

dence of dual atrioventricular pathways. The simultaneous presence of both, which may be developmentally related, is clinically significant, as the former may precipitate recurrent episodes of reentrant supraventricular tachycardia. Although the former may also lead to spontaneous termination of the faster rhythm, digitalis, by altering the conduction properties of the atrioventricular node,¹² is a useful therapeutic modality.

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PARATHYROID ADENOMA AND LIGHT CHAIN MYELOMA

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An elderly patient with simultaneous hyperparathyroidism and light chain myeloma is described. The patient presented with hypercalcemia, bone pain, pathologic fractures, and cystic and lytic bone lesions. The problems of diagnosis and management are discussed. It is recommended that elderly patients with hypercalcemia and bone lesions should have not only diagnostic studies for hyperparathyroidism but also serum and urine immunoelectrophoresis to detect multiple myeloma or

plasma cell neoplastic variants. Surgery is indicated for the hyperparathyroidism. Chemotherapy and localized radiotherapy usually control the myelomatous disease.

Hypercalcemia and symptomatic bone lesions may be found in a variety of disorders including hyperparathyroidism and multiple myeloma. This report concerns the coexistence in the same patient of a parathyroid adenoma and the light chain variant of multiple myeloma. The problems of diagnosis and management are discussed. There are reports in the medical literature of multiple myeloma associated with multiple neoplasms,¹

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dysproteinemia, and hyperparathyroidism,² myeloma associated with hyperparathyroidism,³ and monoclonal gammopathy and hyperparathyroidism.⁴ However a review of the literature did not reveal any clearly described cases or clinical documentation of hyperparathyroidism and light chain myeloma in the same patient.

The authors report a patient who presented with a pathologic fracture, cystic bone lesions, and hypercalcemia. A parathyroid adenoma was removed and two months later hypercalcemia recurred with progressive painful lytic bone lesions and kappa light chains in both serum and urine.

CASE REPORT

An 80-year-old man was admitted with pathologic fracture of his right forearm, multiple osteolytic lesions of all long bones, and a six-year history of polydipsia and polyuria. He also gave a three-month history of increasing fatigue, weakness, and anorexia with an 18-pound weight loss. He was treated for peptic ulcer disease 23 years previously. There was no history of urinary tract infection or urolithiasis.

On admission, respirations were 16 per minute, temperature 99 F, blood pressure 160/90 mmHg, and pulse 70 per minute and irregular. The thyroid was slightly enlarged with a nodule in the left lobe. Cardiac auscultation revealed a grade II/VI holosystolic murmur at the apex that radiated to the axillae, and a grade II/VI systolic ejection murmur loudest in the aortic region with radiation to the neck. There was a cast on the right forearm.

Pertinent laboratory data included hematocrit 37%, hemoglobin 13.5 gm/100 ml, white blood cell count 9,100/cu mm with a normal differential, calcium 13.1 mg/100 ml, phosphorus 3.1 mg/100 ml, total protein 7.3 gm/100 ml, albumin 4.8 gm/100 ml, blood-urea nitrogen (BUN) 36 mg/100 ml, creatinine 2.5 mg/100 ml, alkaline phosphatase 90 units/100 ml, T₃ uptake 32%, and T₄ 6.3 mg/100 ml. Serum protein electrophoresis was normal. Total protein was 6.5 gm/100 ml with albumin 58.7 gm/100 ml, and alpha-1 globulin 4.2%, alpha-2 globulin 10.8%, beta globulin 12.2%, and gamma globulin 14.0%. Serum immunoelectrophoresis revealed low levels of serum immunoglobulins.

