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Short Communication

Development of protein interaction network with vitamin d deficiency in children's and its correlation with dental caries: A bioinformatics approach

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Dental caries in children's remains a significant chronic disease which is affecting up to 41% of 5-year-old children's in India. There is a known association between dental caries and vitamin D deficiency, but this has not been investigated in details. Vitamin D deficiency is the major public health concern, despite being preventable with providing the supplementation. Molecular level understanding of differential protein expression and their interactions in vitamin D deficiency children's reported by scientists will be very beneficial for understanding the role of vitamin D and the problem of dental caries. An interdisciplinary approach is needed to integrate the complex web of molecular information with clinical information, particularly for diseases where the diagnosis is based primarily on clinical findings. Recently, bioinformatics has made it possible to make specialized databases and apply the information for better research. In the present study we have taken the proteins which were up regulated or down regulated in the children's with vitamin D deficiencies and have constructed the network using string software and have observed that active vitamin D3 (1, 25-dihydroxy vitamin D3) interacts with three proteins: vitamin D binding protein (DBP), vitamin D receptor (VDR), and CYP24A1. These results exhibit the positive correlation of vitamin D deficiency and dental caries in children's, further research is underway. As survival rates after hematopoietic cell transplantation (HCT) have risen, attention has increasingly turned toward important aspects of health, including nutritional and metabolic outcomes. Low bone mineral density (BMD) has been reported among long-term survivors of HCT. Children also have a high prevalence of asymptomatic fractures after HCT. Children treated for leukaemia that experience fractures have lower BMD than those without fractures. In adult survivors of childhood acute leukaemia, HCT increased the risk for low BMD compared to chemotherapy alone, when data were adjusted for diagnosis, age at diagnosis, follow-up duration, and corticosteroid use. Since low BMD is likely linked to the development of fractures in children, the timing and duration of bone loss following HCT in children maybe an important consideration in the development of appropriate treatment or preventive interventions. Vitamin D deficiency has been associated with low bone mass and osteoporosis in adult patients after HCT, and is also common in pediatric HCT recipients. While the prevalence of vitamin D deficiency among long-term survivors of pediatric cancer appears to be similar to that of the general population, changes that occur in the immediate post HCT period may coincide with bone loss in this population. In a prospective study of children undergoing HCT, mean loss of BMD was 5% six months after HCT, and in half of the subjects, bone density losses continued through one year after HCT. Acute changes in BMD and biomarkers associated with bone health, such as vitamin D, in the first months after HCT are unknown. Prior studies have not reported bone morbidities at 30 and 100 days after pediatric HCT. We hypothesized that significant bone loss accrues in children during the first 100 days following HCT, and that this bone loss is accompanied by vitamin D deficiency. We performed an ancillary study as part of a multi-centre, randomized, double-blind controlled clinical trial of two approaches to the provision of parenteral nutrition (PN) to pediatric HCT patients: (1) the standard of care in which energy intake was provided in the amount of 140% of estimated basal metabolic rate calculated by standard reference equations, and (2) an alternative strategy in which energy intake was titrated to match resting energy expenditure measured by indirect calorimetric. Study methods and main results have been published. The Institutional Review Boards of Boston Children's Hospital and UCLA Mattel Children's Hospital approved the protocol. The study was registered in ClinicalTrials.gov ID: NCT00115258. The primary outcome of the main study was body composition as measured by dual energy x-ray absorptiometry (DXA). The aim of this ancillary study was to evaluate changes in bone density and bone biomarkers among children undergoing HCT. Preparation for HCT included either total body irradiation (1400 cGy) or busulfan in addition to cyclophosphamide or other chemotherapeutic agents. GVHD prophylaxis included calcineurin-inhibitors, methotrexate and corticosteroids. Standard medications included oral non-absorbable antibiotics for gut decontamination, ursodeoxycholic acid, and vitamin E for venoocclusive disease prophylaxis, and leucovorin calcium for recovery from methotrexate, when used for GVHD prophylaxis. In a prospective study of children undergoing HCT, mean loss of BMD was 5% six months after HCT, and in half of the subjects, bone density losses continued through one year after HCT. Acute changes in BMD and biomarkers associated with bone health, such as vitamin D, in the first months after HCT are unknown. Prior studies have not reported bone morbidities at 30 and 100 days after pediatric HCT. 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panied by vitamin D deficiency. Children who were scheduled for their first myeloablative, matched related or unrelated donor allogeneic HCT (n = 21) or UCLA Mattel Children's Hospital (n = 5) who were greater than or equal to 6 years of age were eligible. Underweight (body mass index [BMI] z-score <−2) and overweight (BMI z-score >2) children were excluded. Subjects with unrelated donors received low dose steroids for graft-versus-host disease (GVHD) prophylaxis.

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