Effect of vitamin D administration in vitamin D-deficient pregnant women on maternal and neonatal serum calcium and vitamin D concentrations: a randomised clinical trial

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Abstract

There are several studies in which a correlation between maternal vitamin D deficiency and serum mineral disorders in the mother and the newborn has been reported. The present randomised clinical trial was designed to investigate the effect of vitamin D administration on maternal and fetal Ca and vitamin D status. The trial was carried out on 160 pregnant women. Vitamin D-deficient (25-hydroxyvitamin D (25(OH)D) < 30 ng/ml) pregnant women were recruited at 26–28 weeks of pregnancy. In the control group, a multivitamin supplement containing 400 IU vitamin D₃/d was given. Patients in the treatment group were treated with 50 000 IU vitamin D₃ weekly for a total duration of 8 weeks. At delivery, maternal and fetal Ca and 25(OH)D levels in both groups were compared. In total, 81% of pregnant women were vitamin D deficient. At the time of delivery, Ca and vitamin D levels were higher in the treatment group compared with the control group (92 (sD 3) *v*. 85 (sD 4) mg/l, respectively, *P*=0.001 for serum Ca; 47.8 (sD 11·1) *v*. 15.9 (sD 6·6) ng/ml, respectively, *P*<0.001 for vitamin D). At the time of delivery, 32.7% of women in the control group had hypocalcaemia, while no hypocalcaemic case was detected in the vitamin D-treated group. Mean neonatal serum 25(OH)D was higher in the treatment group compared with the control group (27.7 (sD 5·2) *v*.10·9 (sD 4·4) ng/ml, respectively, *P*<0.01). The neonatal Ca level in the treatment group was significantly higher than that of the control group (99 (sD 3) *v*. 91 (sD 3) mg/l, respectively, *P*<0.001). The administration of vitamin D to pregnant women were with vitamin D to pregnant women with vitamin D deficiency improves both maternal and neonatal Ca levels.

Key words: Vitamin D deficiency: Pregnancy: Hypocalcaemia

Vitamin D is an essential substance for maintaining the natural bone architecture. Vitamin D deficiency can lead to complications including osteomalacia and secondary osteoporosis⁽¹⁾. During pregnancy, considerable amounts of Ca are transferred from the mother to the fetus, and the preparation of such supply of Ca is mainly achieved by the regulation of increased intestinal Ca absorption. Moreover, during pregnancy, the concentration of 1,25-dihydroxyvitamin D increases two to three times above the normal range mainly because of the increase in renal 1- α -hydroxylase activity⁽²⁾. Theoretically, vitamin D deficiency in mothers could lead to a low production of 1,25-dihydroxyvitamin D due to substrate reduction. Maternal 1,25-dihydroxyvitamin D is transported to the fetus through the placenta, and the cord blood vitamin D level is equal to or at most 25% lower than the maternal concentration⁽³⁾.

There are only a few foods that are good sources of vitamin D, and in countries with no food fortification industries, the generation of vitamin D in the skin is the major source of vitamin D in the body. However, many studies have reported high frequencies of vitamin D deficiency in sunny countries^(4–7).

Vitamin D deficiency is highly common in Iran, and based on several reports, up to 80% of women living in Tehran suffer from mild to severe vitamin D deficiency⁽⁸⁾. Studies on pregnant women have also shown a high prevalence of vitamin D deficiency, particularly in developing countries including Iran^(9–13). Maternal vitamin D deficiency has several disadvantages to fetal outcomes. In cross-sectional studies, a correlation of maternal 25-hydroxyvitamin D (25(OH)D) levels with fetal femur volume⁽¹⁴⁾ and newborn weight⁽¹⁵⁾ has been reported.

Abbreviation: 25(OH)D, 25-hydroxyvitamin D.

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