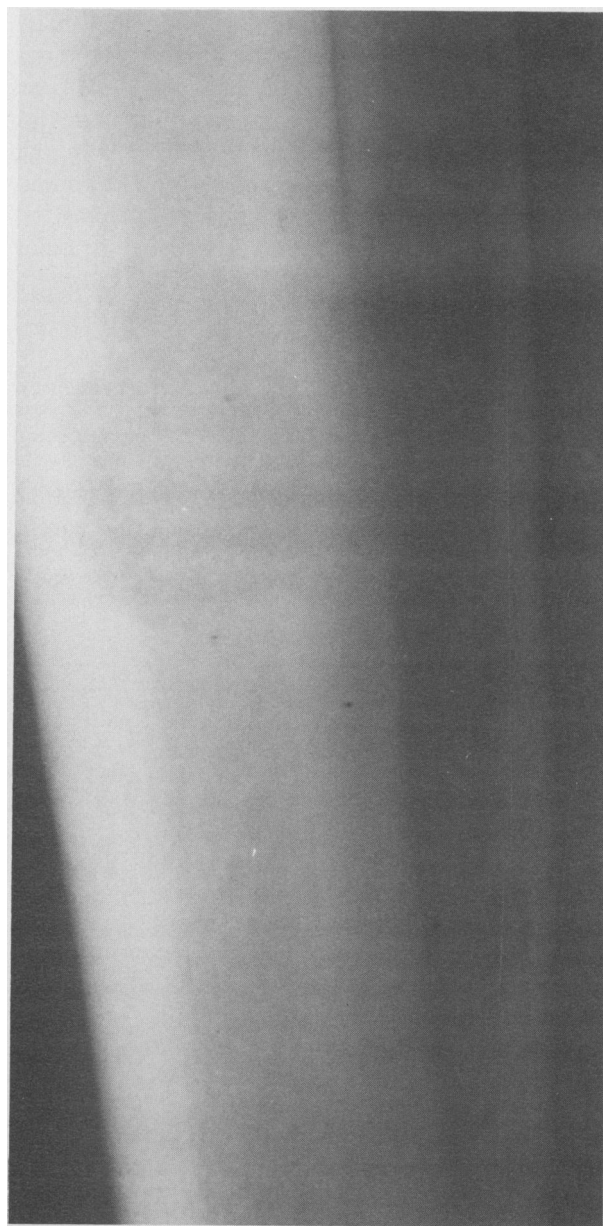


Figure 1. X-ray shows cystic and lytic lesions of tibia and fibula

Parathyroid hormone was elevated and a calcium serum done concomitantly was 12.8 mg/100 ml (normal 8.5-11.0). Urine was negative for Bence Jones protein. Urinary calcium was elevated. Chest x-ray revealed osteolytic lesions of the ribs with mild cardiomegaly. X-ray of the right forearm revealed pathologic fracture of the mid-shaft of the



Figure 2. Lateral view of leg shows multiple osteolytic lesions

radius. Roentgenograms of the hands revealed no subperiosteal resorption or demineralization. X-rays of the legs showed cystic and lytic lesions of both tibia and fibula (Figures 1 and 2). Electrocardiogram revealed multifocal premature ventricular contractions, premature atrial contractions, and left axis deviation. Aspirated bone marrow was hypocellular with no increase in normal or abnormal plasma cells. Attempt at a bone marrow biopsy was unsuccessful. The thyroid scan showed mild thyromegaly with decreased uptake in the left upper pole.

A neck exploration revealed a nodular goiter of the thyroid and in the left upper pole a parathyroid adenoma, chief cell type (Figure 3). The patient did well postoperatively. The calcium and phosphorus returned to normal and the fracture of the right arm showed slow healing.

Two months later, the patient was readmitted with complaints of bone pain in both legs and continued weakness and weight loss. He was lethargic with twitches and tremors of both upper and lower extremities. There was a 3×3 cm nodule over the upper portion of the left anterior tibia. Cardiac examination revealed many extrasystoles.

Serum calcium was 15.3 mg/100 ml, phosphorus 3.8 mg/100 ml, hematocrit 25.2%, hemoglobin 8.6 gm/100 ml, and alkaline phosphatase 65 units/100 ml. Protein electrophoresis showed a flat gamma curve. X-rays of both lower extremities showed a destructive lesion in the distal right femur and multiple cystic and lytic lesions of both tibias. Electrocardiogram revealed multiple ventricular contractions.

The patient received lidocaine and quinidine for the arrhythmia along with hydration and furosemide for the hypercalcemia. Serum immunoelectrophoresis revealed the immunoglobulins to be low. Light chain immunoglobulins, kappa type, were found in the serum and urine. Biopsy of the right lower femur revealed a plasmacytoma (Figure 4). A plasmacytoma was also removed from his anterior chest wall, apparently an extension from the underlying rib lesion. The patient was given a total of 3,000 rads to the right femur and both tibias, which relieved his pain considerably. Specific chemotherapy with melphalan and prednisone was administered for four days every four weeks. Convalescence was complicated by an abscess in the right groin, which responded to incision and drainage plus antibiotics.

