The Investigations of Calcium abnormalities

Essential for understanding this presentation:

1) **Anatomy:** Parathyroid gland, skin, liver, kidney, intestine, bone
2) **Biochemistry:** Hormones produced by the Parathyroid gland & Kidney. Bone metabolism
3) **Physiology:** Regulation and metabolism involving calcium

First then can one start on a journey to investigate abnormal s-calcium levels

The Investigations of Calcium abnormalities

Objectives:

1) Describe the mechanisms behind **hypocalcaemia** and **hypercalcaemia**.

2) Differentiate among **primary**, **secondary** and **tertiary** 'calcium' disorders.

The Investigations of Calcium abnormalities

*Discuss - based on the normal physiology - the **rationale** behind*

3) Symptoms of a dysregulated calcium.

4) The investigations dysregulated calcium.

The Investigations of Calcium abnormalities

Essential for understanding the investigations

1) **Anatomy:**

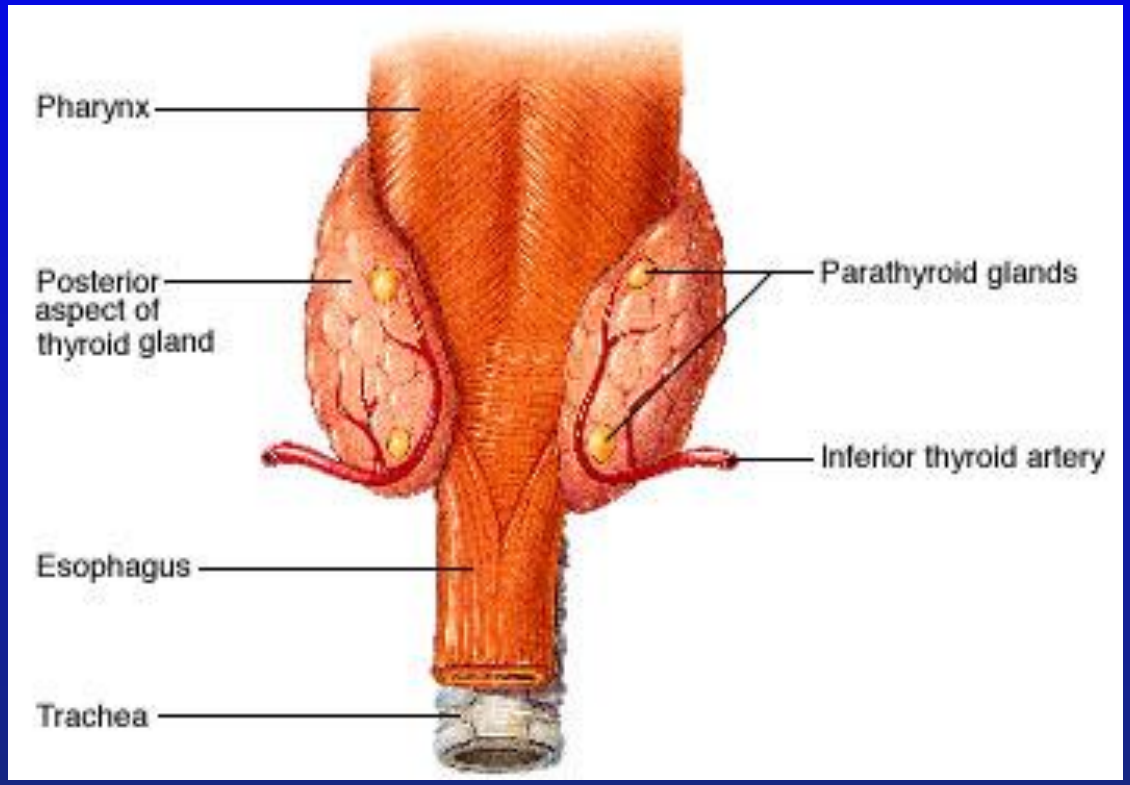
2) **Biochemistry:**

3) **Physiology:**

4) **Diseases**

Essential anatomy

Parathyroid gland



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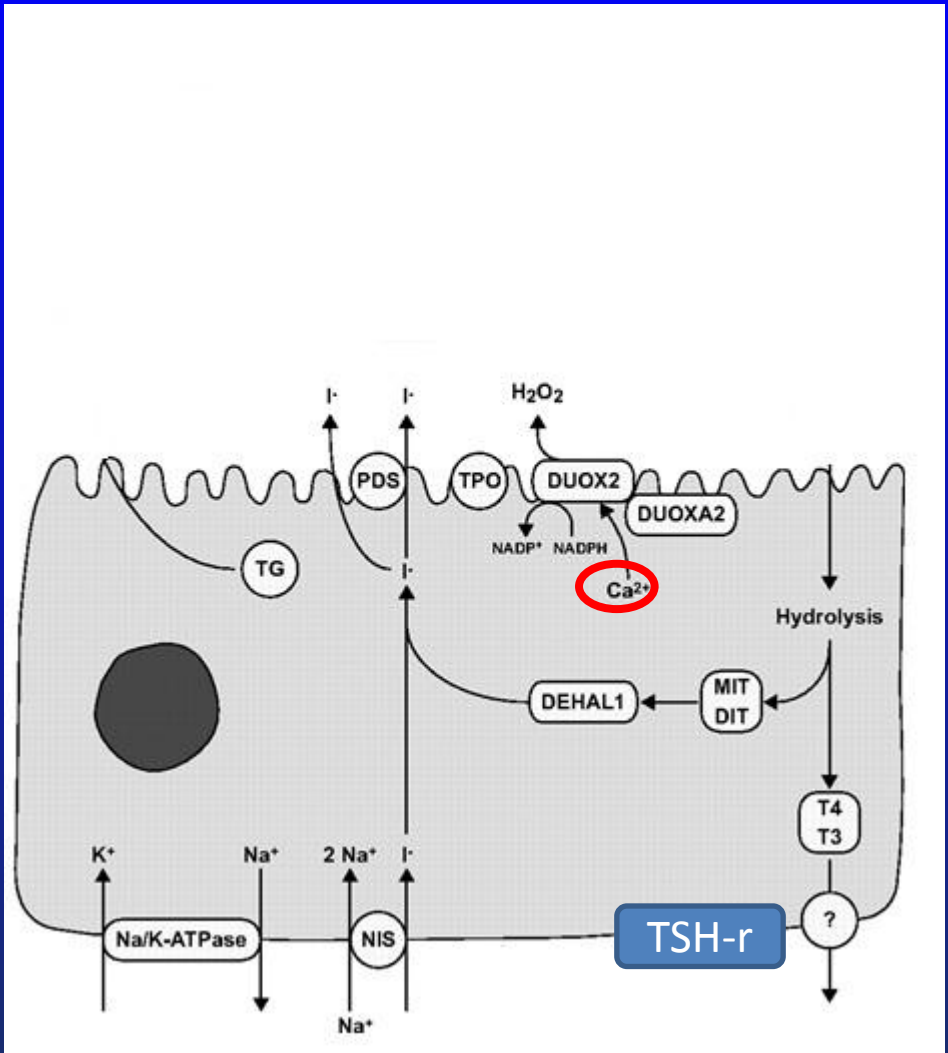
Essential biochemistry



Bizhanova A , Kopp P Endocrinology 2009;150:1084-1090

Remember calcium's role in cell signaling (This one is from the thyroid gland)

Clinical most important functions?

- Heart
- Muscles
- Nervous system
- Immune system
- Coagulation

Essential biochemistry

Calcium bound to proteins – mainly albumin app 50%

Free ionized calcium (Ca2++).
It is the physiologically active fraction

Measurements

- Total calcium
- Free ionized calcium

Essential biochemistry

H^+ affect the binding of calcium to protein
H^+ competes with Ca^{2+} for binding sites

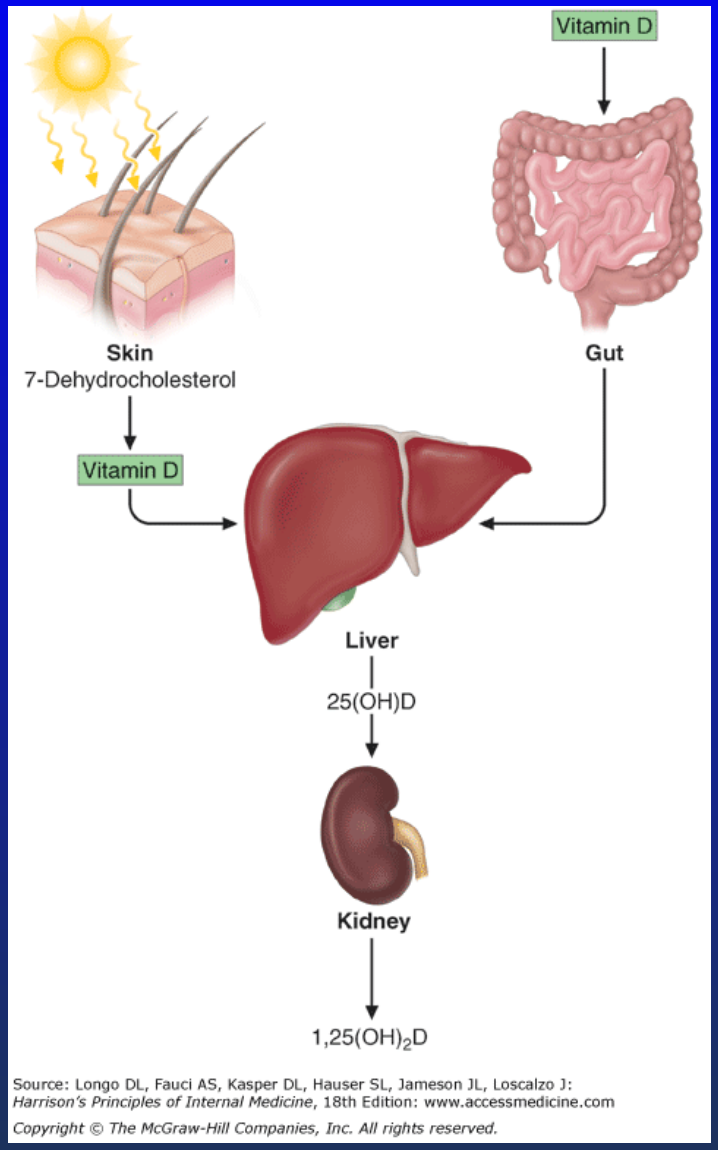
What are the implications?

The total calcium is not affected

If H^+ falls the patient might experience tetany despite a normal total plasma concentration

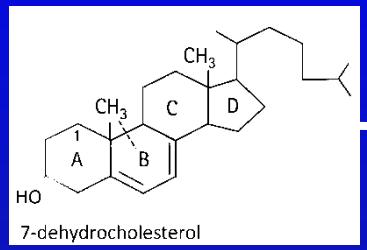
If H^+ increases the patient might loose calcium > osteomalacia

Essential biochemistry



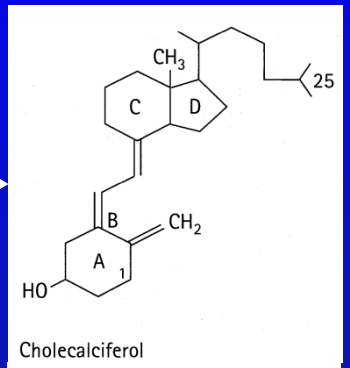
Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine*, 18th Edition: www.accessmedicine.com

Ergocalciferol (vitamin D_2)



7-dehydrocholesterol

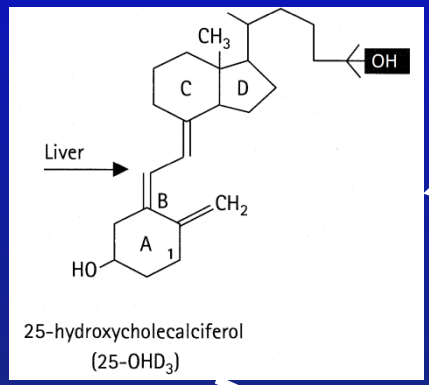
UV



Cholecalciferol

(Viatmin D_3)

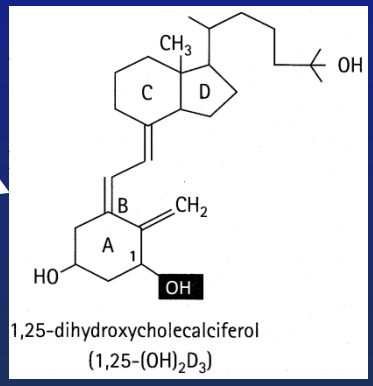
25-hydroxylase



25-hydroxycholecalciferol

(25-OHD_3)

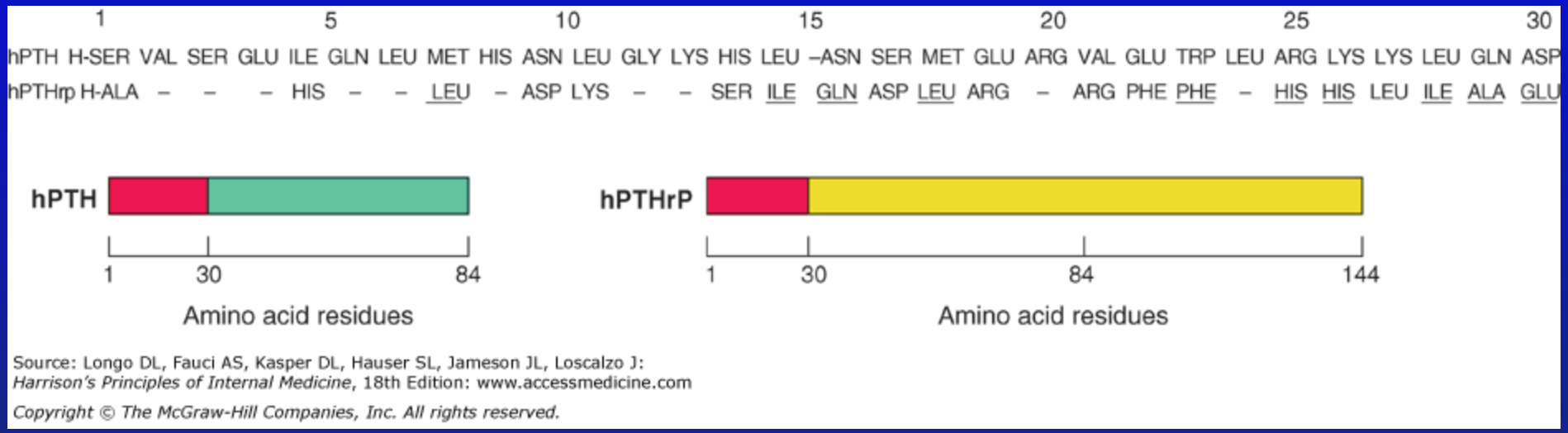
1-α-hydroxylase



1,25-dihydroxycholecalciferol

(1,25-$(OH)_2D_3$)

