Hypercalcaemia and phaeochromocytoma

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Summary

A patient with a phaeochromocytoma associated with hypercalcaemia is described. The hypercalcaemia was corrected by removal of the phaeochromocytoma. Parathyroid hormone-like peptide was isolated from the tumour suggesting that ectopic parathyroid hormone production from the phaeochromocytoma was the explanation of the hypercalcaemia.

Introduction

The association of hypercalcaemia with phaeochromocytoma usually occurs as part of the multiple endocrine neoplasia syndrome type II, which includes medullary carcinoma of the thyroid, phaeochromocytoma and parathyroid adenomas or hyperplasia (Sipple, 1961). However, Swinton, Clerkin and Flint (1972) described a patient with hypercalcaemia which was corrected by removal of a phaeochromocytoma. Further reports of hypercalcaemia reversed by removal of phaeochromocytomas have been made (Kukreja *et al.*, 1973; Ghose *et al.*, 1976; Gray and Gillan, 1976). Kukreja *et al.* (1973) suggested that the hypercalcaemia was due to excess catecholamines stimulating the parathyroid glands to produce excess parathyroid hormone (PTH). This explanation has been challenged by Miller *et al.* (1975).

The authors now describe a further patient with a phaeochromocytoma associated with hypercalcaemia, which was corrected by removal of the tumour.

Case report

A 47-year-old male presented with a 6-month history of thirst and polyuria. He had also noted attacks of nocturnal sweating with palpitations occurring 2 or 3 times per week for 6 months. He had been receiving therapy for hypertension for the past 5 years—initially methyldopa, but for the past 3 years propranolol and a thiazide diuretic. There was no dyspnoea or weight loss. He had one brother who was normotensive.

Examination revealed a well nourished patient. His pulse was regular at 80/min and the blood pressure was 160/100 mmHg. There was no clinical cardiomegaly, oedema, neck vein distension, abdominal tenderness or palpable mass. The fundi were normal.

Investigations

Hb 15.8 g/dl; ESR 35 mm fall in one hr; serum sodium 134 mmol/l; serum potassium 4.2 mmol/l; serum chloride 98 mmol/l; serum calcium 3.54 mmol/l; serum phosphate 0.98 mmol/l; alkaline phosphate 13 KA.U.; urea 4.1 mmol/l; urine calcium excretion whilst taking a normal ward diet was 14.5 mmol/24 hr; total serum proteins 74.4 g/l; serum albumin 41.8 g/l; glucose tolerance test revealed a mild diabetic curve with a fasting blood sugar of 7.8 mmol/l and a 2-hr value of 11.1 mmol/l; serum thyroxine 108 mmol/l (normal 61–165 mmol/l) T_3 uptake 119; free thyroxine index 91 (normal 52–161). The urinary catecholamines were measured on 5 occasions; 24-hr excretion of free catecholamines varied from 100–810 μ g (normal less than 100 μ g) and the vanillyl mandelic acid excretion varied from 80–150 μ mol/24 hr (normal 10–35 μ mol/24 hr). Chest X-ray was normal. No bone change seen on X-ray of hands. The i.v. urogram showed normal pelvi-calyceal systems and normal kidney shadows. There was a round soft tissue shadow above the right kidney. Aortogram revealed a highly vascular tumour 7 cm in diameter above the right kidney.

A diagnosis of hypertension due to phaeochromocytoma was made and initially medical treatment with atenolol 100 mg daily and phenoxybenzamine 10 mg daily was commenced.

Four months after the patient was first seen the tumour was removed and postoperative recovery was uneventful. The blood pressure reading fell to normal levels without hypotensive therapy. The serum calcium returned to normal after surgery (Fig. 1).

The tumour tissue was assayed for PTH-like activity producing a figure of 2.8 μ g/g of dried tissue.