

ADOLESCENT OSTEOMALACIA IN SAUDI ARABIA: A HOSPITAL-BASED STUDY

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ABSTRACT

Background: Osteomalacia is a metabolic bone disorder caused by deficiency of Vitamin D and its active metabolites.

Design and Setting: A retrospective hospital-based study, conducted at King Khalid University Hospital, Riyadh, Saudi Arabia in the period January 1990 and December 2014.

Material and Methods: Medical records were retrospectively reviewed. Various data analysis including detailed history, physical examination, various laboratory investigations and radiological studies. The diagnosis of osteomalacia was based on clinical, radiological and biochemical data.

Results: Twenty-three adolescents were diagnosed with osteomalacia in the period under review. There were various etiologic diagnosis, with nutritional deficiency being the most common. Non-specific symptoms, such as bone pains and aches were the most presenting symptoms and signs. Milk and dairy consumption was generally low. The sun exposure was minimum and the majority of activities are indoor.

Conclusion: A high prevalence of osteomalacia in our adolescent population. Although, nutritional deficiency is being the most common cause which indicates the importance of establishing a preventive measures, however, malabsorption such as celiac disease, or intake of any other drug interfering with Vit. D metabolism, such as anti-epileptic drugs should be seriously considered. Routine serological screening (anti-endomyseal antibodies) for celiac disease, and patients on long-term anti-epileptic medications, should be routinely screened for secondary osteomalacia.

Keywords: Adolescent, osteomalacia, Saudi Arabia.

INTRODUCTION

Osteomalacia is a metabolic disorder caused by deficiency of Vitamin D and its active metabolites either due to inadequate dietary intake of calcium and Vitamin D, malabsorption,

inadequate exposure to sunlight, and impaired Vitamin D metabolism.¹⁻³ In a healthy individual, bone mineralization continues throughout childhood and adolescents until peak bone mass is reached. Peak bone mass is an important factor that determines an individual's risk of developing fractures and there is a relationship between vitamin D status and bone health.⁴⁻⁵

Osteomalacia presents with non-specific symptoms and early recognition requires a high degree of suspicion in the absence of skeletal deformities. The diagnosis usually based on a combination of clinical, exclusion of other diseases, radiological, and biochemical studies, and confirmed by response to treatment.⁶

In this report, we present our clinical experience with adolescent's osteomalacia from a Paediatric Endocrine Clinic, Riyadh, Saudi Arabia, highlighting the Aetiology, and clinical presentation.

MATERIAL AND METHODS

This is a retrospective, hospital-based study, conducted at a Pediatric Endocrine Clinic, King Khalid University Hospital, Riyadh, Saudi Arabia, between the period January 1990 and December 2014 and included adolescents aged 12-18 years. The diagnosis of osteomalacia was based on clinical, radiological and biochemical features as suggested.⁶

Medical records were reviewed and data analysed included age, sex, presenting symptoms and signs, detailed history including dietary practice and medication intake, as well as, clinical examination. Laboratory investigations included complete blood count (CBC), renal, liver and bone profiles and serum concentrations of 25-hydroxy Vitamin D (25-OH Vit. D). Tests for parathyroid hormone (PTH) and 1,25 dihydroxy Vitamin D (1,25 (OH)₂ Vit. D). Serological studies for celiac disease (anti-gliadin, and anti-endomyseal antibodies (Elisa Immunoassay) were conducted if indicated. Small bowel biopsies were done to confirm the diagnosis.⁷ Radiographs of wrists, knees, ankles and long bones were also evaluated.

