Serum Vitamin D Level and Disease Activity in Pediatric Inflammatory Bowel Disease

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Vitamin D deficiency is common in children and adults with Inflammatory Bowel Disease (IBD). Laboratory studies have shown vitamin D exerts potent anti-inflammatory effects and pre-clinical studies have suggested vitamin D deficiency may promote more severe mucosal inflammation. As such, patients with vitamin D deficiency may be more likely to experience more severe clinical disease activity. Our **Aims** were to identify determinants of vitamin D deficiency in pediatric IBD patients and to test for an association with clinical disease activity.

Methods: Subjects with IBD were recruited and serum and diet records were collected through a Crohn's and Colitis Foundation of America funded research project ["Immuno-genetic determinants of linear growth in pediatric IBD"], an ongoing, prospective cohort study of 200 patients at Cincinnati and Toronto. Three-day diet diaries were used to estimate vitamin D intake. The Pediatric Crohn's Disease (Ulcerative Colitis) Activity Index (PCDAI/PUCAI) measures were used to calculate disease activity. Winter was defined as November through February. Serum 25-OH D and Parathyroid Hormone (PTH) were measured by the Cincinnati Children's Hospital GCRC using a commercially available RIA kit; vitamin D deficiency was defined as ≤ 15 ng/mL. Serum Lipopolysaccharide Binding Protein (LBP) was measured using a commercially available ELISA kit. Serum albumin was measured by a clinical lab at the two sites.

Results: 284 25-OH D measurements were obtained from 184 subjects. Of these measurements, 21% were vitamin D deficient. There were no significant differences in serum albumin, PTH, or LBP between the vitamin D deficient and non-deficient groups. However, vitamin D intake was significantly lower in the deficient group (median(IQR): 81(39,177) IU/day vs. 202 (98,354) IU/day, $p < 0.01$). There was also a modest increase in the frequency of vitamin D deficiency during the winter. Within the vitamin D deficient group, 28% had moderate to severe disease activity, compared to 7% in the non-deficient group ($p = 0.001$).

Conclusions: Vitamin D deficiency is strongly associated with reduced intake and moderate to severe disease activity. Better strategies are needed for correcting vitamin D deficiency in pediatric IBD patients. Further study is warranted on whether supplementation of vitamin D would improve the clinical course of IBD.

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