Essential biochemistry

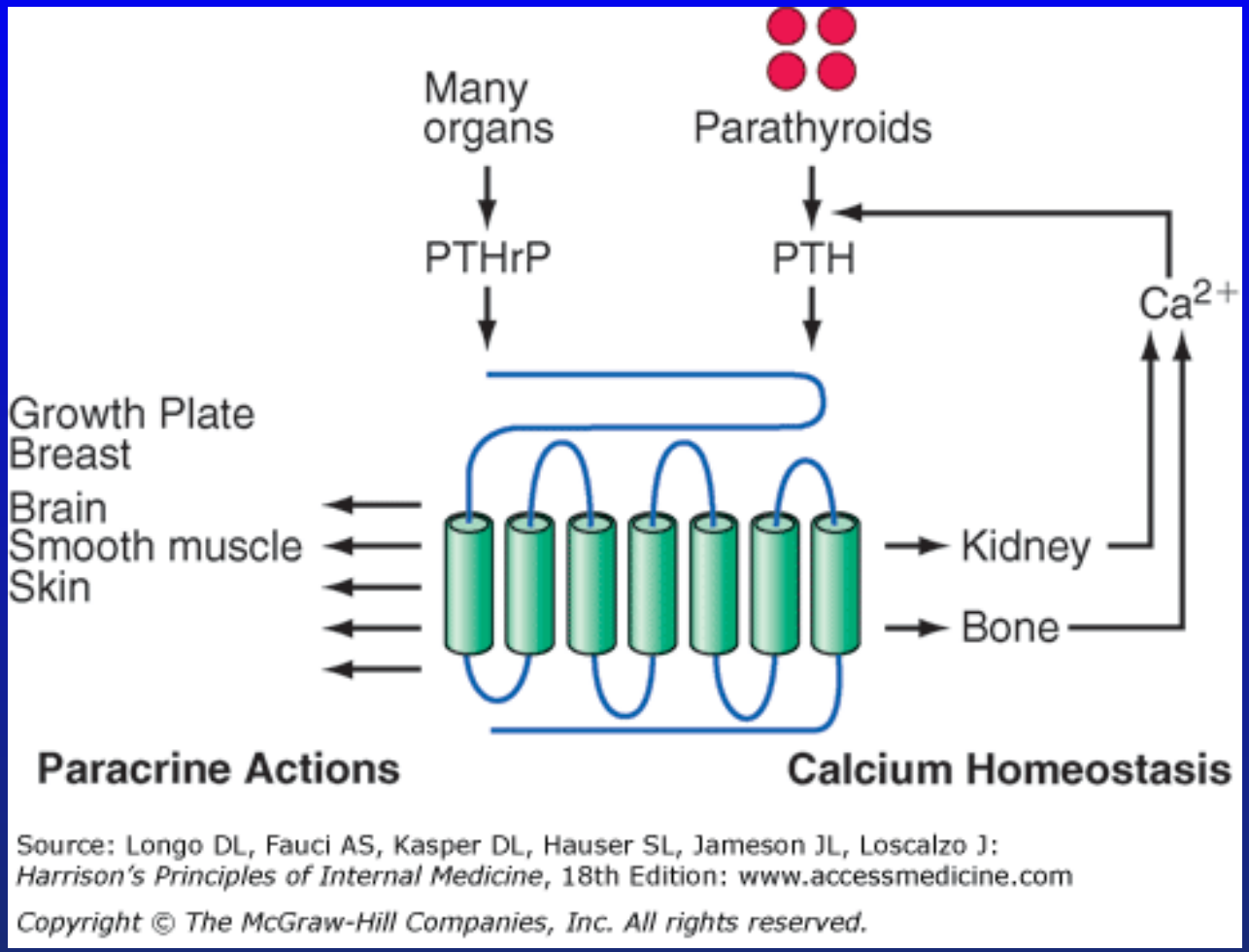


Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine*, 18th Edition: www.accessmedicine.com

hPTH = humane PTH

hPTHrP = humane PTH related peptide

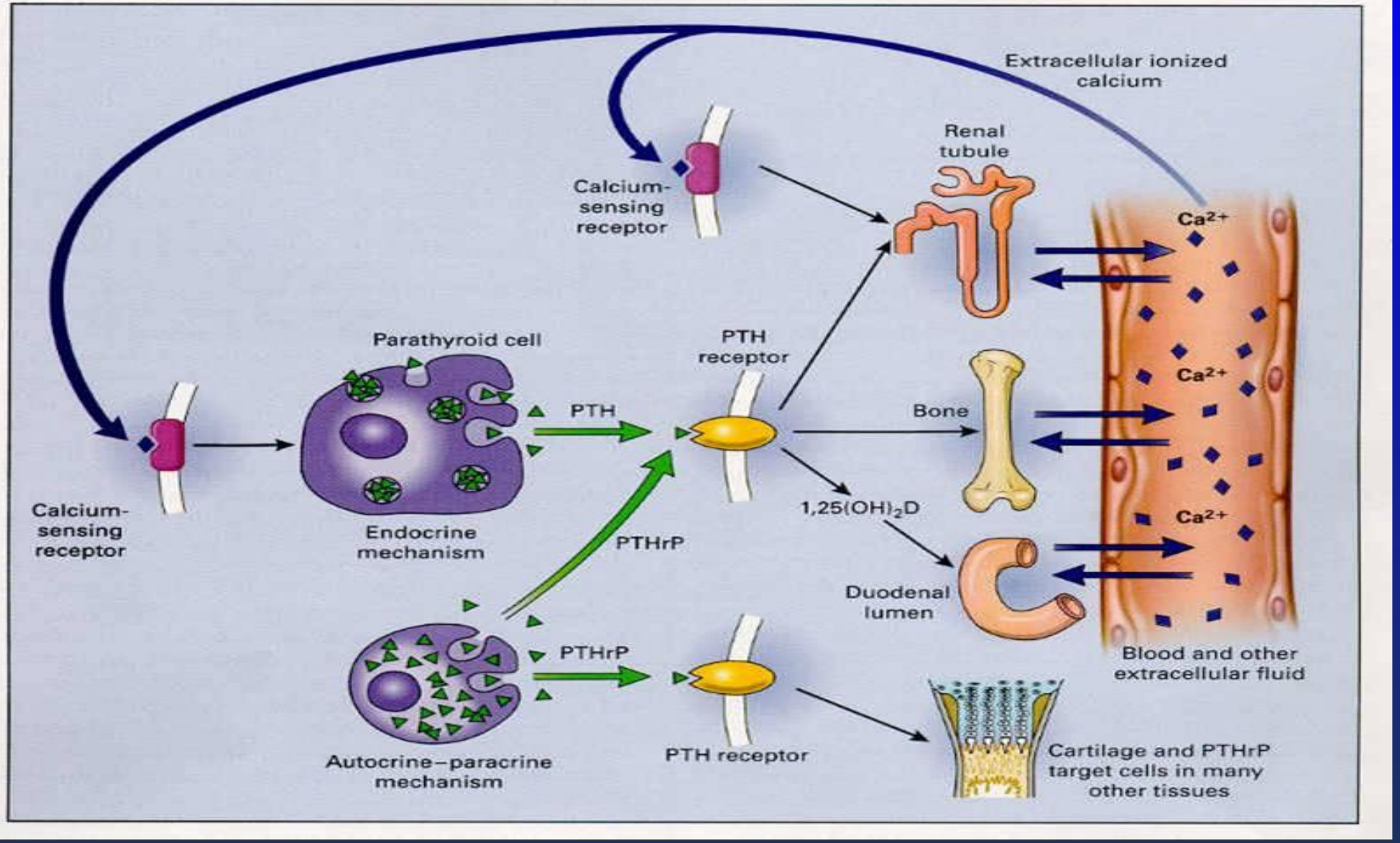
Essential biochemistry



hPTH = humane PTH

hPTHrP = humane PTH related peptide

A complex system



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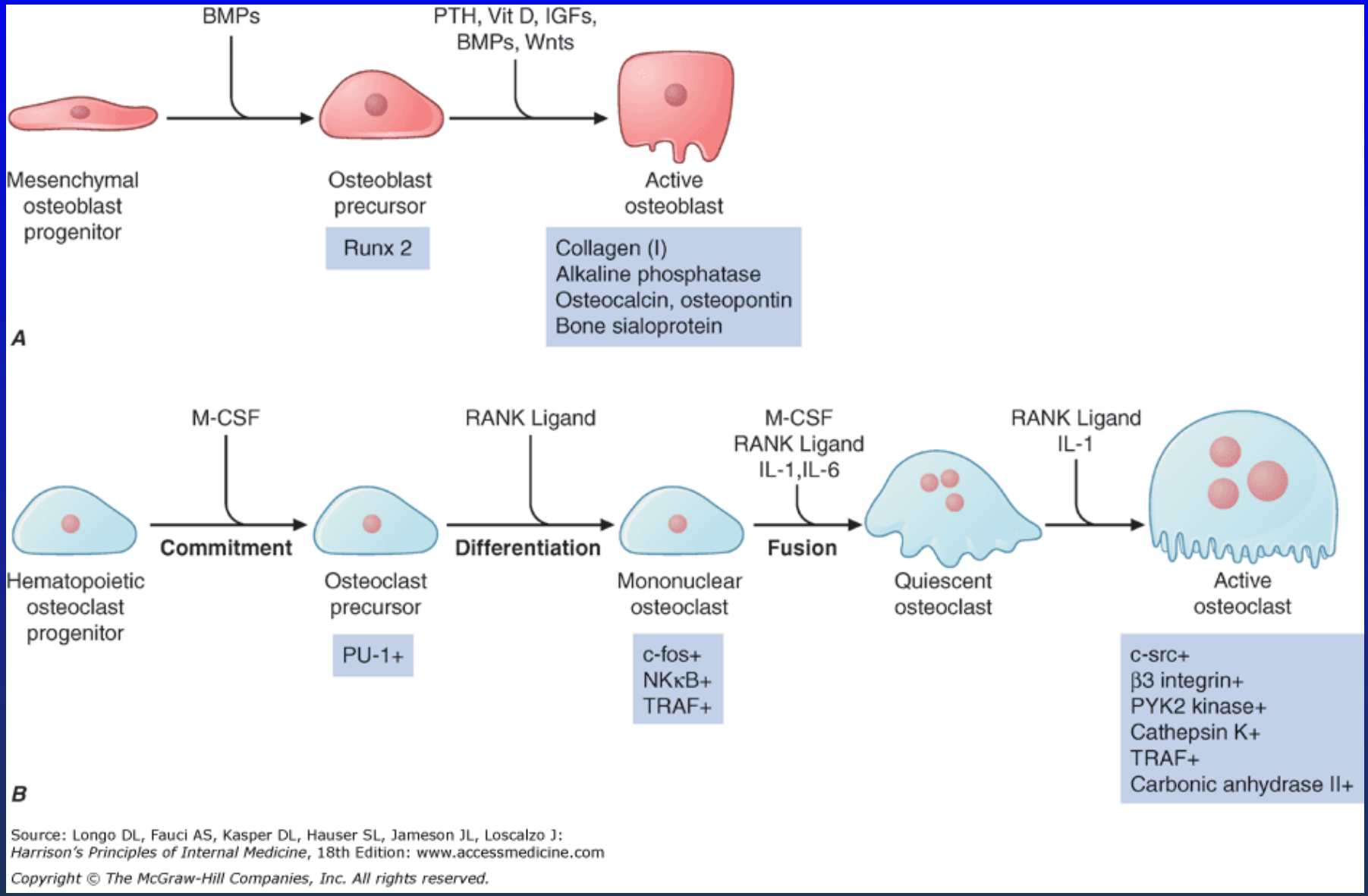
The bone component



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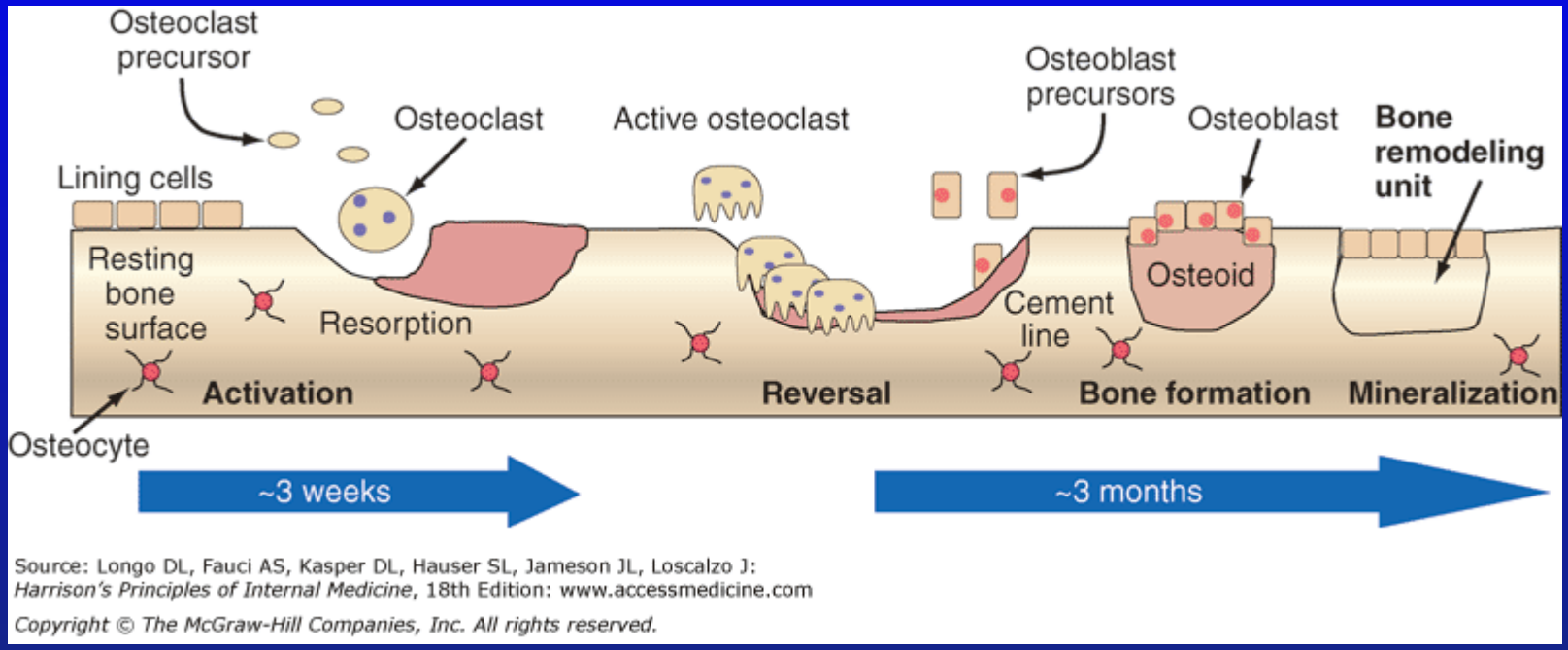
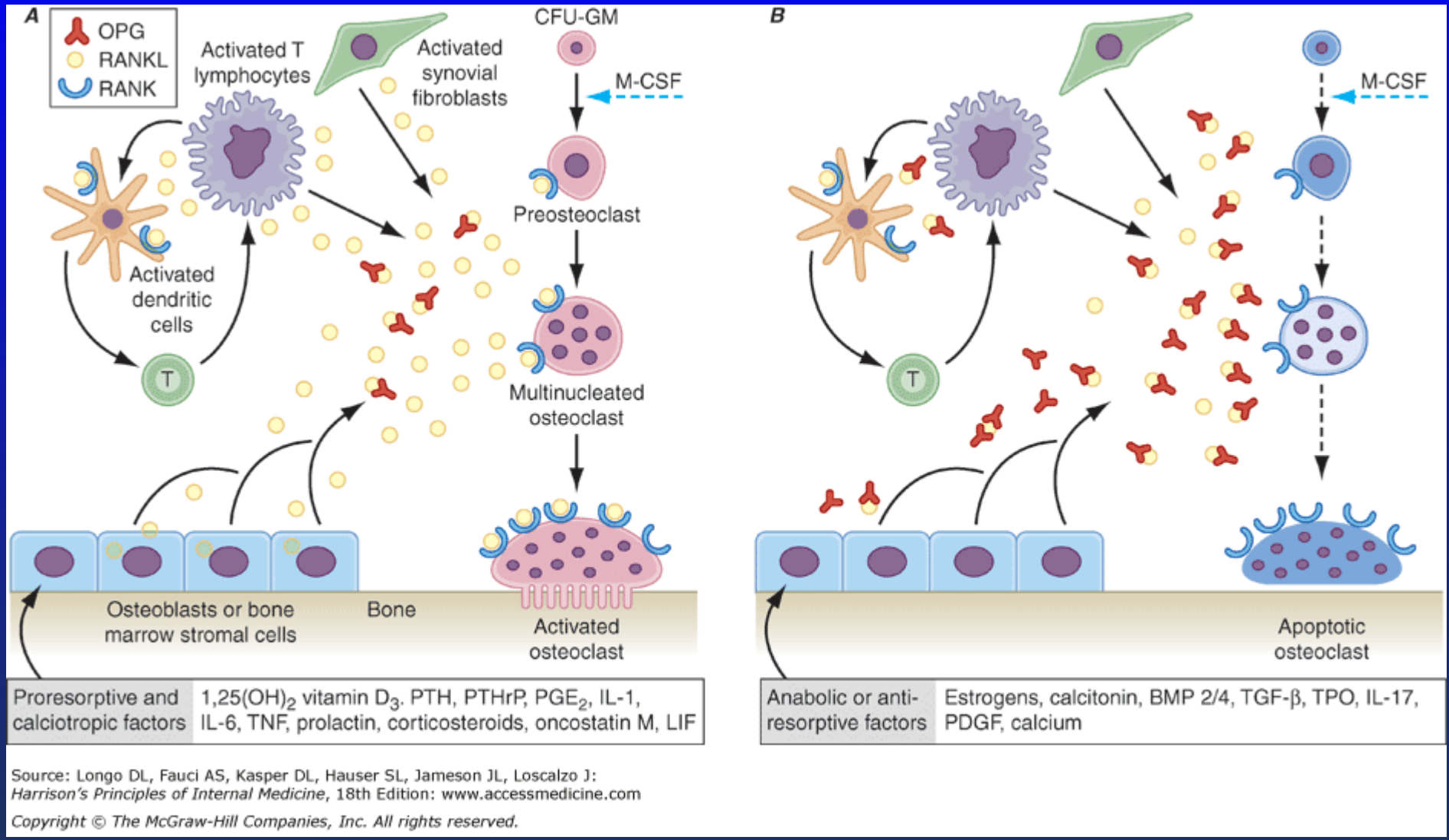
The bone component



Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine*, 18th Edition: www.accessmedicine.com

Don't forget bone remodeling formation is a constant process/ balance

The bone component



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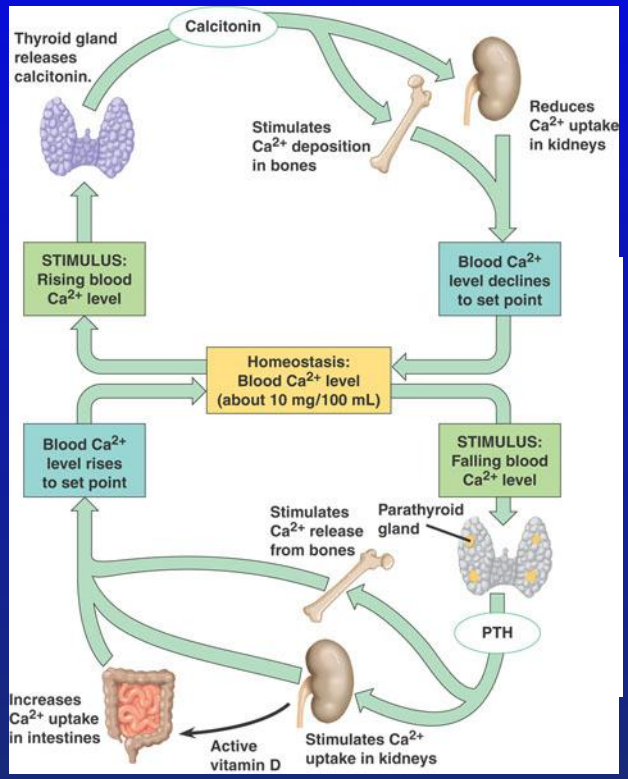
The Investigations of Calcium abnormalities



