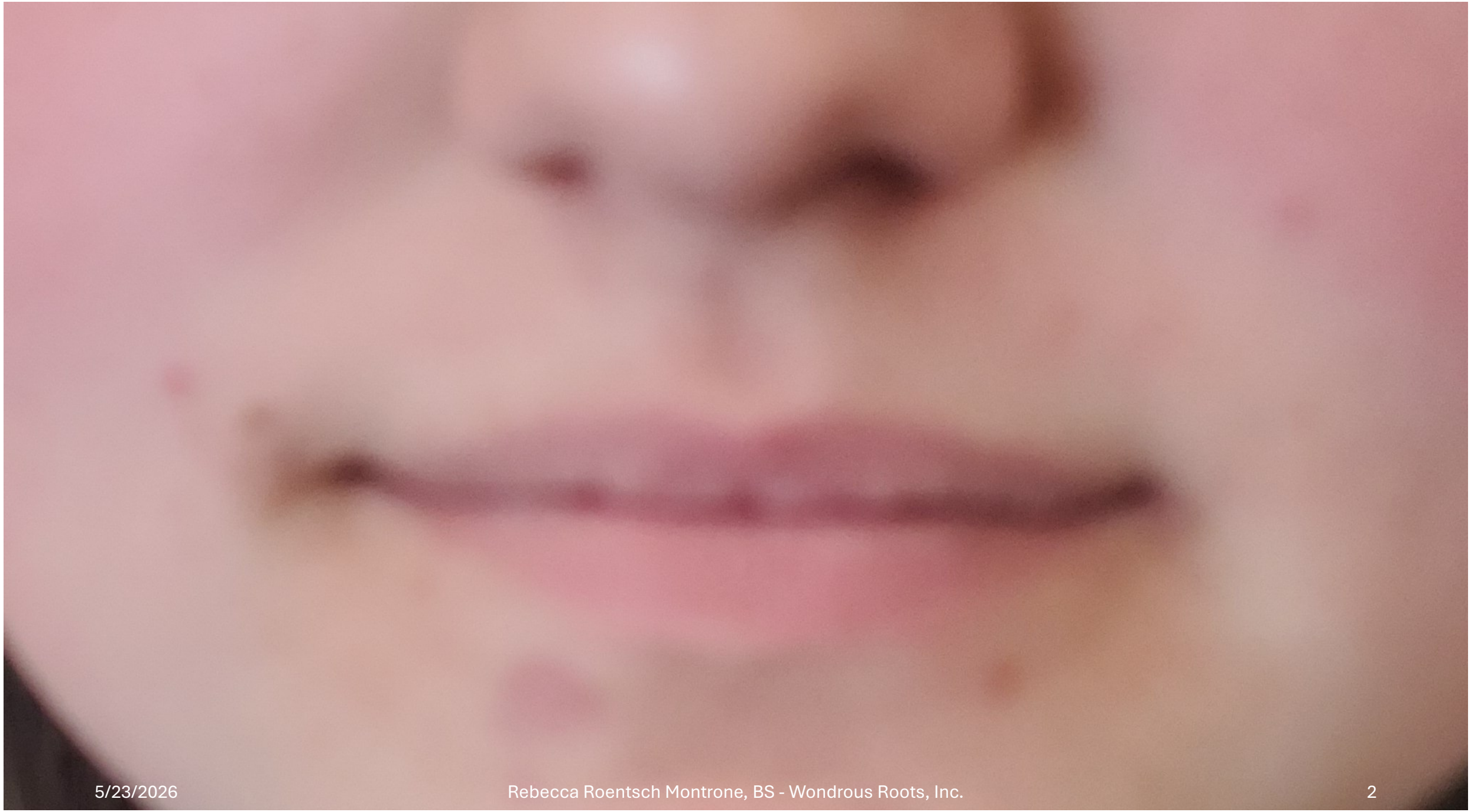
“Why is my skin yellow?”

Carotenemia, Vitamin A, Thyroid Function & Metabolism Explained



From my client notes...

