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Types of Rickets, Dental and Histologic Findings: Review of the Literature

Tipos de Raquitismo, Aspectos Dentários e Histológicos: Revisão de Literatura

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RESUMO

Introdução: O raquitismo, uma doença que ocorre durante a infância, é a falta de crescimento ósseo necessário para a mineralização. Diversas alterações radiográficas esqueléticas podem ocorrer devido a ausência de osteóide calcificado e a formação de cartilagem. A formação óssea adequada requer uma complexa interação de vários órgãos e produtos químicos, e a vitamina D merece uma menção especial, pois qualquer alteração na sua produção, absorção ou no seu metabolismo é fundamental para o desenvolvimento do raquitismo. Acredita-se que a fisiopatologia da doença está no transporte inadequado do fosfato, especialmente na diminuição da reabsorção de fosfato no túbulo renal proximal bem como no intestino. Na maioria dos casos, o diagnóstico é estabelecido com uma história completa e exame físico, e confirmado por exames laboratoriais. O diagnóstico precoce é essencial, pois a morbidade pode ser minimizada se as crianças forem tratadas antes dos oito meses de idade.
Objetivo: Apresentar os vários tipos de raquitismo com suas características clínicas, aspectos dentários, medidas preventivas e tratamento.
Conclusão: O cirurgião-dentista, bem como o pediatra, devem ser informados das características desta desordem para que a intervenção precoce possa evitar seqüelas subsequentes e procedimentos odontológicos mais invasivos.

DESCRITORES

Rickets; Hipofosfatemia; Manifestações bucais; Treatment.

ABSTRACT

Introduction: A disease that occurs during childhood, rickets is the failure of growing bone to mineralize. Many skeletal and radiographic changes can ocur because of the lack of calcified osteoid and the buildup of unossified cartilage. Proper bone formation requires a complex interplay of several organs and chemicals, and vitamin D deserves special mention because any disturbance in its production, absorption, or metabolism is paramount in the development of rickets. The pathophysiology of the disease is thought to be impaired phosphate transport, especially decreased phosphate resorption in the renal proximal tubule, as well as in the intestine. In most cases, the diagnosis is established with a thorough history and physical examination and confirmed by laboratory evaluation. Early diagnosis is essential because morbidity can be minimized if children are treated before eight months of age.
Objective: The aim of this literature review is to present various types of rickets with clinical features and the dental findings, preventive measurements and treatments.
Conclusion: The dentist as well as the pediatrician should be made aware of the features of this disorder so that early intervention can prevent subsequent serious and more invasive dental procedures.

KEYWORDS

Rickets; Hypophosphataemia; Oral manifestations; Therapeutics.

INTRODUCTION

Rickets develops when growing bones fail to mineralize. In most cases, the diagnosis is established with a thorough history and physical examination and confirmed by laboratory evaluation. Early diagnosis is essential because morbidity can be minimized if children are treated before eight months of age[1].

Human beings maintain adequate levels of vitamin D by producing it from cholesterol or by absorbing it from ingested food sources. Sunlight is a vital component necessary for the production of vitamin D, which begins in the skin and ends in the kidney. In skin, vitamin D production begins with conversion of 7-dehydrocholesterol to vitamin D_3 because of ultraviolet light. Another source of vitamin D is dietary intake of vitamins D_2 and D_3. Vitamin D_3 is converted in the liver to 25(OH)D_3 or calcidiol, the major circulating form of vitamin D. The enzyme 25(OH)D_3-1-a-hydroxylase in the kidney converts calcidiol to 1,25$(OH)_2D_3$ or calcitriol, the most active form of vitamin D[2].

Recently, 2 theories on impaired phosphate transport have been proposed. One is an impairment of the sodium-dependent phosphate transporter[3], and the other is a phosphatic factor that increases phosphate excretion[4,5]. However, impaired lα(OH)2D metabolism has also been suggested[6,7]. Various types of rickets with clinical features and treatments defined on Table 1[8,9-15].

The aim of this literature review is to present various types of rickets with clinical features and the dental findings, preventive measurements and treatments.