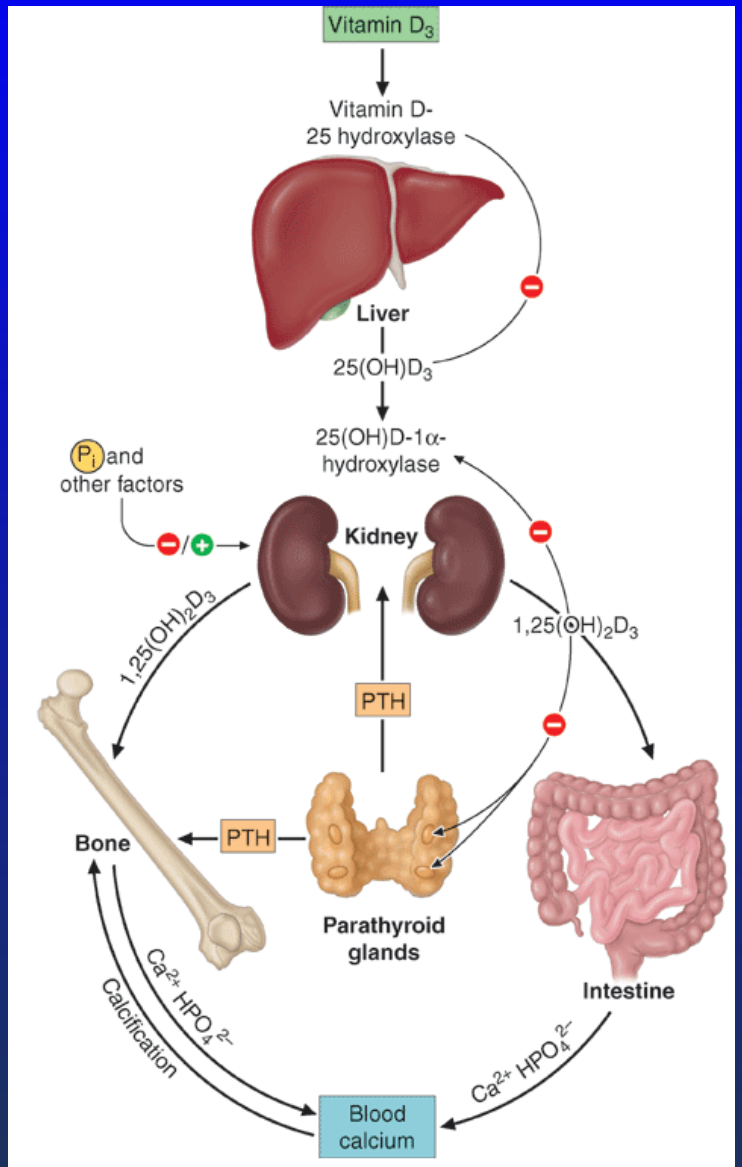
The Calcitonin regulation
Down-regulates calcium

The PTH regulation
Up-regulates calcium

What type of feedback is it ?

Classical negative feedback

The Investigations of Calcium abnormalities



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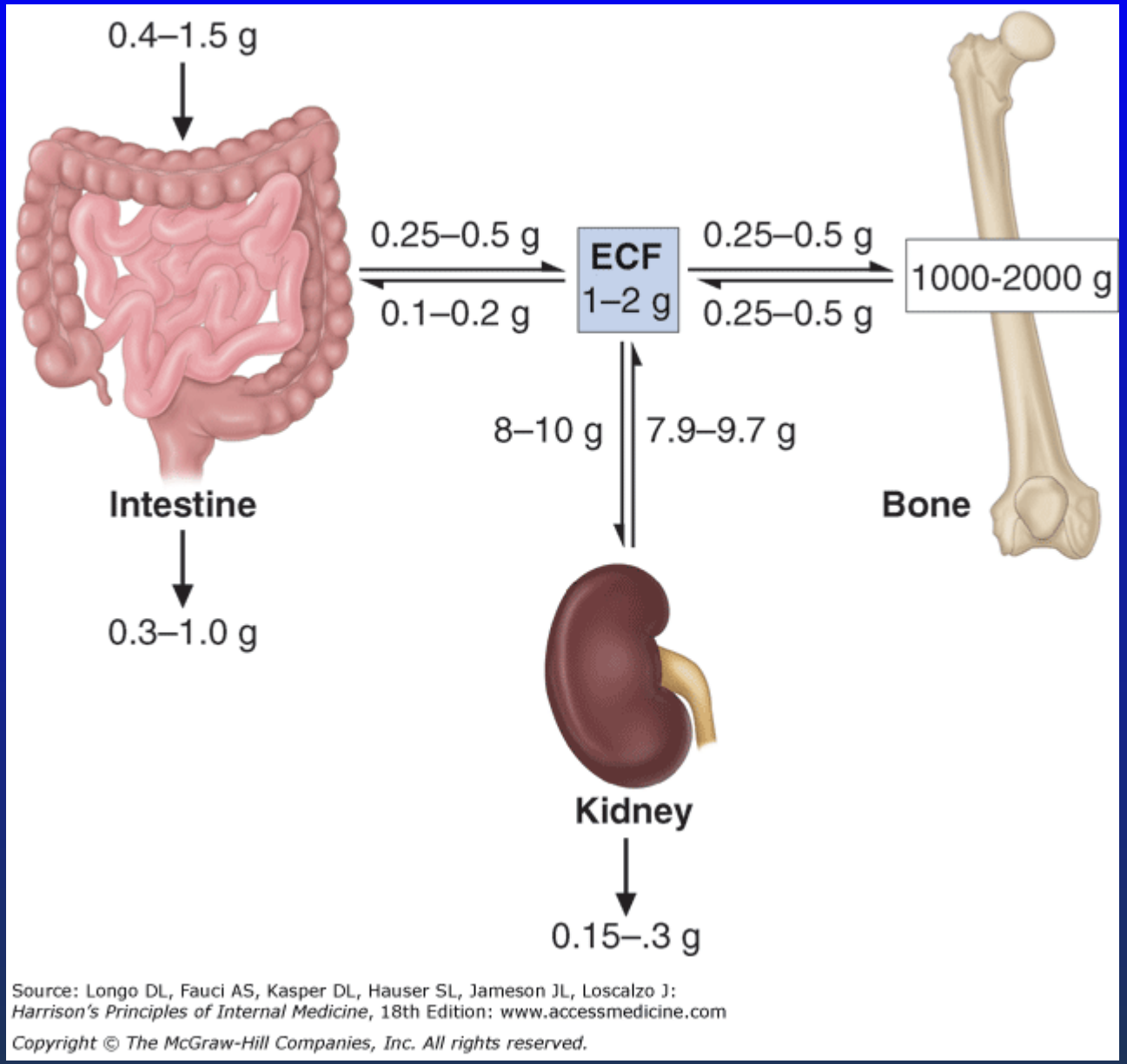
The Investigations of Calcium abnormalities



Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine*, 18th Edition: www.accessmedicine.com

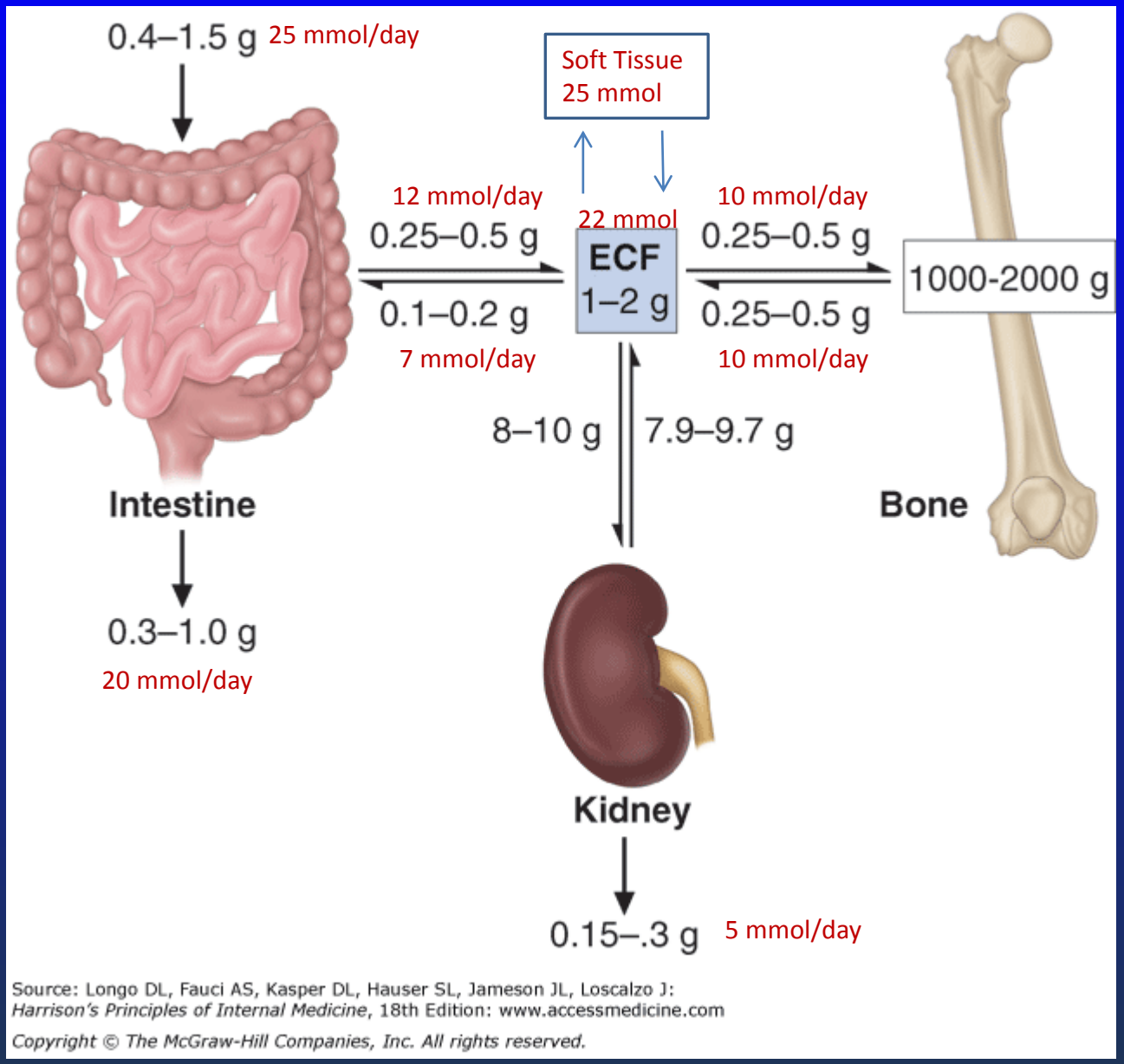
Remember it is a dynamic balance

Homeostasis

Plasma calcium is tightly controlled

2.15 -2.55 mmol/L

The Investigations of Calcium abnormalities



Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine*, 18th Edition: www.accessmedicine.com

With approximate daily turnover in mmol

Plasma calcium is tightly controlled

2.15 -2.55 mmol/L

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Hyper - & Hypo-'states'

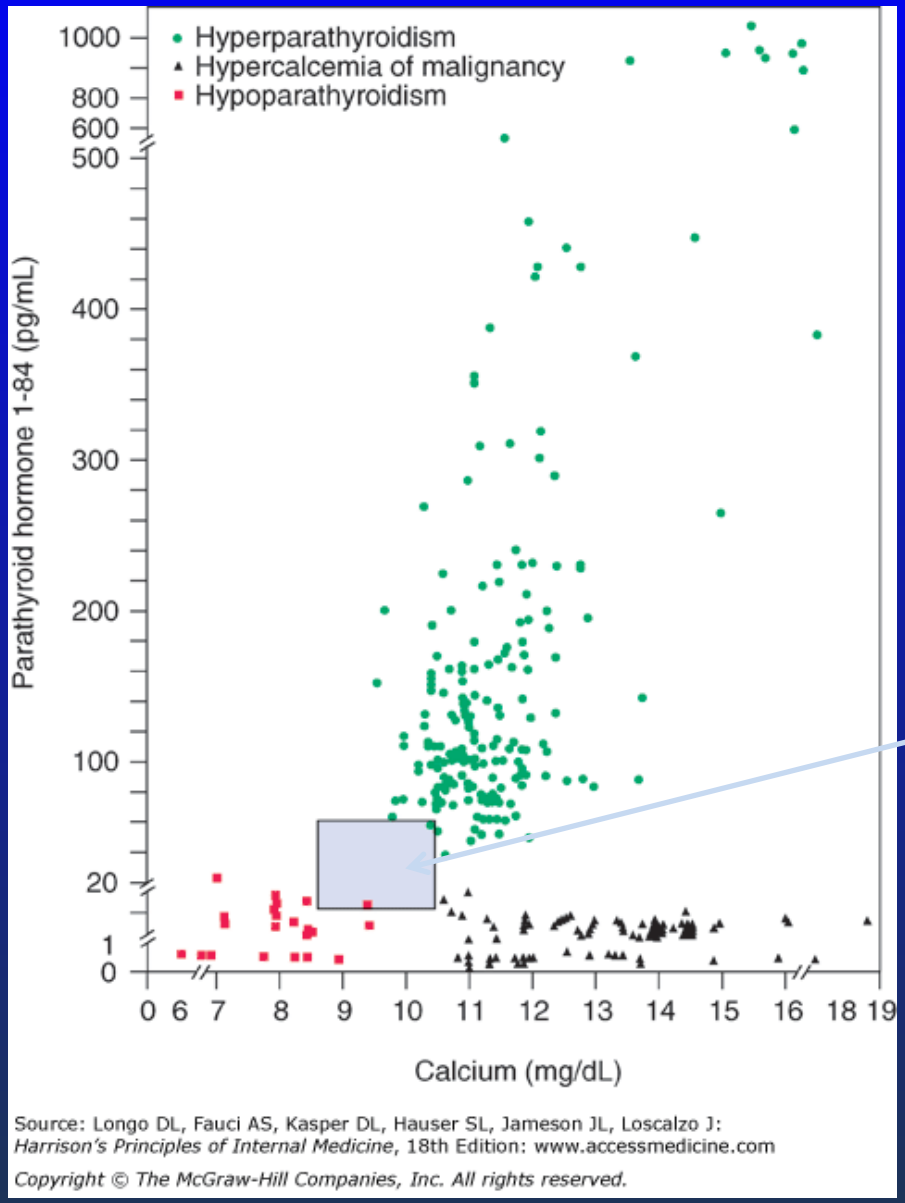
In principle only two things can go wrong:

Increased level (high concentration) of substance: **Hyper**…..aemia

Decreased level (low concentration) of substance : **Hypo**….. aemia

Of cause there can be many underlying causes: Tumor, starvation, infections …….