“She has signs and symptoms of sluggish thyroid with borderline labs, borderline prediabetes, and she has this odd discoloration - looks almost like carotenoid coloring but more yellow like jaundice (but it is not jaundice - sclerae are clear, no other signs of jaundice, and the doctor said that it's just that her cheeks are red so this stands out. It isn't obvious, really, but it is different coloration - more like a different skin tone; like her skin tone otherwise is red-based, and in this area the skin tone is yellow-based. It doesn't itch or crust, there is no rash; it comes and goes - Oh! It really pops out when she eats a carrot or tomato sauce or sweet potato fries, so it is obviously beta-carotene, but not a lot, but when she eats a carrot, it will be very pronounced.”

What IS going on here?

Her doctor said there was no discoloration around her mouth and nose but that because her cheeks were red the “normal” tone stood out. Hmm… I don’t *think* so…

Case Background

This case involved a teenage girl previously seen for significant environmental allergies related to working around hay and barn animals. While her allergy symptoms had improved substantially, a new constellation of findings emerged that appeared interconnected:

Symptoms suggestive of sluggish thyroid function despite only borderline laboratory findings.

- Borderline prediabetes/early glucose dysregulation.
- Menorrhagia.
- A distinctive yellow-orange discoloration around the nasolabial folds and mouth.

The skin finding was particularly interesting because it was not jaundice. The sclerae remained completely clear, there was no itching, crusting, rash, or evidence of liver disease, and the discoloration waxed and waned. Most notably, it became dramatically more apparent after consumption of carotene-rich foods such as carrots, tomato sauce, and sweet potato fries.

Initial Interpretation

The clinical picture was highly suggestive of mild hypercarotenemia (carotenemia), a condition in which beta-carotene accumulates in the bloodstream and skin because it is not being efficiently converted into active vitamin A (retinol).

The discoloration pattern itself provided an important clue. Hypercarotenemia commonly affects:

- Palms
- Soles
- Nasolabial folds
- Areas of thicker keratinized skin

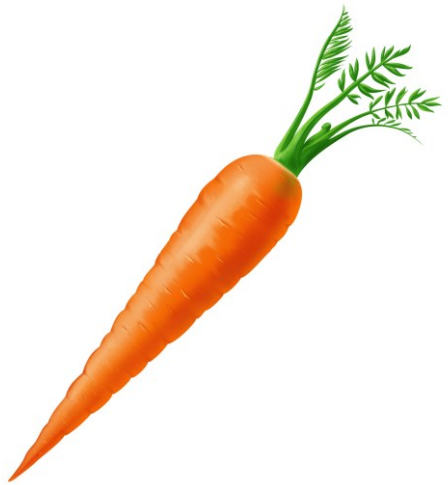
...while sparing the sclerae, which differentiates it from jaundice.

Pathophysiology Discussed

Beta-carotene is not active vitamin A. It must be converted into retinol by the enzyme Beta-Carotene Oxygenase 1 (BCO1).

When this conversion pathway is sluggish:

- Beta-carotene is absorbed normally.
- Conversion to retinol is inefficient.
- Carotene accumulates in circulation.
- Excess carotene deposits in the skin.
- Yellow-orange discoloration develops.



Several factors discussed as potential contributors included:

Thyroid Function

- Hypothyroidism is a well-established cause of hypercarotenemia.
- Low thyroid activity decreases BCO1 activity, reducing conversion of beta-carotene to retinol and allowing carotene to accumulate.
- This fit well with my client's symptoms of sluggish thyroid function despite borderline laboratory values.

Rebecca Roentsch Montrone, BS - Wondrous Roots, Inc.

Iron Status

Iron is required for normal carotene metabolism and thyroid function.

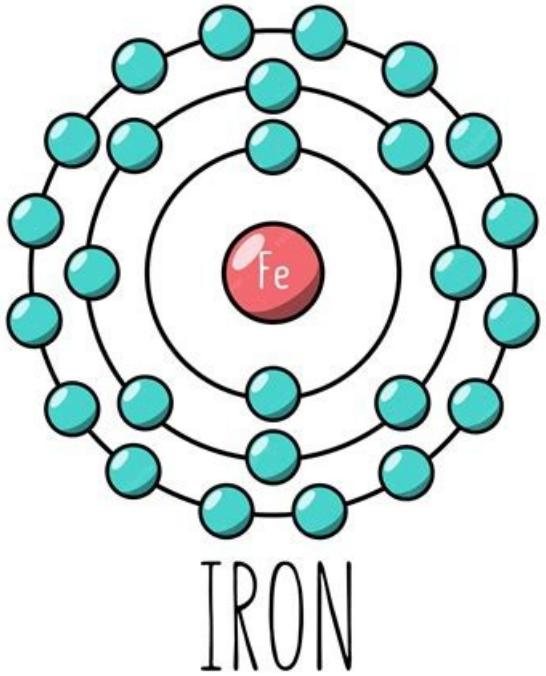
Because my client experiences menorrhagia, iron deficiency was considered a likely contributor.

