

Case report

Gastric carcinoid tumour and parathyroid adenoma

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SUMMARY A case involving a gastric carcinoid in association with parathyroid adenoma is reported and the question of a possible link between these two ailments is discussed.

Case report

A 33 year old woman with no previous history was admitted to hospital with a haematemesis. An emergency upper alimentary endoscopy showed an ulcerated submucosal tumour on the greater curve. The patient complained of flushing of the face during the previous months. 5 HIAA concentrations (5 hydroxy-indol-acetic-acid) were three times the normal average concentration, and the serotonin concentration in her urine was twice normal. Plasma gastrin and thyrocalcitonin were normal. A recurrence of haematemesis prompted an operation, during which a 4 cm protruding tumour was found on the greater curvature, which spurted blood from the pit in its centre. A subtotal gastrectomy was carried out. The liver was macroscopically normal, but a surgical biopsy revealed the presence of a carcinoid tumour. Endoscopic and ultrasound observations done after surgery as well as the 5 HIAA biological concentrations were normal. No serum calcium measurements were taken.

Four years later, the patient was again admitted to hospital suffering from right renal colic caused by a stone. The biological results showed hypercalcaemia (2.88 mmol/l=116 mg/l) and hypophosphataemia (0.69 mmol/l=21 mg/l). The pain persisted and at surgery a nut sized (6×10 mm) oxalic stone was removed. The phosphate calcium abnormalities remained. Radiographs of the skeleton showed demineralisation. Biopsy of the iliac crest and parathormone concentrations confirmed primary hyperparathyroidism (parathormone concentration being

twice the normal value). A cervicotomy involved removal of a 12×15 mm lower left adenoma. Histological examination confirmed the presence of a parathyroid adenoma and calcium concentration returned to normal.

Six years later the patient suffered from hot flushes, headache and diarrhoea. Examination showed several hepatic metastasis in the left lobe. The urinary 5 HIAA and serotonin concentrations were five times the normal. Endoscopy was normal, as were the serum calcium values. In order to decrease the secreting mass, a left hemihepatectomy was carried out. Histology confirmed the diagnosis of several hepatic metastasis (multinodular shape) of a carcinoid tumour.

Further endocrinopathy was sought without result. Four years later the patient complained of moderate flushing linked to the residual carcinoid syndrome. Biochemical tests (calcium, urine, samples, thyrocalcitonin, gastrin) remained normal.

Discussion and review of the literature

This patient suffered from a parathyroid adenoma and a gastric carcinoid tumour. Given the rare occurrence of these two ailments (gastric carcinoids represent 2/1000 of gastric cancers and hyperparathyroidism affects some 1% of the general public), it seems unlikely that they occur simultaneously by chance. Moreover, carcinoid tumours of the stomach account for less than 3% of carcinoid tumours of the digestive tract.¹

One could suggest a carcinoid triggered hyperparathyroidism. The gastric tumour, however, was secreting serotonin and it is difficult to see how this could trigger a compensatory hyperparathyroidism. More-

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over, after removal of the gastric tumour serotonin values in the urine became normal and hyperparathyroidism set in. Finally, after parathyroid adenectomy, calcium concentrations remained normal and there was no appearance of hyperparathyroidism, in spite of the development of several carcinoid hepatic metastasis. The action of serotonin secretion thus does not appear to trigger the development of hyperparathyroidism, and we shall have to place our observations along with type I (MEN I) multiple endocrine neoplasias.

A case identical to this one was not found on reviewing the literature. One report² lists three patients in which primary hyperparathyroidism is associated with a carcinoid tumour of the digestive tract. Unlike our case, however, the carcinoid was ileal in two instances and duodenal in the other. Moreover, the carcinoids secreted calcitonin and not serotonin. Metastasis were present in two patients. There was no family history. The authors concluded that this association fell under Type I MEN. But, contrary to serotonin secretion, could exaggerated calcitonin secretion not promote a hyperparathyroid reaction?³ In this case, when thyroid medullary cancers involve calcitonin secretion, hyperparathyroidism would tend more to be a compensating

mechanism⁴ than part of the Sipple syndrome (MEN 2).⁵ Another publication reports a case of gastric carcinoids associated with hyperparathyroidism.¹ Tumour secretion included gastrin. The authors concluded that this association came under type I MEN.

Hyperparathyroidism/carcinoid association should be seen as a rare form of MEN I and patients with digestive tract carcinoids should systematically be examined for hyperparathyroidism.

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