RESULTS

Twenty-three adolescents with osteomalacia presented during the study under review, 16 female (69.6%) and 7 male (30.4%) patients, aged 12 to 16.5 year with a mean age of 13.6 years. All were Saudis from the central region of Saudi Arabia. There were various etiologic diagnosis of osteomalacia in our series (Table 1), with nutritional deficiency being the commonest cause constituted (73.9%). Serum concentrations of 25 hydroxy Vitamin D were low, ranging between <10 to 45 nmol/L, normal > 50 nmol/L. Milk and dietary consumption of calcium containing products was generally low, with increased consumption of fast food and soft drinks. Sun exposure was minimum and the majority of activities were indoors. Non-specific symptoms, such as bone pains and aches were the most presenting symptoms and signs (Table 2). Muscular weakness was the next most prominent symptoms and was universally present. Four patients (17.4%), all females with proximal myopathy.

Looser zones (pseudofractures) were seen at the upper end of the long bones (figure 1) or ischial rami, associated with irregular pubic symphysis. This was evident in bone scan (figure 2). Brown tumour was also seen in 1 patient who presented with a fracture (figure 3). Osteomalacia as a result of chronic use of medication was diagnosed upon screening in three (13%) adolescents with variable neurological disorders on different anticonvulsant

medication. Two (8.7%) female adolescents were diagnosed as being celiac disease histologically.

DISCUSSION

This is a retrospective study with its limitations being a hospital based study and represent a cohort from a referral rather than a community-based study. These patients are more likely to be under diagnosed. A high index of suspicion should be present as in the majority of adolescents with osteomalacia usually present with non-specific symptoms or signs like generalized bone pain, muscle weakness and fatigue. Thus, it is not uncommon for the diagnosis to be delayed or even missed. Mis-diagnosis of osteomalacia mimicking rheumatologic disease has been reported.^{8,9} Also, Al Otaibi et al¹⁰, reported osteomalacia in three girls who presented with proximal myopathy and seen initially by neurologist where EMG and nerve conduction were done.

In Saudi Arabia, inadequate vitamin D levels were detected in a population-based study, it was shown that vitamin D deficiency is common.¹¹⁻¹⁵ Similar to other studies,^{2,16} were found poor calcium intake, high consumptions of fast food and soft drinks, and minimum sun exposure. In our study, it was noted that in patients who developed osteomalacia were vitamin D deficient.^{2,12-14}

Special consideration should be given to adolescents receiving long-term medications, such as, anti-convulsant or glucocorticoids as these medications interfere with vitamin D metabolism and action by mechanisms that are still poorly understood.^{2,16,17} Celiac disease, due to intolerance to dietary gluten, can also be an important cause.¹⁸

Attention should be given to preventive measures through education and appropriate dietary supplements of Vitamin D and minerals starting from infancy.¹⁹ Sun exposure are to be encouraged through outdoor activities. Patients on anti-convulsant medication should be screened periodically for osteomalacia.

In conclusion, a high prevalence of osteomalacia in our adolescents population. Although nutritional deficiency is being the most common cause which indicates the importance of establishing a preventive measures programme, however, malabsorption. Such as coeliac disease, or intake of any drug interfering with vitamin D metabolism, such as anti-convulsant medications, should be seriously considered. Routine screening for coeliac disease a silent disease, and patients on long term anti-convulsant medications, should be routinely screened for secondary osteomalacia.

