CLINICAL VIGNETTE

Rare Cutaneous Manifestation of Hepatitis C

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Case Presentation

A 60-year-old Hispanic female presented for skin blisters and ulcerations on her hands and face for a few months. She is a retired teacher and spent the past 6 months in Mexico with her husband. She noticed intermittent skin blisters on the back of her hands and her face after sun exposure. The blisters turned into painful ulcers then healed slowly with scarring. She denied skin itching or rash. She went on 30-minute daily walks with her husband and loves the suntan from the walks. There is no muscle pain or joint pain.

Upon review of systems, she mentioned dark colored urine from time to time. She denied urinary burning, frequency, urgency, flank pain, fever, chills, foul smelling urine, nausea or vomiting. She attributed unhealthy food to the dark urine and believed high Glutathione food cleared her dark urine.

She has a history of hepatitis C from blood transfusions after childbirth before 1992 and is otherwise healthy. She takes no medications or supplements and denies tobacco, alcohol or intravenous drug use.

Her vital signs were all normal. She appeared to be much darker than before. Oddly, she was evenly tanned across her entire body without any tan lines. She had 1 cm scabbed ulceration with raised erythema border on left dorsal hand and similar < 1 cm lesion on right dorsal hand. No other skin lesions. The rest of her physical exam was unremarkable.

Initial Laboratory findings were significant for slightly abnormal iron studies:

FE 218
TIBC 260
Transferrin Saturaton 84%,
Positive Cryoglobulin titer
and HCV RNA SER PCR QN 430,965.

Other labs including CBC, LFTs, BUN, Cr, Electrolytes and inflammatory markers, and UA were all normal. Urine culture is negative. Antibodies including ANA, RF, anti-smooth muscle antibody were all negative.

She was referred to dermatology and shave biopsy was performed to rule out Cryoglobulins and Vasculitis. Biopsy showed hypergranulosis with hyperkeratosis. The dermis was fibrotic with only minimal inflammation. There was no vasculitis nor cryoglobulins. The fibrosis was suggestive of a scar. She was given a trial of Triamcinolone cream by Dermatology without improvement.

She continued to make frequent office visits seeking for a definitive diagnosis. Each subsequent visit made me question the "facts" of a tan from sun exposure, without tan lines normally indicative of a natural suntan. Her "suntan" was in reality pathological skin hyperpigmentation. When combining her skin hyperpigmentation, dark colored urine, skin blisters and history of hepatitis C, the situation supported additional testing for Porphyria Cutanea Tarda. Her plasma porphyrins all returned elevated.

Uroporphyrin 17.6 Heptacarboxylporphyrin 23.2 Hexacarboxylporphyrin 1.7 Pentaporphyrins, BLD QN 5.3 Coproporphyrin 1.0 Porphyrins 48.8

She subsequently received phlebotomies every 2 weeks for 6 months and antiviral therapy for hepatitis C. Her serum iron level decreased to from 218 to 22 and Hepatitis C viral load became undetectable. Her skin hyperpigmentation reverted back to her normal skin color. She stopped getting blisters on her hands and face after sun exposure.

Discussion

Porphyria cutanea tarda (PCT) is the most common form of porphyria. Unlike other forms of porphyria, which are inborn errors of metabolism, PCT is usually caused by an acquired liver disease, chief among which are excess alcohol intake, iron overload, chronic hepatitis C, Estrogen therapy and cigarette smoking. Chronic hepatitis C infection decreases hepcidin production by hepatocytes. The decrease in hepcidin leads to increased iron absorption from the gut. Iron loading is usually only mild or moderate in degree (less than that associated with full-blown hemochromatosis). In the liver, iron loading and increased oxidative stress leads to formation of non-porphyrin inhibitor(s) of uroporphyrinogen decarboxylase and to oxidetion of porphyrinogens to porphyrins. The treatment of choice of active PCT is iron reduction by phlebotomy and maintenance of a mildly iron-reduced state without anemia. Low-dose antimalarials (cinchona alkaloids) are also useful as additional therapy.[1]

Hepatitis C cutaneous manifestations are numerous. Common skin conditions like eczema, pruritus, psoriasis can be associated with hepatitis C. Patients with hard to treat skin conditions should prompt hepatitis C testing because they may be the first indication of hepatitis C. In most cases, skin manifestations disappear after appropriate hepatitis C treatment or viral clearance.

REFERENCES

1. **Ryan Caballes F, Sendi H, Bonkovsky HL**. Hepatitis C, porphyria cutanea tarda and liver iron: an update. *Liver Int*. 2012 Jul;32(6):880-93. doi: 10.1111/j.1478-3231.2012.02794.x. Epub 2012 Apr 17. Review. PubMed PMID: 22510500; PubMed Central PMCID: PMC3418709.

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