

METASTASIZING CARCINOMA OF THE PARATHYROID GLAND WITH OSTEITIS FIBROSA CYSTICA AND EXTENSIVE CALCINOSIS *

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The case described in this paper provides one of the few examples of metastasizing carcinoma of the parathyroid gland studied post mortem, and it has unusual interest because of the functional derangements produced by the tumor. Intensive clinical and laboratory studies were made over a prolonged period, and post-mortem examination was complete.

CLINICAL OBSERVATIONS

The patient was a white housewife, 29 years old, who entered the New York Hospital on June 29, 1949, because of the recurrent symptoms of hyperparathyroidism. In 1943, during her first pregnancy, she had developed extreme fatigue, nausea, and vomiting which persisted until the delivery at term of a malformed baby who died at the age of 5 weeks of congenital heart disease. Her health did not improve after delivery. Nausea and vomiting ceased. Her appetite was seriously impaired; fatigue and weakness persisted. Severe pains developed about many joints and in the back. Over a period of 1 year she lost 40 lbs. in weight. In November, 1944, a specimen was taken for biopsy from a cystic lesion in the right mandible and was diagnosed as giant cell tumor and osteitis fibrosa. In July, 1945, roentgenograms revealed numerous cystic lesions involving the ribs and long bones. The level of the blood calcium at this time was 20 mg. per cent; the phosphorus, 4 mg. per cent. Surgical exploration revealed two tumors which were regarded pathologically as adenomas of the parathyroid gland. The operation was followed by apparent restoration of health. The lost weight was regained and strength returned. Five months later the cystic lesions were reported as completely restored on roentgenologic examination and the serum calcium level was normal.

For 2½ years, and even during the course of a completed second pregnancy, she remained entirely well until in March, 1948, with the beginning of a third pregnancy, she experienced nausea, vomiting, weakness, and pain and aching over the right hip and iliac crest. Pregnancy was interrupted at the sixth month by a cesarean section and the fallopian tubes were tied. Her symptoms, however, did not abate following delivery and in March, 1949, roentgenograms revealed recurrence of the cystic lesions. The kidneys showed rather extensive diffuse parenchymal calcification. The serum calcium level was 22 mg. per cent. Her neck again was explored, with the removal of three parathyroid tumors measuring 2 by 1.5 by 1.5, 1.5 by 1 by 0.5, and 1.2 by 1 by 0.5 cm. Postoperatively her symptoms continued. She vomited at frequent intervals, thirst became extreme, constipation was obstinate, and her weakness was profound. After 3 months she entered the New York Hospital. Examination revealed considerable emaciation. Her legs were bowed and she walked with a limp. The thyroid gland was palpable, but no nodules could be detected in the region of the previous operations. Her muscles were extremely lax. Tender points could be demonstrated at various places over the bones of the forearm, the iliac crests, thighs, and legs.

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The urine was of low specific gravity with numerous leukocytes and with cultures positive for *Escherichia coli*. Phenolsulfonphthalein excretion was only 10 per cent in 2 hours. Intravenous pyelograms exhibited diminished renal function. The Sulzberg test for calcium was 4 plus. The blood urea nitrogen was elevated only moderately at 22 mg. per cent. The level of the serum calcium varied between 14.5 and 18.7 mg. per cent. Phosphorus values varied between 3.5 and 4.6 mg. per cent. Alkaline phosphatase was 15.7 units. Roentgenologic examination showed nephrocalcinosis, numerous areas of osteitis fibrosa cystica, and two small, nodular densities in the left lung field. The series of x-ray films taken at the time of admission failed to reveal calcification of vessels except for one small segment of the left axillary artery.

On a diet containing 130 mg. of calcium and 670 mg. of phosphorus over a period of 6 days the urinary excretion varied from 565 to 995 mg. of calcium and 808 to 921 mg. of phosphorus. Increase in calcium intake to 680 mg. and later to 2.026 gm. did not materially increase the excretion of calcium in the urine. On July 14, 1949, at a third operation, the left lobe of the thyroid gland was removed. Within it there were two small, nodular, white, firm, encapsulated masses which were diagnosed microscopically as carcinoma, since metastatic lesions were present in the left lung. Postoperatively the level of blood calcium remained unchanged and that of the urinary calcium was elevated. On July 25, 1949, two metastatic nodules with diameters of 0.9 and 1.2 cm. were removed from the upper and lower lobes of the left lung. Severe bone pain, nausea, vomiting, and anemia continued, together with elevated levels of blood and urinary calcium. Cough became an increasingly troublesome symptom and roentgenograms of the lungs taken during the course of her illness showed constantly expanding opacities which were interpreted clinically as probable carcinomatous extensions (Fig. 1).

On October 4, 1949, estrogen and testosterone therapy was instituted. This was done with the hope that these agents might act to remineralize the skeleton and thereby diminish the bone pain. Testosterone was given in doses of 25 mg. and progynon in doses of 10,000 rat units daily from October 4 to November 21. Calcium values for the week preceding this treatment ranged from 15.8 to 17.0 mg. per cent. Four days after treatment was started, the value was 12.8 mg. per cent, and for the remainder of the course varied between 11.8 and 13.9 mg. per cent. For a short period, pains were less marked and weakness seemed to be less profound. Racking cough, however, caused great distress. On November 17 it was noted that both hands were becoming pallid and cyanotic. Pulsation could not be observed in the radial or ulnar arteries. Roentgenograms revealed extraordinary calcification, not only of these vessels, but of almost all vessels of the extremities. Death occurred on November 24, 1949, approximately 6 years after the onset of symptoms.

FINDINGS AT NECROPSY

The body was that of a well developed but poorly nourished white female. There were well healed surgical scars over the thyroid area and the left 6th rib. All of the teeth were absent except for the right lower premolars. There were superficial ulcers over both knees, 2 cm. in diameter.

The left lobe of the thyroid gland was absent. The right lobe was normal and on the posterior aspect three parathyroid glands measuring 3 to 6 mm. were identified. The cervical and supraclavicular lymph nodes were enlarged, gray, and soft.

The heart weighed 250 gm. The valves were normal except for extensive calcification of the annulus fibrosus of the mitral ring. The right and left coronary arteries were widely patent and only slightly calcified. A small artery within the interventricular septum was extensively calcified as shown by roentgenologic examination. The tips of the papillary muscles in the left ventricle at the insertion of the chordae tendineae were calcified.

The aorta was elastic and showed little atherosclerosis. However, all of its major branches were extensively calcified to their terminal divisions, as revealed by both gross anatomical and roentgenologic examinations (Fig. 2). Several small branches of the pancreaticoduodenal artery were occluded by calcific deposits. Branches of the splenic artery likewise were completely occluded by a calcific thickening near the hilum. There were two anemic splenic infarcts distal to these occlusions, 5 cm. in diameter, which were partially calcified.

