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Case Report

Hypercarotenemia – A rare clinical condition

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ABSTRACT

Hypercarotenemia is a rare clinical condition. Hypercarotenemia is associated with a high intake of carotene-rich diets and is also associated with hypothyroidism, hypopituitarism, liver disease, inborn errors of metabolism, and kidney disease. We present one such case of a 2-year-old child who had deep yellowish discoloration of the palms and soles. This case report is intended to alert pediatricians that yellowish discoloration of palms and soles may develop as a result of a high intake of carotene-rich foods commonly recommended and consumed by children as the richest source of Vit-A.

1. Introduction

Hess and Meyers in 1919 first described carotenemia as a yellow-orange pigmentation of the skin caused by high levels of carotene in the blood.[1] Carotenemia is a rare clinical condition associated with yellowish discoloration of the skin and increased levels of beta-carotene in the blood. In most cases, long-term and excessive consumption of foods rich in carotene, such as carrots, sweet potatoes, and squash, prevent this condition. However, carotenemia is a rare finding in children, which in most cases is not associated with any clinical problem but can often lead to a misdiagnosis of jaundice. This skin coloration can also have significant symbolic meaning.[2] Carotene is a lipochrome that normally gives the skin its yellow color. When blood carotene levels are elevated, the significance of this yellowing increases. This can manifest itself especially when the stratum corneum is thickened or subcutaneous fat has a strong presence. This condition is easily recognized in fair-skinned people and can be manifested primarily by the yellowing of the palms and soles of darker-pigmented individuals. Yellow skin discoloration in carotenemia is more visible under natural light.

Carotene is secreted by the sebaceous glands that's why, the yellowish pigmentation is very noticeable in areas where sweating is prominent. The pigment change is the result of fat-soluble carotene deposition in the stratum corneum. Pigmentation usually appears first on the tip of the nose, palms, and soles, gradually spreading throughout the body[3] and most prominently on the palms, soles, and nasolabial folds. The sclera is always spared, which easily distinguishes carotenemia from jaundice; however, carotenemia may occasionally manifest as palatal discoloration. It is important to note that the conjunctiva and oral mucosa are areas without a stratum corneum.

2. Case Report

Hypercarotenemia is a rare clinical manifestation. Here we present a case of a 2-year-old child who had deep yellowish discoloration of the palms and soles. The mother of a 2-year-old girl complained of yellowish discoloration of the palms and soles, occasional vomiting, and loss of appetite for 15 days during history taking, it turned out that she had

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consumed 25 g of raw carrots and 30 g of spinach per day for the last 3 months. During the examination, a yellowish discoloration of the hands and feet was observed. The sclera appeared normal and the liver and spleen were normal in size Figure 1.

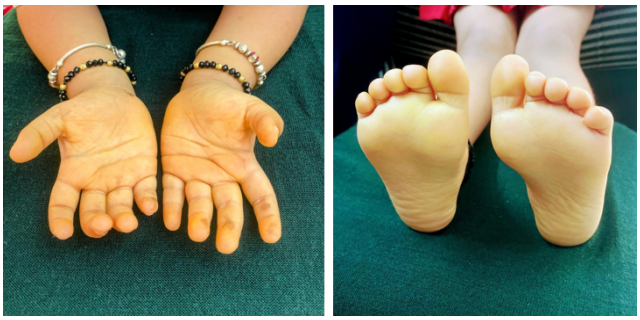

Fig. 1: A 2-year-old girl child presented with yellow discoloration of her palms and soles.

Laboratory Investigations were within normal limits including liver function test.

1. Hb-11.6 g/dl,
2. TLC -9200,
3. DLC- Neutrophil 59%,
   (a) Lymphocytes 34%,
   (b) Eosinophils 05%,
   (c) Monocytes 02%,
   (d) Basophils 0 %
4. RBC count - 4.36 10^{12}/L
5. Platelets Count-2.69 lakh
6. Total bilirubin -1 mg/dl
   (a) Conjugated- 0.6 mg/dl,
   (b) Unconjugated -0.4 mg/dl,
   (c) SGOT- 19 IU/L,
   (d) SGPT- 34 IU/L
   (e) ALK Phosphatase- 77 U/L
7. Total protein-7.6 g/dl,
   (a) S.Albumin- 4 1 g/dl,
   (b) S.Globulin- 3 5 g/dl,
8. High Carotene level in blood

