Seven types of porphyria are recognized. The light sensitivity in the six types with cutaneous lesions is caused by the presence of porphyrin molecules. These wavelengths lie in the 400-nm range, representing long-wave ultraviolet light (UVA) and visible light.
In \textit{erythropoietic porphyria}, a very rare disease that typically develops during infancy or childhood, recurrent vesiculobullous eruptions in skin lesions may appear. 

In \textit{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 manifestation of dermatologic porphyria can occur without any precipitating factor. In most instances, in addition to the inherited enzymatic defect, an acquired factor, such as a drug, can precipitate the disorder.
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 mantle of the arterial walls may be intensified 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 thicker.

The bullae, which are most common in porphyria cutanea tarda and least common in erythropoietic protoporphyria, arise...
Porphyria Cutanea Tarda = اﻠآﺠﻠﺔ اﻠﺠﻠدﻴﺔ اﻠﺒورﻔﻴرﻴﺔ

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 induced by the presence of 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 intracellularly or extracellularly; 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, amorphous material. This material is produced by fibroblasts as an amorphous substance in lipoid proteinosis.

On electron microscopic examination, concentric duplications of the basement membrane around dermal blood vessels are observed. This is interspersed with intermingled filamentous and amorphous material 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 IgG, that are deposited in the skin and are thought to be responsible for the characteristic photosensitivity of porphyria cutanea tarda. This 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 deficiencies lead to the accumulation of porphyrins, which are then excreted in the urine and can cause photodermatitis. The specific enzyme deficiencies vary depending on the type of porphyria, but all lead to increased porphyrin production.
Liver damage is generally mild and chronic in porphyria cutanea tarda. In erythropoietic protoporphyria, liver damage can be more severe.
Porphyria Cutanea Tarda
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Pseudoporphyria Cutanea Tarda
In patients with chronic renal failure who are receiving maintenance hemodialysis, an eruption indistinguishable from...
Pseudoporphyria cutanea tarda may also occur following the ingestion of certain drugs, such as furosemide, and is often seen in patients undergoing chemotherapy for malignancies. The histologic picture in pseudoporphyria is indistinguishable from that seen in mild cases of porphyria. In affected patients, blisters are typically seen above the PAS-positive basement membrane zone.
immunoglobulins are often observed in vessel walls and at the dermal-epidermal junction. Complement is