The lungs weighed 1300 gm. Distributed in the periphery of all three lobes of the right lung there were six triangular, sharply circumscribed, firm, yellowish, gritty, calcific infarcts (Figs. 6 and 7). Calcified thrombi were found occluding the pulmonary arteries supplying two of these areas (Fig. 6). Roentgenologic examination revealed slightly diffuse calcification in the remainder of the right and left lungs. No tumor was present.

The liver weighed 1590 gm. and in the left lobe there was a solitary, gray, circumscribed soft tumor mass, measuring 4 by 4 by 5 cm. (Fig. 10). Roentgenologic examination failed to reveal calcification in the tumor or in hepatic vessels. The pancreas weighed 159 gm. and was of unusually firm consistency. Section revealed many focal cystic areas up to 1 cm. in diameter scattered throughout. The ducts and ampulla of Vater were normal. The gastric mucosa was thickened and granular, with prominent rugal folds (Fig. 13). The granularity was due to diffuse calcium deposition throughout all areas as revealed by roentgenologic examination. The remainder of the gastro-intestinal tract was unchanged.

The kidneys weighed 130 gm. each and the capsules stripped with difficulty from finely granular surfaces with numerous large depressed scars (Fig. 17). Sections revealed a diffuse mottling in the cortex and linear streaks of calcium in the medulla (Fig. 18). The medulla was firm and contained numerous cystic areas from 7 to 8 mm. in diameter. The pelves were normal. The renal arteries were calcified and rigid but the lumina were not significantly narrowed. Roentgenograms confirmed the calcium distribution (Fig. 19).

The vertebral bodies contained numerous brown cysts up to 5 mm. in size in the midportion, whereas the remainder of each vertebral body was firm and dense (Fig. 22). The ribs were soft and fractured easily. On section, extensive fibrosis of the marrow was seen and cystic areas surrounded by dense elastic bone tissue were present (Fig. 23). The calvarium was soft and elastic but no cysts were found. The cortex of the femur was of normal thickness but much softer than is normal.

The right adrenal weighed 15 gm. and the left, 10 gm. The adrenals were not unusual except for the presence of two small cortical adenomata in each.

The brain and the remaining organs were unchanged.

Microscopic Examination

Neoplastic tissue was found microscopically in the liver, a left supraclavicular lymph node, and in the left sternocleidomastoid muscle in the region of the surgical incision (Figs. 11 and 12). In all areas the tumor was similar and consisted of sheets and cords of cells of uniform size, closely resembling chief cells. The rounded nuclei contained a diffuse chromatin network; mitotic figures were uncommon. In some areas the cells contained abnormal nuclei. The cytoplasm was abundant, eosinophilic, slightly granular, and contained a moderate amount of glycogen. A few cysts filled with eosinophilic material were noted.

The smaller branches of the coronary arteries revealed diffuse medial calcification without significant intimal proliferation. Calcium was deposited also on the elastic membranes of some arterioles (Fig. 3). Occasional myocardial fibers stained lavender with hematoxylin and eosin and were surrounded by slight interstitial fibrosis. Microscopic sections disclosed calcification of the annulus fibrosus of the mitral ring and of the tips of the papillary muscles. There was moderate serous atrophy of epicardial fat.

The mesenteric arteries, splenic artery, pancreaticoduodenal artery, gastric arteries, brachial and femoral arteries, as well as small vessels in skeletal muscle showed medial calcification and intimal fibrosis in varying degrees (Figs. 4 and 5). In branches of the pancreaticoduodenal, mesenteric, and splenic arteries, and in a small vessel in the interventricular septum of the heart, the medial calcification and intimal fibrosis significantly narrowed the vessel lumen. The lumina of the larger vessels, however, were widely patent.

Sections through the calcified infarcts revealed that the alveolar

spaces were completely filled with a loose vascular fibrous tissue. The alveolar walls were calcified and a calcific ring encircled the capillaries when viewed in cross section (Fig. 8). The occluding thrombi were partially organized and calcified. The remainder of the lung parenchyma showed patchy calcification of the alveolar walls. A considerable number of multinucleated giant cells and phagocytes often were adjacent to the calcium salts (Fig. 9). There was also calcification of some of the pulmonary arteries and veins, as well as of the bronchi.

There was focal and diffuse scarring of the kidneys. In the cortex many glomeruli were atrophic, fibrotic, and calcified. Many normal appearing glomeruli showed varying degrees of calcium deposition of the capillary basement membrane, Bowman's membrane, and of the efferent and afferent arterioles. Calcium deposits surrounded atrophic and normal proximal and distal tubules (Fig. 20). A considerable number of collecting tubules were dilated and many contained concentric masses of calcium (Fig. 21). A few neutrophils were found in some of the dilated tubules.

Throughout all areas of the stomach the gastric glands were greatly dilated and the mucosa thickened. The lumina of the glands were greatly distended with lamellated masses of inspissated secretion, often partially calcified (Fig. 14). In addition the interstitial tissue was partially calcified. Calcification of individual cells was not present.

The marrow spaces were filled with loose vascular fibrous tissue containing scattered giant cells and hemosiderin-laden macrophages (Fig. 24). The areas of increased density surrounding cystic lesions in the ribs were composed of an increased number of irregular bony trabeculae separated by marked marrow fibrosis. The trabeculae were poorly calcified, had a serrated outline due to osteoclastic resorption, and many were devoid of normal lamellar structure. Some trabeculae showed osteoclasts on one surface and rows of osteoblasts on the opposite surface (Fig. 25). The central areas of the vertebrae contained active marrow, whereas the outer areas showed marrow fibrosis and bony trabeculae as described above. The calvarium was similar.

There was marked pancreatic atrophy with interstitial fibrosis, together with much lymphocytic and a smaller amount of neutrophilic infiltration. Many dilated ducts of medium size contained calcified debris and concentric masses of eosinophilic material, probably inspissated secretion (Fig. 16). The islands of Langerhans were normal. Many muscular arteries showed extensive medial calcification and intimal fibrosis.

Sections from all three right parathyroid glands showed a marked

increase in fibrous tissue, often with isolation of groups of glandular cells. Chief cells composed the bulk of the parenchymal tissue. Many small vesicles filled with a pink colloid material were present (Fig. 15).

The zona reticularis and zona fasciculata were markedly vacuolated due to lipid deposition. The adenomata were composed of normal cortical cells. The medulla was normal.

Diagnoses

The final diagnoses were as follows: Resected carcinoma of the left parathyroid gland with metastases in left lung; metastatic carcinoma in left sternocleidomastoid muscle, left supraclavicular lymph node, and liver; extensive metastatic calcification of the lungs, kidneys, stomach, arteries, and heart; calcific occlusion of splenic arteries with splenic infarction; thrombosis of pulmonary arteries with pulmonary infarction and calcification; generalized osteitis fibrosa cystica involving the ribs, sternum, vertebrae, femur, and calvarium; fibrosis and slight atrophy of right parathyroid glands; chronic interstitial pancreatitis; cortical adenomata of the adrenal glands.