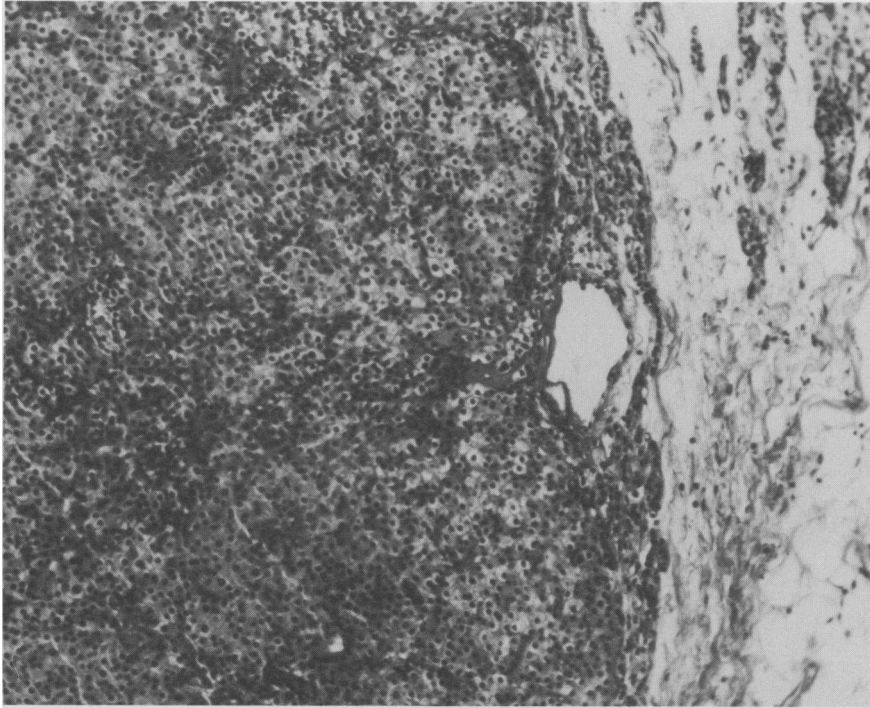


Figure 3. Chief cell adenoma of parathyroid gland that measured 1.4×1.0×0.9 cm. On the right, note the capsule and some entrapped parathyroid lobules (hematoxylin-eosin, ×125)

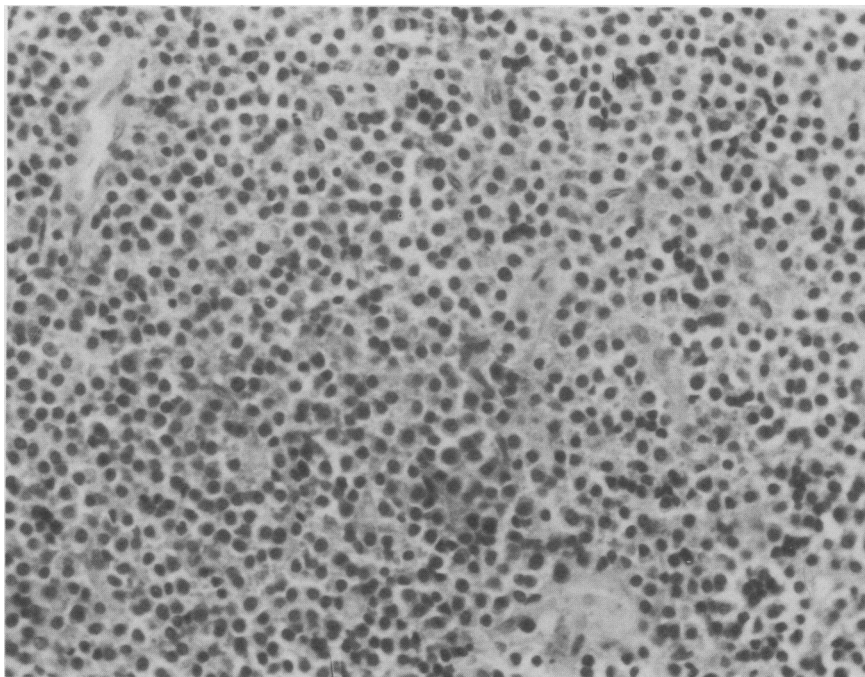


Figure 4. Biopsy of right distal femur showing typical plasmacytic myeloma (hematoxylin-eosin, ×500)

On discharge, the patient was much improved with no pain in his legs and normal serum calcium levels.

He remained asymptomatic and active for 22 months. Melphalan and prednisone were administered for four days every four weeks. Serum immunoglobulins remained normal with the disappearance of free light chains from serum and urine. Approximately two years after the diagnosis of myeloma, the patient developed severe bone pain and new lytic lesions in the right tibia. The painful bone lesions were irradiated and chemotherapy was changed to cyclophosphamide, vincristine sulfate, and carmustine (BCNU). He was finally admitted with progressive bone pain, lethargy, confusion, and severe hypercalcemia. Serum calcium on admission was 21.5 mg/100 ml. Despite vigorous therapy with saline hydration, furosemide, steroids, and mithramycin, the hypercalcemia was refractory. He developed ventricular tachyarrhythmias and expired 48 hours after admission. Permission for autopsy was refused.