Hyper - & Hypo-'states'

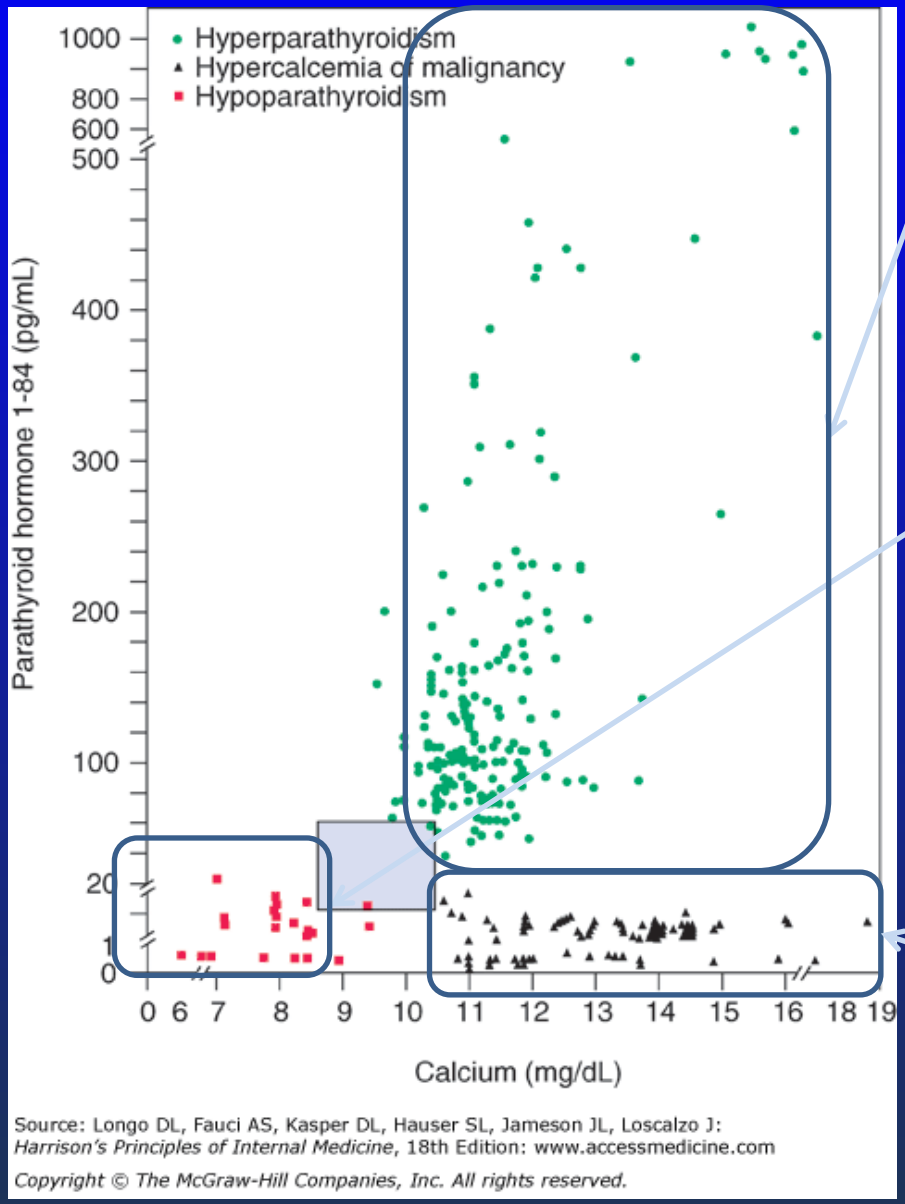


Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine*, 18th Edition: www.accessmedicine.com

Diagnosis using s-calcium and s-PTH

The box represent the upper and normal limits for s-calcium and/or s-PTH

Hyper - & Hypo-'states'



High PTH results in high calcium > hyperparathyroidism

Low PTH results in low calcium > hypoparathyroidism

High calcium with low PTH > the PTH response is adequate hence not a parathyroid problem

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Hypercalcemia

Hypercalcemia

Manifestations of hypercalcemia

Normal Laboratory: Serum calcium (total) 1.12-2.65mmol/L
Serum calcium (ionized) 1.0-1.25mmol/L

Inability to Concentrate Urine and Exposure of Kidney to Increased Concentration of Calcium
- Polyuria
- Increased thirst
- Flank pain
- Signs of acute renal insufficiency
- Signs of kidney stones

Neural and Muscle Effects (Decreased Excitability)
- Muscle weakness
- Ataxia, loss of muscle tone
- Lethargy
- Personality and behavioral changes
- Stupor and coma

Cardiovascular Effects
- Hypertension
- Shortening of the QT interval
- Atrioventricular block

Gastrointestinal Effects
- Anorexia
- Nausea, vomiting
- Constipation

Hypercalcemia

Urgent treatment is required

if - calcium > 3.5mmol/L
- clouding of consciousness or confusion is present.
- hypotension
- severe dehydration causing pre-renal failure

Management

•Rehydrate with iv Na saline (0.9%)

•Diuretics once patient is rehydrated (monitor electrolytes K+ and Mg++)

•If inadequate response give calcitonin, bisphosphonates, steroids.

Some Causes of hypercalcemia

Malignancy

- Bony metastases, e.g. breast, lung, prostate, kidney, thyroid.
- Solid tumors with humoral effects (Parathyroid hormone-related protein (PTHRP)).
- Hematological tumors, e.g. myeloma.

Parathyroid hormone abnormalities

- Primary hyperparathyroidism (adenoma, hyperplasia or associated with multiple endocrine neoplasias).
- Tertiary hyperparathyroidism

High bone turnover

- Thyrotoxicosis
- *Immobilization, e.g. with Paget's disease, long bed rest.*

High level of vitamin D

- Vitamin D toxicity.
- Granulomatous disease, e.g. sarcoidosis, tuberculosis.

Some Causes of hypercalcemia

Drugs

- Thiazides (reduced renal calcium excretion)
- Vitamin A toxicity.
- Milk-alkali syndrome (Burnett syndrome).

Familial hypocalciuric hypercalcemia

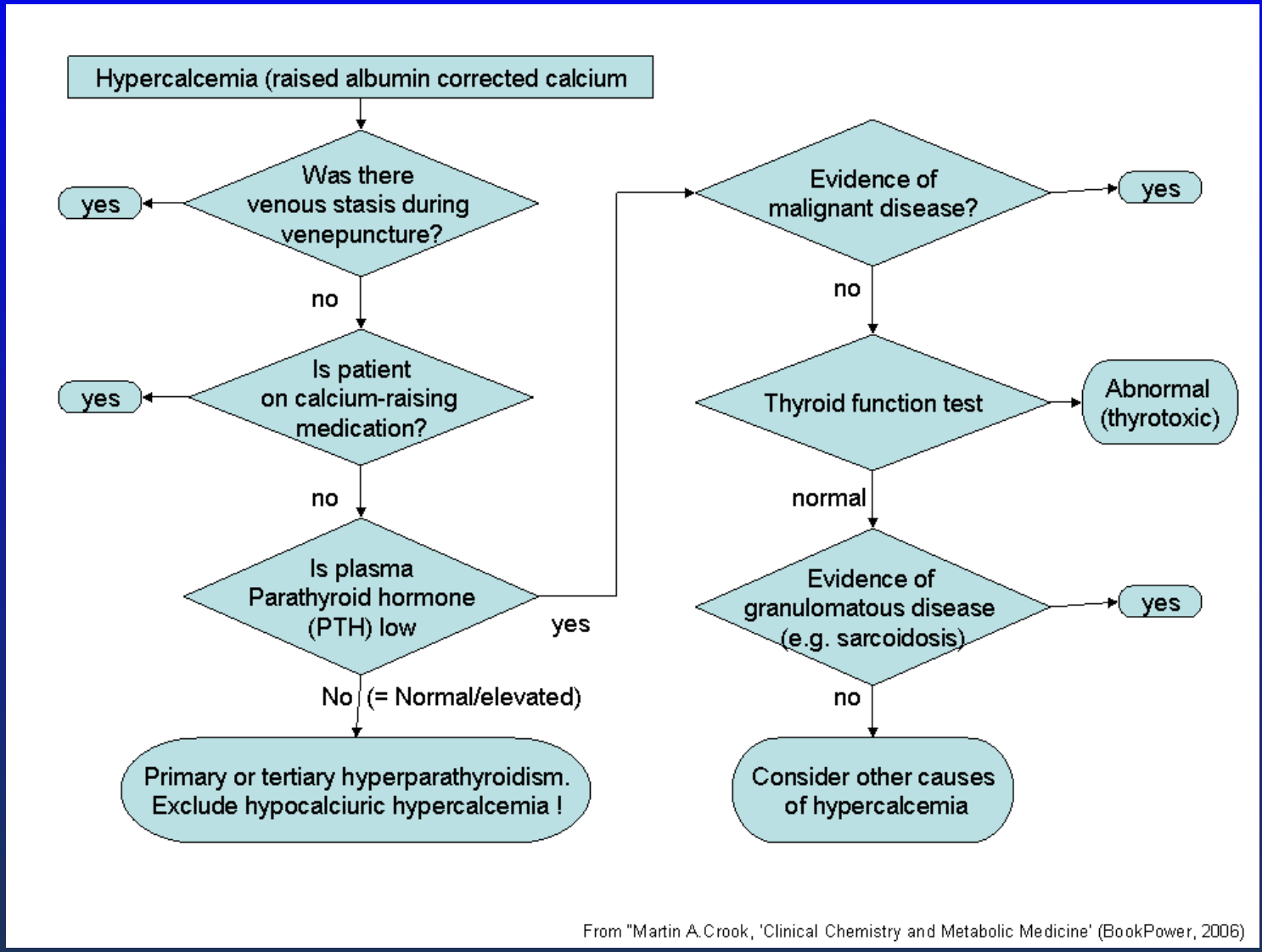
Other endocrine causes

- Adrenal insufficiency
- Acromegaly

Rarer causes, e.g.

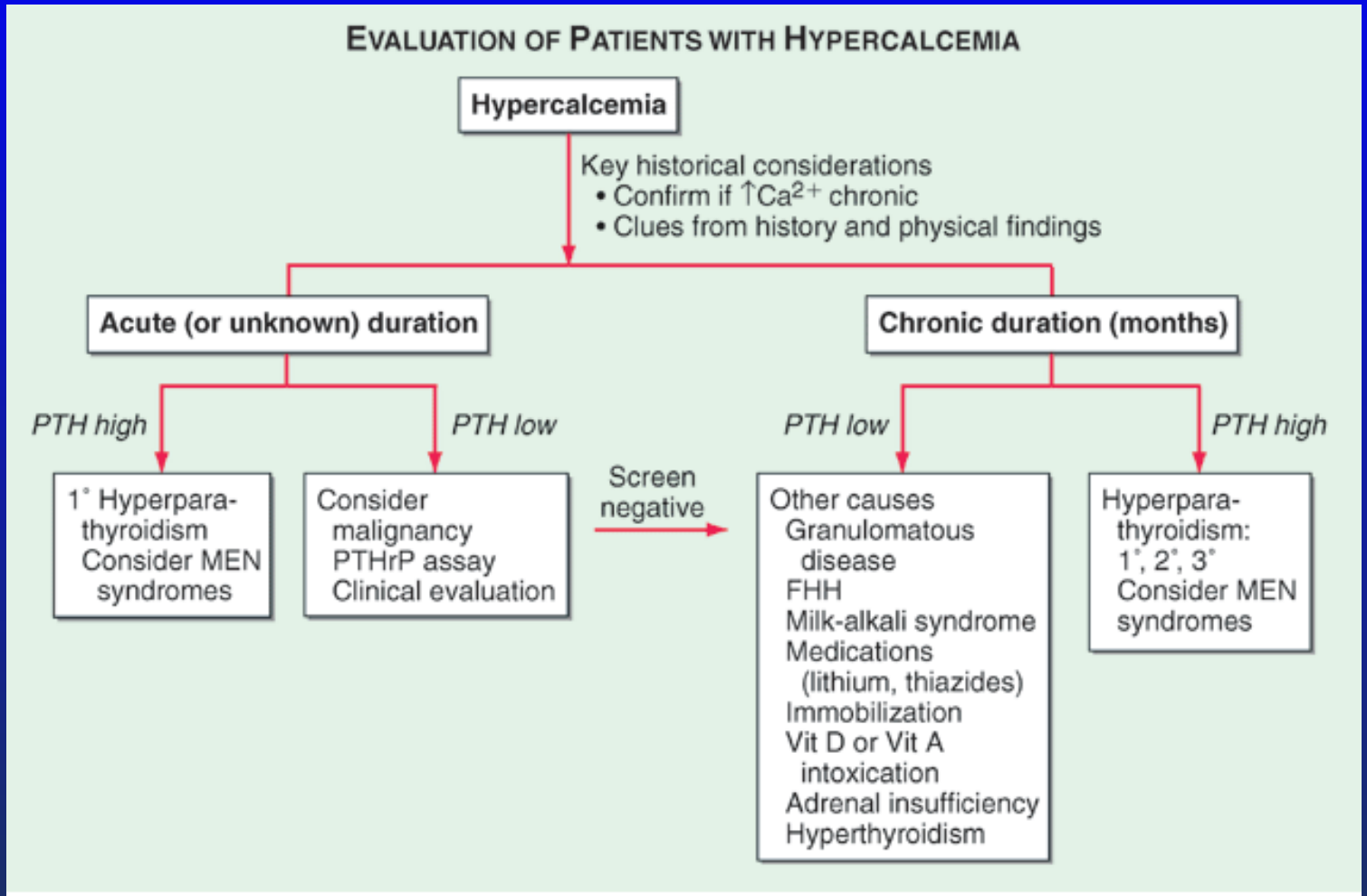
- William's syndrome.
- Human immunodeficiency virus infection.
- Leprosy.
- Histoplasmosis.
- Berylliosis.

Algorithm for hypercalcemia



From "Martin A.Crook, 'Clinical Chemistry and Metabolic Medicine' (BookPower, 2006)

Algorithm for hypercalcemia



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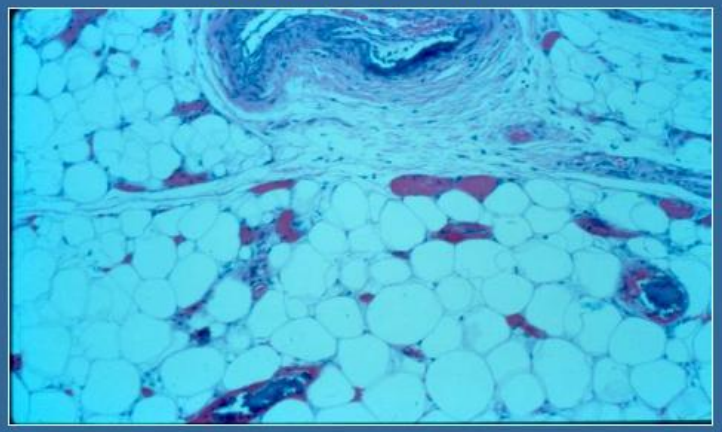
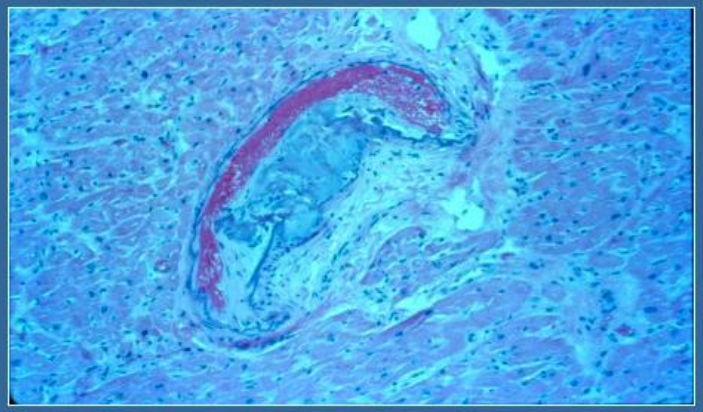
Calcium deposits in various tissues

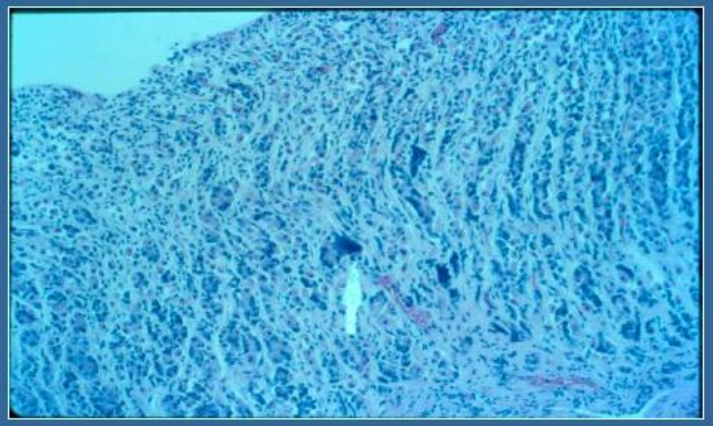
Calcium in skin



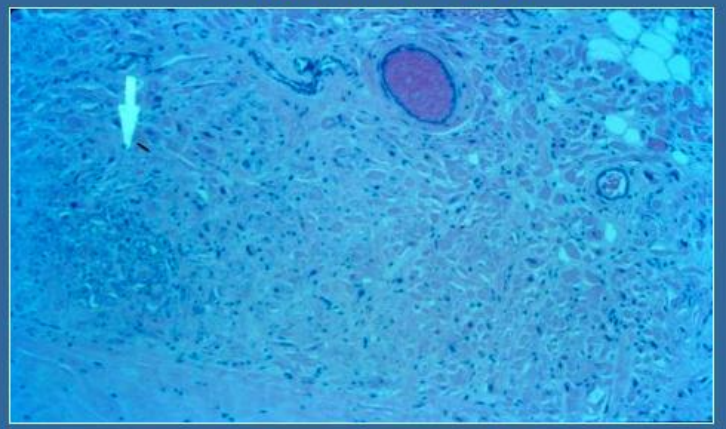
Vessel Calcification



Calcium in gastric mucosa



Calcium in AV node



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Hypercalcemia - Cases 1 to 8

Hypercalcemia: Case 1

Calciphylaxis presents as painful, indurated, focally necrotic, erythematous plaques that usually are on extremities. Secondary hyperparathyroidism resulting from chronic renal failure treated by dialysis is the most common association.

Calcification of small blood vessels, focal calcification of the fat, and vascular thrombosis are seen histopathologically. he degree of necrosis of the dermis and/or lobular panniculitis is variable within a given lesion, and this is reflected in the highly variable pathology seen in multiple biopsies of a lesion or from case to case. Either a large biopsy or multiple biopsies that include subcutis may be necessary to demonstrate the calcification.