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REFERENCES

1. Klein G. Nutritional rickets In: Favus MJ ed. *Primer on the Metabolic Bone Diseases and Disorders of Mineral Metabolism*. Philadelphia : Lippincott Williams and Wilkins 1999;pp315-319.
2. Al Jurayyan NA, El Desouki ME, Al Herbish AS, Al Mazyad AS, Al Qhtani MM. Nutritional rickets and osteomalacia in school children and adolescents. *Saudi Med J* 2002;23(2):182-5.
3. Holick MF. Vitamin D deficiency. *N Engl J Med* 2007;357:266-281.
4. Bachard KL. Making an impact on pediatric bone health. *J Pediatr* 2000;136:137-139.
5. Gloth FM, Gundberg CM, Hollis BW, Haddad JG, Tobin JD. Vitamin D deficiency in home bound elderly person. *J Am Med Assoc* 1995;274:1683-1686.
6. Agarwal A, Gulati D. Early adolescent nutritional rickets. *J Orthopaedic Surgery* 2009;17(3):340-5.
7. European Society of Pediatric Gastroenterology and Nutrition (ESPGN). Revised criteria for diagnosis of celiac disease. Report of the working group ESPGN. *Arch Dis Child* 1990;65(8):909-911.
8. Demirbilek H, Aydogdu D, Ozon A. Vitamin D-deficient. Rickets mimicking ankylosing spondylitis in an adolescent girl. *Turkish J Pediatr* 2012;54:177-179.
9. Onur O, Celiker R, Cetin A, Alikasifoglu A, Ugur O, Basgoze O. Hypophosphalemic rickets with sacroilitis-like presentation in an adolescent. *Scand J Rheumatol* 1997;26:332-335.
10. Al Otaibi HM, Al Jurayyan NA, Mohamed S, Salih MA. Osteomalacia in adolescents presenting as proximal myopathy. *Curr Pediatr Res* 2012;16(1):57-60.
11. Sedrani SH. Are Saudis at risk of developing Vitamin D deficiency. *Saudi Med J* 1986;7:427-433.
12. El Idrissy ATH. Vitamin D deficiency. Rickets in a sunny country: pathogenesis, clinical picture and management. *Ann Saud Med* 1987;7:119-125.
13. Al Turki HA, Sedat-Ali M, Al-Elg AH, Al Mulhim FA, Al-Ali AK. 25-hydroxy vitamin D levels among healthy Saudi Arabian women. *Saudi Med J* 2008;29(12): 1765-68.
14. Abdullah MA, Salhi HS, Bakry LH, et al. Adolescent rickets in Saudi Arabia: a rich and sunny country. *J Pediatr Endocrinol Metab* 2002;15(7):1017-1025.
15. Thomas MK, Demay MB. Vitamin B deficiency and disorders of Vitamin D metabolism. *Endocrinol Metab Clin N Am* 2009;29:611-627.
16. Hazzazi MA, Al Zeer I, Tamimi W, Al Atawi M, Al Alwan I. Clinical presentation and etiology of osteomalacia / rickets in adolescents. *Saudi J Kidney Dis Transpl* 2013;24(5):938-41.
17. Tsukahara H, Kimura K, Todoroki Y, et al. Bone mineral status I ambulatory paediatric patients on long-term anti-epileptic drug therapy. *Pediatr Int* 2002;44:247-253.
18. Al Jurayyan AZN, Al Otaibi HM, Al Jurayyan RN, Al Assiri AM, Al Jurayyan NA. Coeliac disease presenting as rickets in children. *Paediatr Me* 2009;14(3):68-70.
19. Misra M, Pacaud D, Petryk A, Collett-Solberg PF, Kappy M. Vitamin D deficiency in childhood and it's management. Review of current knowledge and recommendations. *Pediatr* 2008;122:398-417.

Table 1: Aetiological diagnosis of osteomalacia in 23 patients

Diagnosis	No. of patients	%
Nutritional	17	73.9
Anti-convulsant medication induced	3	13.0
Celiac disease	2	8.7
Chronic renal failure	1	4.4
Total	23	100

Table 2: Clinical symptoms and signs in 23 adolescents with osteomalacia

Symptoms and signs	No. of patients	%
Bone pain and ache	15	65.2
Muscle weakness	6	26.1
Fracture	1	4.4

Legend to figure 1:

Antero-posterior view of the pelvis showing looser zones (Pseudofracture) (Arrow).



Legend to figure 2:

(A) anterior and (B) posterior views of Tc⁹⁹. Bone scan, demonstrating a high uptake of tracer throughout the skeleton “super scan”. (Multiple stress fractures)

**Legend to figure 3:**

Antero-posterior radiograph of the pelvis showing multiple lytic lesions (brown tumour).

