CAUSES OF HYPERPHOSPHATEMIA

Pseudohyperphosphatemia
Multiple myeloma
Extreme hypertriglyceridemia
In vitro hemolysis

Increased exogenous phosphorus load or absorption
Phosphorus-rich cow's milk in premature neonates
Vitamin D intoxication
PO^3_4-containing enemas
Intravenous phosphorus supplements
White phosphorus burns
Acute phosphorus poisoning

Increased endogenous loads
Tumor lysis syndrome
Rhabdomyolysis
Bowel infarction
Malignant hyperthermia
Heat stroke
Acid-base disorders
Organic acidosis
Lactic acidosis
Ketoacidosis
Respiratory acidosis
Chronic respiratory alkalosis

Reduced urinary excretion
Renal failure
Hypoparathyroidism
Hereditary
Acquired
Pseudohypoparathyroidism
Vitamin D intoxication
Growth hormone
Insulin-like growth factor-1
Glucocorticoid withdrawal
Mg^{2+} deficiency
Tumoral calcinosis
Diphosphonate therapy
Hypophosphatasia

Miscellaneous
Fluoride poisoning
β-Blocker therapy
Verapamil
Hemorrhagic shock
Sleep deprivation

FIGURE 7-25
Causes of hyperphosphatemia. (*From* Knochel and Agarwal [5]; with permission.)

CLINICAL MANIFESTATIONS OF HYPERPHOSPHATEMIA

Consequences of secondary changes in calcium, parathyroid hormone, vitamin D metabolism and hypocalcemia:	Consequences of ectopic calcification:
Neuromuscular irritability	Periarticular and soft tissue calcification
Tetany	Vascular calcification
Hypotension	Ocular calcification
Increased QT interval	Conduction abnormalities
	Pruritus

FIGURE 7-26
Clinical manifestations of hyperphosphatemia.

TREATMENT OF HYPERPHOSPHATEMIA

Acute hyperphosphatemia in patients with adequate renal function	Chronic hyperphosphatemia in patients with end-stage renal disease
Saline diuresis that causes phosphaturia	Dietary phosphate restriction
	Phosphate binders to decrease gastrointestinal phosphate reabsorption

FIGURE 7-27
Treatment of hyperphosphatemia.

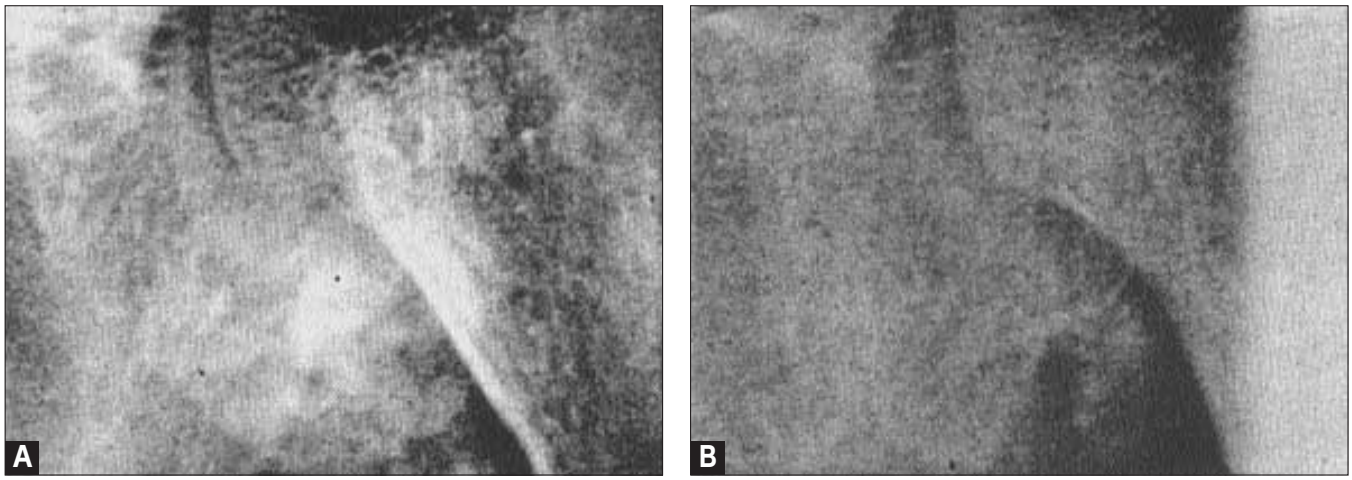

FIGURE 7-28

Periarticular calcium phosphate deposits in a patient with end-stage renal disease who has severe hyperphosphatemia and a high level of the product of calcium and phosphorus. Note the partial resolution of calcific masses after dietary phosphate restriction and oral phosphate binders. Left shoulder joint before (**A**) and after (**B**) treatment. (*From* Pinggera and Popovtzer [17]; with permission.)

