The Egyptian Journal of Hospital Medicine (Apr. 2017) Vol. 67(1), Page 491- 493

Rickets among Saudi Infants in Jeddah, Saudi Arabia

Ibrahim Hussain Muzaffar[1], Mashael Abdullah Abdulsubhan[2], Abdulrahman Mohammedsaeed Baqasi[1] Omar Badr Bawazir[1], Renad Mousa Aljohani[1], Yazeed Waleed O Alfadl[3], Mohammed Taha A Alnoor[3], Akram Abdulwahed G Atbay[3], Manar Ibrahim Y Sangoura[3]

Umm Alqura University[1], Batterjee Medical College[2], Ibn Sina National College[3]

Corresponding author: Ibrahim Hussain Muzaffar, Dr.Ibrahim.muzaffar@outlook.sa - 00966547771242

ABSTRACT

Background: In most developing countries, nutritional rickets is a major health problem. The aim of this study was to explore the magnitude of nutritional rickets among Saudi infants, and the various clinical presentations, as well as to address the possible operating risk factors behind the disease.

Methods: Cross sectional study has been conducted among Infants, aged 4 – 24 months, who were seen and evaluated, at King Abdulaziz Hospital and Oncology center, pediatrics endocrine clinic in Jeddah - Saudi Arabia, during the period from January 2015 to December 2016, and confirmed to have rickets were included.

Conclusion: Infants under 2 years of age are liable to have vitamin D deficiency and rickets, if they are exclusively breast fed and having inadequate exposure to sunlight. Delayed introduction of complementary foods and malnutrition are also important contributing factors in nutritional rickets. Vitamin D deficiency can present with variety of symptoms other than musculoskeletal symptoms like recurrent acute respiratory, diarrhea and seizures.

Keywords: Rickets, Vitamin D Deficiency, Saudi infants.

INTRODUCTION

Vitamin D plays an essential role in calcium homeostasis, normal development and maintenance of human bones, and enhancement of the immune system [1].

Rickets is a term signifying failure of mineralization of growing bone or osteoid tissue with characteristic changes of growth plate cartilage among infants before closure of growth plate [2]. Vitamin D deficiency rickets remains prevalent in developing countries and ranks among the five most common diseases in infants [3-4]. In the Kingdom of Saudi Arabia, despite having economic affluence and adequate sunlight all year round, vitamin D deficiency is fairly common in infants, infants, adolescents, as well as pregnant and lactating Saudi women [5-8]. It is common cultural practice to keep infants and young infants indoors with minimal or total avoidance of direct sunlight. Although there is an awareness of the high prevalence of vitamin D deficiency in Saudi Arabia, a review of the literature revealed a lack of data on the most common presentations.

Rickets typically presents in the 1st 18 months of life with hypotonia and short stature in addition to widening of the ends of long bones, growth retardation, skeletal deformities and delayed developmental milestones [9-11]. There are many causes of rickets; include inadequate dietary intake of calcium and vitamin D, malabsorption, inadequate exposure to sunlight, renal and liver diseases, and medications [12]. Other causes of rickets include calcium and phosphorous deficiencies, inherited forms of hypophosphatemic rickets and Vitamin D metabolism defects, including receptor mutations [13-17]. Patients with rickets usually develop secondary hyperparathyroidism and characteristic changes of the growth plates and metaphyseal bone, which include widening of wrists and ankles, bowing of the legs, craniotabes, ricketic rosary and Harrison sulcus [16-17].

The aim of this study was to explore the magnitude of vitamin-D deficiency rickets among Saudi infants, and the various clinical presentations, as well as to address the possible operating risk factors behind the disease and identify recommendations to prevent its occurrence.

SUBJECTS AND METHODS

169 Infants, aged 4 – 24 months, who were seen and evaluated, at King Abdulaziz Hospital and Oncology center, pediatrics

Received: 22 / 02 /2017
Accepted: 29 / 02 /2017

DOI : 10.12816/0036666

endocrine clinic in Jeddah - Saudi Arabia, during the period from January 2015 to December 2016, and confirmed to have rickets were included. The diagnosis was based on clinical, biochemical and radiological features as suggested: Data were reviewed and analyzed included age, sex, presenting symptoms and signs, housing, sun exposure, color of the skin, nutrition and medication intake, as well as careful physical examination. Laboratory investigations included complete blood count, renal, liver and bone profiles and serum concentrations of 25-OH-Vitmin D (25-OH-Vit D).