DISCUSSION

In contrast with the paucity of reported cases of metastatic carcinoma of the parathyroid glands, Norris,¹ in 1947, cited 322 cases of adenoma, and Black and Ackerman,² in 1950, added 22 cases to the literature. Previously the only reported cases of functioning parathyroid carcinoma with distant metastases which had been carefully studied both clinically and post mortem were those of Meyer and Ragins³ and of King and Wood.⁴ Several cases, however, have been reported with local recurrence and with either infiltration or regional lymph node metastasis.^{2,5-8} In the present case the tumor was obviously malignant as evidenced by local recurrence, regional lymphatic extension, and metastases in lung and liver. It is noteworthy, however, that microscopically it was not possible to distinguish this carcinoma from the parathyroid adenomas reported in the literature.^{1,2,9}

The changes of generalized osteitis fibrosa cystica were entirely typical, but less extensive than those reported in several other cases.¹⁰⁻¹²

The extreme degree of medial calcification of the arterial system was most interesting and the functional results of arterial insufficiency have not been emphasized previously, although somewhat similar findings were reported in 1936 by Magnus and Scott¹³ in a case of chronic nephritis with parathyroid hyperplasia and extensive pathologic calcification of the peripheral arteries. The influence of testosterone and estrogen therapy on the degree of soft tissue calcification cannot be

assessed. It is of interest to note, however, that the institution of this treatment was followed by significant lowering of serum calcium and that the calcium deposit in arteries which had been only slightly evident in the roentgenograms in June, 1949, had become extreme in November, 1949, after 6 weeks of hormone treatment.

The degree of calcium deposition in areas normally supplied by pulmonary arteries which were thrombosed was much more extensive than in the remainder of the lung. The position, shape, and histologic appearance of these regions were consistent with calcification in areas of local tissue damage or infarction. The degree of alveolar calcification in the remainder of the lungs was similar to that described in other cases.^{10,14} No carcinoma was demonstrated in the lungs and the opacities interpreted clinically as carcinoma were in all probability attributable to the calcified infarcts.

Renal calcification was limited to the parenchymal tissue and was not accompanied by the formation of calculi. Functional renal impairment was evident clinically, but, despite the massive calcification and extensive interstitial and glomerular sclerosis, never led to obvious uremia. The calcium deposit was present to some degree in and about all segments of the tubules, the glomeruli, arteries, and arterioles. Roentgenologic examination, however, revealed a greater concentration in the medulla than in the cortex.

Myocardial degeneration is a very striking change in experimental animals dying of excessive doses of parathormone.^{15,16} Here the morphologic findings were rather inconspicuous, consisting of calcification, degeneration of scattered myocardial fibers, and focal fibrosis. Calcification of chordae tendineae also has been noted in animals given excessive doses of parathormone.¹⁶

Metastatic calcification of the stomach usually affects the distal or acid-bearing portion, although in the present case there was diffuse calcium deposition in all areas. Microscopically, calcification of the interstitial tissue and glandular cells has been emphasized hitherto; no cases have been found with the degree of dilatation and calcification of the mass of contained secretion found in this case.

The diffuse fibrosis and slight atrophy of the three remaining parathyroid glands deserve mentioning. It is questionable whether these changes were due to surgical trauma, to an involutionary change resulting from hypersecretion of parathyroid hormone by the tumor cells, or to some other cause. In this relation it is noteworthy that Jaffe and Bodansky¹⁷ described functional involutional atrophy of the parathyroid glands in dogs dying of induced acute hyperparathyroidism, while McJunkin, Tweedy, and Breuhaus¹⁸ found that para-

thyroid hormone inhibited mitotic activity of parathyroid cells when given to rats in amounts insufficient to produce hypercalcemia and destructive lesions of parenchymatous organs.

SUMMARY

In a case of metastasizing carcinoma of the parathyroid gland with osteitis fibrosa cystica and extensive calcinosis, the tumors removed surgically from the neck and lung and those found post mortem in the sternocleidomastoid muscle and liver were alike in histologic appearance. They were composed for the most part of well differentiated cells resembling chief cells, which could not be differentiated by morphologic criteria from the elements of growths generally diagnosed as parathyroid adenomas.

Three remaining parathyroid glands were neither neoplastic nor hyperplastic, but showed fibrosis and atrophy. The possibility was considered that this may have been an involutionary change resulting from hypersecretion of parathyroid hormone by the neoplastic cells.

Osteitis fibrosa cystica was generalized but less extensive than in a number of previously reported cases of hyperparathyroidism.

Other features were a very marked degree of medial calcification of arteries of medium and small size, associated clinically with pallor, cyanosis, and circulatory insufficiency of the extremities; extensive deposition of calcium in infarcted areas of the lungs; widespread renal calcinosis without obvious nephrolithiasis or renal failure; and metastatic calcification of the gastric mucosa, which was not limited to cells engaged in acid secretion.

REFERENCES

1. Norris, E. H. The parathyroid adenoma. A study of 322 cases. *Internat. Abstr. Surg.*, 1947, **84**, 1-41.
2. Black, B. K., and Ackerman, L. V. Tumors of the parathyroid; a review of 23 cases. *Cancer*, 1950, **3**, 415-444.
3. Meyer, K. A., and Ragins, A. B. Carcinoma of the parathyroid gland. *Surgery*, 1943, **14**, 282-295.
4. King, E. S. J., and Wood, B. Parathyroid tumour with visceral metastases. *J. Path. & Bact.*, 1950, **62**, 29-35.
5. Norris, E. H. Carcinoma of the parathyroid glands with a preliminary report of 3 cases. *Internat. Abstr. Surg.*, 1948, **86**, 1-21.
6. Gentile, R. J., Skinner, H. L., and Ashburn, L. L. The parathyroid glands: malignant tumor with osteitis fibrosa cystica. *Surgery*, 1941, **10**, 793-810.
7. Black, B. M. Adenocarcinoma of parathyroid origin with hyperparathyroidism, local recurrence and metastases: report of case. *Proc. Staff Meet., Mayo Clin.*, 1948, **23**, 8-14.
8. Young, J. H., and Emerson, K., Jr. Parathyroid carcinoma associated with acute parathyroid intoxication. *Ann. Int. Med.*, 1949, **30**, 823-837.