LITERATURE REVIEW

Types of Rickets

Nutritional rickets results from inadequate sunlight exposure or inadequate intake of dietary vitamin D, calcium, or phosphorus. A diet deficient in calcium[16], such as one dependent on nonfortified milk substitutes, can lead to rickets[17,18]. Nutritional rickets presents in the first two years of life with short stature, gait abnormality, developmental delay, and characteristic findings (Table 1[8,9-15] and Table 2[9,14,19]). Commonly, infants younger than six months present with hypocalcemic tetany or seizures, whereas older children present with failure to thrive or skeletal deformities[9].

Vitamin D–dependent rickets, type I results from abnormalities in the gene coding for 25(OH)D_3-1-a-hydroxylase[2], and type II is a rare autosomal disorder caused by mutations in the vitamin D receptor. Type II does not respond to vitamin D treatment; elevated levels of circulating calcitriol differentiate this type from type I[20].

The vitamin D–resistant types are familial hypophosphatemic rickets and hereditary hypophosphatemic rickets with hypercalciuria. Rickets refractory to vitamin D treatment may be caused by the most common heritable form, known as vitamin D–resistant rickets or familial hypophosphatemic rickets[10,11].

Hypophosphatemic vitamin D-resistant rickets or X linked hypophosphataemia (XLH) is a hereditary disease manifesting marked hypophosphataemia caused by renal tubular loss of phosphate into urine and an associated decrease in the calcium and potassium ion product. Normal levels of calcitriol are found in this disorder[12,21]. XLH, first reported by Albright et al.[22], is a syndrome showing marked hypophosphatemia, short stature, and rickets. In general, the main abnormality is considered to be a congenital impairment of phosphate transport and hypophosphatemia, resulting from decreased phosphate reabsorption in the brush border membrane on the luminal side of the proximal renal tubule and impaired phosphate reabsorption in the intestine.

X-linked hypophosphataemia (XLH) is also characterized by growth retardation, osteomalacic bone disease and hypophosphataemia[23,24]. Sporadic cases are often initially detected by limb deformity or gait abnormality. The systemic findings of XLH include bowed legs because of a body load showing immature skeletal bone calcification, spinal curvature deformities and beading of the ribs called rachitic rosary[25-27].

Other causes of rickets include renal disease, medications, and malabsorption syndromes (Table 1[8,9-15]).

Clinical Presentation and Diagnosis

General physical examination revealed typical bowing of the legs, marked genu valgum, rachitic rosary and growth retardation. Genu valgum or “knock-knee” deformity results in circumduction, a gait that requires the individual to swing each leg outward while walking to avoid striking the planted limb with the moving limb. Rachitic rosary is the name given to the bead-like enlargements of the costochondral junctions[28].

The diagnosis of rickets is made upon a physical exam, and confirmed by blood tests and X-rays. In children with rickets, complete physical and dental examinations should be performed. The entire skeletal system must be palpated to search for tenderness and bony abnormalities (Table 2[9,14,19]). Bowlegs in the absence of other findings are

relatively common in normal children in the first two years of life; rickets should be suspected in older bowlegged children and in cases associated with asymmetry, pain, or progression in severity. Gait disturbances in the ambulatory child and neurologic abnormalities (such as hyperreflexia) in all children should be sought.

Laboratory and Radiographic Findings

Laboratory investigation may include serum levels of calcium (total and ionized with serum albumin), phosphorus, alkaline phosphatase, parathyroid hormone, urea nitrogen, creatinine, and calcidiol. Urine studies include urinalysis and levels of urinary calcium and phosphorus. The serum level of calcidiol is indicative of the patient's overall vitamin D status[29]. Although calcitriol is the active form of vitamin D it has a short half-life and circulates at a concentration that is 1,000 to 2,000 times less than calcidiol[29]. Depending on the stage of the disease, laboratory values can vary.

An anteroposterior radiograph of rapidly growing skeletal areas, such as the knee or wrist, is most helpful in diagnosing rickets. The skeletal changes caused by rickets usually are most pronounced at the knees, wrists, and anterior rib ends (rachitic rosary)[19]. Classic radiographic findings include widening of the distal physis, fraying and widening of the metaphysis, and angular deformities of the arm and leg bones.

Table 1. Various Types of Rickets with Clinical Features and Treatments.