All patients were treated with oral vitamin D preparation, calcium and appropriate dietary restriction if needed. Diagnosis of inherited forms of rickets and conditions mimicking rickets were based on criteria suggested.

The study was done after approval of ethical board of King Abdulaziz Hospital and oncology center.

RESULTS

The age and sex distribution of infants, as well as their demographic character are summarized in Table 1. Out of the total 169 infants with the rickets, 74 (43.8%) were male and 95 (56.2%) were female. Mean age was 14±10 months with mean age of male infants at 12±5.5 months female at 13±8 months. The number of infants in the age group 4 – 14 months was 32 (18.9) and the age group 15 – 24 months 137 (81.1%). As regards skin color 31 (18.3%) were have tanned color were 102 (60.4%), with those belonging to the groups of light and dark skin being 31 (18.3%) and 36 (21.3%) respectively. Overall, 142 infants (84.1%) were exclusively on breast-feeding (73 males, 69 females) with no supplementation. In majority of the cases (97.6%) infants had sunlight exposure less than 30 min/week and only 4 (2.4%) had the required proper sun exposure.Rickets was more prevalent in families residing in apartments with limited or no sun exposure.

Table 1: demographic characters:

demographic character		Number (N=169)	Percentage
Age	4 - 14 months	32	18.9%
	15 – 24 months	137	81.1%
Sex	Male	74	43.8%
	Female	95	56.2%
Skin color	Light	31	18.3%
	Tanned	102	60.4%
	Dark	36	21.3%
Nutrition	Exclusive Breast feeding	142	84.1%
	Formula	66	39.1%
	Family diet	15	8.8%
Housing	Closed flat	162	95.9%
	Open house	7	4.1%
Sun exposure	less than 30 min/week	165	97.6%
	More than 30 min/week	4	2.4%

Main clinical presentation revealed gross motor delay for 94 (55.6%) cases, hypocalcemic convulsions in 12 (7.1%), acute gastroenteritis in 40 (23.7%) and pneumonia in 23 (13.6%) (Table 2).

Table 2: Clinical Presentations:

Clinical Presentation	Number (N=169)	Percentage
Gross motor delay	94	55.6%
Hypocalcemic convulsions	12	7.1%
Acute gastroenteritis	40	23.7%
Pneumonia	23	13.6%

The most common clinical signs were bowing of leg, wide wrist, and craniotabes (Table 3).

Table 3: Physical Examination:		
Physical Examination	**Number**	**Percentage**
Wide wrists	137	81.1%
Wide anterior fontanel	26	15.4%
Frontal bossing of skull	3	2.4%
Rachitcrosary	32	18.9%
Bowing of legs	151	89.3%
Kyphosis	4	2.4%
Craniotabes	141	83.4%
Caput Qaudratum	4	2.4%
Harrison's grove	11	6.5%

DISCUSSION

Presentation of rickets in the present study seems to be the tip of the iceberg as 169 cases with apparent signs of rickets were documented in a 2 year period at single hospital.

In the present study majority of the cases (81.1%) involved infants in the second year of life. Kreiter in USA [18] reported high prevalence of rickets in 5-25 month age. In Pakistan Khan *et al.* [19] reported that 74% of rachitic infants were aged below 12 months. The reason for increased incidence in this age group is the increased metabolic demand due to rapid growth. In the present study there was a male to female ratio of 1.1. A male predominance has been reported in previous studies conducted in Australia, Ethiopia, and Sydney [20-22] comparatively a Copenhagen based study depicted a large female predominance [23], whereas a 1:1 ratio was reported from Saudi Arabia [8]. The reason of high incidence of nutritional rickets in female sex is not clearly understood, but study by Siddiqui commented that Saudi females tend to have less sun exposure due to socio-cultural reasons and lack of an awareness of the importance of sun exposure for bone health as well as cosmetic reasons or because it is thought to be harmful. Additional research is required to identify the reasons for the disparity [21].

