



## **Bone Deformity due to Rickets and Osteomalacia in Children and Adolescents**

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### **Authors' contributions**

*This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.*

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### **ABSTRACT**

Rickets is a worldwide bone disease that is associated with disorders of calcium and phosphate homeostasis and can lead to short stature and joint deformities. Osteomalacia is a major metabolic bone disease that results from a chronic and severe deficiency of vitamin D or phosphate from any cause after growth has stopped. A deficiency of vitamin D or phosphate leads to defective bone mineralization and generalized or localized vague bone pain in various parts of the skeleton and / or proximal muscle weakness. Rickets and osteomalacia are two different clinical diseases with impaired bone mineralization. Rickets occurs throughout the growing skeleton in infants and children, while osteomalacia occurs in adults after fusion of the growth plates. Rickets and osteomalacia are increasingly common in Saudi Arabia, with vitamin D deficiency being the most common etiological cause. Early skeletal deformities can occur in infants, such as soft, thin skull bones, a condition known as craniotabes. In adults, as a result of demineralization, the bones become less rigid (soft bone) with pathological fractures. The diagnosis of both diseases is based on the medical history and physical examination, radiological characteristics, and biochemical tests. Management depends on the underlying etiology.

*Keywords: Bone deformity; rickets; osteomalacia; children; adolescents.*

## 1. INTRODUCTION

Rickets is a worldwide bone disease that is associated with disorders of calcium and phosphate homeostasis and can lead to short stature and joint deformities. Rickets can be diagnosed based on the medical history and physical examination, radiologic features, and biochemical tests. It can be divided into 2 main groups according to the phosphate or calcium content: phosphopopes and calcipene. Knowing the categorization of the type of rickets is essential for a rapid diagnosis and proper management [1].

Nutritional rickets remains the most common form. Other causes of rickets include calcium and phosphorus deficiencies, inherited forms of hypophosphatemic rickets, and vitamin D metabolic defects, including receptor mutations [2].

In most developing regions around the world, nutritional rickets is a major health problem, probably because risk factors are still in place. In the Kingdom of Saudi Arabia, vitamin D deficiency is quite common among infants, children, adolescents, and pregnant and lactating mothers, despite economic prosperity and sufficient sun exposure throughout the year [3].

Osteomalacia is a major metabolic bone disease that results from a chronic and severe deficiency of vitamin D or phosphate from any cause after growth has stopped. A deficiency of vitamin D or phosphate leads to defective bone mineralization and generalized or localized vague bone pain in various parts of the skeleton and / or proximal muscle weakness [4]. Osteomalacia is more common in regions where sun exposure is low due to long winters with early sunsets or wearing traditional long clothing. These regions include: Middle Eastern and Asian countries such as Saudi Arabia. Due to the various clinical manifestations of this condition in its mild and early stages, it can be misdiagnosed with a variety of musculoskeletal disorders such as osteoporosis, especially in the elderly, and sometimes with bone biopsy that may be necessary. Early diagnosis requires a high suspicion of osteomalacia and there is no specific single-screen blood test for diagnosis [5].

Rickets and osteomalacia are two different clinical diseases with impaired bone

mineralization. Rickets occurs throughout the growing skeleton in infants and children, while osteomalacia occurs in adults after fusion of the growth plates. Rickets and osteomalacia are increasingly reported in Saudi Arabia, with vitamin D deficiency being the most common etiologic cause [6].

### 1.1 Objectives

The study aims to summarize the updated evidence regards: epidemiology, causes, types, effects on bone deformity, diagnosis and management.