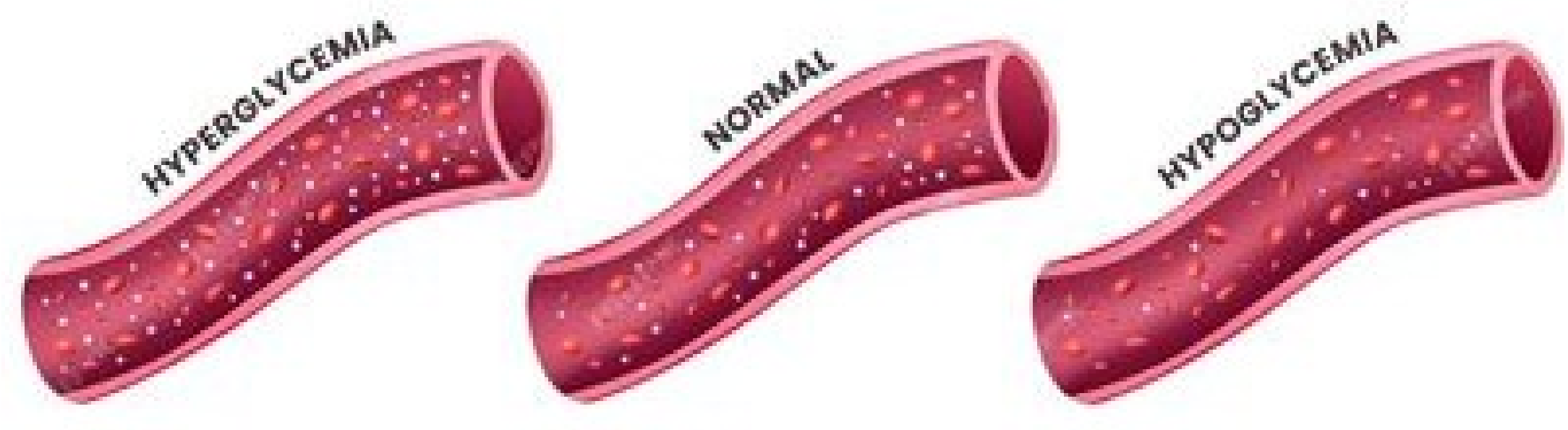
Low iron can impair:

- Thyroid peroxidase activity
- Thyroid hormone production
- Carotene-to-retinol conversion
- Cellular energy production

Iron deficiency therefore provides a plausible link between:

- Menorrhagia
- Thyroid symptoms
- Carotenemia
- Metabolic dysfunction



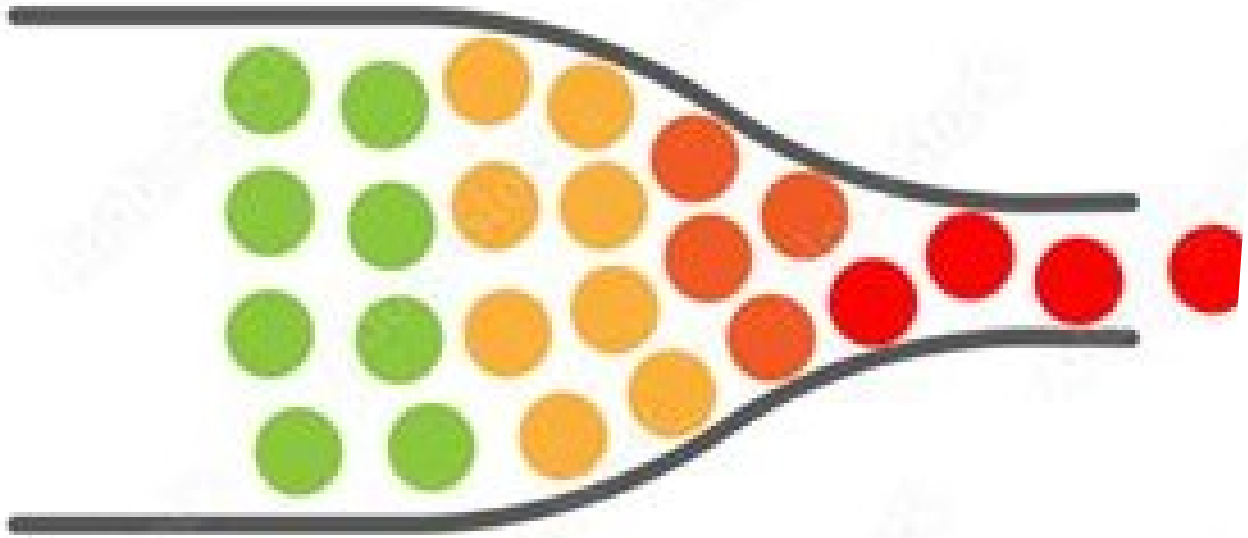


Glucose Dysregulation

- Borderline prediabetes may also contribute to impaired carotenoid metabolism.
- Insulin resistance and altered metabolic signaling have been associated with higher circulating carotene levels and reduced conversion efficiency.

Genetic Factors

- The literature review revealed that common BCO1 genetic polymorphisms can reduce conversion efficiency by as much as 50%.
- While genetic testing was not discussed as immediately necessary, the possibility was acknowledged that my client may naturally be a poorer carotene converter than average.



Why Retinol Supplementation Was Recommended

Retinol does not directly "fix" the conversion pathway. Rather, it ***bypasses the bottleneck***.

By supplying active Vitamin A directly:

- The body's reliance on carotene conversion decreases.
- Retinol status improves.
- Excess circulating carotene gradually declines.
- Skin discoloration should diminish over time.

In practical terms, the body no longer has to depend so heavily on an inefficient conversion pathway.

Protocol Considerations

The proposed additions included:

- Fish-oil sourced vitamin A (retinol) 20,000 IU daily
- Iodine
- Iron bisglycinate 25–50 mg daily

Existing supports already included:

- Selenium (via Thorne Basic Nutrients 2/Day)
- Zinc (15 mg daily)
- Magnesium
- Omega-3 fatty acids
- S-acetyl-L-glutathione
- Quercetin

Discussion centered on the practical challenge of supplement timing.

Because:

- Iron competes with minerals.
- Iodine is ideally separated from vitamin C.
- Compliance is more important than theoretical perfection.

The conclusion was that combining:

- Moducare
- Iodine
- Iron

...into the same dosing window was a reasonable compromise and preferable to creating a complicated schedule that would be difficult to follow consistently.

