A CASE OF HYPERPARATHYROIDISM WITH THYROTOXICOSIS

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Primary hyperparathyroidism can present itself in the following manner (Keynes, 1961):

1. Renal stones, with pain, secondary infection of the urinary tract or renal hypertension.
2. Bone diseases. Osteoporosis with or without cyst and tumour; pathological fracture; bone deformities such as collapse of the vertebrae, alteration of the shape of the hands or tumours in the jaw.
3. Combination of the above two.
4. Symptoms due to hypercalcaemia. Vague symptoms of fatigue, lassitude, weakness, hypotonia, bradycardia, anorexia and constipation. Mental disturbances. Polyuria and polydipsia.
5. Peptic ulceration. St. Goar (1957) found that 4 out of 45 cases of hyperparathyroidism had peptic ulceration (8.8%). The mechanism is not known.
6. Pancreatitis and pancreatic lithiasis due to pancreatic calcification.
7. Presenting as part of a syndrome of multiple endocrine adenomas involving also the pituitary (chromophobe or chromophile adenomas), the pancreas (non-insulin producing islet cell tumour or insulin producing islet cell tumour), the thyroid (with or without hyperthyroidism) and/or the adrenals.

Underdahl et al (1953) reviewed all cases of multiple endocrine adenomas in literature. In the 14 cases reviewed, either two or all three of the following were involved: the pituitary, the pancreas and the parathyroids. Of these cases, the thyroid was involved in 4 and only one case presented with clinical hyperthyroidism. This was a female of 22 (Claud and Bandonim, 1911), who also had acromegaly. At autopsy, an eosinophilic adenoma (48 gm.), 4 enlarged parathyroid glands (2 size of kidney bean and 2 size of small hazel nut), enlarged adrenals (17 gm.) and an enlarged thyroid gland (190 gm.) were found. In his own series of 8 cases, the total experience of the Mayo Clinic up to 1953, there was only one case in which both the thyroid and the parathyroids were involved. This was a man of 38 presenting with hyperinsulinism; an islet cell tumour 3.5 cm. in diameter was removed at operation, and at autopsy, two more islet cell tumours (size 1.5 cm. and 4 cm. in diameter respectively, the latter being cystic), three parathyroid adenomas (total weight 3.88 gm.), and a cyst 1.5 cm. in diameter in the right lobe of the thyroid gland were found. The kidneys were normal thereby ruling out secondary hyperparathyroidism. In a series of 10 cases of hyperparathyroidism, Cope et al (1958) described a form of parathyroid hyperplasia involving all four glands, which he called chief cell hyperplasia. In four of the 10 cases, the thyroid gland was involved. Two of the four thyroid lesions were adenomas, one showed squamous cell metaplasia, and the other nodular enlargement. None of these, however, showed clinical hyperthyroidism.

The object of this paper is to present a case of primary hyperparathyroidism with an enlarged thyroid probably toxic.

She was a Chinese female, aged 62, admitted to medical unit II of general hospital, Singapore, on 29.11.60 with complaints of dyspnoea and cough for one week. Dyspnoea was progressive, and she was orthopnaoeic on admission. Cough was productive with whitish thick sputum. She noticed a nodule over the front of her neck for the past 48 years. The mass had remained stationery in size and had never been painful or tender. There were no symptoms of thyrotoxicosis. 10 years ago, she had a fall and fractured one of her legs. While staying in the hospital, she had another fall resulting in a fracture of the other leg. She was able to move about with the aid of crutches at discharge a few months later, but since then there was a progressive deformity of her lower limbs. For the past $1\frac{1}{2}$ years, she had swelling of legs off and on for which she never sought treatment. Bowel and micturition habits were normal. Her appetite remained fair and there was no marked loss of weight. Her diet consisted of average Chinese diet with rice, porridge, vegetables, fish and occasionally meat.

On examination: General condition fair. Thin and small built. Temperature 100°F. Orthopnoaeic and slightly dehydrated. Neck veins engorged. Mild pitting ankle oedema. Thyroid gland diffusely enlarged and nodular, firm in