Type	Causes	Inheritance pattern	Clinical features	Treatment*
Nutritional rickets or vitamin D-deficiency rickets	Vitamin D deficiency, phosphorus or calcium deficiency (rare), inadequate sunlight exposure, secondary to malabsorption syndromes (IBD, celiac disease, cystic fibrosis [rarely])	NA	Skeletal findings, abnormal gait, hypocalcemic tetany/seizures, developmental delay, failure to thrive	Replace the deficient nutrient orally; may need to administer vitamin D intramuscularly if rickets secondary to malabsorption.
Vitamin D-dependent rickets				
Type I or pseudovitamin D-deficiency rickets	Deficiency of renal 25(OH) D_3-1-alpha-hydroxylase	Autosomal recessive	Younger than two years, hypocalcemic tetany, severe bony changes, seizures	Calcitriol (Rocaltrol)
Type II or hereditary 1 alpha, 25 dihydroxyvitamin D-resistant rickets	Defective interaction between calcitriol and receptor	Autosomal recessive	Younger than one year, severe bony changes, alopecia	Massive doses of calcitriol and calcium
Vitamin D-resistant rickets				
Familial hypophosphatemic rickets or X-linked hypophosphatemic rickets	Impaired proximal renal tubular reabsorption of phosphorus and inappropriately normal calcitriol levels	X-linked dominant	Short stature, leg bowing, dental abnormalities	Oral phosphate and calcitriol
Hereditary hypophosphatemic rickets with hypercalciuria	Impaired proximal renal tubular reabsorption of phosphorus and increased calcitriol	Autosomal recessive, autosomal dominant	Bone pain, muscular weakness	Oral phosphate
Miscellaneous				
Renal rickets or renal osteodystrophy	Loss of functional renal parenchyma caused by chronic renal failure leads to mineral derangements and decreased calcitriol production	NA	Bone pain, arthralgias, fractures, muscle weakness, failure to thrive	Vitamin D and phosphate-binding compound
Rickets of prematurity	Multifactorial	NA	Osteopenia, fractures	Replace dietary deficiencies and minimize iatrogenic causes.
Tumor-induced or oncogenic rickets	Tumor-induced inhibition of renal 25(OH)D_3-1-alpha-hydroxylase	NA	Fractures, bone pain, muscle weakness	Treat underlying malignancy.

IBD = inflammatory bowel disease; NA = not applicable; PTH = parathyroid hormone. *Must closely monitor serum calcium, phosphorus, and alkaline phosphatase levels; renal function; urine calcium levels; and radiographic results.

Table 2. Skeletal and Radiographic Findings Associated with Rickets.

Bowing or widening of physis	Flaring of wrists
Costochondral beading (rachitic rosary)	Fractures
Craniotabes	Fraying and cupping of metaphysis
Delayed closure of anterior fontanel	Frontal bossing of skull
Dental abnormalities	Genu valgum or varum
Flaring of ribs at diaphragm level (Harrison's groove)	Lordosis/kyphosis/scoliosis
	Osteopenia

Dental and Histologic Findings

This phenomenon is associated with well documented oral and dental findings[27,30-34]. Hypophosphataemic vitamin D-resistant rickets have been attributed to the enlarged coronal pulp spaces and to the grossly defective dentine allowing ingress of micro-organism to the dental pulp once attrition has removed the overlying protective enamel[35,36]. The enamel in some affected individuals has been described variously as relatively thin, hypocalsified or hypoplastic, although it is not always obvious from the text which dentition is affected by these changes[36]. In general, both primary and permanent teeth have dentinal dysplasia[33]. The teeth usually show taurodontism, poorly defined lamina dura and a hypoplastic alveolar ridge[36,37].

Spontaneous periapical abscess formation is also often observed in patients with XLH without dental caries or traumatic injury[30,38]. Because the teeth of patients with XLH are often associated with high pulp horns, large pulp chambers, and dentinal clefts, it is believed that the abscesses are caused by pulpal infection that was caused by bacterial invasion through enamel cracks and dentinal microcleavage of the teeth[33]. Dentists have diagnosed a few cases in which the systemic features were mild and dental abscesses were the first presenting sign[31].

Although odontoblast function is normal, hypophosphataemia leads to dysplastic and poorly mineralized dentin with areas of interglobular dentin. Because enamel and dentin formation occur between 4 months in utero and 11 months of age, the defects in the primary dentition can usually not be prevented. However, permanent teeth form after birth and their development could possibly be improved by medication started soon after birth. Abnormal dental development and dentin formation may persist despite therapy. The sequence of formation of abscesses usually appears to follow the sequence of eruption[35,39]. Hypophosphataemic patients have also been reported to display large interglobular spaces in the circumpulpal dentin[33,40], whereas the mantle dentin is unaffected[41].