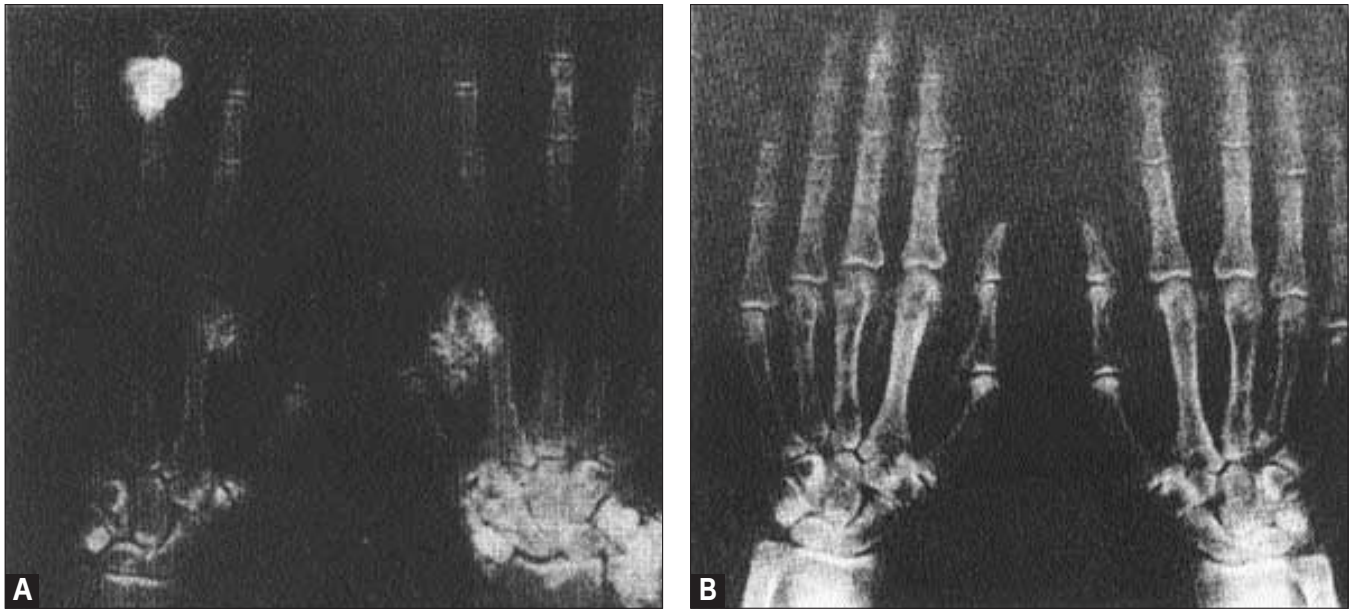

FIGURE 7-29

Resolution of soft tissue calcifications. The palms of the hands of the patient in Figure 7-28 with end-stage renal disease are shown before (**A**) and after (**B**) treatment of hyperphosphatemia. The patient has a high level of the product of calcium and phosphorus. (*From* Pinggera and Popovtzer [17]; with permission.)

