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Rickets

Is nutritional rickets returning?

J Allgrove

See original article by Ladhani *et al* (pp 781–4)

Mineralisation of osteoid tissue of bone is dependent on a suitable supply of mineral, both calcium and phosphate, to that tissue. Failure to provide sufficient mineral results in osteomalacia which, in growing bone with its attendant growth plates and unfused epiphyses, is manifest as rickets. Although vitamin D deficiency is not the only cause of rickets (nutritional calcium deficiency has also recently been proposed as an important factor[1]), it has historically been a major cause of morbidity.[2]

Vitamin D is mainly derived from the action of sunlight on 7-dehydrocholesterol in the skin, and vitamin D deficiency is more likely to occur in individuals with darker skins or in those whose skin is extensively covered.[3] Vitamin D is converted via two enzymatic reactions to the active metabolite, 1,25-dihydroxyvitamin D (1,25-$(OH)_2D$), whose principal actions are to stimulate absorption of calcium by the gut and promote mineralisation of bone. Failure to do so adequately may therefore result in osteomalacia and rickets; it also limits peak bone mass[4] and contributes to involutional osteoporosis.[5] Vitamin D deficiency in pregnant mothers also limits fetal growth.[6] In addition, vitamin D may have an important part to play in preventing other diseases such as hypertension, various cancers, and type 1 diabetes.[5]

Rickets is not a new disease. It was first described by an English physician, Dr Daniel Whistler, in 1645, although it is known to have existed well before that.[7] It has long been recognised that lack of exposure to adequate amounts of sunlight was a major cause of vitamin D deficiency rickets which, following the industrial revolution, became a significant cause of morbidity especially in the urbanised cities of the north of England and Scotland. As the causes of so called ''nutritional'' rickets became better understood, and particularly following the second world war when the privations of food rationing necessitated a closer examination of minimal nutritional requirements, nutritional rickets virtually disappeared in the UK, particularly with the introduction of vitamin D supplementation in the form of cod or halibut liver oil and the fortification of foods.

Rickets is a relatively rare condition in western societies and, when it presents, is often associated with some form of metabolic disturbance of vitamin D metabolism, which may be primary (for example, vitamin D dependent rickets type 1, an inborn error of metabolism of vitamin D; or vitamin D dependent rickets type 2, end organ resistance to the action of vitamin D) or secondary (for example, associated with liver or kidney disease), or a defect in renal tubular function (for example, hypophosphataemic vitamin D resistance rickets).[8] However, over the past few years several reports have appeared which have suggested that nutritional rickets is reappearing in the UK,[9–11] Europe,[12–20] and North America[21–26] in a variety of ethnic groups. Other reports have suggested, perhaps surprisingly, that rickets is also prevalent in sunnier climes,[13 27–33] although here it seems that malnutrition may be a contributing factor[34] in some instances.

Most, though not all, of the nutritional rickets that occurs in the UK is seen in patients of black or Asian origin and it is well recognised that the darker skin colour of these ethnic groups is a contributing factor to the high incidence of nutritional vitamin D deficiency. Darker skinned individuals are just as capable as those with lighter skins of synthesising vitamin D, but require greater exposure to ultraviolet light to do so.[3] Other factors, such as nutritional deficiency associated with macrobiotic,[12 35] vegetarian,[16 36 37] strict vegan,[38] or ''health food'' milk alternatives[39] diets, and low exposure to sunlight, either by staying indoors or covering the skin are also important.[40] Breast milk contains little vitamin D and breast fed infants should receive vitamin D supplements.[41]

Vitamin D deficiency classically presents with symptoms of bony deformity such as bowed legs, swelling of the wrists, a ''rickety rosary'', and muscle weakness. If the rickets is severe enough, fractures may ensue[24 42] and may simulate child abuse.[43] However, a significant proportion of these patients have symptoms of hypocalcaemia which may cause convulsions, stridor, and neuromuscular irritability.[33 44–46] In addition, surveys of at risk populations have shown that a significant proportion have subclinical vitamin D deficiency.[47] Radiological evidence of rickets is not always present in these patients[33 46] and this can lead to diagnostic confusion, such as with hypoparathyroidism or pseudohypoparathyroidism, unless the possibility of vitamin D deficiency is considered.[48 49] For instance, we describe in this issue 65 cases of vitamin D deficiency in northeast and southeast London.[46] Forty five per cent (n = 29) of these had hypocalcaemic symptoms, of whom 55% (n = 17), more than a quarter of the total, had no radiological evidence of rickets. These patients were all either under the age of 2 years or in adolescence and we speculate that this is because growth is most rapid at these ages when demand for calcium by bone is so high that hypocalcaemia develops before rickets can ensue. Vitamin D deficiency must therefore always be excluded before the cause of hypocalcaemia can be defined.

Treatment of vitamin D deficiency is usually straightforward and consists of oral supplementation with vitamin D. Shaw and Pal[50] recommend 3000 units daily for infants under 6 months and 6000 units for older children. We have found that half this dose is sufficient. However, we have also seen a number of infants who have presented with vitamin D deficiency despite being fed with formula milk preparations, which are fortified with vitamin D, or who have been given vitamin supplements in recommended amounts (400 units daily). It seems that the mothers of these infants are also vitamin D deficient[14 16 51 52] and that, while 400 units daily of vitamin D is sufficient to prevent deficiency in replete individuals, this may be insufficient to correct deficiency if this is already present at birth. Nevertheless, oral supplementation with vitamin D is an effective way

of both preventing and treating vitamin D deficiency.

