CLINICAL REPORTS

Pancreatic polypeptide and calcitonin secretion from a pancreatic tumour-clinical improvement after hepatic artery embolization

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Summary

We present a case in which plasma pancreatic polypeptide and calcitonin were found to be raised in association with an islet cell tumour of the pancreas and its hepatic metastases. In this patient, no specific endocrine syndrome was found. Therapeutic hepatic artery embolization improved the general health of the patient with no change in plasma pancreatic polypeptide, but a fall in calcitonin.

Key words: pancreatic polypeptide, calcitonin, hepatic artery embolization, islet cell tumour.

Introduction

Pancreatic polypeptide (PP), a 36 amino acid peptide, is found principally in small granules in the islets of Langerhans and, to a lesser extent, in the exocrine parenchyma. Its release by food is under both vagal and hormonal control and it is likely to play an important part in the physiological control of pancreatic and gallbladder function (Greenberg et al., 1978). Over 50% of pancreatic endocrine tumours secrete pancreatic polypeptide in addition to another hormone. In this situation, PP does not seem to contribute to the clinical features of these tumours (Polak et al., 1976).

Calcitonin, a 32 amino acid peptide, found mainly in the C cells of the thyroid gland, has no clearly defined physiological role in man. It is thought to be important in regulating bone resorption during times of physiological calcium stress such as growth, pregnancy and lactation (Stevenson, 1980). This action may explain its elevation in patients with a variety of tumours.

Case report

A 58-year-old man presented with a 6 week history of constant dull epigastric pain unrelated to meals. He was constipated, anorexic and had lost 14 kg in weight. Past history included mild Type II diabetes and angina for 7 years. On examination, he was cachectic and the liver enlarged 5cm below the right costal margin. Fibreoptic endoscopy revealed a normal oesophagus, stomach and pylorus. The duodenal cap was scarred, but no active ulcer was present. The duodenum was narrowed by an extrinsic mass, and ultrasound demonstrated a large solid lesion in the region of the head and body of the pancreas, with liver metastases. The histology of a liver biopsy taken at laparoscopy was compatible with a tumour of APUD-cell origin. Plasma pancreatic polypeptide was found to be elevated at 40000–60000 pmol/litre (normal, under 200), but there was no elevation of other pancreatic peptides. To characterise the molecular form of pancreatic polypeptide, gel chromatography was carried out using a Sephadex G50 superfine column. All the tumour-secreted PP coeluted in a single peak in the position of human PP (Kav 0·55). Plasma calcitonin was 0·74 μg/litre (normal <0·08). Serum calcium and phosphate were normal. Selective coeliac axis arteriography and indirect splenoportography confirmed the presence of extensive hepatic metastases,
Calcitonin release has been reported from a wide variety of tumours (Coombes et al., 1974) and the diarrhoea of medullary carcinoma of the thyroid has been attributed to it. Our patient, however, had constipation, despite a grossly elevated plasma calcitonin concentration. This may be explained by PP masking the effect of calcitonin on the gut, since PP has recently been shown to produce a net fluid absorption in the rat small intestine (Mitchener et al., 1981). The patient's other symptoms were those commonly associated with neoplasia, namely malaise, anorexia, and cachexia. These improved considerably after embolization, at a time when his plasma calcitonin had fallen to almost normal, perhaps suggesting an association between the two.

References


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