IMAGES IN CLINICAL MEDICINE

Lindsey R. Baden, M.D., Editor

Cutaneous Melanosis in Peripheral Cortisol Resistance





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47-YEAR-OLD-MAN PRESENTED WITH A 3-MONTH HISTORY OF ORTHOstatic hypotension and cutaneous hyperpigmentation. He was infected with the human immunodeficiency virus (clinical stage 4, according to World Health Organization criteria), with a CD4 T-cell count of 50 cells per cubic millimeter and associated cryptococcal meningitis, pulmonary tuberculosis, and lung abscess. Physical examination revealed cachexia and melanodermia, predominantly on the cheeks, nose, and chin and around the mouth (Panels A and B). The patient was awake and alert and had a temperature of 38°C and a pulse of 90 beats per minute. His blood pressure was 105/65 mm Hg when he was supine and 85/50 mm Hg when he was standing. Laboratory results included a 24-hour urinary free cortisol level of 409 μ g (1128 nmol; normal range, 50 to 190 μ g [138 to 524 nmol]), a plasma cortisol level of 42 µg per deciliter per 8 hours (1159 nmol per liter; normal range, 4 to 19 μ g per deciliter [110 to 524 nmol per liter]), a plasma cortisol level of 29 μ g per deciliter per 16 hours (800 nmol per liter; normal range, 3 to 17 μ g per deciliter [83 to 469 nmol per liter]), and a plasma corticotropin level of 103 ng per liter (27 nmol per liter; normal range, 10 to 48 ng per liter [2 to 11 nmol per liter]). The serum sodium level was 128 mmol per liter, and the potassium level was 4 mmol per liter. Hydrocortisone was administered to address the peripheral resistance to cortisol. The patient received antiretroviral therapy with tenofovir, emtricitabine and efavirenz. His condition improved, and he was discharged 4 weeks after hospital admission. With proper therapy, the evolution of peripheral cortisol resistance can be slowed, and this slowing is reflected in the gradual decrease of abnormal skin pigmentation, which occurred in this patient.

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