Calcinosis Cutis

There are four forms of calcinosis cutis: metastatic calcinosis cutis, dystrophic calcinosis cutis, idiopathic calcinosis cutis, and subepidermal calcified nodule.
Metastatic calcification develops as the result of hypercalcemia or hyperphosphatemia. Hypercalcemia results from increased bone resorption or decreased renal excretion of calcium. Increased serum calcium stimulates parathyroid secretion, leading to secondary hyperparathyroidism and to resorption of calcium.
and phosphorus from bone. The demineralization of bone causes both osteodystrophy and metastatic calcinosis cutis.
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 have varied from asymptomatic roughening of the skin to deep ulcerations and subcutaneous masses. Clinical features include: variably sized, firm, subcutaneous nodules; firm, subcutaneous plaques; or small papules or nodules from which a granular, white, curdled material can be expressed.

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, or to renal allograft. 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 with PTAH. Many granulomas are described which may 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 lumina. 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 arteries and arterioles.
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
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Calcinosis universalis occurs as a rule in patients with dermatomyositis, but exceptionally it has also been observed in patients with systemic diseases which involve the tendons. In dermatomyositis, if the patient survives, the nodules of dystrophic calcinosi gradually resolve.

Calcinosis circumscripta
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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 the CREST syndrome, because...
Lupus erythematosus is only rarely associated with dystrophic calcinosi 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 calcinosi cutis, the calcium in dystrophic calcinosi cutis usually
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 of a slowly growing mass of bone-like material.
Histopathology.

Tumoral calcinosis shows in the subcutaneous tissue large masses of calcium salts, which are 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 childhood or in early adult life, increase in size and number, and sometimes break down to discharge their chalky contents.
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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 cysts. As the cysts age and calcify, lose their cyst walls. It is likely that authors who found no cyst walls were examining old lesions.
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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 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.