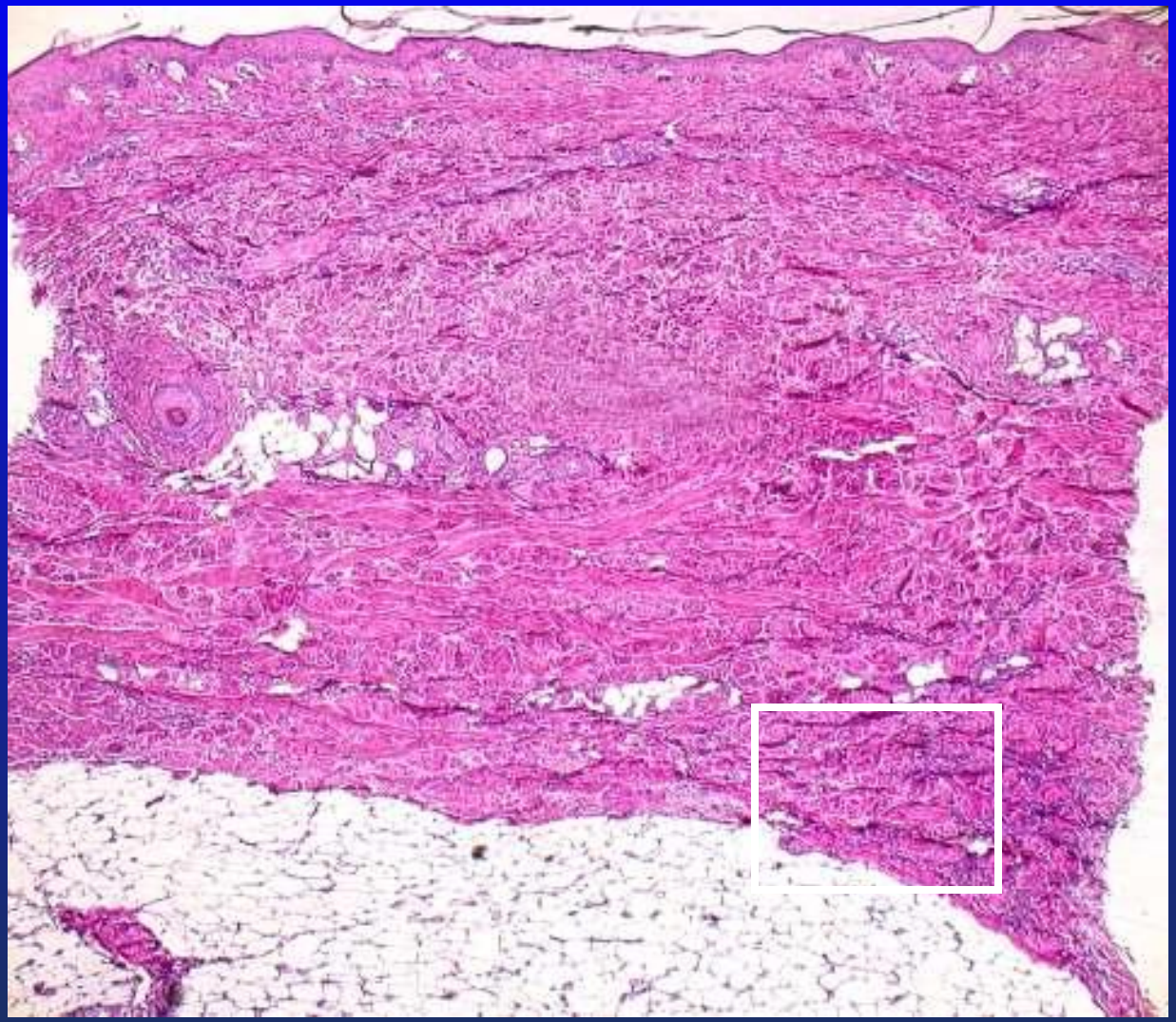
Necrosis of the dermis and/or the vasocentric nature of the calcification in calciphylaxis help distinguish this from the calcification sometimes associated with pancreatic fat necrosis involving the subcutis.

From **http://www.bweems.com/calciphylaxis.html**

Calciphylaxis - Case 1

This 66 year old male, who has been on dialysis for renal failure, developed bilateral, indurated, erythematous, painful plaques about 10 cm. in diameter on the thighs. Focal necrosis was noted clinically. This patient had secondary hyperparathyroidism, and he also had focal superficial penile ulcers. The penis was not biopsied, but an x-ray set for soft tissue density demonstrated calcification within it.

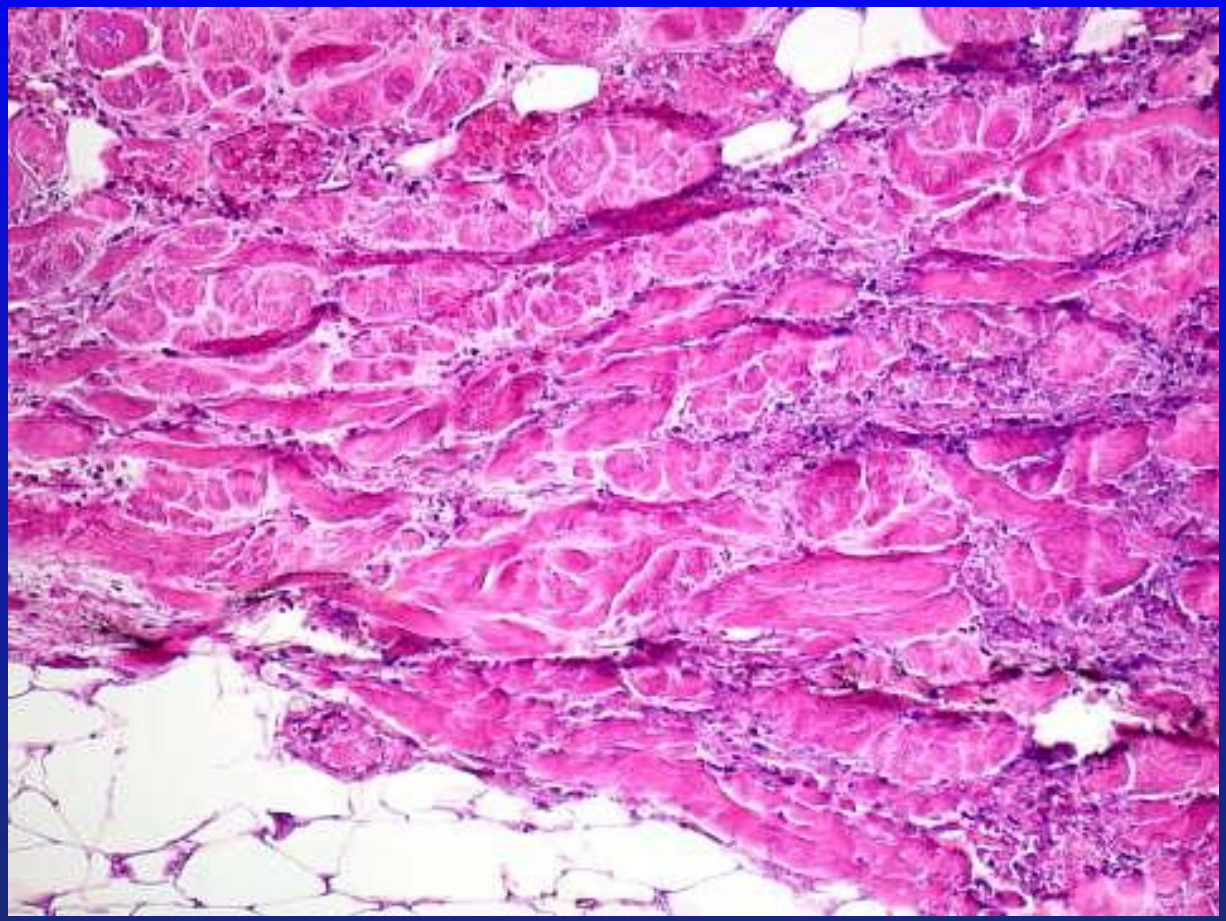
Calciphylaxis - Case 1



Scan power view. The skin does not look too remarkable at this magnification, but there is some-thing abnormal in the lower right side of the reticular dermis.

From **http://www.bweems.com/calciphylaxis.html**

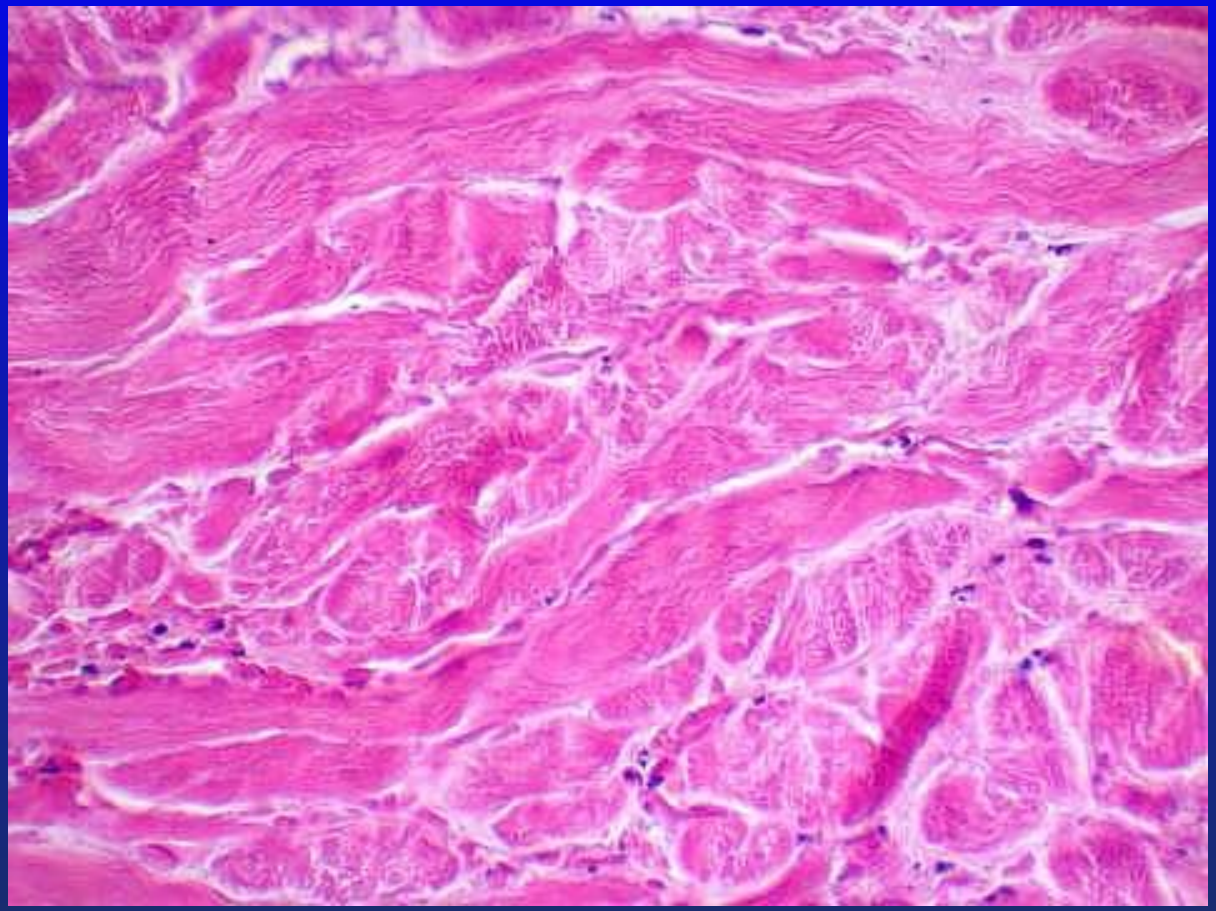
Calciphylaxis - Case 1



From **http://www.bweems.com/calciphylaxis.html**

A low power view of the lower right side of the reticular dermis. This biopsy was taken near the edge of a focus of clinical necrosis. The PMN's and nuclear debris scattered between collagen bundles are consistent with the reaction on the edge of a necrotic zone.

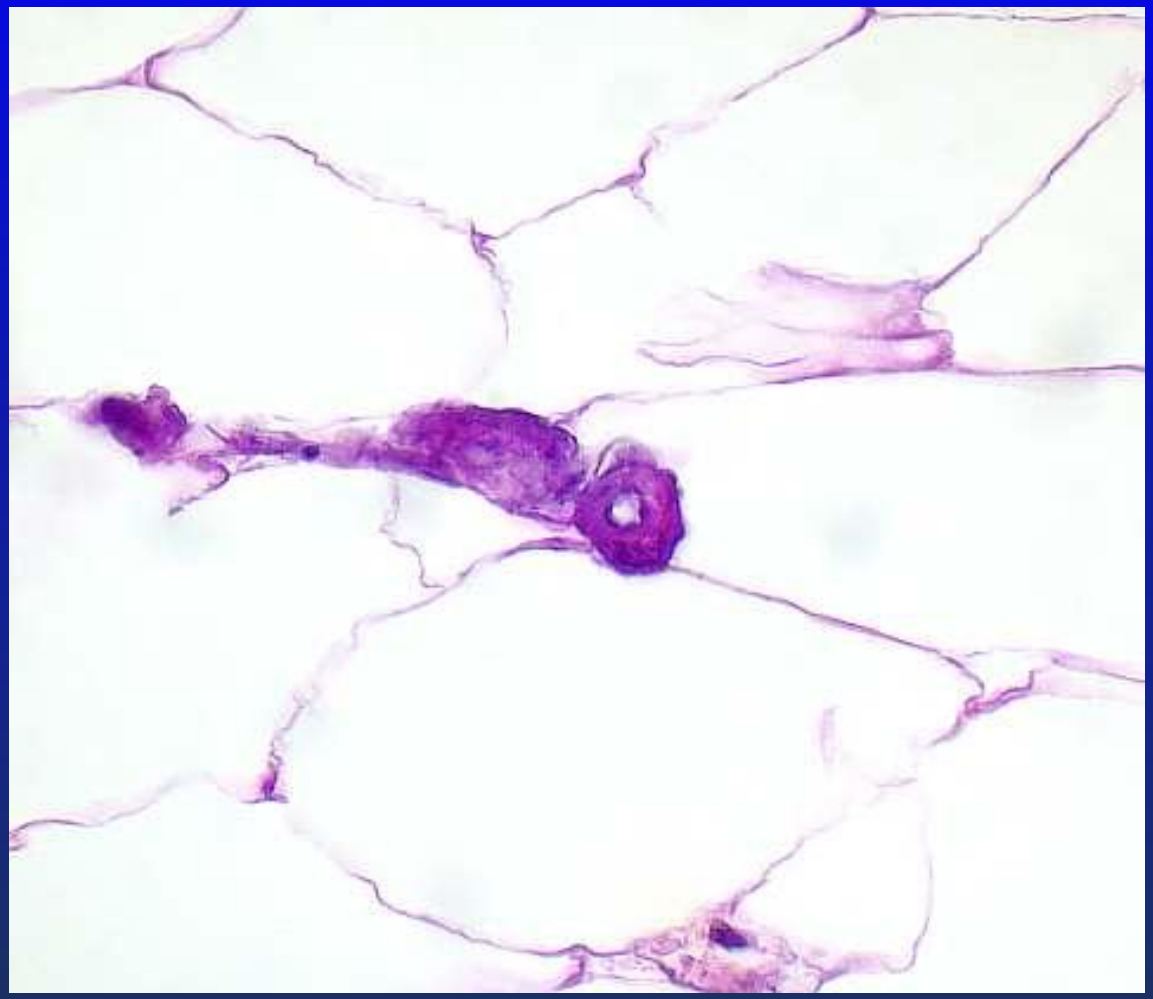
Calciphylaxis - Case 1



From **http://www.bweems.com/calciphylaxis.html**

A medium power view above the zone shown above. The collagen bundles are swollen, and there is a marked diminution in the number of fibroblasts that would normally be present. This can be seen in dermis that has a diminished blood supply.

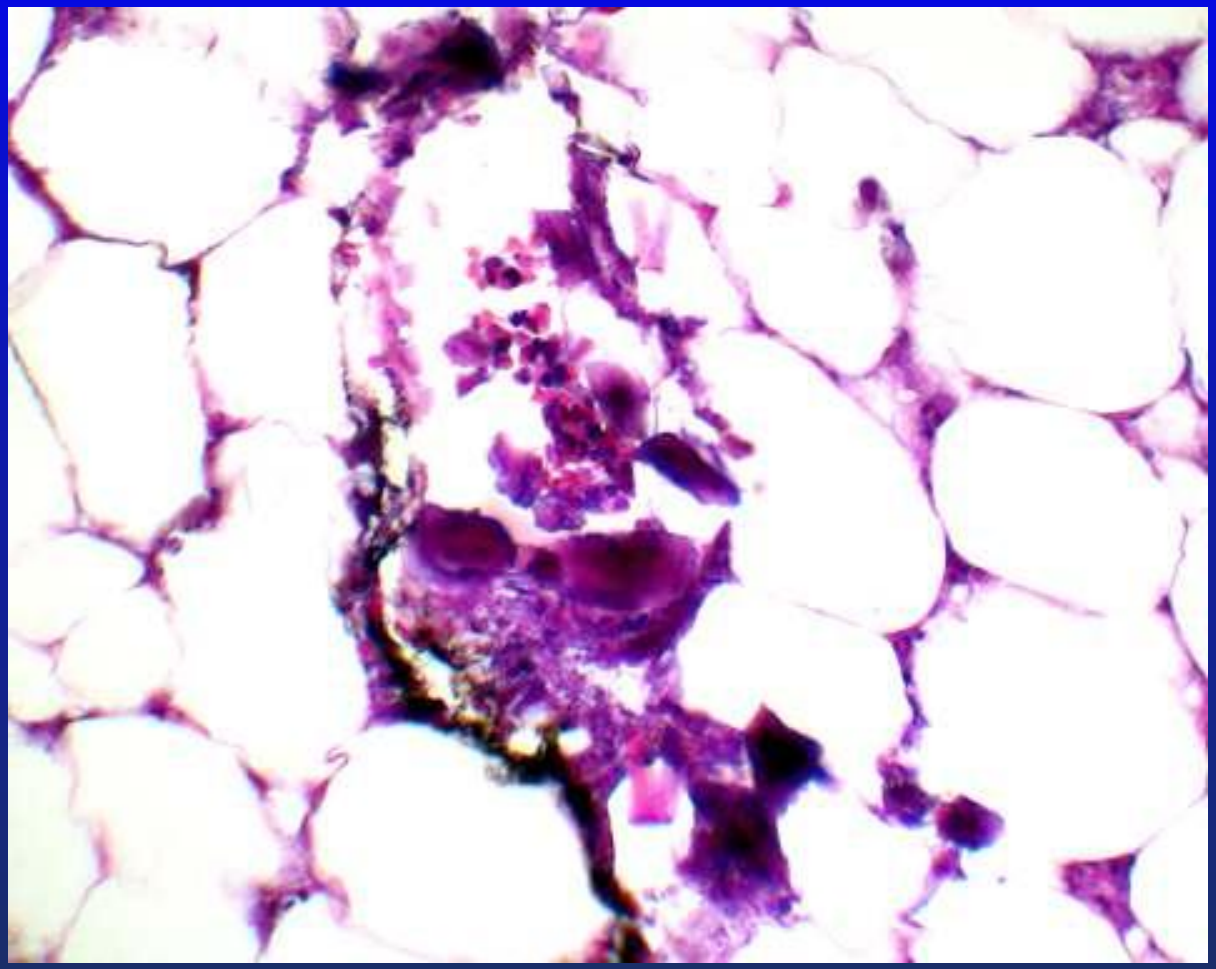
Calciphylaxis - Case 1



From **http://www.bweems.com/calciphylaxis.html**

A high power view of a calcified, tiny blood vessel. Very few small blood vessels having this finding were demonstrated.

