Pediatr Radiol (1981) 10: 173–174

Pediatric Radiology

Short Reports

Development of Hypervitaminosis A in a Patient on Long-term Parenteral Hyperalimentation

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Abstract. A four and one-half year old boy with intestinal pseudo-obstruction who had been maintained on parenteral hyperalimentation for one year developed classical signs of hypervitaminosis A. This diagnosis was confirmed radiographically; serum vitamin A levels were five times normal. The patient had a dramatic response to withdrawal of vitamin A from the hyperalimentation fluid.

Key words: Total parenteral nutrition – Hypervitaminosis A – Intestinal pseudo-obstruction

Radiologists have become increasingly aware of the complications of total parenteral nutrition in children. Numerous catheter related complications have been documented [1, 2]. Skeletal changes related to copper and vitamin D deficiency have also been reported [3, 4]. The precise parenteral requirements of the two fat soluble vitamins, A and D, are still not known. Decimal errors in the addition of the vitamins have been indicated in cases of hypo- and hypervitaminoses. O'Tuama et al. believed that subclinical hypervitaminosis A and D may be produced with even the usual vitamin dosage [5, 6]. We report classical clinical and radiographic findings of hypervitaminosis A in a patient on hyperalimentation who inadvertently received large amounts of vitamin A.

Case Report

T. W. is a four and one-half year old white male with Chronic Idiopathic Intestinal Pseudo-Obstruction Syndrome. The diagnosis was established at the Arkansas Children's Hospital during a prolonged admission of six months. Except for the first week, his fluid and nutritional status were maintained via central venous alimentation. Because of gastrointestinal losses of approximately 5 liters per day, he received parenterally 6 liters per day of fluid. Each liter contained 10,000 IU of vitamin A.

When gastrointestinal losses decreased to approximately 200 cc per day, he was discharged on a home parenteral nutrition program. Daily vitamin supplementation included 10,000 IU of vitamin A. He did well until six weeks after discharge, when gastrointestinal losses increased to 2 liters per day necessitating additional parenteral fluid replacement. This was given as 0.45 normal saline and he continued to receive 10,000 IU of vitamin A daily. During the next two weeks progressive scalp hair loss and scaly lesions on an erythematous base were observed around the mouth, in the axilla, and in the perineal area. Serum zinc level was 0.33 mg/L (normal 0.80–1.24 mg/L). Zinc supplements were increased from 5 mg/day to 20 mg/day. The skin lesions healed and hair growth was evident over the next four weeks. Serum zinc level rose to 0.90 m/L. Stool losses also decreased during this period, averaging approximately 200 cc per day.

The child did well for three more months. Although there was no change in gastrointestinal losses, once again scalp hair loss and scaly lesions on an erythematous base were observed in the axillary areas and in the groin. Zinc deficiency was again suspected. Serum zinc level was 0.60 mg/L. Zinc supplementation was increased to 40 mg/day. Over the next two weeks the dermatitis became worse, involving a larger surface area. A severe chelosis also developed. The parents reported that the child was more irritable and refusing to walk. Except for the chelosis, hair loss, and dermatitis, the physical examination was unchanged. Serum zinc level was 0.45 mg/L and zinc supplementation at a total dose of 40 mg/day was continued.

The child was again seen one month later when scalp hair loss was almost complete. The dermatitis involved even larger areas than on the previous visit. According to the parents the child was extremely irritable and refused to walk because it was painful. On physical examination, in addition to the alopecia and dermatitis, the chelosis appeared to be worse and gingivitis was present. There was no hepatosplenomegaly. The proximal and distal portions of both extremities were swollen and extremely tender to palpation. No abnormal neurologic signs were found.