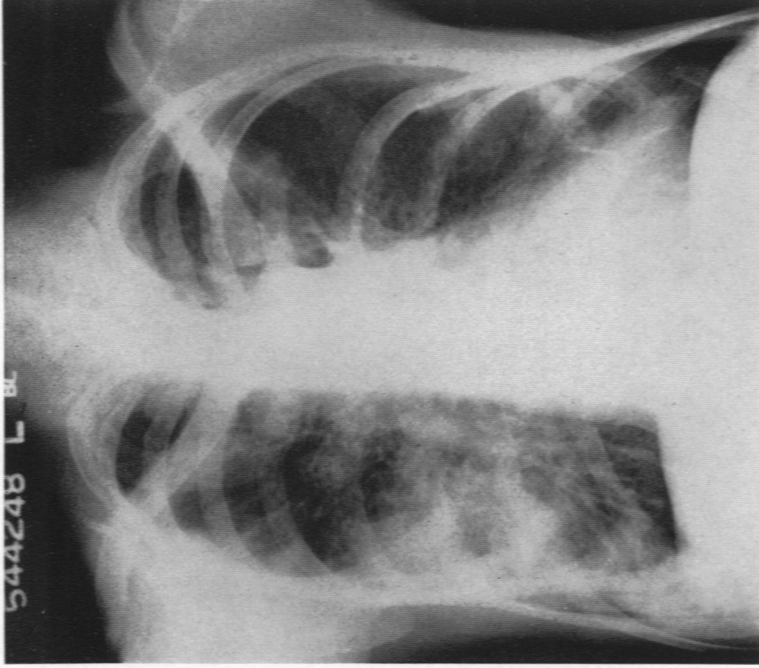
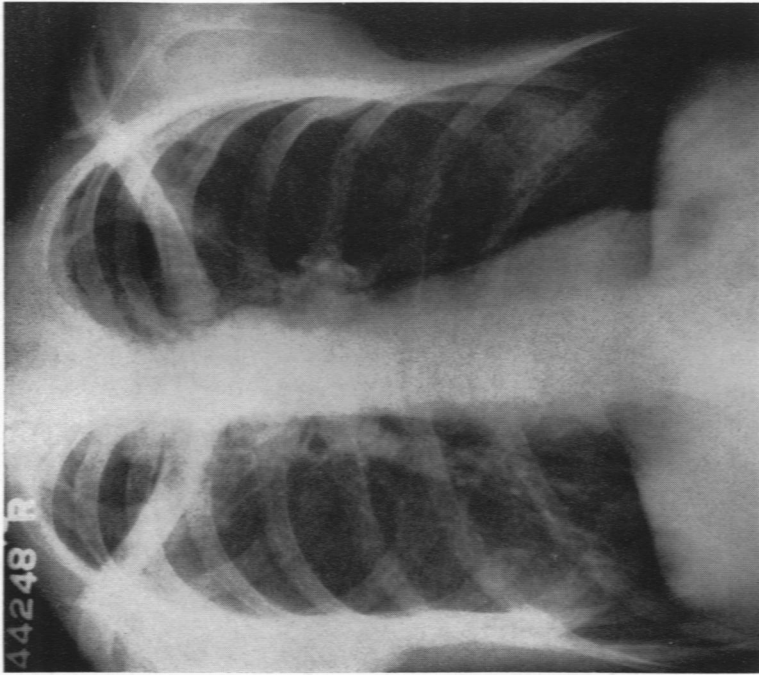
9. Castleman, B., and Mallory, T. B. The pathology of the parathyroid gland in hyperparathyroidism; a study of 25 cases. *Am. J. Path.*, 1935, 11, 1-72.
10. Dawson, J. W., and Struthers, J. W. Generalised osteitis fibrosa with parathyroid tumour and metastatic calcification. *Edinburgh M. J.*, 1923, 30, 421-564.
11. Jaffe, H. L. Hyperparathyroidism. *Bull. New York Acad. Med.*, 1940, 16, 291-311.
12. Hunter, D., and Turnbull, H. M. Hyperparathyroidism: generalized osteitis fibrosa. *Brit. J. Surg.*, 1931-32, 19, 203-284.
13. Magnus, H. A., and Scott, R. B. Chronic renal destruction and parathyroid hyperplasia. *J. Path. & Bact.*, 1936, 42, 665-672.
14. Barr, D. P., and Bulger, H. A. The clinical syndrome of hyperparathyroidism. *Am. J. M. Sc.*, 1930, 179, 449-476.
15. Cantarow, A., Stewart, H. L., and Housel, E. L. Experimental acute hyperparathyroidism. *Endocrinology*, 1938, 22, 13-27.
16. Hueper, W. Metastatic calcifications in the organs of the dog after injections of parathyroid extract. *Arch. Path.*, 1927, 3, 14-25.
17. Jaffe, H. L., and Bodansky, A. Experimental fibrous osteodystrophy (osteitis fibrosa) in hyperparathyroid dogs. *J. Exper. Med.*, 1930, 52, 669-694.
18. McJunkin, F. A., Tweedy, W. R., and Breuhaus, H. C. The parathyroid hormone. *Arch. Path.*, 1932, 14, 649-659.

[*Illustrations follow*]

DESCRIPTION OF PLATES

PLATE 70

FIG. 1. Roentgenograms of chest (A) taken on July 5, 1949, shortly after admission, showing indistinctly in the left lung two discrete areas which were shown to be carcinomatous metastases; (B) taken on October 22, 1949, showing extensive opacity.



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Carcinoma of the Parathyroid Gland