In the USA, Weisberg *et al*. reviewed the cases reported between 1980 and 2003 and concluded that osteomalacia and rickets are still prevalent in the US [24]. In a study from the UK conducted in 2002, Shaw reported that vitamin D deficiency in Asian families is a continuing problem [25]. Although the aforementioned studies are from countries that are prone to have a high prevalence of rickets and osteomalacia due to their limited sun exposure, there are many reports from countries with adequate or high sun exposure, like Saudi Arabia [26] and Australia [21]. These studies show that rickets and osteomalacia are still a major health burden. These studies show that rickets are still a major health burden. In Saudi Arabia, inadequate vitamin D levels were detected in a population-based study, and it was shown that vitamin D deficiency osteomalacia/ rickets is common [27-29].

Karrar found that most cases of rickets were asymptomatic and, if present, the symptoms were non-specific [29]. Al Jurayyan *et al.* [5] reported that the majority of patients in their study presented with nonspecific symptoms such as bone pain, which caused difficulty in making a clinical diagnosis in the less-severe cases. Similar findings were reported by Siddiqui, indicating that most patients presented with non-specific symptoms or were asymptomatic. Similarly, most of our patients presented with non-specific symptoms like bone pain or were asymptomatic.

Whereas most infants with rickets are easily recognized and treated, some can have lasting bone deformities or mild-to-severe neurological symptoms. Deficiency can easily be prevented by routinely supplementing infants with vitamin D.

CONCLUSION

Infants under 2 years of age are liable to have vitamin D deficiency rickets if they are exclusively breast fed and having inadequate exposure to sunlight. Delayed introduction of complementary foods and malnutrition are also important contributing factors in nutritional rickets. Vitamin D deficiency can present with variety of symptoms other than musculoskeletal symptoms like recurrent acute respiratory, diarrhea and seizures. These are under recognized features of vitamin D deficiency. It is therefore recommended that rickets should be investigated for patients reporting these complaints. It is also possible for clinical signs and symptoms to be present with no radiographic evidence of rickets.

RECOMMENDATIONS

Special attention should be given to preventive measures through education and appropriate dietary supplements of vitamin D

and minerals. Outdoor activities with direct or indirect exposure of sunlight are to be encouraged and supervised. We advise that all patients on either anticonvulsants or steroids be screened periodically for osteomalacia and, if they are at a high risk, should be started on vitamin D and calcium supplements as prophylaxis as Vitamin D deficiency rickets can result in significant infant morbidity and mortality.