Histologic findings of teeth in XLH include enlarged pulp chambers, wide predentin, marked globular dentin, and tubular dentinal defects extending from the pulp to the enamel. Enamel hypoplasia may or may not be present[42,43].

Prognosis and Therapy

Treatment goals are to relieve symptoms and correct the cause of the condition. With regard to nutritional rickets, the most important role of the primary care physician is helping parents prevent it. Along with sun protection advice, measures needed to prevent nutritional rickets must be stressed to the child's caregivers. Besides all exclusively breastfed infants, some older children also may need vitamin D supplementation[20]. Researchers have suggested an appropriate amount of sunlight exposure for infants (i.e., 30 minutes per week if only in a diaper and two hours per week if fully clothed)[44], but the exact amount needed for a particular child is not known.

Because vitamin D–dependent rickets, type I is caused by lack of production of calcitriol, treatment requires the replacement of that active product. The treatment of type II is more complex[45], and consultation with a children's nephrologist is advised.

Familial hypophosphatemic rickets is treated with oral phosphorus and calcitriol (Rocaltrol), whereas hereditary hypophosphatemic rickets with hypercalciuria requires replacement of oral phosphorus alone.

Investigators stress that treatment begun early in life lessens the disease burden[46]. To ensure early treatment, infants of affected parents must be screened often for hypophosphatemia and increased levels of serum alkaline phosphatase.

After treatment initiation, all patients will require careful monitoring of serum calcium, phosphorus, alkaline phosphatase, and calcidiol levels and of urine calcium and phosphorus levels. A spot urine calcium to creatinine ratio should be followed to detect hypercalciuria. Adjustments to medications are made to accommodate any abnormal fluctuations in serum or urine values. The earliest biochemical change after treatment initiation is a rise in the level of phosphorus followed by calcium within the first week. Radiographic changes may be evident within a week, and physical examination findings may normalize within six months. No matter which treatment course is chosen, the physician has to closely monitor the child's progress[20].

Dental care of these patients should consist of periodic examinations, topical fluoride applications, pit and fissure sealants and maintenance of good oral hygiene. Some authors advocate extraction of teeth that present periradicular abscesses and eventual restoration with

implants; however, endodontic and restorative treatment may not be able to maintain asepsis. The incompletely mineralized dentin exists in the form of calcospherites, which trap microorganisms and also impede mechanical endodontic cleaning[47,48]. The practitioner must conclude that the occurrence of spontaneous abscesses following a shallow cavity preparation necessitates aggressive preventive dental procedures.

Application of prefabricated metal or polycarbonate crowns for primary teeth without caries has been reported to be effective for prevention of attrition and enamel microfracture[37]. Prophylactic coverage of teeth of rickets cases with stainless steel crowns on molars and composite resin on the other teeth should be applied. In addition, the thin dentin layer perforates easily and does not support restorative posts for prosthetic crowns. This treatment should be carried out with caution and crown preparation should be minimal to avoid inadvertent pulp exposure. Another critical factor is the loss of vertical dimensions if multiple posterior primary teeth need to be extracted. Thus, there is a delicate balance between the benefits and possible risks of using stainless steel crowns. However, this aggressive preventive method cannot be adopted in all patients with XLH, because not all the pulp tissue is infected and iatrogenic pulp infection may occur during the tooth crown preparation.

In patients with Rickets, the dentition is highly susceptible to dental caries or attrition, and bacteria can invade easily from the oral cavity to dental pulp by menas of structural defects in enamel and dentin, resulting in pulpitis. Therefore, pit and fissure sealants are useful when the teeth are erupting as they prevent ingress of bacteria into the enamel microfractures as well as initiation of caries in the deep pits and fissures. Also, in patients with this disorder, professional dental care consisting of periodic examinations, topical fluoride applications and maintenance of good oral hygiene is imperative.

CONCLUSION

The dentist as well as the pediatrician should be made aware of the features of this disorder so that early intervention can prevent subsequent serious and more invasive dental procedures.

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