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## Carotenoderma in Metabolic Carotenemia

Rita Christopher  
G.R. Rangaswamy  
N. Santhoshkumar\*  
K. Taranath Shetty

Carotenoderma is a yellowish to orange skin discoloration usually caused by the ingestion of excessive amounts of carotene-

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*From the Department of Neurochemistry, National Institute of Mental Health and Neuro Sciences, Bangalore 560 029 and \*Amarjyothi Nursing Home, Indiranagar, Bangalore 560 038.*

*Reprint requests: Dr. Rita Christopher, Associate Professor, Department of Neurochemistry, NIMHANS, Post Bag 2900, Bangalore 560 029.*

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containing foods. Carotenoderma is occasionally associated with hypothyroidism, diabetes mellitus or nephrosis. The areas most often involved are the palms, soles and face. The sclera is not affected. This disorder is asymptomatic and non-injurious(1). We describe a child with carotenemia and carotenoderma probably due to an inherited defect in the metabolism of carotenes.

### Case Report

An 18-month-old female child presented with a history of a yellowish discoloration of the skin especially of the palms and soles which had been observed since the age of six months. She had no other symptoms. There was no history of hemolytic episodes, jaundice or of excessive consumption of carotene-containing foods including red palm oil. Her birth and neonatal history and developmental milestones, both motor and mental, were normal.

On examination an orangish yellow, discoloration of the skin especially around the nasolabial folds, forehead, anterior axillary folds, palms and soles, were noticed. The sclerae and oral mucosa appeared normal. She had no organomegaly. A detailed systemic examination including a cardiac, respiratory and neurological evaluation did not reveal any abnormality.

The routine laboratory investigations performed on this child including liver, renal and thyroid function tests were within normal limits. The peripheral blood showed a normocytic, nonochromic blood picture. Her serum total carotenoids, estimated by the method of Bradley and Hornbeck(2), was 390  $\mu\text{g}/\text{dl}$  compared to the normal range of 50 to 150  $\mu\text{g}/\text{dl}$ . The serum total carotenoids of the parents were within the normal range (Table I). The child was placed on a carotene-deficient diet with vitamin A supplementation for 6 months. The yellowish discoloration of the palms and soles reduced substantially and the serum total carotenoid level came down to 76  $\mu\text{g}/\text{dl}$ .

### Discussion

The carotenoids which are the most important of the various classes of pigments in living organisms are ubiquitous in nature and are efficient precursors of vitamin A. They include alpha, beta, gamma carotenes and cryptoxanthine which contains only one beta-ionone ring, and are not equally potent in their ability to form retinol. Beta carotene, the most effective provitamin, is cleaved into two molecules of retinol by the NADH dependent enzyme, carotene dioxygenase present in the intestinal mucosal cells and liver. Bile salts

TABLE I – Laboratory Findings in the Patient and Parents

Investigation	Normal range	Patient	Father	Mother
Serum bilirubin	0.2-0.8 mg/dl	0.5	0.8	0.7
SGOT	6-40 U/L	51	31	35
SGPT	8-40 U/L	26	19	22
GGT	0-37 U/L	17	12	19
Thyroxine	5-12 $\mu\text{g}/\text{dl}$	12.0	11.2	8.5
Tri-iodothyronine	0.7-2.0 $\mu\text{g}/\text{ml}$	1.8	1.2	0.8
TSH	0.5-5.0 $\mu\text{U}/\text{ml}$	2.3	1.9	1.5
Total carotenoids	50-150 $\mu\text{g}/\text{dl}$	390	103	76

are necessary for the absorption of the provitamin. Foods rich in carotenes include carrots, spinach, parsley, turnip tops, watercress, lettuce, tomatoes, papayas and mangoes.

Hypercarotenemia is usually of dietary origin and it disappears when the intake is reduced to normal limits. The time taken to produce clinically appreciable pigmentation depends to some extent on the dietary consumption. Children develop carotenoderma more readily than adults. Excess carotene is in part excreted in sweat, and reabsorbed by the horny layer of the skin. Deposition occurs predominantly in the nasolabial folds and over the forehead where sebaceous glands abound and palms and soles of the skin where the horny layer is thickest. It is found to a lesser degree on the upper eyelids, inner canthi, ears and anterior folds of axillae and over the areas subject to pressure. Mucous membranes including sclera are not stained by carotene.

The conversion of beta-carotene to two molecules of vitamin A is accelerated by thyroxine and hyperthyroidism. The characteristic yellow tint of the skin in hypothyroidism is due to hyperbeta-carotenemia. In a study of 36 patients with thyroid dysfunction, the serum level of beta-carotene in the hypothyroid group was significantly higher in relation to the euthyroid controls, and the hyperthyroid group showed significantly lower values(3). This may be related to the known function of the thyroid hormone in facilitation of carotene conversion.

In diabetes mellitus there is frequently raised serum carotene levels but carotenoderma develops in only 10% of cases. It is due to impaired conversion of carotene to vitamin A.

Metabolic carotenemia due to a genetic defect in the conversion of carotenes to retinol by the enzyme, carotene dioxygenase,

present in the intestinal mucosal cells and liver, has been reported in a 3-year old girl with longstanding yellow discoloration of her skin and a three-fold increase in plasma carotenoids(4). Another plausible explanation for hypercarotenemia without any secondary cause could be the facilitated uptake of carotenes through the intestinal wall. The patient reported here had no history of excessive consumption of carotene-containing foods and showed a normal blood chemistry, with no diabetes mellitus or hypothyroidism. The clinical findings of clear sclerae and oral mucosa and the laboratory findings of normal liver function tests ruled out the presence of jaundice due to any cause. There was no maternal carotenemia. The carotenemia was probably due to a defective conversion of carotenes to retinol by carotene dioxygenase. Confirmation of the enzyme defect by the assay of carotene dioxygenase in the intestinal mucosa or liver was not done as it involves a highly invasive procedure which is unnecessary in a child due to the benign nature of the condition.

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