An alternative, equally effective, treatment strategy is to use stosstherapy, a single large dose of vitamin D (300 000 to 600 000 units) as a single dose given either orally or parenterally.[53] This is particularly useful if there are concerns about compliance with treatment. Alphacalcidol, a synthetic analogue of vitamin D which is converted to 1,25-$(OH)_2D$, has no part to play in the treatment of nutritional vitamin D deficiency as large doses have to be given initially to mimic the supraphysiological rise in 1,25-$(OH)_2D$ which occurs naturally at the start of treatment with vitamin D.[13 54] Not only does a poor response often result from the use of ''physiological'' doses, but it also does not address the underlying issue of vitamin D deficiency. If hypocalcaemia is the presenting symptom, initial treatment with calcium supplementation, by intravenous infusion if necessary, will be required, and oral calcium supplements in addition to vitamin D increases the rate of recovery.[55]

Nutritional rickets has always been with us but, following the recognition of its principal cause, was largely eradicated in Western society. Large scale immigration from the West Indies and Indian subcontinent resulted in its reappearance, but systematic campaigns of vitamin D supplementation, such as the Glasgow Rickets Campaign of the 1970s, were successful in reducing its incidence. The initial phase of this, a campaign of voluntary free supplementation accompanied by a health education programme aimed mainly at community health workers and the Asian community,[56] was successful in reducing the number of admissions to hospital with symptoms of vitamin D deficiency, which had been increasing before 1973.[57] In 1979 an official campaign was launched to supplement all children at risk, and this produced a significant reduction in hospital admissions with rickets.[58]

However, there is now good evidence to suggest that, since such campaigns have been relaxed, a third wave resurgence of vitamin D deficiency is now being seen.[59–62] Some of this increase may be related to the recognition that vitamin D deficiency may cause problems other than rickets but it is, nevertheless, a serious indictment of community services that this is happening. Little is known of the long term effects of vitamin D deficiency on bone development, but it is probably one of the factors which contributes to ''involutional'' osteoporosis in later life.[5] In addition, vitamin D deficiency may have an important part to play in other diseases of adults. Since this is a common condition with a high morbidity, it is incumbent on us as paediatricians to try to minimise it as much as possible.

There is good agreement that vitamin D deficiency can be prevented by dietary supplementation, but there is disagreement about how much should be given to which individuals. The American Association of Pediatrics has recently recommended that a reduced dose of 200 units be given to all infants, children, and adolescents.[63] In the UK the Department of Health recommends 400 units for pregnant and lactating women, 340 units for infants under 6 months, and 280 units for children under 4 years. No recommendations are given for older children,[64] but the importance of ensuring that vulnerable groups—that is, infants, young children, and pregnant women from Asian families as well as young African-Caribbean children being reared on strict exclusion diets, receive these supplements is stressed. In practice, both of the vitamin preparations that are available free of charge in the UK provide adequate amounts of vitamin D and can be provided by general practitioners and health visitors. There is probably no place for universal vitamin D supplementation because of the potential dangers of hypercalcaemia in a few individuals.

It seems, therefore, that nutritional rickets is not returning, but has always continued to be with us. It is increasing in frequency again, partly because it is being recognised in its different manifestations but mainly because vitamin D supplementation is no longer regarded as essential for ''at risk'' individuals. It is almost certainly the case that the clinically apparent cases of vitamin D deficiency represent the ''tip of the iceberg''. Unfortunately, this is one iceberg which, despite global warming and the attendant increase in sunshine, is not diminishing in size. The time has come to mount a national campaign to promote awareness of the risks of vitamin D deficiency, particularly among susceptible populations in the UK, and to eliminate it as a cause of morbidity.

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Substance abuse

Substance abuse by children and young people

P McArdle

A contemporary disease

Use of illicit substances by significant numbers of young people has been of concern for at least a generation in Western Europe and North America.[1] This is reflected in official statistics, police sources and surveys, and particularly school based anonymous self-reports that have shown a substantial increase in consumption over that time with a further surge during the 1990s.[1] In addition, the rates of substance use (alcohol and drugs) in the UK currently outstrip those reported elsewhere in Europe.[2] Furthermore, there is now evidence of illicit substance use by significant numbers of pre-adolescent UK children;[3] up to 5% of preteens currently report use of illicit substances and an appreciable number hard drugs such as heroin; apparently an entirely new development.

Despite this exposure to toxic substances, children and young people are not referred in large numbers to health services as a consequence primarily of substance related disorders. In part this is because they present in other ways: through intoxication, accidental or violent injury, self-harm, sexually transmitted disease, teenage pregnancy, and psychiatric disorder. It may be too that traditional services are unprepared for them or for adapting existing skills and resources to attempt to recognise or meet their needs.

A further problem concerns confusion of concepts and terminology. For instance, the WHO has identified ''disorders due to psychoactive substance use'' as ''intoxication'', ''dependence syndrome'', and ''harmful use''. However, because they are in general likely to have been using substances for a relatively short time, dependence among young people is probably less common than among adults. Also, the definition of ''harmful use'' specifically excludes ''socially negative consequences'', an important type of harm for developing children and youth. DSM IV, the classification system of the American Psychiatric Association, describes a range of ''substance related disorders'', including ''substance use disorders'', of which ''substance abuse'' is a subcategory. This is characterised by ''a maladaptive pattern of substance use manifested by recurrent and significant adverse consequences related to the repeated use of substances''. This includes:

> ''… failure to fulfil major role obligations at work, school or home … recurrent … legal problems (and) repeated substance use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the substance …''

In addition, a large US survey[4] has reported that among 15–16 year olds who had used illicit drugs more than five times (''… two thirds of those who had ever used''), almost 80% of boys and 70% of girls had been ''drugged or high'' at school and over half had been