Acne, Hypervitaminosis A and Hypercalcaemia

A Case Report

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SUMMARY

The ingestion of vitamin A, which is often prescribed for the treatment of acne, may lead to hypervitaminosis A. This syndrome has a wide spectrum of clinical features, hypercalcaemia being of special note since it has been reported in 4 previous cases only. Hypervitaminosis A has been described as resulting from excess ingestion of vitamin A for prevention of sunburn and treatment of minimal brain dysfunction. With the present glut of health foods, this condition should be borne in mind when patients present with symptoms of hypercalcaemia and liver dysfunction.


Vitamin A is a fat-soluble vitamin stored predominantly in the liver. Dietary sources include dairy products, eggs, liver and fish-liver oils. Its precursor is carotene, which is present in carrots and tomatoes. The daily requirement is 5 000 IU, but for women in the third trimester of pregnancy and for lactating women 8 000 IU/day are recommended. The clinical picture of chronic hypervitaminosis A is varied, but it is well known to cause desquamation, hair loss, weakness, anorexia, nausea, vomiting, musculoskeletal pain, headaches and hepatomegaly. Associated hypercalcaemia, which occurred in our patient, has been noted in the literature on only 4 previous occasions.

CASE REPORT

A 17-year-old male was referred with documented hypercalcaemia for investigation. The calcium level was 3,4 mmol/l, inorganic phosphate 1.55 mmol/l and uric acid 0,58 mmol/l. He had been in good health until a year previously when he started taking vitamin A 150 000 units/day for his acne. He began to feel lethargic. Three weeks before admission he noticed recurrent epistaxis and thereafter experienced anorexia, nausea, vomiting, constipation, frontal headaches, dizziness, nocturia, lower back pain, and pain below his shoulder blades and in the region of his right triceps muscle. Also, his hair began to fall out. At this stage he spontaneously stopped all medication.

On examination he was found to be an alert and a well-built young man. His pulse rate was normal, blood pressure was 110/70 mmHg and jugular venous pressure was not raised. The heart and lungs were normal. The skin of his lips was cracked, there was marked exfoliation of the skin of his palms and soles (Fig. 1), and there were white lines at the proximal ends of all his nails (Fig. 2). Abdominal examination revealed a 1-cm hepatomegaly and a 1-cm splenomegaly.

Fig. 1. Complete exfoliation of skin on sole, with a small amount of skin left on heel.

Investigations showed the following: normal blood count, prothrombin index 67%, sodium 141 mmol/l, potassium 3,5 mmol/l, chloride 104 mmol/l, glucose 5,94 mmol/l, urea 11,45 mmol/l, creatinine 114,92 µmol/l, calcium 3 mmol/l, inorganic phosphate 0,969 mmol/l, uric acid 0,413 mmol/l, total bilirubin 42,75 µmol/l, and direct bilirubin 32,49
Alkaline phosphatase was 304 mU/ml (normal range 30 - 100 mU/ml), SGPT 24 mU/ml (normal range 2 - 35 mU/ml), and SGOT 67 mU/ml (normal range 2 - 35 mU/ml). The parathyroid hormone assay, 25-dihydroxycholecalciferol and carotene levels were normal, and the vitamin A level was 805 IU/100 ml (normal range 60 - 200 IU/100 ml).

The first set of calcium levels was measured by the atomic flame spectrophotometer, while the second and subsequent levels were measured by the o-cresolphthalein complexone method. (These methods are comparable, although the former usually gives slightly higher values.)

Radiographic examination of the skeleton was normal, except for a suggestion of a periosteal reaction on the proximal third of the medial side of the right humerus. The excretory urogram was normal. The ECG showed a shortened QT interval. On discharge, urea, creatinine and calcium levels had all returned to normal, splenomegaly had disappeared and the prothrombin index was 100%.

The patient was lethargic, but otherwise asymptomatic. Exfoliation of his skin and hair loss continued.

**DISCUSSION**

The features described are consistent with those of chronic hypervitaminosis A. The syndrome has been found to occur after 6 weeks of massive doses, but more frequently occurs after 18 months on 50 000 IU/day. Toxic levels have been attained by excess ingestion for treatment of acne, prevention of sunburn, treatment of minimal brain dysfunction, and by eating meat of the polar bear, which has a high vitamin A content in the liver, during Arctic expeditions.

Two features which should be emphasized are hypercalcaemia and hepatic involvement. The hypercalcaemia accounts for lassitude, nausea, vomiting, anorexia, headaches, impaired renal function and a shortened QT interval on ECG. The hypercalcaemia is independent of parathyroid hormone, and is the result of a direct effect of vitamin A on the bone. According to Jowsey and Rigs there is an increase in the extent of the bone-resorbing surface, enlarged osteocyte lacunae, and osteocytes which behave like osteoclasts and resorb the bone. Hepatic dysfunction is shown by a low prothrombin index, hepatomegaly and raised bilirubin levels. Reports by Farrel et al. indicate that on liver biopsy the ratio of fat cells to liver cells is increased. Splenomegaly was attributed to portal hypertension.

In view of daily requirements, doses of 150 000 IU/day are excessive. Further, it is questionable whether vitamin A is of any value in the treatment of acne, and certainly, if it is used, very careful monitoring for vitamin A toxicity should be instituted by the physician. There is no definitive treatment for hypervitaminosis A, but abrupt cessation of vitamin A ingestion is indicated, after which all features will regress over a period of months.

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**REFERENCES**