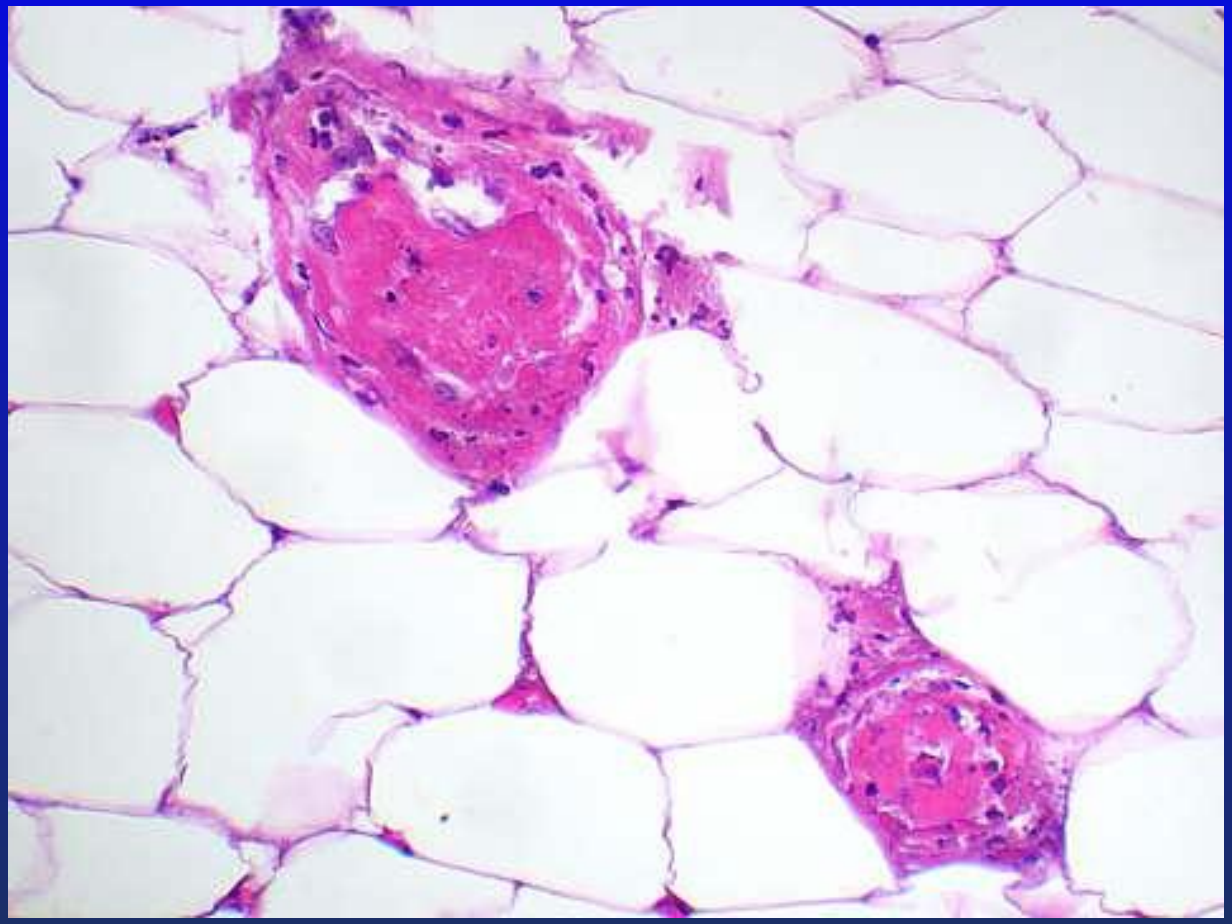
Calciphylaxis - Case 1



From **http://www.bweems.com/calciphylaxis.html**

A high power view of a larger focus of calcification, possibly of a blood vessel. Foci such as this were not plentiful in the biopsy.

Calciphylaxis - Case 1



A medium power view of two thrombosed blood vessels

From **http://www.bweems.com/calciphylaxis.html**

Calciphylaxis - case 2

A 53-year-old woman with diabetes mellitus and end-stage renal disease who was undergoing hemodialysis presented with fever, painful finger necrosis, and nonhealing ulceration of the legs. Hand radiographs showed no evidence of osteomyelitis but did show severely calcified arteries and the loss of soft tissue at the tips of the index and middle fingers (visible in the image, along with an intravenous catheter at the wrist). The results of skin biopsy were consistent with calciphylaxis.

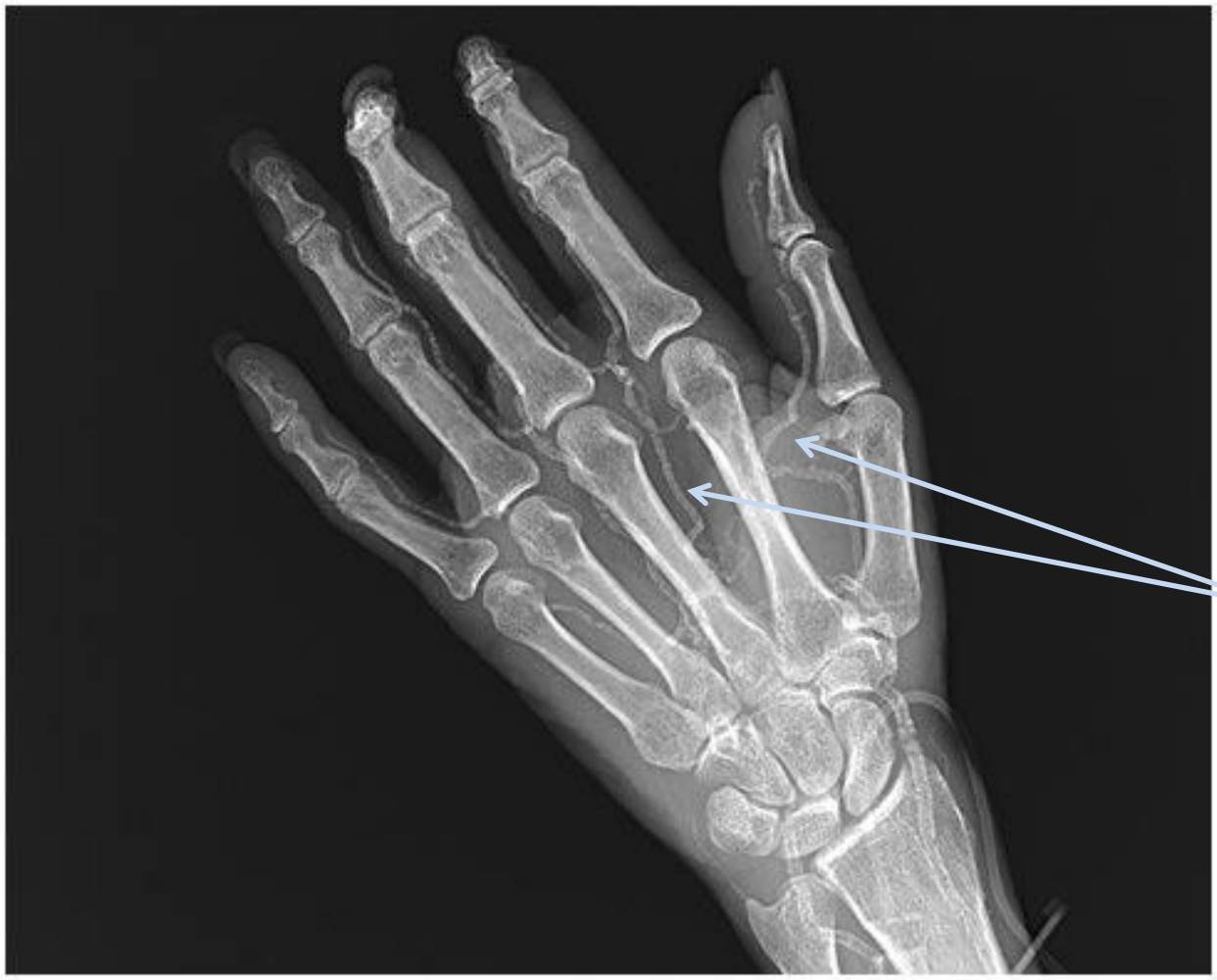
Calciphylaxis is a syndrome of arterial calcification and tissue necrosis that most often occurs in patients with end-stage renal disease who are undergoing hemodialysis. The mortality rate is as high as 80%, with death usually from associated wound infection and sepsis. The pathogenesis of calciphylaxis remains unclear.

Calciphylaxis - case 2

The patient was found to have two associated risk factors: hyperparathyroidism (parathyroid hormone level, 1113 pg per milliliter) and an elevated calcium–phosphorus product (90 mg per deciliter).

She was treated with broad-spectrum antibiotics, cinacalcet, low-calcium dialysis baths, phosphate binders, and sodium thiosulfate. The serum parathyroid hormone, calcium, and phosphorus levels improved, but the skin necrosis and clinical status worsened. The patient died after a 1-month hospitalization.

Calciphylaxis - case 2



Plain x-ray (without contrast)

See how all the arteries are calcified

Calciphylaxis - case 3

A 38-year-old man who had undergone renal and pancreatic transplantation five years earlier was referred because of acral gangrene of both hands. The patient also had type 1 diabetes and was a nonsmoker. Within the previous month, painful paraungual blue spots had developed on three of the patient's fingers, including the fourth digit of his left hand (Panel A); ulcers had then developed on the affected fingers. His renal function had deteriorated, a fact attributed to the effects of infection with polyomavirus type BK. The systemic blood pressure was 130/80 mm Hg, and the segmental arterial pressures in the upper limbs were more than 300 mm Hg, with normal pulse signals. The serum calcium level was 2.52 mmol per liter, the phosphorus level was 1.04 mmol per liter, and the creatinine level was 2 mg per deciliter (177 µmol per liter).

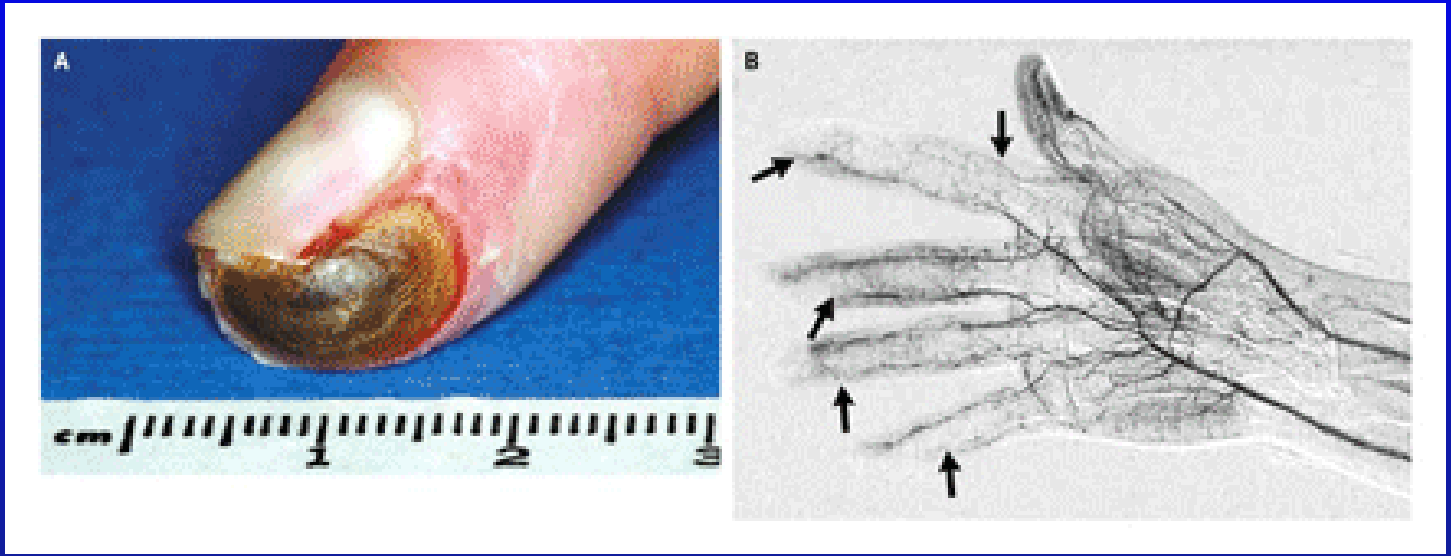
Joyal and Margaroli 352 (26): e24, Figure 1 June 30, 2005

Calciphylaxis - case 3

The patient was treated with calcium antagonists, antiplatelet therapy, and heparin. However, within six months, ischemia had progressed in all digits except for the thumbs, and he underwent partial amputation of the second, third, and fourth fingers of both hands. Microscopical examination of an amputated digit showed ischemic necrosis with inflammatory infiltration, medial calcifications of medium-sized arteries, and intimal hyperplasia with luminal thrombosis. Despite the amputations, the patient continues to have ischemic pain, and only the thumbs have adequate perfusion.

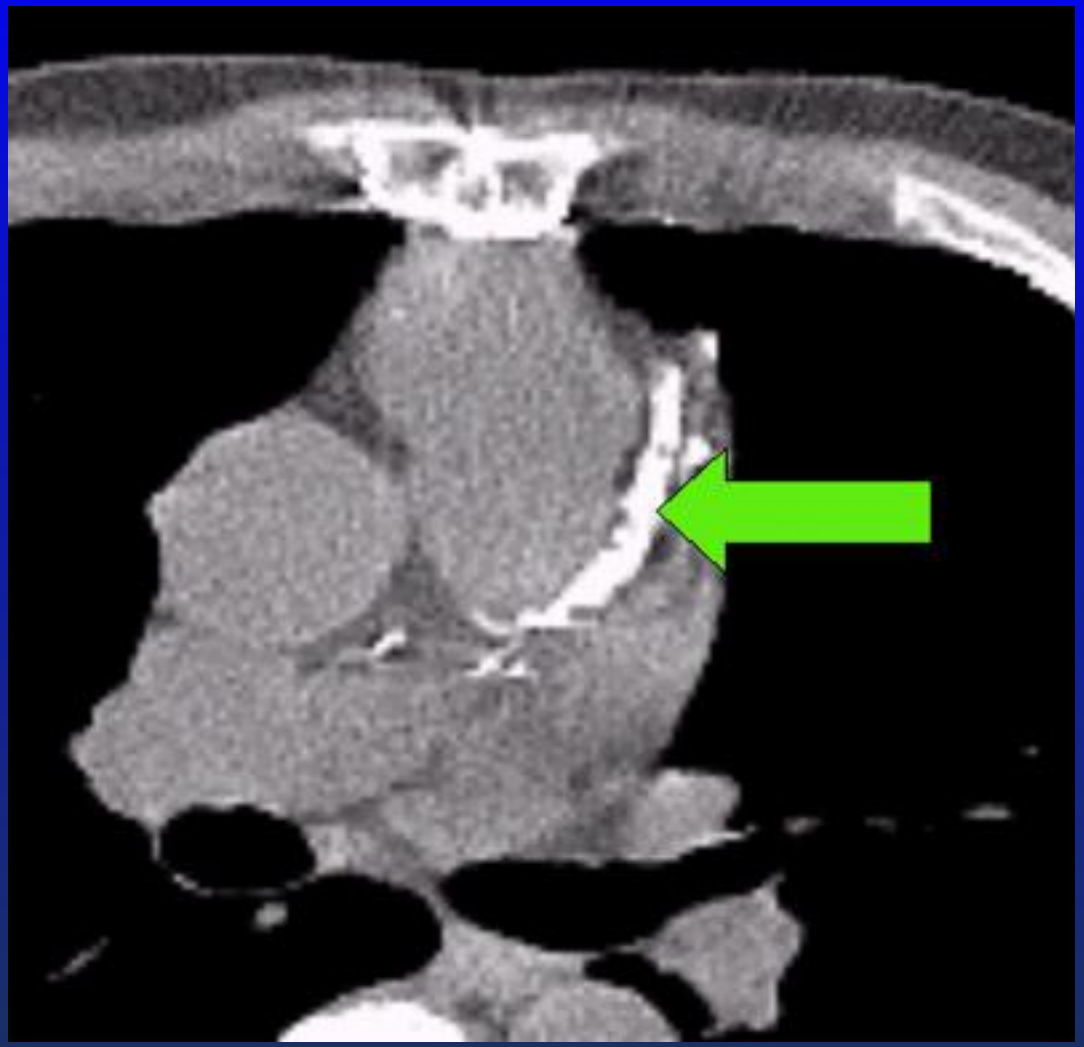
Joyal and Margaroli 352 (26): e24, Figure 1 June 30, 2005

Calciphylaxis - case 3



Arteriography revealed diffuse arterial occlusions (arrows)

Calcification - case 4



Calcification of the left coronary artery in a patient with chronic kidney disease receiving dialysis as seen on a computerized tomography (CT) scan. The extensive deposition of mineral (arrowed) results in a radio-opaque vessel with a density similar to that of bone (compare with the sternum, seen in cross section at the top of the image).

