix of bones and teeth, likely contributing locally to cause matrix hypomineralization (osteomalacia).[1][2][3][4][5]

The most common cause of osteomalacia is a deficiency of vitamin D, which is normally derived from sunlight exposure and, to a lesser extent, from the diet.[6] The most specific screening test for vitamin D deficiency in otherwise healthy individuals is a serum 25(OH)D level.[7] Less common causes of osteomalacia can include hereditary deficiencies of vitamin D or phosphate (which would typically be identified in childhood) or malignancy.

Vitamin D and calcium supplements are measures that can be used to prevent and treat osteomalacia. Vitamin D should always be administered in conjunction with calcium supplementation (as the pair work together in the body) since most of the consequences of vitamin D deficiency are a result of impaired mineral ion homeostasis.[8]

Nursing home residents and the homebound elderly population are at particular risk for vitamin D deficiency, as these populations typically receive little sun exposure. In addition, both the efficiency of vitamin D synthesis in the skin and the absorption of vitamin D from the intestine decline with age, thus further increasing the risk in these populations. Other groups at risk include individuals with malabsorption secondary to gastrointestinal bypass surgery or celiac disease, and individuals who immigrate from warm climates to cold climates, especially women who wear traditional veils or dresses that prevent sun exposure.[9]

1 Signs and symptoms

Osteomalacia is a generalized bone condition in which there is inadequate mineralization of the bone. Many of the effects of the disease overlap with the more common osteoporosis, but the two diseases are significantly different. There are two main causes of osteomalacia: (1) insufficient calcium absorption from the intestine because of lack of dietary calcium or a deficiency of, or resistance to, the action of vitamin D; and (2) phosphate deficiency caused by increased renal losses.

- Diffuse joint and bone pain (especially of spine, pelvis, and legs)
- Muscle weakness
- Difficulty walking, often with waddling gait
- Hypocalcemia (positive Chvostek sign)
- Compressed vertebrae and diminished stature
- Pelvic flattening
- Weak, soft bones
- Easy fracturing
- Bending of bones

Osteomalacia in adults starts insidiously as aches and pains in the lumbar (lower back) region and thighs before spreading to the arms and ribs. The pain is symmetrical, non-radiating and accompanied by sensitivity in the involved bones. Proximal muscles are weak, and there is difficulty in climbing up stairs and getting up from a squatting position.

As a result of demineralization, the bones become less rigid. Physical signs include deformities like triradiate pelvis[10] and lordosis. The patient has a typical "waddling" gait. However, these physical signs may derive from a previous osteomalacial state, since bones do not regain their original shape after they become deformed.

Pathologic fractures due to weight bearing may develop. Most of the time, the only alleged symptom is chronic fatigue, while bone aches are not spontaneous but only revealed by pressure or shocks.It differs from renal osteodystrophy, where the latter shows hyperphosphatemia.

2 Causes

The causes of adult osteomalacia are varied, but ultimately result in a vitamin D deficiency:

- Insufficient nutritional quantities or faulty metabolism of vitamin D or phosphorus
- Renal tubular acidosis
- Malnutrition during pregnancy
- Malabsorption syndrome
- Hypophosphatemia[11]
- Chronic kidney failure
- Tumor-induced osteomalacia
- Long-term anticonvulsant therapy[12]
- Celiac disease[13]
- Cadmium poisoning, itai-itai disease

3 Diagnosis

3.1 Biochemical findings

Biochemical features are similar to those of rickets. The major factor is an abnormally low vitamin D concentration in blood serum.Major typical biochemical findings include:[14]

- Low serum and urinary calcium
- Low serum phosphate, except in cases of renal osteodystrophy
- Elevated serum alkaline phosphatase (due to an increase in compensatory osteoblast activity)
- Elevated parathyroid hormone (due to low calcium)

Furthermore, a technetium bone scan will show increased activity (also due to increased osteoblasts).

3.2 Radiographic characteristics

Radiological appearances include:

- Pseudofractures, also called Looser's zones.
- Protrusio acetabuli, a hip joint disorder

4 Treatment

Nutritional osteomalacia responds well to administration of 10,000 IU weekly of vitamin D for four to six weeks. Osteomalacia due to malabsorption may require treatment by injection or daily oral dosing[15] of significant amounts of vitamin D.

5 Etymology

Osteomalacia is derived from Greek: *osteo-* which means "bone", and *malacia* which means "softness". In the past, the disease was also known as **malacosteon** and its Latin-derived equivalent, **mollities ossium**. Osteomalacia is associated with increase in osteoid maturation time.

6 See also

- Osteopetrosis

7 References

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