

Carotenoderma Associated With a Diet Rich in Red Palm Oil

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PRACTICE POINTS

- Carotenoderma is a cutaneous manifestation of elevated serum β -carotene levels and classically localizes to fatty tissues and areas rich in sweat glands.
- Carotenoderma caused by a diet rich in β -carotene is a benign condition in which a diet low in β -carotene is implicated for treatment.

To the Editor:

Carotenoderma is a cutaneous manifestation of elevated serum β -carotene levels and classically localizes to fatty tissues and areas rich in sweat glands. We present a case of carotenoderma associated with a diet rich in red palm oil, a common food additive in parts of the world outside of the United States.

A previously healthy 8-year-old boy who recently immigrated to the United States from Liberia was hospitalized for treatment of a febrile illness that subsequently was attributed to a viral syndrome. On physical examination by the dermatology department, the patient was noted to have marked orange discoloration on the palms and soles (Figure). Laboratory workup revealed elevated serum β -carotene levels of 809 $\mu\text{g}/\text{dL}$ (reference range, 10–85 $\mu\text{g}/\text{dL}$). Testing of hemoglobin/hematocrit levels and liver, thyroid, and kidney function was normal, and systemic examination revealed no further abnormalities. Upon further inquiry by the dermatology department, the patient's family reported frequent addition of red palm oil to all of the child's meals. The patient subsequently was diagnosed with carotenoderma and was instructed to limit inclusion of red palm oil in his diet.

Red palm oil is a rich source of β -carotene and is commonly used outside the United States as a dietary supplement or food flavoring. Excessive consumption of red palm

oil or other sources rich in carotenes can result in elevated serum carotene levels or hypercarotenemia. An elevation in serum β -carotene levels may be recognized from 4 to 7 weeks after starting a β -carotene-rich diet.¹



Orange discoloration of the palms (A) and soles (B) characteristic of carotenoderma in an 8-year-old boy with a diet rich in red palm oil.

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While dietary consumption of carotenes is the most common cause of carotenoderma, others include kidney or liver disease, hyperlipidemia, porphyria, diabetes mellitus, hypothyroidism, and anorexia nervosa.²⁻⁴ Moreover, since carotenoids are enzymatically converted to vitamin A in the small intestine, a mutation of the gene of the conversion enzyme β -carotene 15,15'-monooxygenase 1 (BCMO1) also can cause be a rare cause of hypercarotenemia.³

Carotenoderma, the clinical cutaneous manifestation of hypercarotenemia, occurs as a result of β -carotene deposits in the skin when serum concentration exceeds 250 $\mu\text{g}/\text{dL}$. More specifically, β -carotene accumulates mainly in the lipid-rich stratum corneum as well as in sweat and sebum, which explains the localized discoloration in fatty tissues and areas rich in sweat glands (eg, nasolabial folds, palms, soles).^{3,4} The sclerae of the eyes are not affected by the surplus of β -carotene in carotenoderma, which helps distinguish it from jaundice.⁵

The differential diagnosis of yellow discoloration of the skin includes jaundice, encompassing the prehepatic, hepatocellular, and posthepatic categories.⁴ Also noteworthy in the differential diagnosis is lycopeneemia, which occurs as a result of eating lycopene-rich foods (eg, tomatoes), resulting in a deeper orange-yellow pigmentation when compared to the cutaneous manifestation of hypercarotenemia.^{2,4,6} Several drugs also have been reported to induce yellow discoloration of the skin, including sunitinib,⁷ sorafenib,⁸ quinacrine, saffron supplements, santonin, fluorescein, 2,4-dinitrophenol, canthaxanthin, tetryl and picric acids, and acriflavine.^{2,4}

Carotenoderma caused by a diet rich in β -carotene is a benign condition in which a diet low in β -carotene is implicated for treatment. Contrary to popular belief, vitamin A toxicity does not occur in the presence of a surplus of β -carotenes because the enzymatic conversion of β -carotene to vitamin A is strictly regulated.⁹ Although acknowledging the various causes of carotenoderma is important, a simple history and laboratory testing for elevated serum β -carotene levels can eliminate further unnecessary testing and allow for prompt recognition of the condition. Appropriate dietary modifications also may be warranted.

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