REFERENCES

1. **Wedad Z Mostafa, Rehab A Hegazy (2014):** Vitamin D and the skin: Focus on a complex relationship: A review. http://doi.org/10.1016/
2. **Pitt MJ (1991):** Rickets and osteomalacia are still around. Radiol Clin North Am., 29: 97–118.
3. **T R Welch, W H Bergstrom, R C Tsang (2000):** Vitamin D-deficient rickets: the reemergence of a once-conquered disease. J Pediatrics, 137 (2): 143–145.
4. **C L Wagner, F R Gree (2008):** Prevention of rickets and vitamin D deficiency in infants, infants, and adolescents. American Academy of Pediatrics Section on Breastfeeding; American Academy of Pediatrics Committee on Nutrition. Pediatrics,122(5):1142-1152.
5. **Al-Jurayyan NA, El Desouki ME, Al-Herbish AS, Al-Mazyaz AS, Al Qthani MM (2002)**: Nutritional rickets and osteomalacia in school infants and adolescent.Saudi Med J., 23(2): 182-5.
6. **Narchi H, El-Jamil M, Kulyalat N (2001)**: Symptomatic rickets in adolescent. Arch Dis Child, 84(6):501-3.
7. **Sedrani SH, Al-Arabi K, Abanmy A, Elidrissy A (1992):** Are Saudi infants at risk of developing vitamin D deficiency rickets? Saudi Med J.,13:430–3.
8. **Erfan AA, Nafie OA, Neyaz AH, Hassanein MA (1997):** Vitamin D deficiency rickets in maternity and infants's hospital, Makkah, Saudi Arabia. Ann Saudi Med.,17(3):371-373.
9. **Thacher TD, Fischer PR, Strand MA, Pettifor JM (2006)**: Nutritional rickets around the world: causes and future directions. Ann Trop Paediatr, 26:1–16.
10. **Wagner CL, Greer FR (2008)**: Prevention of rickets and vitamin D deficiency in infants, infants, and adolescents. Pediatrics,122:1142-52.
11. **Nield LS, Mahajan P, Joshi A, Kamat D (2006):** Rickets:Not a Disease of the past. Am Fam Physician,74:619-26.
12. **Cashman KD. (2007):** Vitamin D in childhood and adolescence. Postgrad Med J.,83:230-5.
13. **Pfitzner M, Thacher T, Pettifor J, Zoakah A, Lawsan T (1998).** Absence of Vitamin D deficiency in young Nigerian. J Pediatr.,133:740-744.
14. **Fischer PR, Rahman A, cimma JP, Kyaw-Myint TO, Kabir AR, Talukder K (1999):** Nutritional rickets without Vitamin D deficiency in Bangladesh. J Trop Paediatr.,55:291-293.
15. **Tacher T, Glew RH, Isichei CI, Lawaon JO, Scariano JK, Hollis BW (1999)** Rickets in Nigerian infants: response to calcium supplementation. J Trop Paediatr.,45:202-207.
16. **Thomas MK, Demay MB (2000):** Vitamin D deficiency and disorders of Vitamin D metabolism. EndocrinolMetabClin N Am.,29:611-627.
17. **Miller WL, Anthony AP (1999):** Genetic disorders of Vitamin D biosynthesis. EndocrinolMetbClin N Am.,28:825-840.
18. **Kreiter SR, Schwartz RP, Kirkman HN Jr, Charlton PA, Calikoglu AS, Davenport ML (2000)**: Nutritional rickets in African American breast fed infants. J Pediatr.,137:153-157.
19. **Khan HI, Abdullah A, Kazi MY, Afzal MF (2006)**: Hypocalcemia and nutritional rickets in infants: Common etiological factors. Ann King Edward Med Coll., 12:29-32
20. **Lulseged S (1990)**: Severe rickets in a infants's hospital in Addis Ababa. Ethiop Med J.,28:175
21. **Robinson PD, Högler W, Craig ME, Verge CF, Walker JL, Piper AC,*et al*. (2006)**: The re-emerging burden of rickets: a decade of experience from Sydney. Arch Dis Child, 91;564-568.
22. **Nozza JM, Rodda CP (2001)**: Vitamin D deficiency in mothers of infants with rickets. Med J Aust,175:253
23. **Pedersen P, Michaelsen KF, Molgaard C (2003)**: Infants with nutritional rickets referred to hospitals in Copenhagen during a 10-year period. ActaPaediatr., 92:87–90.
24. **Weisberg P, Scanlon KS, Li R, Cogswell ME (2004)**: Nutritional rickets among infants in the United States: Review of cases reported between 1986 and 2003. Am J Clin Nutr.,80(6):1697-705S.
25. **Shaw NJ, Pal BR (2002)**: Vitamin D deficiency in UK Asian families: Activating a new concern. Arch Dis Child, 86:147-9.
26. **Al-Mustafa ZH, Al-Madan M, Al-Majid HJ, Al-Muslem S, Al-Ateeq S, Al-Ali AK (2007)**: Vitamin D deficiency and rickets in the eastern province of Saudi Arabia. Ann Trop Paediatr., 27:63-7.
27. **Sedrani SH, Al-Arabi K, Abanmy A, Elidrissy A (1992)**: Vitamin D status of Saudis: I. Effect of age, sex and living accommodation. Saudi Med J., 13: 1510158.
28. **Sedrani SH. Vitamin D status of Saudis: III (1992):** Prevalence of inadequate plasma 25 hydroxy-vitamin d concentrations. Saudi Med J., 13: 214-9.
29. **Karrar ZA (1998)**: Vitamin D deficiency rickets in developing countries. Ann Trop Paediatr., 18 (l):S89-92.