

Carotenaemia in Infancy

R. McCarthy, E. Dempsey

Cork University Maternity Hospital, Cork City, Ireland.

Abstract

Presentation

Carotenaemia in infancy can develop due to excess dietary carotenoids, resulting in a yellow-orange discolouration of the skin. These changes are more commonly seen over the palms, soles, and nasolabial folds, with sparing of the sclera.

Diagnosis

This is based on a combination of clinical findings, occasionally aided by specific lab investigations such as beta-carotene levels.

Treatment

Specific interventions are not typically required, as skin changes tend to self-resolve as diet naturally evolves.

Discussion

We identified this condition in an infant, whose diet was rich in carotenoids since commencing pureed and solid foods. Whether this increases the chances of developing carotenaemia has not been definitively confirmed, but we will discuss the potential pathophysiology behind this infrequently seen condition.

Introduction

A 9-month-old girl was reviewed for gross motor delay. Delivered at term, with a birth weight of 3.14kg, she had an unremarkable neonatal period and medical history. In addition to mild hypermobility and hypotonicity in her lower limbs, she was noted to have a yellowish discolouration of her skin, primarily affecting her palms, soles, and nasolabial folds (Image 1 and 2). Her sclera remained unaffected, and there were no further developmental concerns.



Image 1



Image 2

Case Report

A detailed dietary history revealed a significant intake of pureed vegetables containing high levels of carotenoids, such as carrots, sweet potatoes, and pumpkin, the so called 'Orange Diet'. Specific blood tests were requested, primarily looking at levels of Beta Carotene. The parents subsequently introduced a varied diet over time, and within 9 months her skin tone had returned to normal.

Discussion

Carotenaemia is a well-documented condition but can be difficult to diagnose when unfamiliar with its typical pattern and aetiology. A key feature that distinguishes it from jaundice is the absence of scleral icterus, in addition to the palmoplantar and nasolabial fold distribution, the latter being due to carotenoid tendency to accumulate in regions with additional sweat glands ¹.

Carotenoids are naturally occurring and are found in many of our food products. Some of the most common sources are carrots, tomatoes, green vegetables, and food colouring. These include alpha- and beta-carotene, lutein, lycopene, beta-cryptoxanthin, and zeaxanthine ². The primary carotenoid involved in carotenaemia is beta-carotene, which is converted to Vitamin A through the actions of Beta-carotene 15,15'-dioxygenase ³. However, elevated levels of carotenoids do not typically result in hypervitaminosis A ⁴, due to intrinsic inhibitory mechanisms, and Vitamin A toxicity secondary to elevated beta-carotene has not been widely reported ⁵. Interestingly, we did see a slight elevation in Vitamin A levels of 1.47µmol/L.

Carotenaemia in infancy is typically of dietary origin. Use of pureed vegetables for feeding is known to increase the bioavailability of carotenoids, thus increasing the possibility of developing this condition ^{6,7}.

These changes are both benign and reversible, and in a case series by *Karthik et al*, they found there was a complete recovery in skin colour in several infants diagnosed with carotenaemia between the ages 6 to 11 months, without any dietary interventions ⁶. This may be due to a natural progression in diet and would reassure many parents.

It should also be noted that although diet is the most common contributing factor in the majority of children, there are some who have an increased susceptibility to carotenaemia as a result of conditions such as diabetes mellitus, or hypothyroidism, the underlying mechanisms of which are thought to be related to a decreased conversion of carotene to its Vitamin A metabolite ⁸, in addition to hypercholesterolaemia, which increases the binding site availability for carotenoids ⁷.

This case in-particular is a good example of how a diet consisting primarily of vegetables rich in carotenoids can lead to dietary related, or primary carotenaemia. It emphasises the importance of a detailed feeding history and being aware of the typical dermatological distribution of this condition, to avoid unnecessary investigations and anxiety. As was the case with those children followed up by *Karthik et al* ⁶, our patient showed a spontaneous resolution in skin colour at 18 months, without the need for significant dietary interventions. It is likely that as the diet varies it would contain less carotene rich sources, leading to an improvement in skin tone.

Declaration of Conflicts of Interest:

The authors of this paper have no conflicts of interest to declare.

Patient Consent: Received.

Corresponding Author:

Dr Robert McCarthy,
Cork University Maternity Hospital,
Cork City,
Ireland.
MRCN: 415057, 30/09/2020
Email: robertmccarthy0105@gmail.com

References:

1. Prince MR, Frisoli JK. Beta-carotene accumulation in serum and skin. *AM J Clin Nutr* 1993;57:175-181.
2. Saldana-Chaparro R, Cara E, Barron JL. Hypercarotenaemia or hypercarotenoidaemia. *Annals of Clinical Biochemistry* June 2003, 40(Pt 3):280-2
3. Keown K, Bothwell J, Jain S. Nutritional implications of selective eating in a child with autism spectrum disorder. *BMJ Case Rep.* 2014 Mar 20;2014
4. Sharman IM. Hypercarotenaemia. *Br Med J* 1985;290:95-96.
5. Institute of Medicine, Food and Nutrition Board Beta-carotene and other carotenoids. Dietary reference intakes for vitamin C, vitamin E, Selenium, and carotenoids. Washington, DC: National Academy Press, 2000:325–400

6. Karthik SV, Campbell-Davidson D, Isherwood D. Carotenemia in Infancy and its Association with Prevalent Feeding Practices. Pediatr Dermatol. 2006 Nov-Dec;23(6):571-3.
7. Nyekiova M, Ghaderi S, Han TS. Carotenoderma in a young woman of normal body mass index with hypothalamic amenorrhoea: a 2-year follow-up case report. Eur J Clin Nutr. 2014 Dec;68(12):1362-4
8. Aktuna D, Buchinger W, Langsteger W et al. Beta-carotene, vitamin A and carrier proteins in thyroid diseases. Acta Med Austriaca 1993;20:17-20.