Serum electrolytes, SGOT, SGPT, total protein, and albumin, calcium, phosphorus, magnesium and copper levels were normal. Serum zinc level was 0.35 mg/L (normal 0.80–1.24 mg/L). Vitamin levels in the serum were as follows: A 1116 ug/dl (normal 50–220 ug/dl), 25 0H D 35 ng/ml (normal 0.2–20 ng/ml). Lumbar puncture showed an opening pressure of 410 cm H_2O. Analysis of

0301-0449/81/0010/0173/$01.00

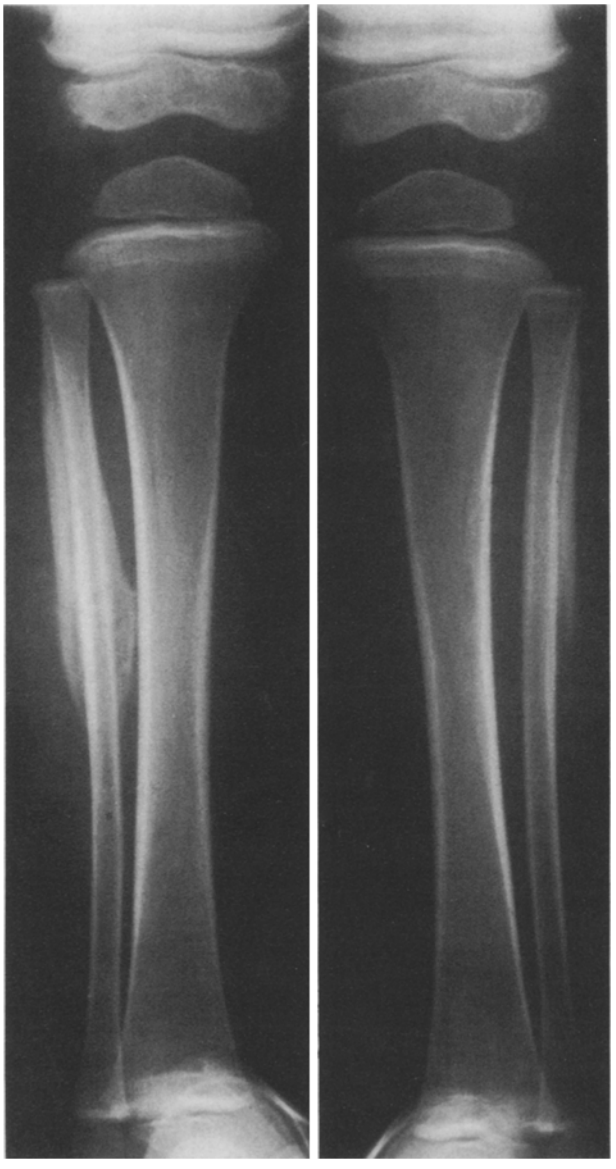

Fig. 1. AP of both lower extremities shows the periosteal new bone formation about the proximal portion of both fibulae with dense metaphyses in the fibulae, tibiae, and distal femurs

the CSF showed no RBC's or WBC's, glucose 58 mg/dl and protein 18 mg %. Films of the upper and lower extremities showed thick periosteal new bone formation with soft tissue swelling about the proximal diaphyseal region of the femurs, ulnas, and the fibulas bilaterally. Also, there was increased density in the metaphyses (Fig. 1).

On the basis of the radiographic studies and elevated serum vitamin A level, it was decided that this child's symptoms were due to hypervitaminosis A. Vitamin A supplementation was discontinued. Over the next seven days the dermatitis, chelosis and gingivitis completely resolved. The child's personality returned to normal and he began to walk without complaint. Serum zinc remained low, 0.25 mg/L, in spite of supplementation of 40 mg/day. Vitamin supplementation of 10,000 IU of vitamin A, 2 days per week, was resumed.

The patient has continued to do well over the ensuing six month period with no recurrence of symptoms. His scalp hair has completely regrown and his ambulation has improved to the point that he is now able to run. Laboratory studies show a serum vitamin A level of 182 ug/dl, and zinc 0.35 mg/L. Repeat radiographic studies after two months showed significant improvement in the skeletal abnormalities.

Discussion

This patient demonstrated typical clinical and radiographic findings described by Caffey of chronic hypervitaminosis A [7]. The combination of periosteal new bone formation and dense metaphyses is typical for this hypervitaminosis. The usual site of periosteal new bone formation was present with involvement of the ulnae, tibiae, fibulae, femurs and clavicles. The matatarsals, however, were spared. The prompt clinical response to withdrawal of vitamin A was dramatic. Radiologists should become aware of the possibility of the development of hypervitaminosis A in a patient who is receiving large amounts of parenteral nutrition.

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Date of final acceptance: May 27, 1980

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