## 2. EPIDEMIOLOGY

The prevalence of rickets has increased in both developed and developing countries. However, it is generally higher in developing countries than in developed countries. [7] Countries in Africa, the Middle East and Asia have a wide prevalence rate from 10% to 70%. A cross-sectional study was carried out on the knowledge of the prevalence of rickets in children living in Saudi Arabia with 864 participants on various social networks. platforms during the period from February to April 2018. Results: 15.3% of the children were diagnosed with rickets, 50.5%. % of the children diagnosed were between 1 and 5 years old [8]. Regarding risk factors, 41.9% of children drink soft drinks and 15.4% of them are obese. Only 55% of the participants breastfed their children, 35.9% of the participants know what rickets is, 45.3% request medical help after their child has been diagnosed and 70.5% of the Participants thought rickets with vitamin D can prevent [9]. At the end of this study, there were an increased number of children diagnosed with rickets in Saudi Arabia.

For osteomalacia there are reports that the prevalence of post-mortem disease histologically in adults is up to 25% [10]. However, the true incidence of osteomalacia is greatly underestimated worldwide. People at risk include people with dark skin, frequent users of full-length clothing, limited sun exposure, low socioeconomic status, and poor diet [11]. These risks vary around the world and depend on geographic location, cultural preferences, and ethnic origin. Healthcare providers should consider these factors, as well as other relevant clinical findings, when deciding to conduct further studies or recommend vitamin D supplements [12].

## 2.1 Causes

Rickets and osteomalacia are two diseases characterized by defective mineralization of bone and cartilage. Thus, children have rickets and osteomalacia, while adults have only osteomalacia. Rickets and osteomalacia have several causes:

Primary vitamin D deficiency, classic vitamin D deficiency - infants and puberty [13]. Vitamin D plays an essential role in skeletal health by regulating normal levels of calcium and phosphorus in the blood [14]. There are two main forms of vitamin D: vitamin D2 (ergocalciferol) and vitamin D3 (cholecalciferol). Vitamin D2 is obtained mainly from plant sources. Vitamin D3 is not only found in foods such as fish, eggs, milk, and cod liver oil, but is produced naturally through the conversion of dehydrocholesterol to cholecalciferol in the skin through sunlight (ultraviolet B in the range 290-315 nm) (fifteen). Vitamin D binds to the vitamin D-binding protein and is transported to the liver for hydroxylation and is converted by hydroxylase to calcidiol, which is then absorbed in the proximal tubule of the kidney by the endocytic receptors megalin and cubillin and by enzyme 1. Alpha hydroxylase.

is hydroxylated to form the active vitamin D metabolite, calcitriol. 1,25-dihydroxy vitamin D acts on the vitamin D receptor in intestinal cells to increase the absorption of calcium in the intestine by upregulation of the calcium channel. There is a complex interaction between the hormones produced by the kidneys (1,25 dihydroxy vitamin D), the bones (FGF23), and PTH. Understanding these interactions is essential to adequately manage rickets and / or osteomalacia [15].

Other common causes are exclusive breastfeeding, adult immigrants in industrialized countries, decreased sun exposure, use of sunscreen, age, residential and institutionalized groups, pregnancy and lactation, decreased food intake, morbid obesity, Secondary vitamin D deficiency, partial gastrectomy, small bowel malabsorption syndromes (eg, celiac disease) [16], hepatobiliary disease, pancreatic failure, chronic kidney failure, metabolic acidosis.

Medications and toxins such as anticonvulsants, antacids that are phosphate binding (eg, aluminum hydroxide), calcium deficiency, hyperparathyroidism deficiency

- chromosomal, autosomal dominant and recessive forms), proximal renal tubule diseases (Fanconi syndrome), distal renal tubule diseases (renal rickets with nephrocalcinosis and dwarfism) and hypophosphatasia [17].

## 2.2 Effects of Rickets on Bone Deformity

Early skeletal deformities can occur in infants, such as soft, thin skull bones, a condition known as craniotabes, which is the first sign of rickets; There may be humps on the skull and delayed closure of the fontanelle [18].

Young children may have bent legs and thickened ankles and wrists; older children may have knee joints. There may be spinal curvatures due to kyphoscoliosis or lumbar lordosis. The pelvic bones can become deformed. A condition known as rickety rosary can develop as a thickening due to the formation of nodules in the costochondral joints [19]. This appears as a visible lump in the center of each rib in a line on each side of the body. This looks a bit like a rosary, that's why it got its name. The deformity of a pigeon's chest can result in the presence of Harrison's Groove [20].