Dietary Considerations

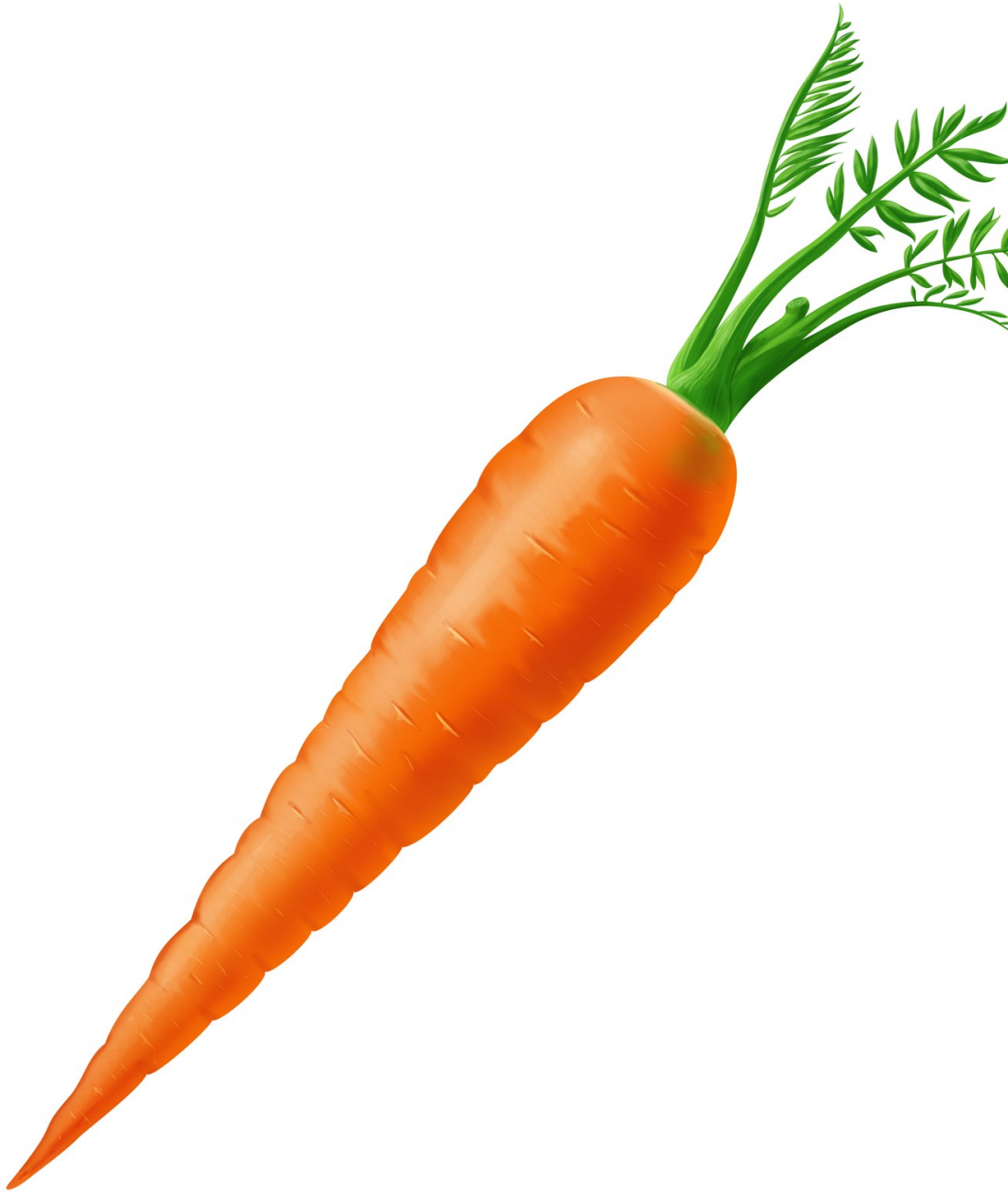
A key question was whether carotene-rich foods should be avoided.

The conclusion was:

- **No evidence of harm from carotene accumulation itself**
- Hypercarotenemia is generally considered benign.
- There is no evidence that the carotene accumulation itself is toxic.

However, temporary moderation may be helpful

For the time being, as vitamin A stores are being replenished and conversion is improving, it may be helpful to moderate intake of high-carotenoid foods such as carrots, sweet potatoes, and tomato-based products. This is not because these foods are harmful, but because the body is currently not converting beta-carotene efficiently, allowing it to accumulate temporarily in the skin. As nutrient status and metabolism improve, this sensitivity should resolve.



Key Clinical Takeaways

The central insight from the case was that the yellow discoloration was not merely a cosmetic issue or a consequence of eating too many carrots.

Rather, it served as a visible marker of an underlying metabolic pattern involving:

- Impaired carotene-to-retinol conversion
- Borderline thyroid dysfunction
- Menorrhagia and likely iron insufficiency
- Early glucose dysregulation
- Possible genetic predisposition

In this sense, the carotene accumulation functioned as a useful clinical clue rather than a disease process itself.

The lesson? Sometimes the shortest path to the root cause begins with following a carrot. 🥕😊

Once again, this case reminds us of the importance of looking beyond isolated symptoms and "getting to the root" of what the body is trying to communicate. My client's physician reassured her that there was nothing abnormal about the skin around her mouth, suggesting that it only appeared different because the surrounding skin was naturally quite red. Yet that seemingly insignificant patch of "normal" skin turned out to be a valuable clue. Rather than being unrelated, it pointed toward a pattern involving thyroid function, iron status, vitamin A metabolism, and blood sugar regulation. What initially appeared to be a cosmetic quirk became a thread that helped tie together multiple findings into a coherent picture. This is one of the aspects of clinical work I find most fascinating: sometimes the body leaves subtle breadcrumbs, and when we take the time to follow them, seemingly unrelated symptoms begin to make sense as parts of a larger story. In this case, the carrot-colored clue around the mouth may have been one of the most informative findings of all. 🍂🥕🔍