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

Metastatic calcification develops as the result of hypercalcemia or hyperphosphatemia. Hypercalcemia may result from (a) increased intestinal absorption of calcium, (b) increased calcitonin, or (c) increased bone resorption. Hyperphosphatemia may result from (a) increased phosphate intake, (b) increased phosphate reabsorption, (c) decreased renal excretion, or (d) decreased bone resorption.
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. This condition is characterized by the formation of calcium deposits in the walls of these blood vessels, leading to reduced blood flow and tissue ischemia.

Clinically, the lesions present as a panniculitis or vasculitis. Bullae, ulcerations, or a livedo reticularis-like pattern may be observed. The disease is associated with a high mortality rate, with complications such as gangrene, sepsis, pancreatitis, and multisystem organ failure contributing to the overall mortality of more than 60%.
Instances of cutaneous metastatic calcinosis. Most reports have concerned patients with renal hyperparathyroidism, but symptoms of cutaneous metastatic calcinosis have also been reported in patients with primary hyperparathyroidism.

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.
Histopathology

Calcium deposits are recognized easily in histologic sections, because they stain deep blue with H&E. They stain black with alcian blue, and may also appear with the von Kossa and silver impregnation techniques. Calcium deposits 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 disease process leads to lumina occlusion or severe distortion of the vascular lumen. Mural calcification often is most pronounced in the internal elastic membranes of arteries or arterioles.

The histologic changes in calciphylaxis include calcium deposits in the subcutis, chiefly within the walls of small- and medium-sized blood vessels. The evolution of the lesion can include areas of necrosis with a clean background or accompanied by neutrophils.
It is particularly important that these findings be recognized in order that appropriate therapy, which often includes parathyroidectomy, might be instituted immediately.
Dystrophic Calcinosis Cutis

In dystrophic calcinosis 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 lupus erythematosus and tendons. In dermatomyositis, if the patient survives, the nodules of dystrophic calcinosis gradually resolve.

Calcinosis circumscripta
Calcinosis Cutis = اﻠﺠﻠدﻲ اﻠﻜﻠاﺲ
occurs as a rule in patients with systemic scleroderma; rarely, however, it may be observed in patients with widespread... of calcinosis, systemic scleroderma manifests itself as acrosclerosis. The association of acrosclerosis and calcinosis is 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...
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 form in early childhood, increase in size and number, and sometimes break down to discharge their chalky contents.
Calcinosi Cutis = اﻠﺠﻠدﻲ اﻠﻜﻠاﺲ
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 scrotal sebaceous cysts.
Subepidermal Calcified Nodule

In subepidermal calcified nodule, also referred to as cutaneous calculi, usually a single small, raised, hard nodule is present. Occasionally, however, there are two or three nodules, and in some cases, there may be even innumerable nodules. Most patients are children; however, in some patients, a nodule is present at birth or even innumerable nodules.
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.