There are four forms of calcinosis cutis: metastatic calcinosis cutis, dystrophic calcinosis cutis, idiopathic calcinosis cutis, and subepidermal calcified nodule.
**Metastatic Calcification**

Metastatic calcification develops as the result of hypercalcemia or hyperphosphatemia. Hypercalcemia may result from (a) primary hyperparathyroidism, (b) renal failure, or (c) malignancy.
and phosphorus from bone. The demineralization of bone causes both osteodystrophy and metastatic calcification.
Metastatic calcification most commonly affects the media of the arteries and the kidneys. In addition, other visceral organs, such as the myocardium, the stomach, and the lungs, may be involved.

Metastatic calcification in the subcutaneous tissue is occasionally observed in association with renal hyperparathyroidism, in uremia, in hypervitaminosis D, and as the result of excessive intake of milk and alkali but rarely in primary hyperparathyroidism. Palpable, hard nodules, occasionally of considerable size, are located mainly in the vicinity of the large joints. With an increase in size, the nodules may become fluctuant.
Calciphylaxis is a life-threatening condition in which there is progressive calcification of small- and medium-sized vessels of the skin. Glycoproteins (matrix G1a protein and glycopontin) likely play a role in the development of vascular calcification.

Clinically, the lesions present as a panniculitis or vasculitis. Bullae, ulcerations, or a livedo reticularis-like pattern of gangrene, sepsis, pancreatitis, and multisystem organ failure contribute to an overall mortality of >60%.
Instances of cutaneous metastatic calcinosis. Most reports have concerned patients with renal hyperparathyroidism and osteodystrophy. The cutaneous lesions are usually small, asymptomatic, and scaly. They may be confused with psoriasis or lichen planus. In some cases, the lesions are more extensive, involving large areas of the skin or even the entire body. The cutaneous lesions may be associated with other systemic manifestations of hyperparathyroidism, such as osteitis fibrosa, bone pain, and hypercalcemia.

Mural calcification of arteries and arterioles in the deep dermis or subcutaneous tissue occurs rarely in primary hyperparathyroidism but somewhat more frequently in secondary hyperparathyroidism subsequent to renal disease. This may lead to occlusion of these vessels and to infarctive ulcerations, especially on the legs.
Histopathology

Calcium deposits are recognized easily in histologic sections, because they stain deep blue with H&E. They stain black in silver stain preparations. Calcification can evoke a foreign body reaction; thus, giant cells, an inflammatory infiltrate, and fibrosis may be present around them.
In areas of infarctive necrosis, as a result of calcification of dermal or subcutaneous arteries or arterioles.

The histologic changes in calciphylaxis include calcium deposits in the subcutis, chiefly within the walls of small- and medium-sized arteries or arterioles.
It is particularly important that these findings be recognized in order that appropriate therapy, which often includes parathyroidectomy, might be instituted immediately.
**Dystrophic Calcinosiis Cutis**

In dystrophic calcinosiis cutis, the calcium is deposited in previously damaged tissue. The values for serum...
Calcinosis universalis occurs as a rule in patients with dermatomyositis, but exceptionally it has also been observed in patients with systemic sclerosis. In dermatomyositis, if the patient survives, the nodules of dystrophic calcinosis gradually resolve.

Calcinosis circumscripta
Calciosis Cutis = اﻠﺠﻠدﻲ اﻠﻜﻠاﺲ occurs as a rule in patients with systemic scleroderma; rarely, however, it may be

often referred to as the Thibierge-Weissenbach syndrome or as the CREST syndrome, because...
Lupus erythematosus is only rarely associated with dystrophic calcinosis cutis. In addition to occurring in subcutaneous fat necrosis of the newborn and, rarely, in the subcutaneous nodules occurring in Ehlers-Danlos disease.

**Histopathology.**

As in metastatic calcinosis cutis, the calcium in dystrophic calcinosis cutis usually...
Idiopathic Calcinosis Cutis

Even though the underlying connective tissue disease in some instances of dystrophic calcinosis cutis may... remain cases of idiopathic calcinosis cutis that resemble dystrophic calcinosis cutis but show no underlying disease.

One entity is regarded as a special manifestation of idiopathic calcinosis cutis: tumoral calcinosis. It consists... the resemblance of tumoral calcinosis to the dystrophic calcinosis universalis observed with dermatomyositis is great.
Tumoral calcinosis shows in the subcutaneous tissue large masses of calcium surrounded by a foreign body reaction. Discharge of calcium may take place through areas of ulceration or by means of transepidermal elimination.
Pathogenesis

Two authors have studied lesions of idiopathic calcinosis cutis by electron microscopy.
Idiopathic Calcinosis of the Scrotum

Idiopathic calcinosis of the scrotum consists of multiple asymptomatic nodules of the scrotal skin. The nodules begin to ... or in early adult life, increase in size and number, and sometimes break down to discharge their chalky contents.
Calcinosi Cutis = ﺗﺤﻠل ﻣﺎدة ﻣﺤﻠل
Histopathology
At one time, the accepted view was that some of the calcific masses in calcinosis...
according to this view, calcinosis of the scrotum represents the end stage of dystrophic calcification of skin cysts. It is likely that authors who found no cyst walls were examining old lesions.
Calcinosis Cutis = اﻠﺠﻠدﻲ اﻠﻜﻠاﺲ
Subepidermal Calcified Nodule

In subepidermal calcified nodule, also referred to as cutaneous calculi, usually a single small, raised, hard nodule is present. Occasionally, there are two or three nodules, and in some patients, a nodule is present at birth or even innumerable nodules. Most patients are children; however, in some patients, a nodule is present.
does not appear until adulthood. In most instances, the surface of the nodule is verrucous, but it may be smooth. The most common location of the nodule is the face.

**Pathogenesis.** The primary event seems to be the formation of large, homogeneous masses that undergo calcification and break up into numerous calcified globules. The origin of the homogeneous masses is obscure. It is not likely that they originate from a specific preexisting structure, such as sweat ducts or nevus cells as has been assumed.