## 2.3 Effects of Osteomalacia on Bone Deformity

Osteomalacia begins slowly with pain in the lower back and thighs before spreading to the arms and ribs. The pain is symmetrical, not radiated, and is accompanied by tenderness in the affected bones. The proximal muscles are weak [21].

Demineralization makes the bones less rigid (soft bones). Physical signs include deformities such as triple pelvis [10] and lordosis. The patient has a typical "waddle" gait. However, these physical signs may be the result of a previous osteomalacial condition, as the bones do not regain their original shape after being deformed. Pathological fractures can result from stress [22].

## 2.4 Diagnosis of Rickets

A detailed medical history and a complete physical examination are essential to diagnose patients with rickets. The medical history should include the gestational age of the child, details of sun exposure, nutritional history, including use of dietary supplements, development / growth history, and relevant family history [23]. A positive family history of skeletal abnormalities,

growth retardation, alopecia, dental abnormalities, and parental blood relationships may suggest a genetic cause of rickets. Physical examinations should include a detailed skeletal examination (taking into account tenderness, deformities, softening, asymmetry, and neurological abnormalities), as well as a detailed dental examination. The medical history and physical examination often provide clues to diagnose rickets. However, the absence of clinical signs of rickets does not rule out this diagnosis, especially in the early stages [24].

The clinical manifestations of rickets vary depending on the underlying etiology, severity, and duration of the disease. Rickets is commonly seen in children between the ages of 6 months and 2 years. Children often have some bony clinical manifestations (often found at sites of rapid bone growth) such as:

Lower rib cage, which occurs when the diaphragm pulls the soft rib cage at its insertion site [25].

2. Skull, craniotables, softening of the skull bone, in infants older than three months. Frontal bumps and wide fontanelles are noted.

3. The extremities, primarily in childhood, may appear with weight-bearing limb deformities that primarily affect the rapidly growing bones. Children who crawl may have upper limb deformities [26]. However, when the child begins to walk, the deformities in the lower extremities will be noticed. Possible lower extremity deformities include bowed legs (genu varus), bumpy knees (genu valgus), and joint swelling (knees and ankles), while upper extremity deformities include widening of the wrist. The ulna grows relatively rapidly and is therefore significantly affected [27].

If rickets is suspected clinically, biochemical tests and radiographic imaging are the next steps to confirm the diagnosis.

## 2.5 Laboratory Investigations

The main laboratory marker for diagnosing rickets is serum alkaline phosphatase (ALP), which is usually high because it is a disease with abnormal mineralization and increased osteoblastic activity. [28].

Serum 25-hydroxyvitamin D level is another laboratory marker that helps diagnose rickets,

particularly vitamin D deficiency known as Fanconi syndrome associated with phosphaturia. [29].

Other biochemical tests include blood urea nitrogen (BUN) / creatinine levels to check the status of the kidneys and liver enzymes to check liver function.

## 2.6 Imaging

Radiological images must include; The fastest growing parts of the long bones are in the upper limb (away from the elbow) and lower limb (near the knee), and chest imaging is also helpful. The appearance of radiolucent lines at the junction between the epiphysis and the metaphysis and the expansion of the epiphyseal plate due to the accumulation of non-mineralized osteoid is the earliest radiological change [30]. The rickets also include cupping, spreading, fraying, and trabecular formation of the metaphysis. The formation of the epiphyseal center may be delayed or appear small. The bony cortex can be thin and osteopenic. Half-body images show a rickety rosary and an expansion of the costochondral connections. In advanced stages, angular deformities and pathological fractures of the bones of the upper and lower extremities can be detected [31].

## 2.7 Diagnosis of Osteomalacia

When evaluating osteomalacia, a medical history, including a surgical history, should be obtained. Other relevant questions should focus on activity level, hobbies, diet (i.e. vegetarian), and assessment of socioeconomic status [32].