Calcification - case 5

A 6-year-old boy with a long-standing history of anemia and failure to thrive had a 2-year history of end-stage renal disease of undetermined etiology requiring peritoneal dialysis. One year before death, the patient was noted to have hyperparathyroidism (parathyroid hormone, 512 pg/mL; reference range, 10-55 pg/mL) and hypercalcemia (calcium, 10.2 mg/dL) that resulted in bony degeneration. Elevated levels of parathyroid hormone and calcium normalized after subtotal parathyroidectomy. Eight *months before the patient's death, he presented with a 4*-month history of increasing fatigue, diaphoresis, and abdominal distention. Chest radiography revealed cardiomegaly, and echocardiography showed a large left ventricle with globally decreased systolic function and abnormal diastolic function. Previous chest radiographic and echocardiographic findings had been normal.

Calcification - case 5

Markedly elevated levels of serum urea nitrogen (62 mg/dL) and creatinine (6.4 mg/dL), hypercalcemia (calcium, 12.8 mg/dL), and hyperphosphatemia (phosphorus, 9.1 mg/dL) were observed, with attendant renal failure requiring hemodialysis. Serum levels of 1,25-dihydroxyvitamin D were decreased (10 pg/mL; reference range, 24-65 pg/mL). The patient was treated with captopril, and his blood pressure was monitored closely. Follow-up echocardiography showed improved cardiac function.

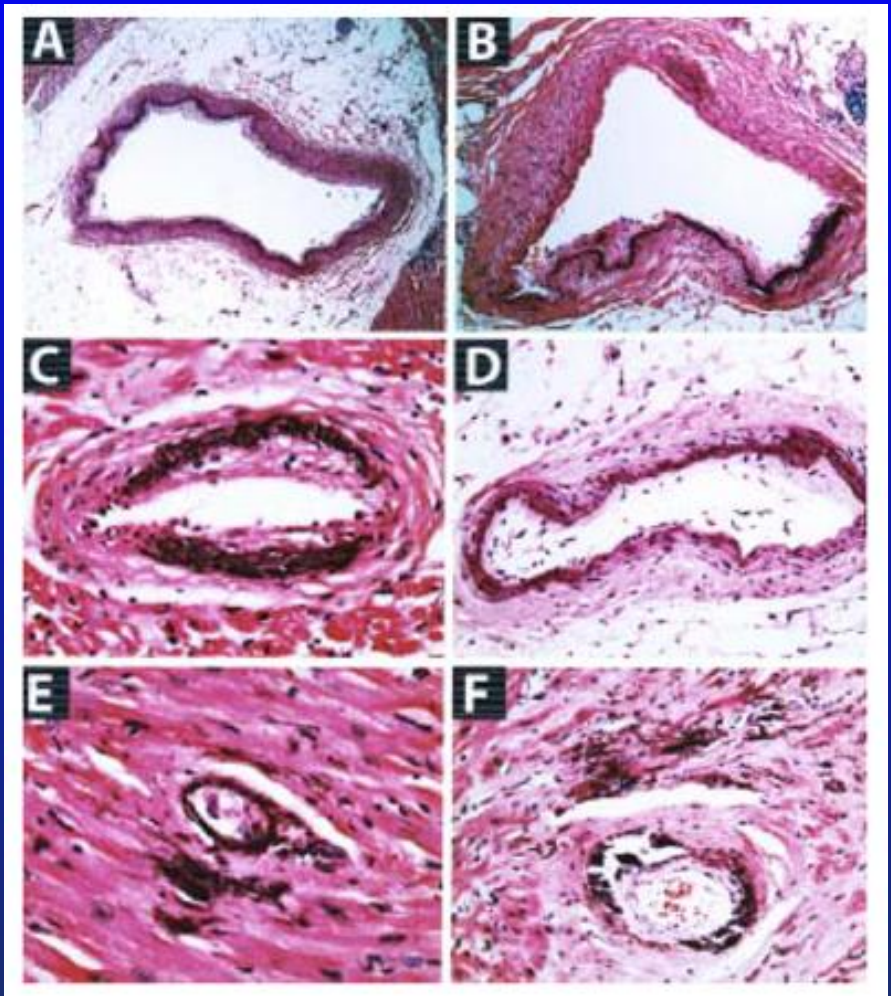
Calcification - case 5

Follow-up echocardiography showed improved cardiac function. Nevertheless, during the next 2 months, the patient exhibited persistent diaphoresis and shortness of breath, especially when supine. While receiving an intravenous infusion of pamidronate, the patient had a hypotensive episode along with chest pain and body aches. His heart sounds became extremely faint, and cardiac arrest ensued. Resuscitative measures were unsuccessful.

Calcification - case 5

Autopsy gross examination showed a heart weighing 130 g with dilated ventricles. The microscopic sections showed multifocal calcifications of myocardial fibers including the papillary muscles. Calcification was also present in the annulus of the mitral valve. The aorta was free of calcification, but the medium and large coronary vessels showed diffuse medial calcification. Microscopic foci of calcification were seen occluding the small coronary vessels of the myocardium. The coronary arteries showed only mild intimal proliferation, with no evidence of atherosclerotic plaque. Autopsy also revealed small kidneys with extensive calcifications.

Calcification - case 5



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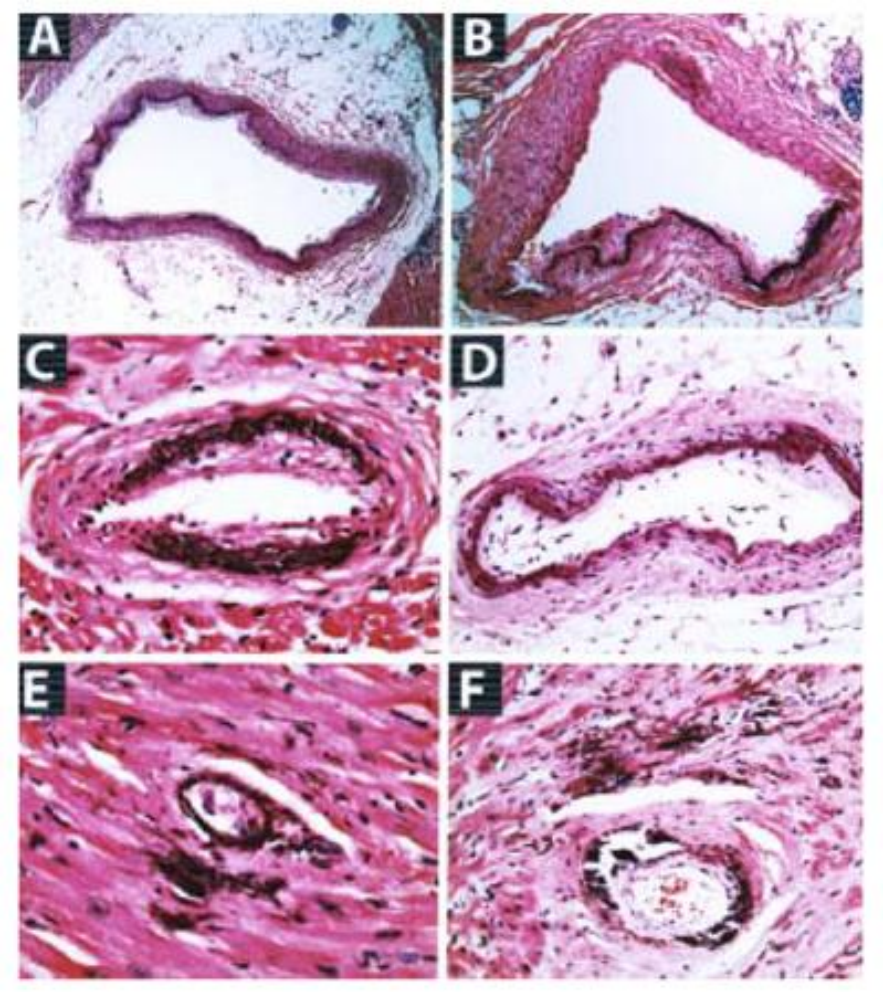
Medial coronary artery calcification in the absence of intimal calcification or atherosclerotic plaque.

A and B, A large epicardial coronary artery is seen with diffuse medial calcification and minimal intimal fibromuscular hyperplasia. The artery is surrounded by epicardial adipose tissue (white region). At least one third of the circumference of the arterial tunica media is calcified (dark stained region). Intimal hyperplasia is present just internal to the medial calcification, but no foam cells are seen. Such intimal hyperplasia is composed predominantly of fibromuscular cells and proliferating myofibroblasts and does not represent atherosclerotic plaque.

Calcification - case 5



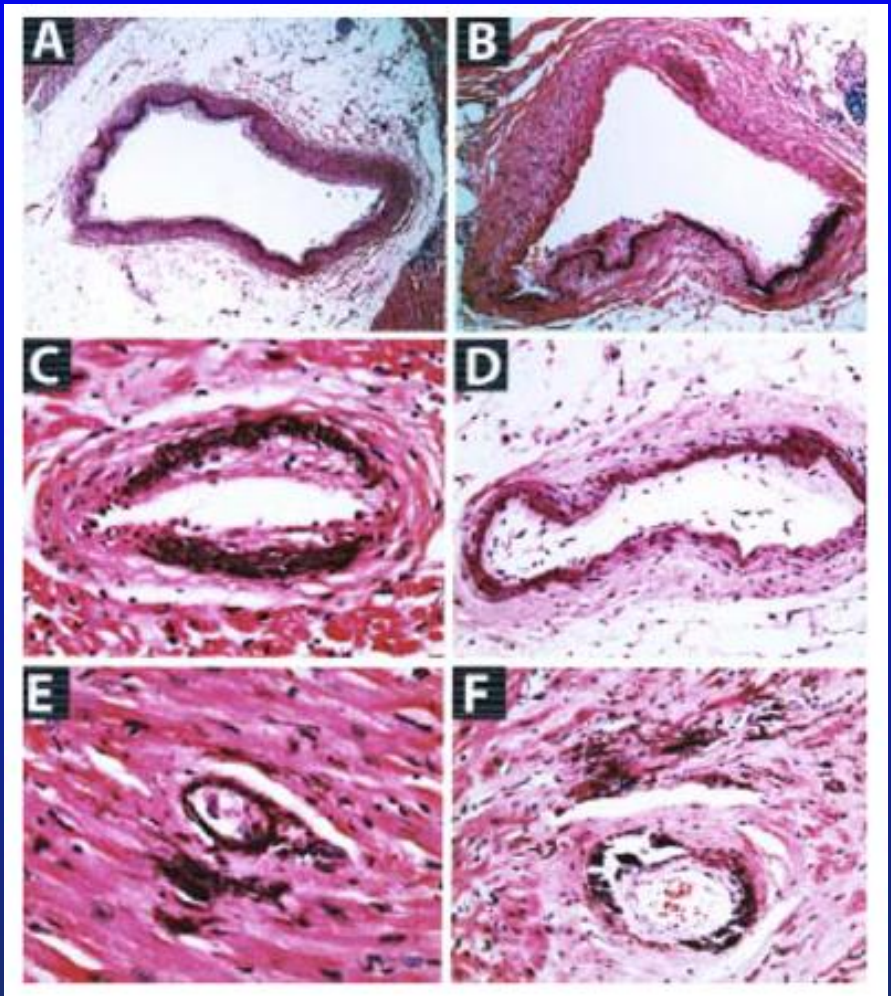
Medial coronary artery calcification in the absence of intimal calcification or atherosclerotic plaque.

C and D, Medium-sized intramyocardial coronary artery with prominent medial calcification that is almost circumferential.

C, Myocardial tissue (red-pink) surrounding the artery.

D, The artery is adjacent to adipose tissue (white).

Calcification - case 5



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Medial coronary artery calcification in the absence of intimal calcification or atherosclerotic plaque.

E, A small, distal intramyocardial coronary artery with prominent medial calcification and dystrophic myocyte *calcification (dark stain at 7 o'clock* position). The lumen is almost totally occluded.

F, Circumferential medial calcification of a distal intramyocardial coronary artery with marked perivascular and interstitial fibrosis and dystrophic myocyte calcification (hematoxylineosin, original magnifications: A and B, ×40; C and D, ×200; E and F, ×400).

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Calcification - case 6

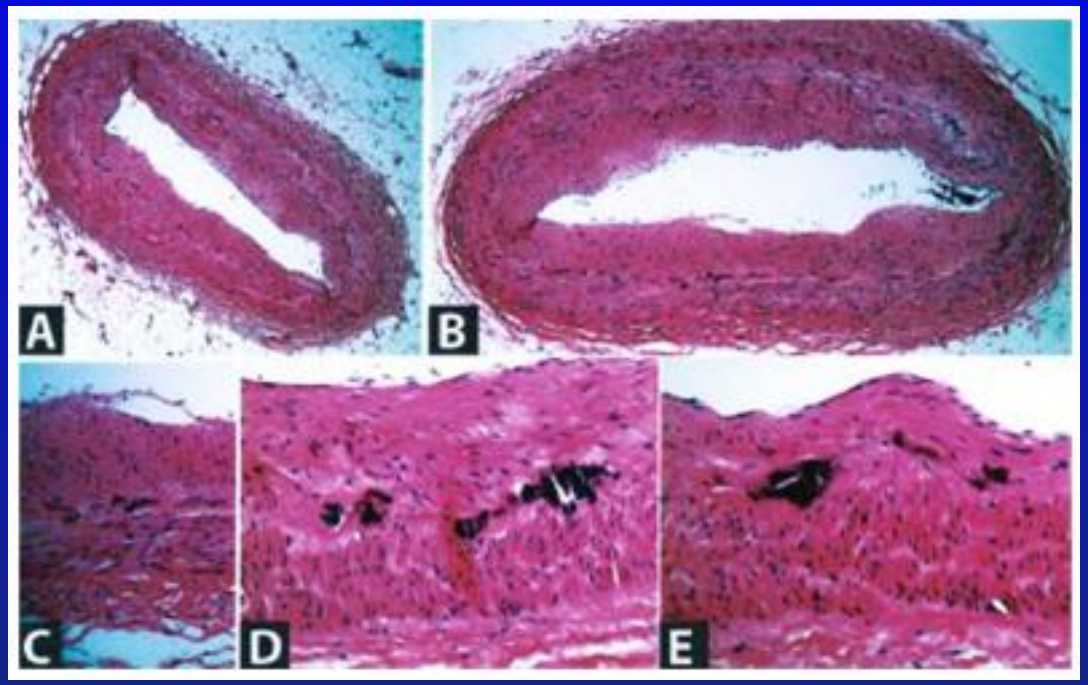
A 62-year-old woman had mild nonobstructive coronary artery disease, idiopathic cardiomyopathy with left ventricular systolic dysfunction, an ejection fraction of 15%, and moderate mitral *and tricuspid regurgitation. The patient's medications included* angiotensin-converting enzyme receptor blockers, angiotensin-*converting enzyme inhibitors, and β*-blockers during the past 2 years, and she had developed progressive symptoms of shortness of breath and intermittent lower extremity edema. Her medical history was remarkable for mild systemic hypertension for 15 to 20 years and type 2 adult-onset diabetes mellitus (non–insulin-dependent diabetes mellitus) for 5 years. The patient had experienced a stroke 4 years previously. She also had undergone orthotopic heart transplantation for her severe left ventricular systolic dysfunction.

Calcification - case 6

Laboratory test results revealed hyperglycemia (glucose, 306 mg/dL), elevated levels of serum urea nitrogen (49 mg/dL) and creatinine (1.8 mg/dL), and hyperphosphatemia (phosphorus, 5.9 mg/dL).

Gross examination of the explanted heart revealed concentric hypertrophy of the left ventricle with slight dilatation. The right coronary artery showed mild atherosclerotic lesions with 20% stenosis. The left anterior descending coronary artery was free of atherosclerosis. Microscopic examination showed evidence of cardiomyopathy with myofiber hypertrophy, diffuse interstitial fibrosis, and patchy replacement fibrosis. Coronary arteries exhibited fibromuscular intimal hyperplasia and mild atherosclerosis. Surprisingly, diffuse medial calcification was present in the epicedial coronary arteries

Calcification - case 6



A and B,
Coronary artery sections from a patient with diabetes mellitus and renal dysfunction.

