Cyclosporine has been reported to be associated with lesions of the pilosebaceous unit including hypertrichosis, epidermal cysts, keratosis pilaris, acne, folliculitis, and sebaceous gland hyperplasia as well as cutaneous malignancies. Bencini et al. studied 67 renal transplant patients, seven of whom were found to have sebaceous hyperplasia and none were reported to have a basal cell carcinoma. Sebaceous hyperplasia is a common finding in the general population; whether these cases occurred out of proportion to what would be expected is not clear. In our patient the sebaceous hyperplasia was florid; in addition, basal cell carcinoma is an unusual finding in a Hispanic patient. We thus believe it is important to realize that cyclosporine is a potential cause of rapidly occurring, extensive sebaceous hyperplasia.

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REFERENCE

Vibrio vulnificus infection and iron overload

To the Editor: We read with interest the article “Vibrio Vulnificus Septicemia in Korea: Clinical and Epidemiologic Findings in Seventy Patients” by Park et al. (J AM ACAD DERMATOL 1991;24:397-403) in which they noted the increased incidence of this infection in patients with liver disease and/or alcohol consumption. We hope to help clarify an important mechanism of increased susceptibility to V. vulnificus in these patients.

Hemochromatosis, or primary iron overload, is an essentially autosomal recessive disorder with a carrier (heterozygote) frequency in the North American white population of 1 in 10 and a homozygous frequency of 1 in 400. Although virtually all homozygotes exhibit the clinical phenotype of hemochromatosis regardless of ethanol intake, heterozygotes may also express clinical hemochromatosis particularly under the hepatotoxic influence of chronic alcohol ingestion.1

Patients with iron overload are at an increased risk for V. vulnificus infection and are more liable to experience poor outcome relative to infected patients without iron overload. The increased risk in these patients is caused by their inability to remove iron from the serum and render it unavailable to V. vulnificus.

In vitro and in vivo experiments strongly support the theory that iron plays a major role in the pathogenesis of V. vulnificus infections. Regardless of the cause of serum iron elevation, the V. vulnificus inoculum size required to kill mice in an experimental model is inversely correlated with serum iron level. The effect is profound; immediately after injection of 80 μg of ferric ammonium citrate, the LD₉₀ in mice drops from 10⁶ bacteria to one bacterium.

Although the diagnosis of iron overload can be suggested on the basis of clinical findings, it must be confirmed by evidence of iron accumulation in the blood such as a marked increase in transferrin saturation, serum ferritin, and plasma iron.2 Prussian blue staining of liver biopsy specimens is diagnostic when iron overload and pigment cirrhosis are seen; all affected tissues including skin will show similar evidence of iron overload with this staining technique.

Prompt diagnosis and appropriate intervention in the early or precirrhotic phase of hemochromatosis provides major benefits for affected patients.4 Certainly in North America, patients with V. vulnificus infections need to be examined for hemochromatosis and we suggest that Park and colleagues continue their impressive research by evaluating their series of patients for iron overload and hemochromatosis.

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Pityriasis rubra pilaris: The problem of its classification

To the Editor: I read with interest the letter of Gelmetti and Cerri and the reply by Cohen and Prystowsky (J AM ACAD DERMATOL 1990,23:1186-8) on the taxonomy of pityriasis rubra pilaris (PRP) and would like to make some comments. The ideal classification of a disorder or group of disorders is based on the cause of the disease. In many of the cutaneous diseases we are unable to achieve this ideal and are obliged to rely on an alternative system