Diagnosis of Hypercarotenemia was made

2.1. Treatment

Carrot and spinach were completely stopped. The yellowish discoloration of the palms and soles start fading after one month and completely disappeared after two months

3. Discussion

Carotene is the precursor of vitamin A in humans. After ingestion of food, carotene in the body is absorbed mainly in the proximal small intestine into the portal circulation. Roughly 10% of ingested carotene is absorbed in the body without conversion and reaches the liver via portal circulation.[4] Fortunately, because the body converts a limited amount of carotene into vitamin A each day, excessive carotene intake does not lead to hypervitaminosis A[5] Carotene has a high affinity for fat and is deposited in the cornea, resulting in the yellowing of the skin if blood levels are excessively high, but the mucous membranes are not affected.[6] Hypercarotenemia is associated with a high intake of carotene-rich foods such as pumpkin, carrots, spinach, lettuce, tomatoes, and mangoes.[7–9] Hypercarotenemia has also been found to be associated with other medical conditions such as hypopituitarism, hypothyroidism, inborn errors of metabolism, and liver and kidney disease.[10] The development of hypercarotenemia is usually not influenced by the age and gender of the patient but occurs more often in infants and young children.[11] In infants and children with hypercalcemia, the doctor usually notices a yellowish discoloration of the palms and soles, which is caused by the deposition of carotenoids in adipose tissue.[11] It is absolutely necessary to distinguish it from jaundice because, in the case of hypercarotenemia, the sclera is not affected at all.

Many other factors are associated with hypercarotemia and include mutations in the enzyme responsible for converting dietary provitamin A carotenoids to vitamin A.[12] Carotenemia can also be caused by hyperlipidemia, restricted dietary habits, or insufficient conversion of carotene to vitamin A in the liver.

The underlying mechanism of carotenemia in hypothyroidism is an incomplete conversion of carotene to vitamin A. Thyroid hormone is antagonistic to vitamin A and mediates the rate of its catabolism. Vitamin A consumption must be increased in hypothyroidism because the rate of conversion of carotene to vitamin A is slowed. Anorexia nervosa is another medical condition associated with carotenemia.[13] Carotenemia in patients with anorexia nervosa is associated with hypercholesterolemia, a reversible defect in the conversion of carotene to vitamin A. In addition, it can also be attributed to a normal dietary intake of carotene in the presence of a reduced need in the body.

A good history and physical examination are usually sufficient to help narrow the differences, and laboratory tests are generally not necessary to make a diagnosis of diet-related carotenemia. Laboratory testing usually shows elevated serum carotene levels, in the range of 250 to 500 micrograms/dL, with normal or mildly elevated liver enzymes. Measurement of skin carotene levels could also be facilitated by the use of resonance Raman spectroscopy

and reflectance spectroscopy, which are non-invasive optical quantitative techniques for measuring carotenoid antioxidants in human skin in vivo. Plasma carotenoid status can also be predicted from skin carotenoid status.[14,15] The bilirubin oxidase method is not reliable and has been reported to misdiagnose β-carotenemia as jaundice.[16] The level of vitamin A is usually normal. range except in the rare case of hereditary enzyme deficiency where levels will be low.[17,18] Evaluation must also include liver enzymes with bilirubin, thyroid function test, and screening for diabetes.

The basis of treatment is to reduce the amount of carotene in the diet. Reducing carotene intake will eventually lead to the resolution of skin pigmentation.[19] Parents of affected children should be advised that various green vegetables such as green beans and spinach are rich in carotene and should therefore be avoided. Consultation with a nutritionist and the preparation of a menu will be useful.

4. Conclusion

Hypercarotenemia can be caused by elevated serum carotenoid levels or mildly elevated vitamin A. The patient reported here had a history of excessive consumption of carotene-containing foods and a normal blood count, without diabetes mellitus or hypothyroidism. Clinical findings of clear sclera and oral mucosa and normal liver tests excluded the possibility of jaundice.

5. Conflict of Interest

None.

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