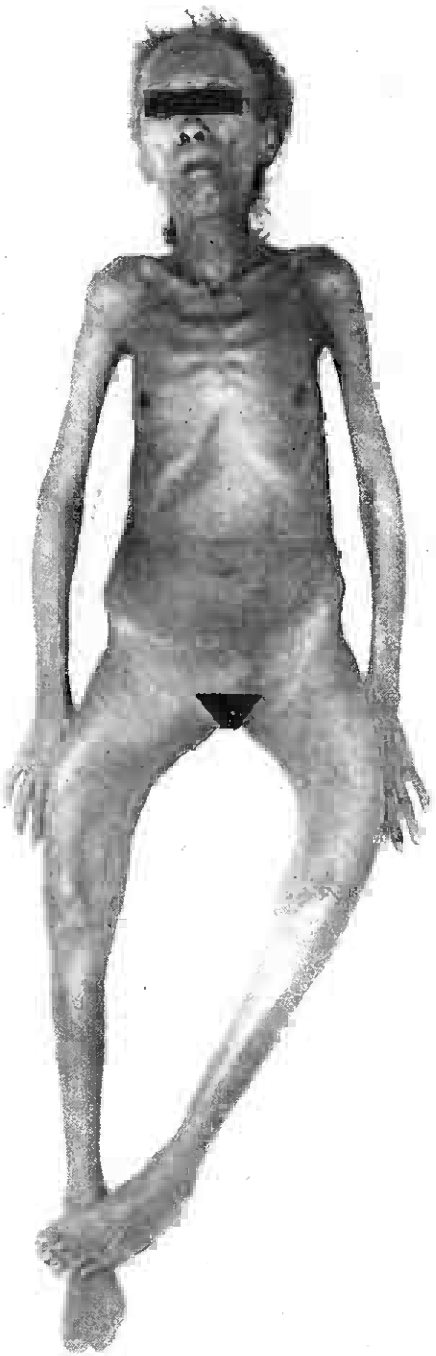

Fig. 1

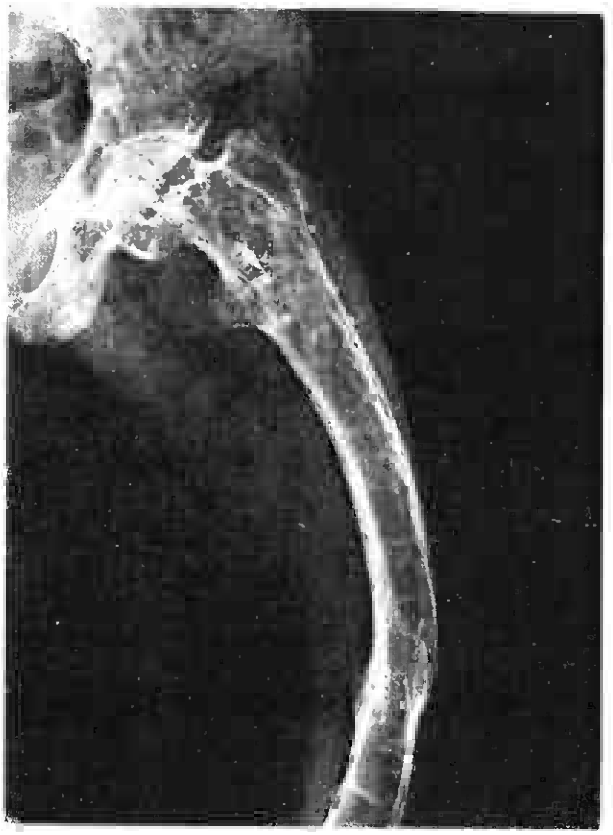

Fig. 2

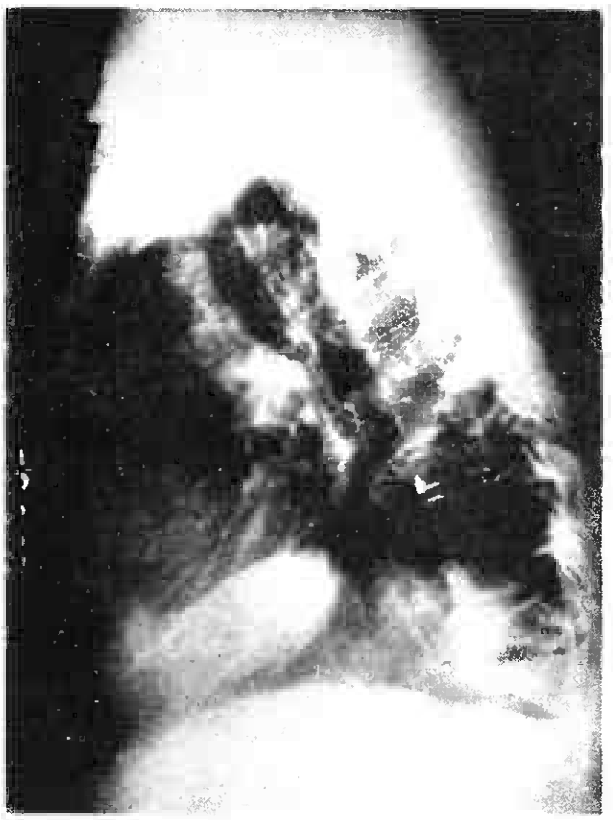

Fig. 3

consistency with no thrill or bruit and no retrosternal extension; a hard nodule 1" in diameter present over isthmus of thyroid gland with no signs of local inflammation. No exophthalmos. No tremor of hands. Pulse 110, regular. B.P. 150/50. Heart not enlarged, dual rhythm, systolic murmur all over precordium. Trachea central. Percussion note impaired, air entry diminished, tactile fremitus and vocal resonance increased over right base; fine crepitations over both bases. Abdomen: soft; liver 1 f.b.; spleen and kidneys not palpable. C.N.S.: N.A.D. Skeletal system: marked kyphosis mainly over thoroco-lumbar spine; scoliosis of thoracic spine; bowing deformity and flattening of long bones of both limbs; old healed fractures were felt over shafts of both femora (Fig. 1).

A preliminary diagnosis of bronchopneumonia with failure was made, and the patient improved with digitalis, penicillin and diuretics. A B.M.R. was done on 8.12.60: 53% and it came down to 29% on 30.12.60 after the exhibition of neomercazole.

Laboratory investigations: Hb. 50%; total white count 4,300; P. 63%, L. 16%, M. 3% E. 8%. B.S.R. 63 mm. Urine normal, no Bence Jones protein. Sputum: negative for acid fast bacilli and malignant cells; no bacterial growth on culture. Serum calcium: 12 mg%; serum inorganic phosphate: 2-2.5 mg%; blood alkaline phosphatase: 25 K.A. units. Blood urea: 38 mg%; blood creatinine: 1 mg%. Urinary calcium: 7-23 mg in 24 hours; inorganic phosphate: 176 mg in 24 hours. (Normal: calcium 150 mg and inorganic phosphate 1-5 gm in 24 hours). Urea clearance: 68%; creatinine clearance: 58 L/hr. Urine concentration and dilution test: normal. Sternal puncture: marrow normal.

