Seven types of porphyria are recognized. The light sensitivity in the six types with cutaneous lesions is caused by the presence of a neurotoxic porphyrin molecule. These wavelengths lie in the 400-nm range, representing long-wave ultraviolet light (UVA) and visible light.
In *erythropoietic porphyria*, a very rare disease that typically develops during infancy or childhood, recurrent vesiculobullous eruptions in the skin are the primary feature. Hypertrichosis and brown-stained teeth that fluoresce are additional features.

In *erythropoietic protoporphyria*, the usual reaction to light is erythema and edema followed by thickening and superficial scarring of the skin. In rare instances, fatal liver disease develops quite suddenly, usually in persons of middle age but occasionally in patients only in the second decade of life.
In *porphyria variegata*, different members of the same family may have either cutaneous manifestations identical to those of porphyria cutanea tarda. Also, a sharp fluorescence emission peak at 626 nm is specific for the plasma of porphyria variegata.
Three forms of porphyria cutanea tarda can be distinguished: sporadic, familial, and hepatoerythropoietic. In the sporadic form, only the hepatic activity of uroporphyrinogen decarboxylase is decreased. Almost all patients are adults, and no clinical symptom occurs typically. In the familial form, symptoms typically begin in childhood and occur earlier than in the sporadic form. In some cases, no precipitating factor exists; in most instances, in addition to the inherited enzymatic defect, an additional factor can occur without any precipitating factor.
Porphyria Cutanea Tarda =اﻠآﺠﻠﺔ اﻠﺠﻠدﻴﺔ اﻠﺒورﻔﻴرﻴﺔ

In hereditary coproporphyria, a very rare disorder, there are episodic attacks of abdominal pain and a variety of neurologic and psychiatric symptoms. There are also cutaneous manifestations indistinguishable from those of porphyria cutanea tarda and porphyria variegata.
Histopathology

The histologic changes in the skin lesions are the same in all six types of porphyria. In mild cases, homogeneous, pale, eosinophilic deposits are limited to the immediate vicinity of the blood vessels in the papillary dermis. These deposits are best visualized with a PAS stain, being PAS positive and diastase resistant.
In severely involved areas, which are most common in erythropoietic protoporphyria, the perivascular mantles of ... with Sudan IV or Sudan black B. In addition, the PAS-positive dermal-epidermal basement membrane zone may be thickened.
In areas of sclerosis, which occur especially in porphyria cutanea tarda, the collagen bundles are thickened.

The bullae, which are most common in porphyria cutanea tarda and least common in erythropoietic protoporphyria...
dermal papillae often extend irregularly from the floor of the bulla into the bulla cavity (104,122). This phenomenon, referred to as festooning, is explained by the rigidity of the upper dermis. Eosinophilic material within and around the capillary walls in the papillae and the papillary dermis.

The epidermis forming the roof of the blister often contains eosinophilic bodies that are elongate and sometimes intracellular or extracellular; and (c) electron-dense material thought to be of basement membrane origin (124).
Pathogenesis. The substance around dermal vessels has the appearance of hyalin because it consists of homogeneous, eosinophilic material, which is absent in large amounts in the dermis of lipoid proteinosis and produced by fibroblasts as amorphous material.

On electron microscopic examination, concentric duplications of the basement membrane around the dermal blood vessels are observed. Intermingled filamentous and amorphous material is seen throughout the upper dermis and even in the mid dermis.
Proof that the perivascular material in porphyria represents excessively synthesized basement membrane material.
In the majority of patients, direct immunofluorescence testing has revealed the presence of immunoglobulins, particularly IgM, in skin biopsies. The result is not the result of a typical immune complex phenomenon; rather, they are the result of “trapping” of immunoglobulins and complement in the filamentous material.

The enzymatic defect that causes each form of porphyria is known. Enzyme determination can be carried out on cultured skin fibroblasts, erythrocytes, or liver tissue.
Liver damage is generally mild and chronic in porphyria cutanea tarda. In erythropoietic protoporphyria, liver function tests may show abnormalities, including elevated levels of transaminases, bilirubin, and alkaline phosphatase. In patients with normal liver function tests, biopsy of the liver may show portal and periportal fibrosis.
Porphyria Cutanea Tarda = اﻠْآﺠﻠﺔ اﻠﺠﻠدﻳﺔ اﻠﺒورﻔﻳرﻳﺔ
Porphyria Cutanea Tarda
Pseudoporphyria Cutanea Tarda
In patients with chronic renal failure who are receiving maintenance hemodialysis, an eruption indistinguishable from...

Porphyria cutanea tarda = اﻠآﺠﻠﺔ اﻠﺠﻠدﻴﺔ اﻠﺒورﻔﻴرﻴﺔ
Pseudoporphyria cutanea tarda may also occur following the ingestion of certain drugs, such as furosemide, cimetidine, or chloroquine. In these cases, withdrawal of the drug is curative.

Histopathology

In patients with pseudoporphyria, the histologic picture is characteristic and easily recognizable. The histologic picture is indistinguishable from that seen in mild cases of porphyria. The dermis shows a dense, chronic infiltrate of lymphocytes and plasma cells around the blood vessels. In addition, there may be an expanded reticular dermis with festooned dermal papillae. Blisters are usually situated above the PAS-positive basement membrane zone.
immunoglobulins are often observed in vessel walls and at the dermal-epidermal junction. Complement is
Porphyria Cutanea Tarda
Porphyria Cutanea Tarda