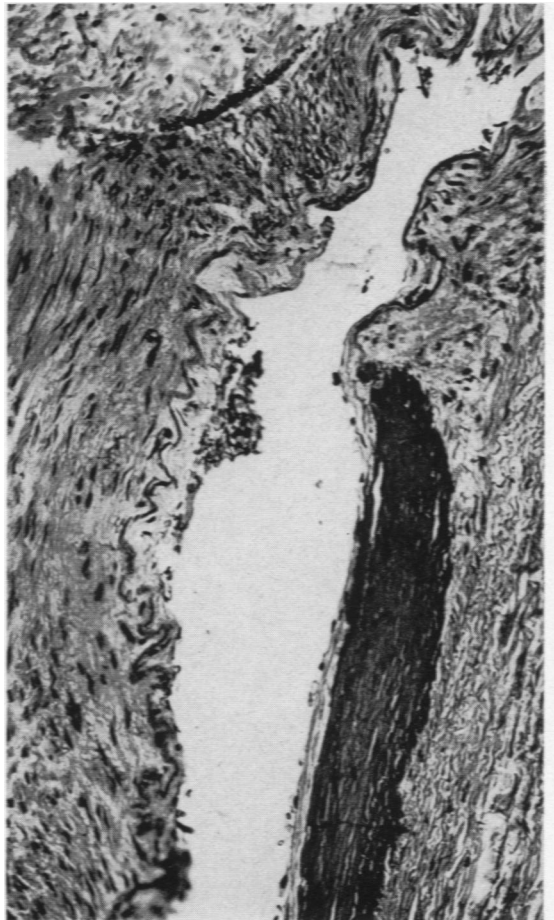
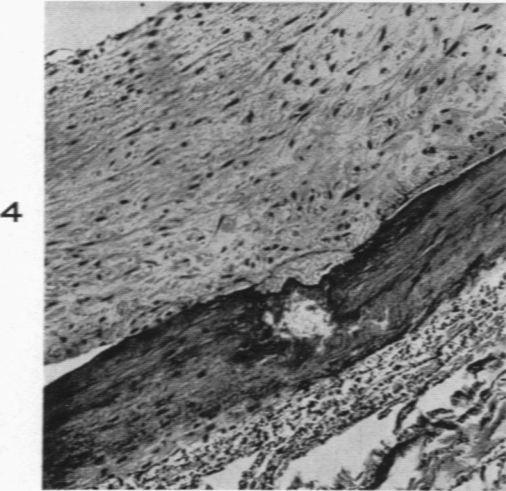
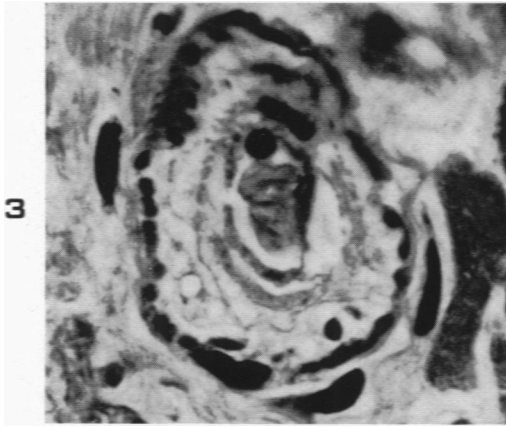
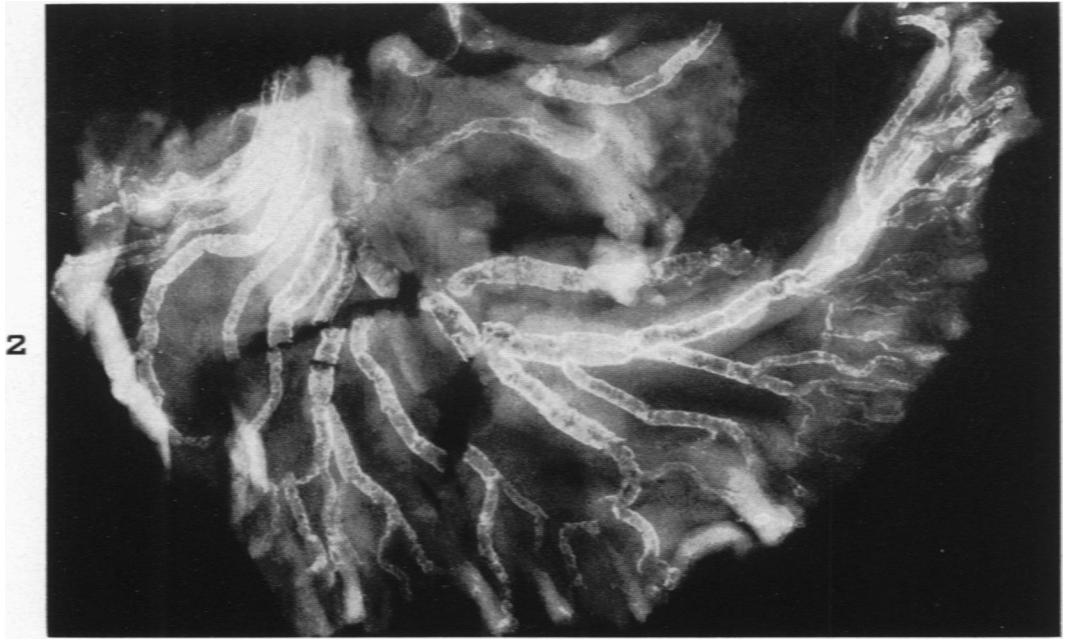
PLATE 71

FIG. 2. Roentgenogram, showing diffuse calcification of the superior mesenteric artery and its branches.

FIG. 3. Calcification of elastica of arteriole of heart. Hematoxylin and eosin stain. $\times 1260$.

FIG. 4. Medial calcification and intimal fibrosis of mesenteric artery. Hematoxylin and eosin stain. $\times 140$.

FIG. 5. Calcification of media of small artery in skeletal muscle. Hematoxylin and eosin stain. $\times 190$.



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PLATE 72

FIG. 6. Thrombus in a branch of the pulmonary artery, with pyramidal area of calcification in the distribution of the artery.

FIG. 7. Higher magnification of another calcified infarct. There is emphysema and moderate calcification of adjacent lung parenchyma.

FIG. 8. Organizing infarct showing calcification largely restricted to alveolar walls and capillaries. Hematoxylin and eosin stain. $\times 180$.

FIG. 9. Calcification of the type found scattered diffusely throughout the lungs. Of note are the multinucleated giant cell reaction and the alveolar fibrosis. Hematoxylin and eosin stain. $\times 120$.