Radiological findings: Abdomen: no radio-opaque stone seen. Chest: right lower pneumonitis and heart failure. Skull: On the thick side; sella turcica of normal size. Skeleton: Gross thinning of bone with bowing of femora and old fractures at mid shafts; no cysts present, anterior wedging of most of the vertebrae D11 and D12 are compressed (Figures 2, 3 and 4).

She was diagnosed as a case of primary hyperparathyroidism, but unfortunately she refused to have surgical interference, and went home after a period of rest in hospital.

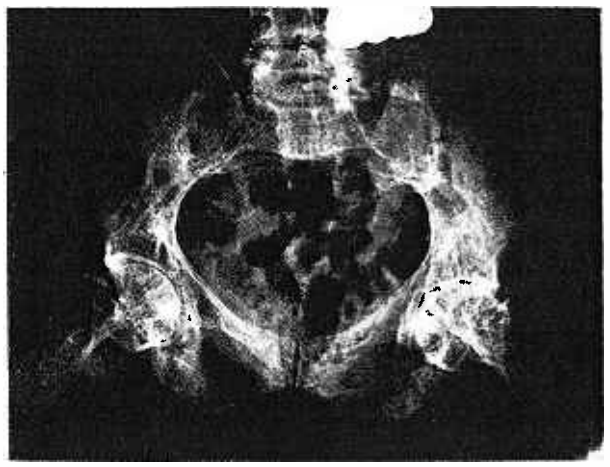

Fig. 4

This is therefore a case of generalised osteoporosis of long standing in view of the history of fracture 10 years ago. The blood biochemical findings of a high calcium, low inorganic phosphate and moderately increased alkaline phosphatase are indicative of hyperparathyroidism. However, the low calcium and inorganic phosphate excretions in urine do not correspond with that of hyperparathyroidism in which the outputs are increased. This could be explained by the presence of mild impairment of renal function in this patient as shown by the very low creatinine clearance, low urea clearance, normal urine concentration and dilution test, normal blood urea and normal blood pressure. Mild renal impairment can arise as a result of the hypercalcaemia. It is unlikely that the hyperparathyroidism is secondary to renal dysfunction because of the high serum calcium level. The enlarged thyroid and the therapeutic response to neomercazole indicate the presence of hyperthyroidism. This case is therefore one of multiple endocrine adenomas involving both the thyroid and parathyroid glands.

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