No laboratory finding is specific for osteomalacia. However, patients with osteomalacia often present with hypophosphatemia or hypocalcemia. Furthermore, increased alkaline phosphatase activity is typically characteristic of diseases with impaired osteoid mineralization. In fact, some sources believe that hypophosphatemia or hypocalcemia and increased levels of alkaline phosphatase in the bones are necessary even if osteomalacia is suspected [33].

Radiographic findings may include looser areas or pseudofractures, and this is a classic finding in osteomalacia. They may represent poorly repaired insufficiency fractures and are visible as transverse bright spots perpendicular to the bone cortex [34]. They generally occur bilaterally and

symmetrically in the femoral necks, shaft, pubic rami, and ischial. In addition, radiographs may show decreased clarity of the vertebral body trabecula due to inadequate mineralization of the osteoid. Although not necessary for diagnosis, studies have shown a decrease in bone mineral density in the spine, hip, and forearm [35].

Iliac crest biopsy is considered the gold standard for diagnosis, but should be reserved for non-invasive methods if there is any doubt about the diagnosis or if the cause of the osteomalacia is unclear [36].

### 3. TREATMENT AND MANAGEMENT

#### 3.1 Rickets

Treatment strategies for rickets depend on the underlying etiology. There are several treatment regimens to treat rickets, all of which involve some form of administration of vitamin D, vitamin D2 (ergocalciferol), or vitamin D3 (cholecalciferol) followed by monitoring for healing [37]. The intensive phase of vitamin D treatment is given for two to three months along with a calcium supplement (500 mg through food or dietary supplements) for children with insufficient dietary calcium [38].

In a study at King Khalid University Hospital, Riyadh, Kingdom of Saudi Arabia, the diagnosis was based on clinical, biochemical and radiological data. Skeletal deformities in schoolchildren and adolescents were the symptoms in only 6.6% of the participants [39]. In a study that examined the prevalence of rickets in children in Saudi Arabia, it was found that 9.8% of rickety children suffer from bowlegs [40].

#### 3.2 Osteomalacia

After diagnosing osteomalacia, it is essential to evaluate the etiology. Treatment should focus on reversing the underlying condition and then correcting vitamin D and other electrolyte deficiencies. If vitamin D deficiency is the underlying cause, treatment through replacement therapy can lead to a significant improvement in strength and relief of bone tenderness within weeks [41]. Serum and urinary calcium levels should be monitored initially after 1 and 3 months and then every 6 to 12 months until 24-hour urinary calcium excretion is normal. Serum 25 (OH) D can be measured 3 to 4 months after initiation of therapy. In the presence of hypercalcemia or hypercalciuria, the dose can be

adjusted to prevent an excessive dose of vitamin D [42].

In a study to determine the causes and clinical symptoms of rickets in Riyadh, Saudi Arabia, the prevalence of osteomalacia deformities in adolescents with osteomalacia, defined as alkaline phosphatase levels  $\geq 500$  IU / L, was 7.4% of which [43]. Osteomalacia in girls can cause pelvic deformities and obstruction of labor later in life [44].

### 4. CONCLUSION

Rickets occurs throughout the growing skeleton in infants and children, while osteomalacia occurs in adults after fusion of the growth plates. Rickets and osteomalacia are becoming more common in Saudi Arabia, with vitamin D deficiency being the most common etiological cause. Early skeletal deformities can occur in infants, such as soft, thin skull bones, a condition known as cranio-tabes. In adults, as a result of demineralization, the bones become less rigid (soft bone) with pathological fractures. The diagnosis of both diseases is based on the medical history and physical examination, radiological characteristics, and biochemical tests. Management depends on the underlying etiology.

### CONSENT

It is not applicable.

### ETHICAL APPROVAL

It is not applicable.

### COMPETING INTERESTS

Authors have declared that no competing interests exist.

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