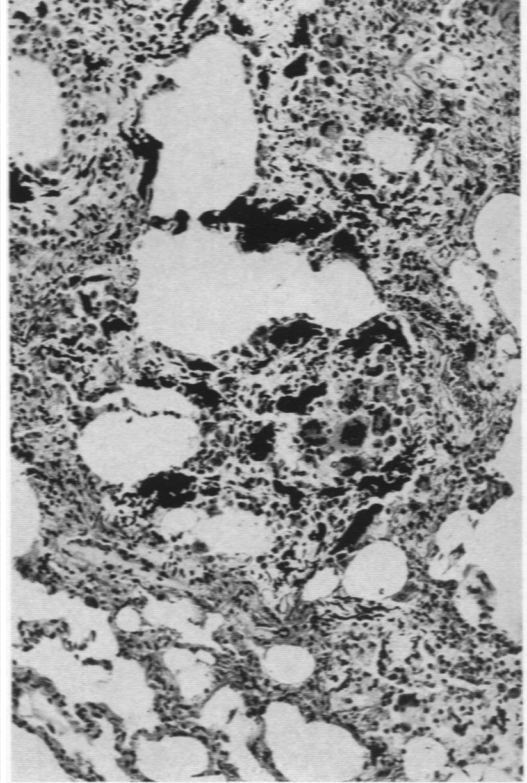
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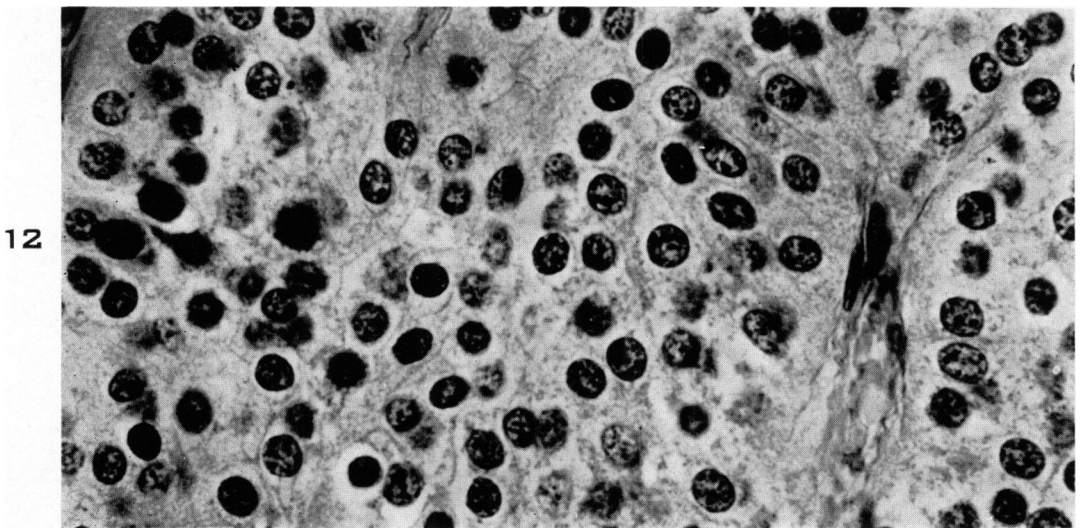
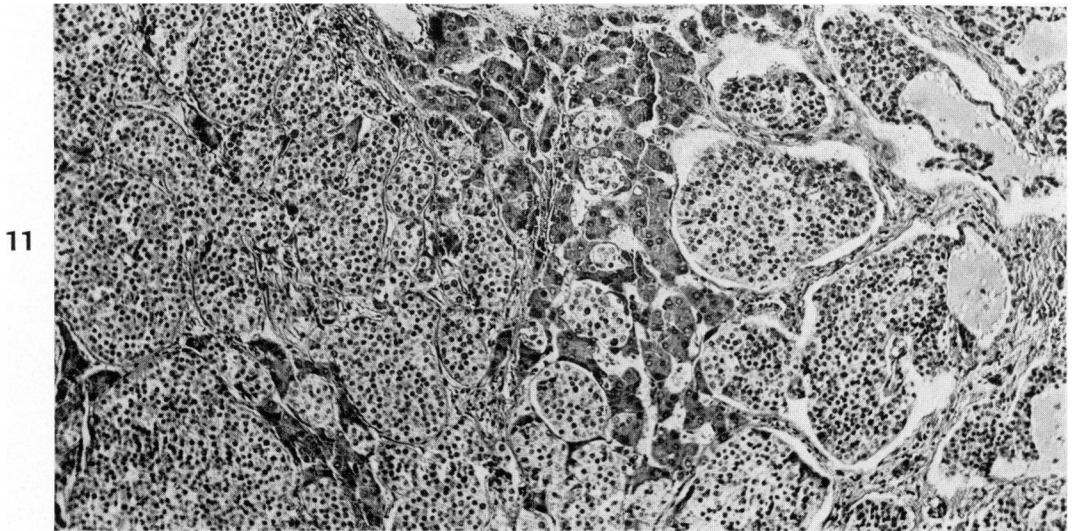
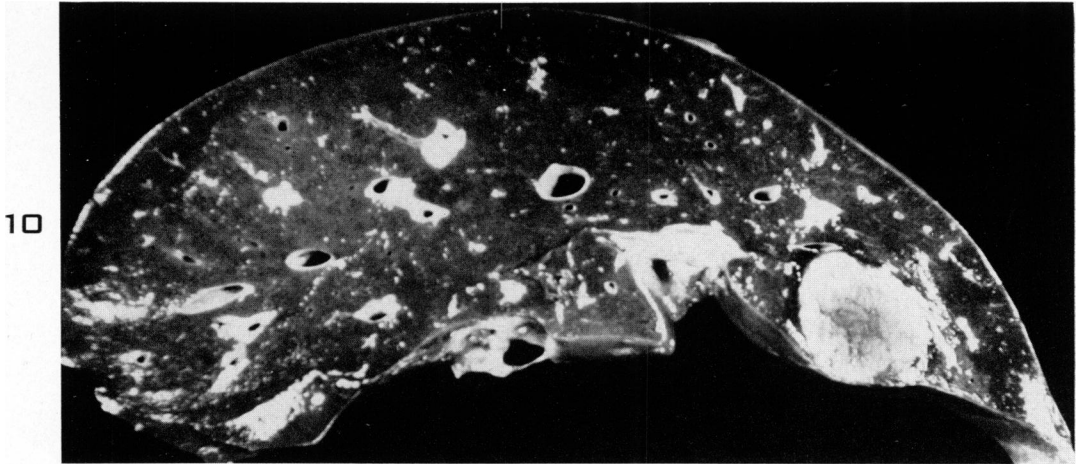
Carcinoma of the Parathyroid Gland

PLATE 73

FIG. 10. Solitary, circumscribed metastasis of carcinoma in the left lobe of liver.

FIG. 11. Microscopic detail of the margin of the hepatic metastasis, showing colloid-filled spaces at the right. Hematoxylin and eosin stain. $\times 130$.

FIG. 12. Higher power of carcinoma in left supraclavicular lymph node. Hematoxylin and eosin stain. $\times 300$.

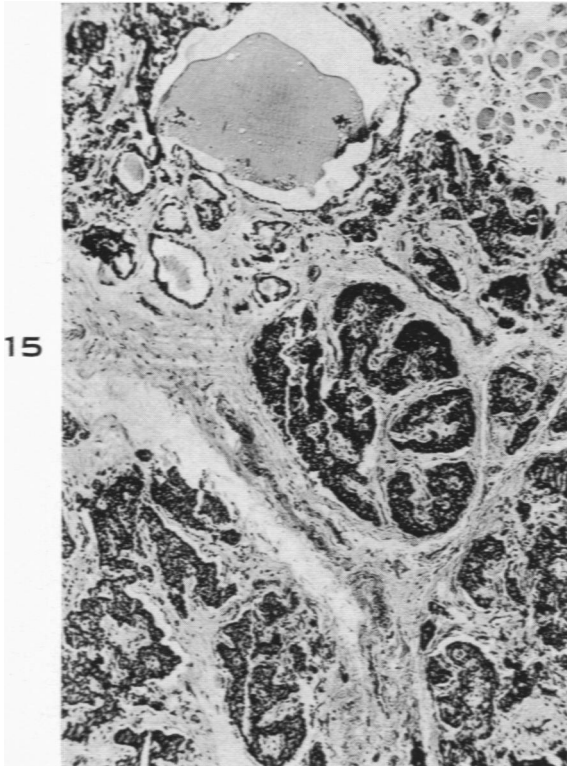
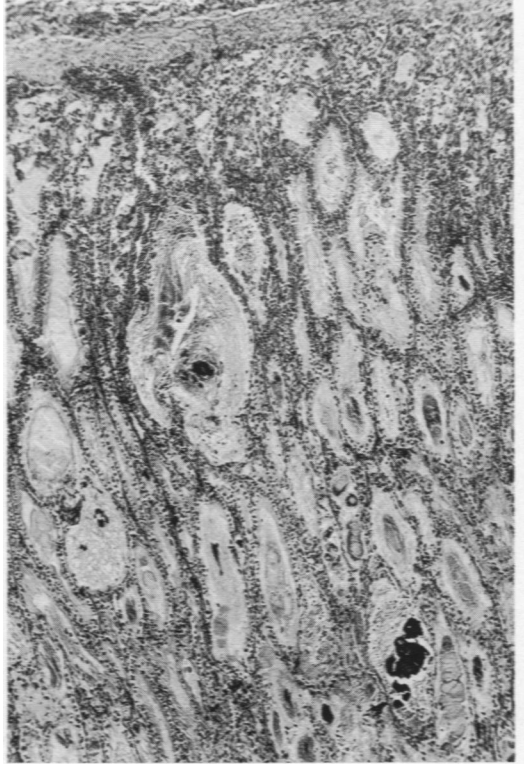
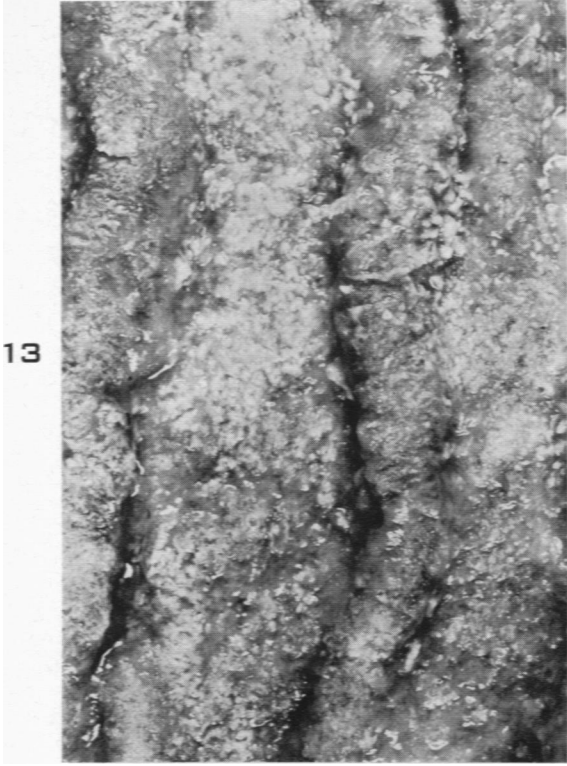


