Case Report

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Hypercalcemic Crisis, Can be Iatrogenic

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Abstract

The recommended upper limits for long term vitamin D intake are 1000 IU for children <1 year old and 2000 IU for older children and adults. Vitamin D intoxication can occur with long term high intake or with a substantial acute ingestion. Excess 25 (OH) D causes bone resorption and intestinal absorption of calcium leading to hypercalcemia. Hypercalcemia is defined as a serum calcium concentration that is greater than two standard deviations above the normal mean. Hypercalcemia is generally considered to be mild, moderate, and severe for total serum calcium concentrations <12 mg/dL (3.00 mmol/L), between 12 and 14 mg/dL (3.00 to 3.50 mmol/L), and >14 mg/dL (3.50 mmol/L), respectively. Children with vitamin D intoxication present with symptoms of hypercalcemia, such as poor appetite, weight loss, abdominal pain, vomiting, constipation, polyuria, and polydipsia, and in severe cases, life-threatening dehydration. We report a case of hypercalcemic crisis presenting in emergency department.

Keywords: Hypercalcemia; Intoxication; Vitamin-D.

Introduction

Vitamin D intoxication is a rare event, however, the exact incidence is unclear because there are no systematic studies that have addressed this question. Vitamin D intoxication from dietary sources has been reported from United Kingdom due to excessive milk fortification [1]. Excess 25 (OH) D causes bone resorption and intestinal absorption of calcium leading to hypercalcemia. Hypercalcemia is defined as a serum calcium concentration that is greater than two standard deviations above the normal mean. Hypercalcemia is generally considered to be mild, moderate, and severe for total serum calcium concentrations <12 mg/dL (3.00 mmol/L), between 12 and 14 mg/dL (3.00 to 3.50 mmol/L), and >14 mg/dL (3.50 mmol/L), respectively [2]. Children with vitamin D intoxication present with symptoms of hypercalcemia, such as poor appetite, weight loss, abdominal pain, vomiting, constipation, polyuria, and polydipsia, and in severe cases, life-threatening dehydration [3,4]. We report a case of hypercalcemic crisis presenting in emergency department.

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Case Details

A 6 yr old boy presented with fever for 2 months, vomiting and pain in abdomen for 1 month, and generalised body pain for 15 days. Child had taken treatment outside and received intramuscular injection weekly for 6 weeks. History of polyuria, polydipsia, constipation, was present and child was completely immunised. Family history of neurofibromatosis was present in mother (Fig. 1). On examination child was conscious, irritable, with cold extremities, feeble pulses, tachycardia (130/min), tachypnoea (40/min), with a BP of 80/60 mm Hg with a urine output of 6 ml/kg/hr. Head to toe examination was positive for cachexia, sunken eyes, pallor, bitots spots in bilateral eyes, angular cheilosis, multiple café-au-lait spots (>6) (Fig. 1), and bilateral lisch nodules in slit lamp examination. Anthropometry was suggestive of severe undernutrition. Systemic examination was normal except flat abdomen with dilated loops, visible peristalsis. Lab investigation reveals raised ionic calcium (2.45 mmol/l), hypercalcemia (14.9 mg/dl), hypokalemia, hypercalciuria decreased serum i PTH (2.8 pg/ml) and increased 25-OH vitamin D (374 ng/l). USG KUB was suggestive of early medullary nephrocalcinosis (Fig. 2). Bone marrow examination and X-ray chest, max, CECT abdomen were normal. Child was managed for shock and other life-threatening complications. Hypercalcemia was managed by giving 1.5 times of normal maintenance fluid 5% DNS, furosemide at 1 mg/kg/day (after normal potassium), and prednisolone at 1 mg/kg/day for 2 weeks followed by tapering over next 2 weeks. Infollow, up at 2 months child showed weight gain, and decreased 25-OH vitamin D (110 ng/l) and serum calcium (11 mg/dl).

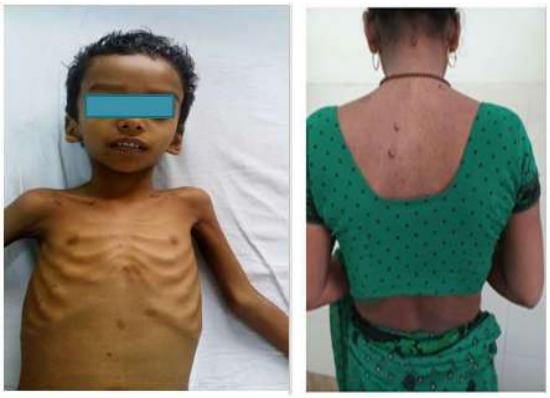

Fig. 1: Pictures of child (multiple café-au-lait spots>6) and mother (neurofibroma) with neurofibromatosis type 1.