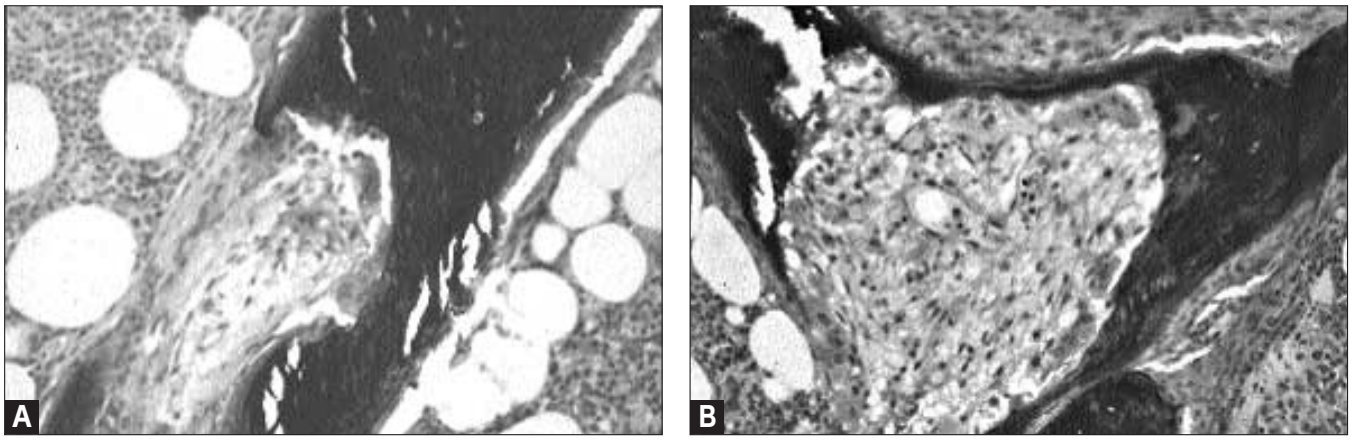

FIGURE 7-30

A, **B**, Bone sections from the same patient as in Figures 7-28 and 7-29, illustrating osteitis fibrosa cystica caused by renal secondary hyperparathyroidism with hyperphosphatemia.

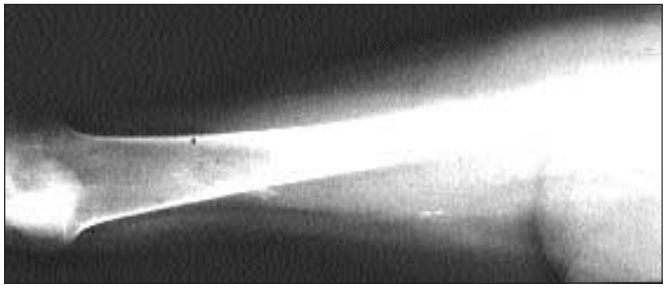

FIGURE 7-31

Roentgenographic appearance of femoral arterial vascular calcification in a patient on dialysis who has severe hyperphosphatemia. The patient has a high level of the product of calcium and phosphorus.

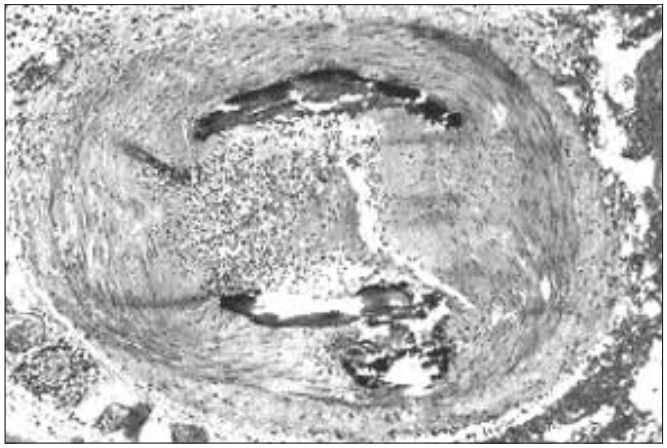

FIGURE 7-32 (see Color Plate)

Microscopic appearance of a cross section of a calcified artery in a patient with end-stage renal disease undergoing chronic dialysis. The patient has severe hyperphosphatemia and a high level of the product of calcium and phosphorus. Note the intimal calcium phosphate deposit with a secondary occlusion of the arterial lumen.

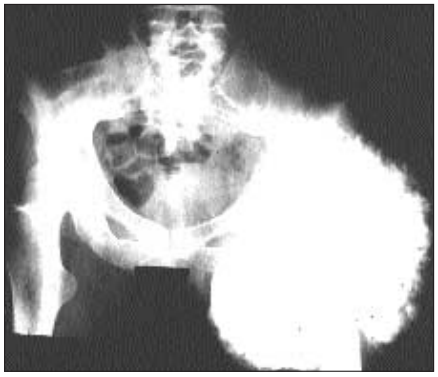

FIGURE 7-33

Massive periarticular calcium phosphate deposit (around the hip joint) in a patient with genetic tumoral calcinosis. The patient exhibits hyperphosphatemia and increased renal tubular phosphate reabsorption. Normal parathyroid hormone levels and elevated calcitriol levels are present. The same disease affects two of the patient's brothers.