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PLATE 74

- FIG. 13. Mucosa of stomach showing coarsened rugal pattern with fine granularity due to plugging of glands with calcified material.
- FIG. 14. Dilatation of gastric glands containing calcified debris and secretion. There is moderate calcification of interstitial connective tissue between the glands. Hematoxylin and eosin stain. $\times 50$.
- FIG. 15. Fibrosis and slight atrophy of one of the remaining parathyroid glands. Hematoxylin and eosin stain. $\times 800$.
- FIG. 16. Dilatation of pancreatic ducts containing lamellated contents. There is marked interstitial fibrosis of the surrounding glandular tissues, with infiltration by lymphocytes. Hematoxylin and eosin stain. $\times 50$.



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Carcinoma of the Parathyroid Gland

PLATE 75

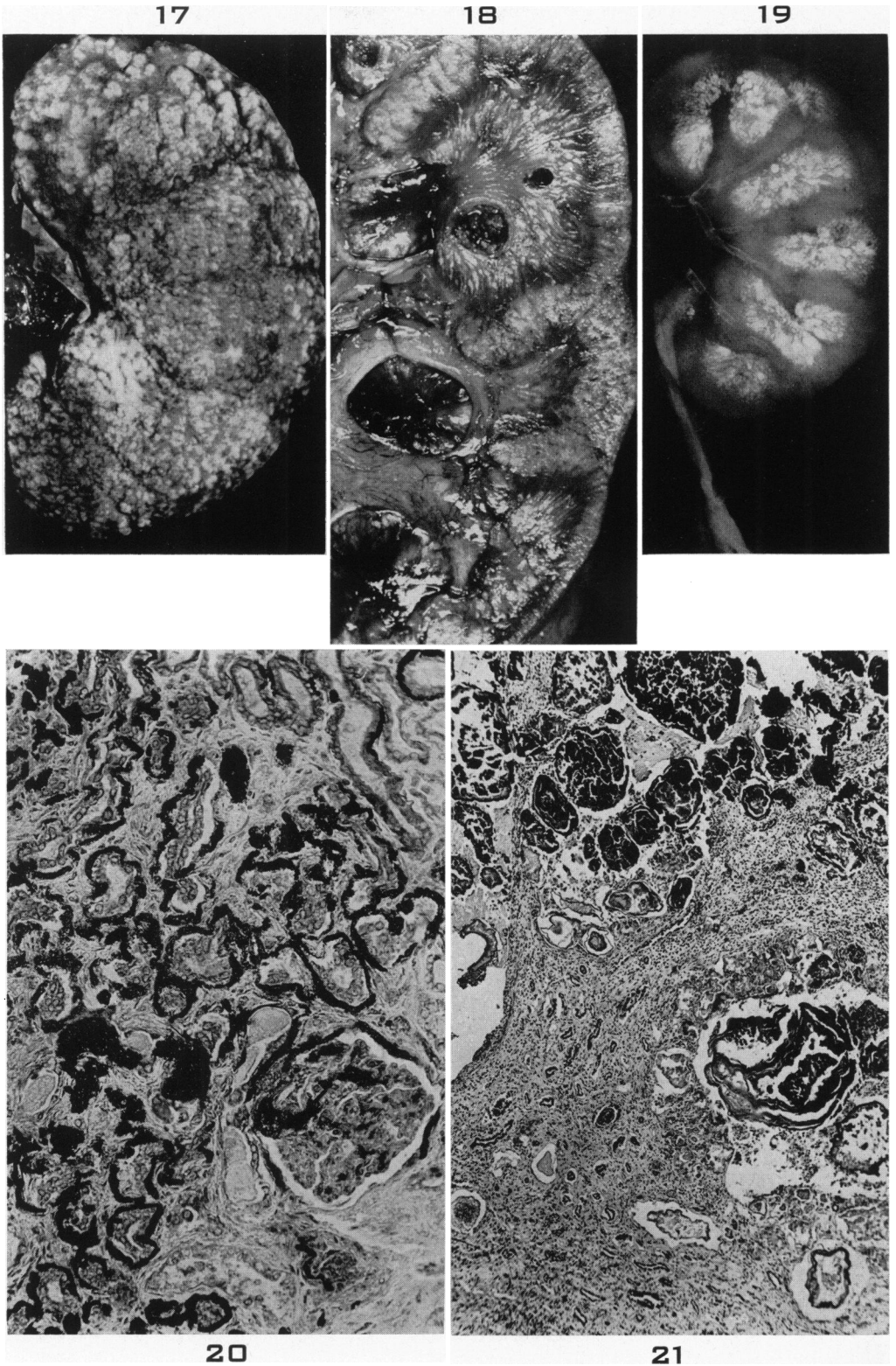
FIG. 17. Diffuse granularity of kidney surface, with larger depressed scars at the upper pole.