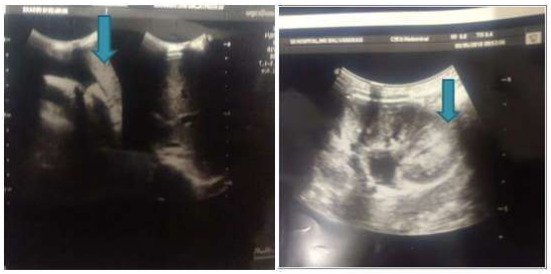

Fig. 2: Pictures of Ultrasound KUB showing features of Early Medullary Nephrocalcinosis.

Discussion

Hypervitaminosis D can occur with long term high intake or with a substantial acute ingestion. The recommended upper limits for long term vitamin D intake are 1000 IU for children <1 year old and 2000 IU for older children and adults. The diagnosis of vitamin D intoxication is based on elevated serum 25OHD concentrations, which are associated with hypercalcemia or hypercalciuria, and PTH is suppressed with evidence of nephrocalcinosis. Vitamin-D intoxication can occurs after use of over fortified formula feeds [5], or dosing errors because of parent misinterpretation of the prescribed doses [6,7]. Clinical features of intoxication manifest at or more serum levels of vitamin D (25OHD) 150 ng/dl and it because of hypercalcemia. Treatment required in case of symptomatic hypercalcemia and start with removal of source of vitamin and management of hypercalcemic crisis. The first line of therapy of hypercalcemia used in this case was hydration with normal saline at 1.5–2.5 maintenance to increase the glomerular filtration rate and calcium excretion with furosemide at 1–2 mg/kg/d, as divided doses every 4–6 hours, Thiazides should be avoided as it increases calcium reabsorption. Despite this hypercalcemia persists, we use Prednisone at 1 mg/kg/d, given as divided doses every 4 hours up to 2 weeks, then tapered over in next two weeks [8]. The response to therapy seen after 72 hours of steroid.

Conclusion

Clinical features of vitamin D intoxication manifest at or more serum levels of vitamin D (25OHD) 150 ng/dl and it because of hypercalcemia. Hypercalcemia, clinically manifests as poor appetite, weight loss, abdominal pain, vomiting, constipation, polyuria, and polydipsia, and in severe cases, life-threatening dehydration and hypovolumic shock. Hydration, and steroid were treatment options.

References

1. Abrams SA, Hawthorne KM, Chen Z. Supplementation with 1000IU vitamin D/d leads to parathyroid hormone suppression, but not increased fractional calcium absorption, in 4–8-y-old children: adouble-blind randomized controlled trial. Am J Clin Nutr. 2013;97:217–23.
2. Bushinsky DA, Monk RD. Electrolyte quintet: calcium. Lancet. 1998;352(9124):306–11.
3. Lietman SA, Germain-Lee EL, Levine MA. Hypercalcemia in children and adolescents. Curr Opin Pediatr. 2010;22(4):508–15.
4. El Saleeby CM, Grottkau BE, Friedmann AM, Westra SJ, Sohani AR.Case records of the Massachusetts General Hospital. Case 4-2011. A 4-year-old boy with back pain and hypercalcemia. N Engl J Med. 2011;364(6):552–62.
5. Samuel HS. Vitamin Das a public health problem. Br Med J. 1964;1:1654–55.
6. Barrueto F Jr, Wang-Flores HH, Howland MA, Hoffman RS, Nelson LS. Acute vitamin D intoxication in a child. Pediatrics. 2005;116:e453–e456.
7. Ezgu FS, Buyan N, Gündüz M, Tümer L, Okur I, Hasanoglu A.Vitamin D intoxication and hypercalcaemia in an infant treatedwith pamidronate infusions. Eur J Pediatr. 2004;163:163–65.
8. Sezer RG, Guran T, Paketçi C, Seren LP, Bozaykut A, Bereket A. Comparison of oral alendronate versus prednisolone in treatment of infants with vitamin D intoxication. Acta Paediatrica. 2012;101:e122–e125.