C-E, Regions of calcification (black) involving the internal elastic lamina and the media. Note that although there is a typical amount of intimal thickening, no atherosclerotic plaque is present (hematoxylin-eosin, original magnifications: A and B, ×40; C-E, ×200).

Prevalence of Coronary-Artery Calcification

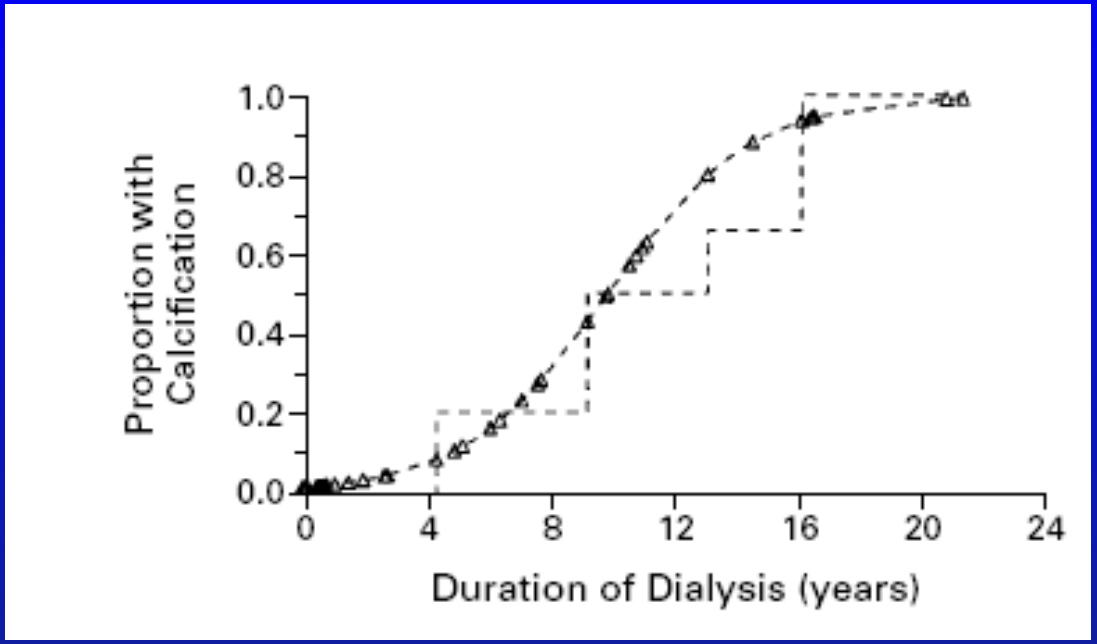


Figure 2.
Prevalence of Coronary-Artery Calcification among 39 Patients with End-Stage Renal Disease, According to the Duration of Treatment with Dialysis. Coronary-artery calcification was assessed by electron-beam computed tomography. The stepped dashed line indicates the proportion of patients with evidence of coronary-artery calcification within each interval of approximately four years.
The curved line reflects estimates derived by logistic-regression analysis. All patients were 30 years of age or younger when they were first evaluated by electron-beam computed tomography. The duration of dialysis excludes intervals of adequate renal function as a result of renal transplantation in 27 patients.

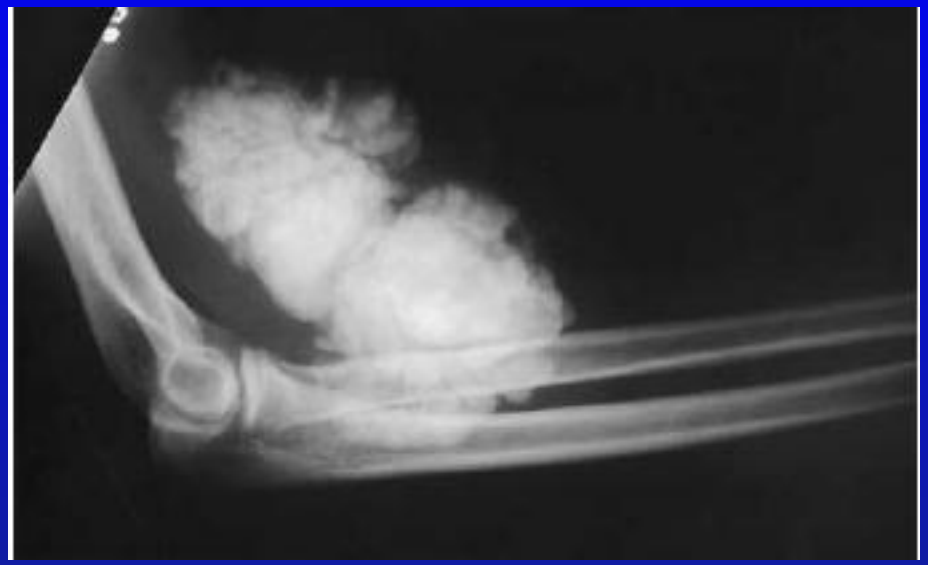
CORONARY-ARTERY CALCIFICATION IN YOUNG ADULTS WITH END-STAGE RENAL DISEASE WHO ARE UNDERGOING DIALYSIS, NEJM, 2000, Volume 342 Number 20 p 1478 - 1483

Calcification - case 7

A 46-year-old man with end-stage renal disease due to diabetic nephropathy had been undergoing hemodialysis for almost two years. Fourteen months after beginning dialysis, he reported pain below the right antecubital fossa, the site of an arteriovenous graft. A plain radiograph showed a nonpalpable calcification measuring 2 cm by 3 cm in the soft tissue. The patient had not been compliant in taking aluminum-containing phosphate binders and had been treated intermittently with intravenous calcitriol. His calcium–phosphate product (with both substances measured in milligrams per deciliter) ranged from 74 to 116 in the ensuing nine months (mean, 93). At the end of that time he presented with a several-day history of pain and swelling in the same antecubital fossa. A mass measuring 5 cm by 5 cm with mild overlying erythema that had not been present at previous dialysis sessions was palpable on the anterior surface of the forearm overlying the arteriovenous graft

Calcified Mass in a Patient on Long-Term Hemodialysis NEMJ, 1998, Volume 338 Number 20 p 1427

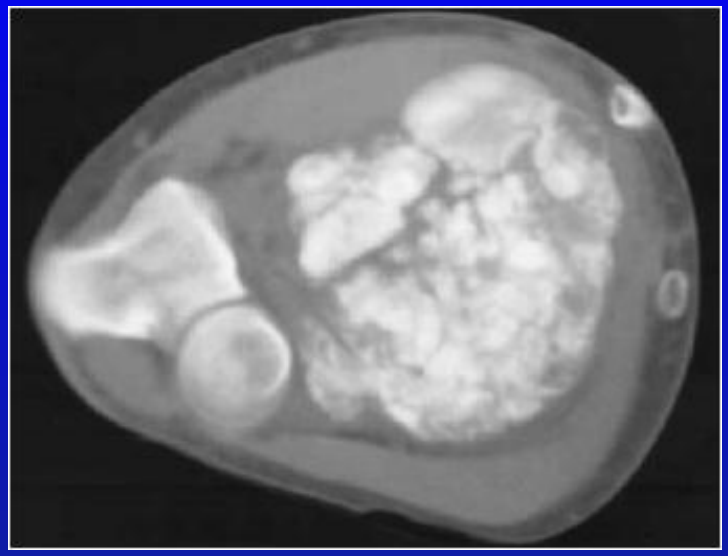
Calcification - case 7



A plain radiograph (Panel B) and a computed tomographic scan (Panel C) revealed a large soft-tissue calcification in the antecubital fossa. Laboratory evaluation revealed the following values: serum calcium, 9.2 mg per deciliter (2.3 mmol per liter); phosphate, 8.5 mg per deciliter (2.7 mmol per liter); alkaline phosphatase, 60 U per liter; and intact parathyroid hormone, 185 pg per milliliter (normal, up to 54)

Calcified Mass in a Patient on Long-Term Hemodialysis NEMJ, 1998, Volume 338 Number 20 p 1427

Calcification - case 7



Calcification presumably resulted from the presence of extravasated blood and softtissue injury caused by repeated venipuncture. During the *following six months, the patient's compliance in taking aluminum-*containing phosphate binders improved, and the mean calcium–phosphate product fell to 70. One year later the mass was smaller and the local pain and erythema had resolved, but computed tomographic scanning showed that the size of the calcification was unchanged. At no time was the function of the arteriovenous graft affected.

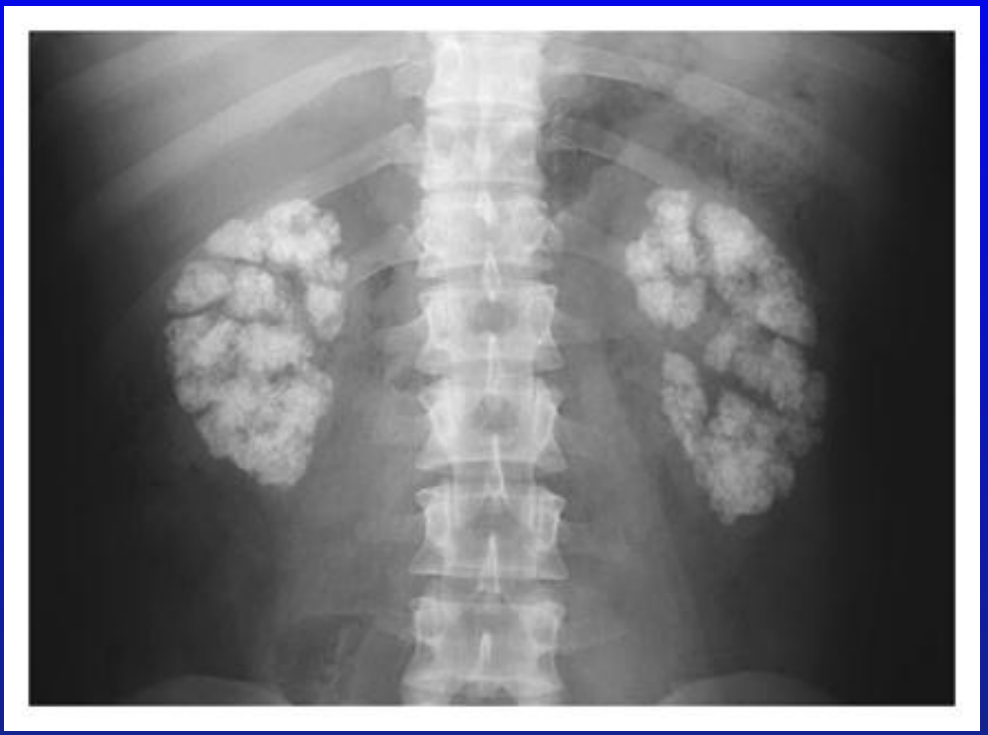
Calcified Mass in a Patient on Long-Term Hemodialysis NEMJ, 1998, Volume 338 Number 20 p 1427

Calcification - case 8

A 37-year-old man was referred for evaluation of distal renal tubular acidosis. Laboratory evaluation revealed a serum potassium level of 3.3 mmol per liter, a bicarbonate level of 16 mmol per liter, a calcium level of 9.3 mg per deciliter (2.3 mmol per liter), a phosphate level of 2.1 mg per deciliter (0.7 mmol per liter), a creatinine level of 3.0 mg per deciliter (265 µmol per liter), a parathyroid hormone level of 62 pg per milliliter, and an estimated glomerular filtration rate of 25 ml per minute per 1.73 m2 of body-surface area. He had been given a diagnosis of renal tubular acidosis at 9 years of age on the basis of metabolic acidosis with a high urinary pH and hypokalemia associated with nephrocalcinosis. At that time, there was evidence of bilateral nephrocalcinosis on plain abdominal radiography. The patient was treated with sodium bicarbonate and potassium supplementation and had normal growth but did not undergo medical follow-up or treatment between 15 and 37 years of age..

Serrano A., Batle D.: **Images in clinical medicine. Bilateral kidney calcifications.** N Engl J Med. 2008 Jul 3;359(1):e1

Calcification - case 8



The plain film of the abdomen obtained during the referral visit (see figure) revealed bilateral symmetric calcification of the renal parenchyma, sparing only the renal pelvis. This finding contrasts with those classically associated with type 1 distal renal tubular acidosis, in which nephrocalcinosis is present but is limited to the renal medulla. Three years after sodium bicarbonate and potassium supplementation was restarted, the patient's renal function has remained stable

Serrano A., Batle D.: **Images in clinical medicine. Bilateral kidney calcifications.** N Engl J Med. 2008 Jul 3;359(1):e1

The Investigations of Calcium abnormalities

Essential for understanding the investigations

1) **Anatomy:**

2) **Biochemistry:**

3) **Physiology:**

4) **Diseases**

Hypercalcemia - Cases 1 to 8

Hypocalcemia

Hypocalcaemia

- Drugs and chemicals
  - Furosemide
  - Enzyme-inducing drugs eg phenytoin
  - Ethylene glycol overdose

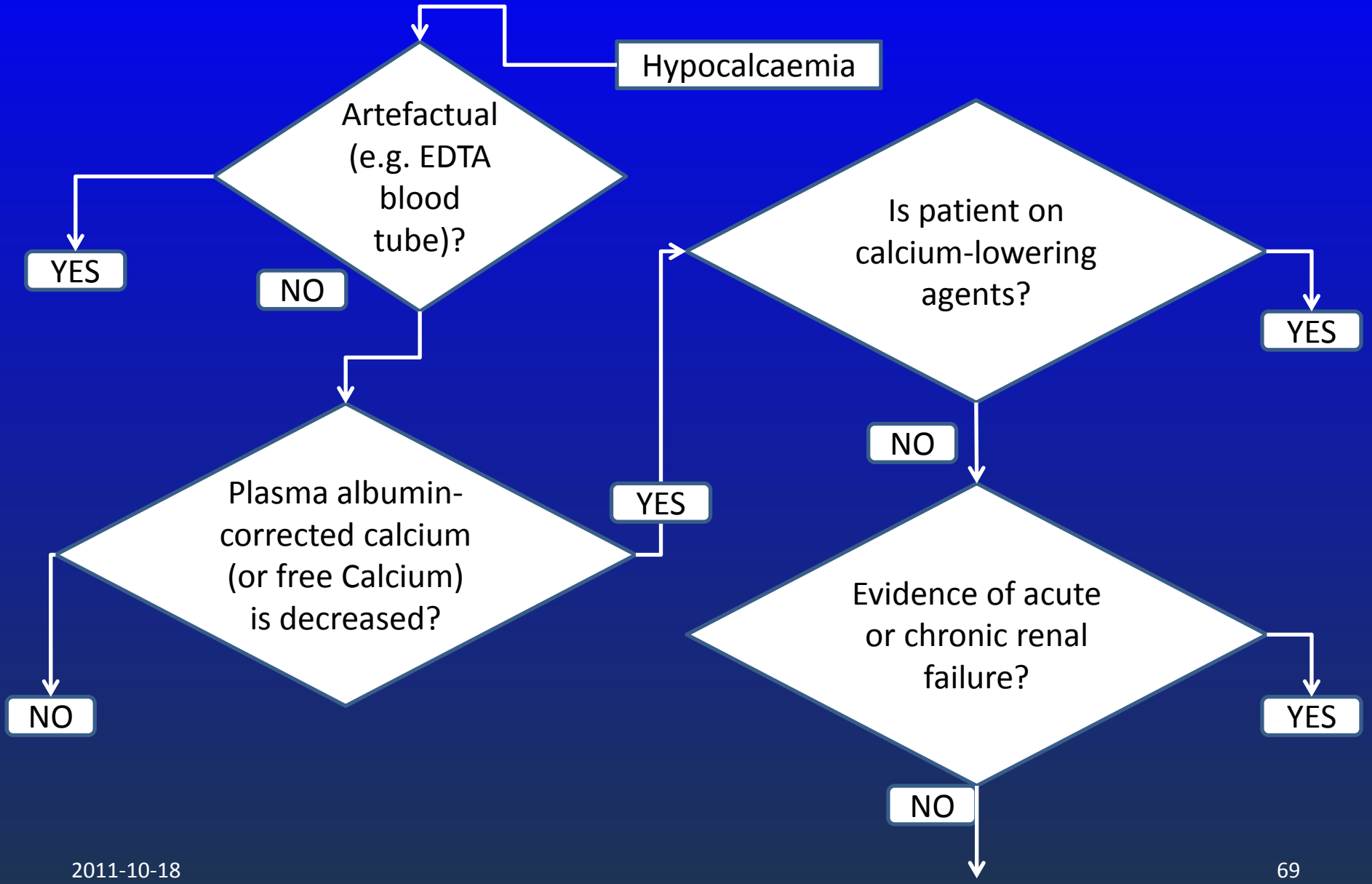
- Hypocalcaemia usually with hypophosphatemia
  - Vitamin D deficiency
    - Ricktes
    - Osteomalacia
  - Malabsorption states

Hypocalcaemia

- Hypocalcaemia usually with hyperphosphatemia
  - Chronic renal failure
  - Hypoparathyroidism (low PTH levels)
    - Idiopathic or autoimmune
    - Surgical removal of parathyroids
    - Congenital absence of parathyroids
    - Infiltration of parathyroids
  - Pseudohypoparathyroidism
  - YH resistance

- Rarer causes
  - Acute pancreatitis, sepsis, high calcitonin levels, rhabdomyolysis

Algorithm for Hypocalcaemia



Algorithm for Hypocalcaemia