FIG. 18. Cut surface of kidney illustrating diffuse cortical calcification, linear streaks of calcium in medullary rays, and medullary cysts. The renal pelvis is normal.

FIG. 19. Roentgenogram of kidney. Calcium deposition is greatest in medulla. The renal arteries are diffusely outlined by calcium.

FIG. 20. Calcification of glomerular arterioles, Bowman's membrane, and peritubular membranes, with occlusion of tubules by calcium. Von Kossa's stain. $\times 180$.

FIG. 21. Concentric masses of calcium in renal medulla, with tubular dilatation and interstitial fibrosis. Hematoxylin and eosin stain. $\times 50$.



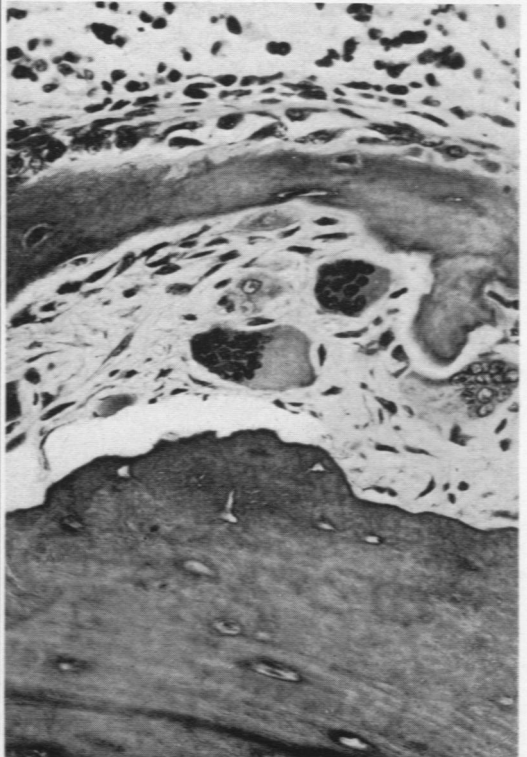
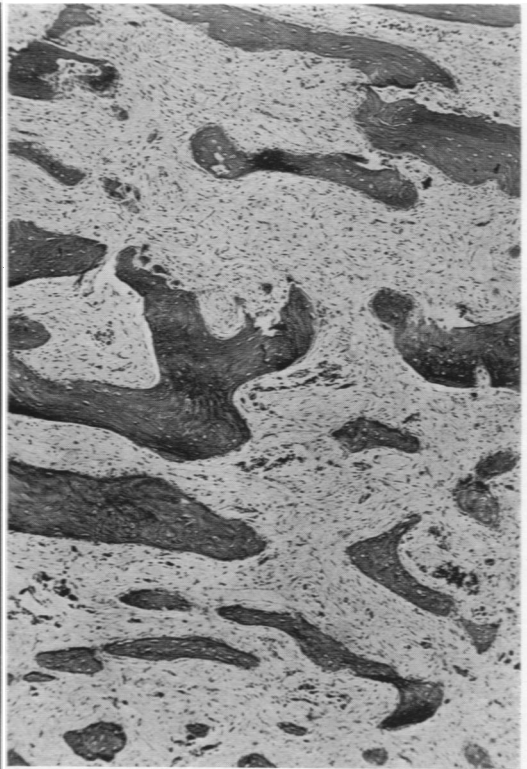
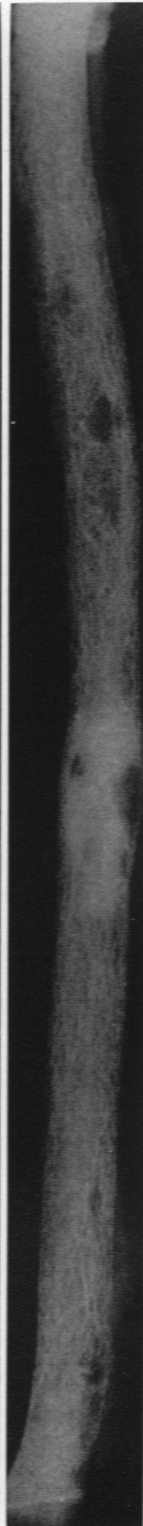
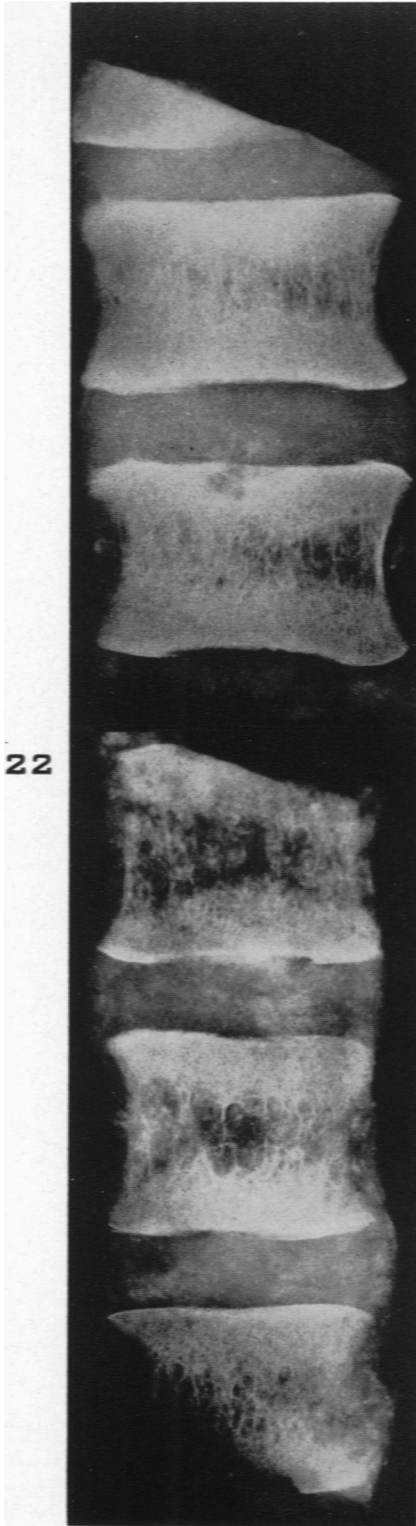
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PLATE 76

- FIG. 22. Roentgenograms of vertebrae. Osteoporosis with disappearance of central trabeculae and condensation of remainder of the vertebral body.
- FIG. 23. Roentgenogram of rib illustrating multiple areas of cyst formation.
- FIG. 24. Rib. Resorption of old bone with complete fibrosis of the marrow spaces. Hematoxylin and eosin stain. $\times 50$.
- FIG. 25. Vertebra. Bony trabeculae undergoing absorption. Narrow bridge of new bone with absence of lamellar pattern surfaced on lower side by syncytial osteoclastic giant cells and on the upper side by proliferating osteoblasts. Hematoxylin and eosin stain. $\times 280$.

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Carcinoma of the Parathyroid Gland