COMMENTS

Hyperparathyroidism is being increasingly recognized in patients over 60, especially with the availability of obtaining serum calcium in routine blood studies. A prevalence of 100 to 200 cases per 100,000 has been reported in various clinic and hospital populations.⁵ One study of the community of Rochester, Minnesota, revealed that since the addition of calcium to the 12 unit serum automated chemistry group, the number of known cases of primary hyperparathyroidism increased fourfold.⁶ The most common presentations of symptomatic hyperparathyroidism are hypercalcemia, azotemia, and osteitis fibrosa cystica. The bone lesions may appear as cystic expanding focal lesions in the extremities with areas of trabeculations.⁷ Other roentgenographic manifestations of hyperparathyroid bone disease, such as loss of the lamina dura, erosion of the distal ends of the clavicles, and subperiosteal resorption of the phalanges, were not present in this patient. Multiple myeloma also is a disease of the elderly. The annual incidence is about 3 per 100,000 and the rate increases with age.⁸ M proteins containing only light chains, kappa or lambda, constitute about 5 to 15 percent in most series. Light chain disease, a variant of multiple myeloma, is fre-

quently characterized by hypercalcemia, multiple "punched out" bone lesions, pathologic fractures, and renal insufficiency. The serum immunoelectrophoresis most often demonstrates hypogammaglobulinemia with the presence of free kappa or lambda light chains.⁹ The serum protein electrophoresis usually shows a flat or low gamma curve without evidence of a monoclonal spike. It should, therefore, not be surprising if both diseases appear in an elderly patient. In this case, the patient presented with painful bone lesions, pathologic fractures, and hypercalcemia, all of which occur in both hyperparathyroidism and myeloma. However, although the diagnostic work-up clearly indicates the presence of hyperparathyroidism, the diagnosis of multiple myeloma was overlooked because the serum immunoglobulin assay and electrophoresis were reported as normal and the qualitative test for Bence Jones protein in the urine was negative. After the parathyroid adenoma was removed, the patient's symptoms returned; this time, however, the serum immunoelectrophoresis revealed hypogammaglobulinemia and kappa light chains. Urine immunoelectrophoresis also revealed kappa light chains.

The association of these two diseases may represent merely a chance occurrence. Various theories have been proposed to explain the relationship,²⁻⁴ but at the present time there is no clear or proven explanation. Regardless of the mechanisms involved, the report of this case has practical clinical implications. The authors propose that elderly patients with hypercalcemia should have protein and immunoelectrophoresis done on serum and urine, in addition to the diagnostic studies for hyperparathyroidism. If these patients also have symptomatic bone lesions, the absence of monoclonal gammopathy and Bence Jones protein should not deter one from a serious search for light chains in serum and urine. When both diseases are present, surgical resection of the adenoma will correct the hyperparathyroidism. Chemotherapy and localized radiotherapy will control the myeloma.

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ACUTE GALLSTONE PANCREATITIS WITH PSEUDOCYST AS A COMPLICATION

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Gallstone disease as the etiology of pancreatitis is much more common in private hospital patients than was once described. Common duct stones (choledocholithiasis) have been proven not to coexist in the majority of cases. The objectives of surgery for gallstone pancreatitis therefore should be adequate drainage of the pancreas, evaluation of the common duct, and cholecystectomy. Common duct exploration usually is not warranted or advised.

A pseudocyst may occur subsequent to the acute phase of pancreatitis, or subsequent to surgery for pancreatitis if the pancreas is not adequately and widely drained. The collection of fluid adjacent to or within the pancreas must be determined to be either a pancreatic abscess or a pancreatic pseudocyst. The management of the pseudocyst, which is usually diagnosed by the ultrasonographic finding of a thickened wall, is adjacent internal

drainage. By contrast, the pancreatic abscess must have wide, radical, external drainage.

Mature judgement must be exercised in the approach to, the timing of, and the management of surgery for gallstone pancreatitis or pseudocyst formation.

Acute pancreatitis was first described by Sir Reginald Fitz in 1889, and has served as a continuing challenge to the clinician. The incidence varies with the hospital setting and the patient population. Reportedly and interestingly, pancreatitis associated with gallstones is more common in private hospitals, whereas pancreatitis associated with alcoholism has a greater incidence in charity hospitals. Other causes include metabolic disorders such as hyperlipidemia and hyperparathyroidism, trauma, mumps, and certain drugs, ie, thiazides and sulfonamides. There is also a rare hereditary type of pancreatitis. Postoperative pancreatitis occasionally occurs after surgical procedures involving the biliary tract or the stomach. Less than ten percent of pancreatitis is thought to be idiopathic. Cholelithiasis and excessive alcohol consumption are the two most common etiologies of pancreatitis.

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