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Vitamin D deficiency rickets due to soybean milk

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Abstract: We describe vitamin D deficiency rickets in an infant nursed with soybean milk (not specifically designed for infants) instead of cow's milk-based infant formula. Symptoms included irritability and carpopedal spasm when crying. The infant's condition improved with nutrition by vitamin-enriched cow's milk-based infant formula, oral administration 1 alpha-hydroxy vitamin D3 and exposure to sunlight. Content analysis of the milk showed very low calcium, phosphate, magnesium and vitamin D levels compared to cow's milk-based infant milk formulas. This case highlights the unsuitability of soybean milk as the sole provider of infant nutrition and demonstrates the false perception that soybean milk is a healthy food for infants. It is necessary to be cautious about not only health claims for soybean milk, but also today's health and natural food booms. Social enlightenment and correction of such misperceptions are necessary.

Key words: atopic dermatitis; breast-feeding; soybean milk; sunlight exposure; vitamin D deficiency rickets.

Rickets due to vitamin D deficiency is now a rare nutritional disorder,[1] but rickets can develop even in industrial countries.[2] Recently reported etiologies for rickets are unusual dietary practices, extreme vegetarianism and lack of sunlight exposure.[2] In the present report, we describe a Japanese infant with rickets who received only soybean milk that is not specifically designed for infants as daily nutrition. The infant also lacked sunlight exposure.

CASE REPORT

This 5-month-old boy was referred to our hospital because of stiffness of limbs. He was born at 38 weeks gestation, delivered after an uncomplicated pregnancy, weighing 2215 g. Apgar scores were 9 and 10 at 1 and 5 minutes, respectively. His family had no history of allergies or other diseases. He had been fed soybean milk not designed for infants instead of cow's milk-based infant formula from 4 weeks after birth. The infant was diagnosed at 3 weeks with infantile eczema, which the parents had interpreted as being due to the cow's milk-based formula he had been receiving. His parents believed soybean milk to be healthy. The milk was not labelled as suitable or unsuitable for infants. In fact, his parents had been addicted to the soybean milk, and they had consumed as much as one litre per day. In addition the baby had not been exposed to sunlight as his family believed sunlight was bad for his skin.

On admission, he was of small physique, being 62.5 cm in height (–1.5 SD) and 5.6 kg in weight (–2.2 SD). He was hypersensitive and irritable to the surroundings. Carpopedal spasm appeared, especially when he was crying. Deep tendon reflexes were accelerated in both upper and lower limbs. He did not have craniotabes or rachitic rosary. He was pale with truncal eczema. Blood examination showed decreased serum level of calcium and phosphate, 5.3 mg/dL and 5.0 mg/dL, respectively. Serum level alkaline phosphatase (ALP) and lactic acid dehydrogenase (LDH) were increased at 2303 IU/L and 1359 IU/L, respectively. Creatine phosphokinase (CK) was increased at 2805 IU/L. Endocrinological investigation showed reduced 1,25 dihydoxy vitamine D3 (11.6 pg/mL) and 25 hydroxy vitamine D3 (8 ng/mL) and increased parathyroid hormone (PTH) at 2400 pg/mL. X-ray revealed cupping and fraying deformations in the epiphysis of radial, ulnar, tibial and fibral bones. After intravenous administration of 8.5% calcium gluconate, the tetanic posture soon disappeared, and irritability and hypersensitivity vanished. Oral administration of 1α-hydroxyvitamin D3 syrup 0.03 μg/kg per day was subsequently started, and daily exposure to sunlight was undertaken. The infant was commenced on a Vitamin D-enriched cow's milk-based infant formula.

Content analysis of the soybean milk showed a markedly low level of calcium (28.9 mg/dL), and undetectable levels of both magnesium and vitamin D. Two months after the admission, the serum levels of ALP, PTH, 1, 25-dihydroxyvitamin D3 and 25-hydroxyvitamin D3 were normal and oral administration of 1α-hydroxyvitamin D3 syrup was ceased. Bone X-ray findings with the cupping and fraying were not seen 2 months after the administration of vitamin supplementation. Daily sunlight exposure was encouraged at the outpatient clinic. Two years later his height and weight have shown catch-up increase (to –0.4 SD and + 0.1 SD, respectively) and his developmental progress is normal.

DISCUSSION

Up to the early part of the 20th century, rickets was one of the most commonly encountered disorders in children and adults. In the periods of industrialization, deficiency of vitamin D readily developed as results of insufficient exposure to sunlight, as well as malnutrition due to poverty.[3] In the late 1920s, formulas for infants were supplemented with vitamin D, and the incidence of rickets became markedly lower.[4] However, this nutritional disorder is still seen even in industrialized countries.[1] According to Nishikura *et al.*[2] who reviewed paediatric etiologies of rickets in Japan, 67% of the cases were related to the mother's inappropriate diet[5–7] and 80% were due to insufficient exposure to sunlight.[8] Another possible etiology is prolonged breast feeding without weaning.[1,4]

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Table 1 Standard table of infant formula and soybean milk in Japan[9]

	Energy	Calcium	Phosphate	Magnesium	Vitamin D
Infant formula	514 kCal	370 mg	220 mg	40 mg	9μg
Soybean milk	46 kCal	15 mg	49 mg	25 mg	0μg

(rate/100 g)

In this case, the parents gave the soybean milk as the sole food to their infant as they believed soybean milk to be healthy and they believed his atopic dermatitis was caused by the cow's milk-based formula he had been receiving. This case shows that the exclusive use of 'natural health foods' not specifically designed for infants may result in severe nutritional deficiencies. The exclusive intake of soybean milk (not soybean milk infant formula) and minimal sunlight exposure was responsible for the rickets and hypocalcemic tetany in this infant.

The vitamin D level in soybean milk that is not specifically designed for infants distributed in Japan is nil, and the level of calcium, phosphate and magnesium is also low (Table 1).[9] It is clear that the exclusive intake of soybean milk, as well as little exposure to sunlight is the most plausible etiology for rickets and hypocalcemia in this patient. To our knowledge, only one case of infant rickets due to the exclusive intake of soybean milk has been published in Japanese, and none in English literature. Tamai *et al.*[10] reported a 4-month-old girl with hypocalcemia and tetany due to intake of home-made soybean milk. The patient improved soon after changing from soybean milk to regular formula, which was supplemented with calcium and vitamin D.

This case demonstrates the risk of preventable nutritional deficiencies from feeding infants inappropriate milk. Soybean milk is viewed as a natural health food, but it is important for the community to be aware that it is not a suitable infant milk without modification.

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