Evaluation of vitamin D levels in patients with acute rheumatic fever

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Acute rheumatic fever (ARF) is a nonsuppurative complication of pharyngitis caused by group A beta-hemolytic streptococcus (GAS) whose underlying pathological mechanisms have not yet been fully elucidated (1). Vitamin D deficiency is a global health problem, particularly in the pediatric age group. Some immunomodulator and anti-inflammatory effects of vitamin D on various infectious and autoimmune diseases have also been reported. In limited number of pediatric studies low serum 25(OH)D levels have been reported in children with autoimmune diseases (2). Incidences of GAS infection and ARF make a seasonal peak at winter and spring months when 25(OH)D levels are expected to be lowest because of the lack of exposure to sunlight (3, 4).

In addition to the high risk of developing ARF, children in the age group of 5–15 years are growing up rapidly, with increased vitamin D requirement. In present study, we evaluated the 25(OH)D levels in children with ARF who had lower 25(OH)D levels. The results of present study suggested that vitamin D deficiency (6). Echocardiographic findings of ARF cases was assessed as absence of valvulitis, presence of mild, moderate, and severe regurgitation either in mitral or aortic valve. Carditis was classified into mild, moderate, and severe (7). Data was analyzed using SPSS software (version 16.0; SPSS Inc., Chicago, IL). There was no statistically significant differences between the groups in terms of age, sex, and BMI z-score (Table 1). None of subject from study group and controls had a BMI z-score (>2 SD) suggesting obesity. Distribution of the diagnostic criteria for ARF patients are shown in Table 2. Serum 25(OH)D levels were significantly lower in the study group than in the control group (14.56±8.31 ng/mL vs. 25.41±1.38 ng/mL, p=0.002). When considering 20 ng/mL as the cutoff for vitamin D deficiency, 23 out of 30 patients with ARF (77%) and 8 out of 16 controls (50%) had vitamin D deficiency (p=0.066). Thirteen of 29 patients with carditis (44.8%) had single valve involvement, 16 (55.2%) had both mitral and aortic valve involvement. Although there was no statistically significant difference among 25(OH)D levels of patients with mild (n: 20) (14.4±7.9 ng/mL), moderate (n: 5) (13.1±5.1 ng/mL), and severe (n: 4) (15.4±14.6 ng/mL) carditis (p=0.335), 25(OH)D levels of patients with carditis was lower than those with no carditis, including controls (25.1±13.5 ng/mL; p=0.054). The underlying biochemical and immunological mechanisms triggering ARF following GAS pharyngitis have not been fully elucidated (1, 8). It may result from endothelial injury in genetically predisposed subjects, which causes molecular mimicry and subsequent activation of cellular and humoral immune systems (1). Furthermore, vitamin D has also been defined as an immunomodulator agent decreasing T helper 1-mediated autoimmune response and symptoms of autoimmune diseases (9). To the best of our knowledge, this is the first study evaluating the vitamin D status in children with ARF who had lower 25(OH)D levels. The results of present study suggested the potential role of inflammatory and/or immune-mediated mech-
There is a lack of food fortification and vitamin D replacement for the spring season when the risk of vitamin D deficiency is highest, consistent with increased frequency of ARF during winter and controls were 77% and 50%, respectively. These findings were limits sunlight exposure, non-white ethnicity, older age, puberty, obesity (10). Vitamin D deficiency detected in our study group mechanisms associated with vitamin D deficiency in ARF pathogenesis. However, vitamin D deficiency was not correlated with carditis severity. The risk factors for vitamin D deficiency in pediatric population include winter season, lack of outdoor activities, which limits sunlight exposure, non-white ethnicity, older age, puberty, and obesity (10). Vitamin D deficiency detected in our study group and controls were 77% and 50%, respectively. These findings were consistent with increased frequency of ARF during winter and spring seasons when the risk of vitamin D deficiency is highest. There is lack of food fortification and vitamin D replacement for the age group between 5 and 15 years in which ARF incidence is highest. In addition, the overlap of seasons with highest incidence of vitamin D deficiency and ARF may support our hypothesis stating that vitamin D deficiency may predispose to development of ARF. In conclusion, vitamin D deficiency thereby decrease in its immunomodulator effect may trigger immune response and predispose developing ARF. Studies evaluating larger number of ARF patients with and without carditis are required to further elucidate the role of vitamin D deficiency in the pathogenesis of ARF and its impact on the disease phenotype.

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References