Loop diuretics even in a single dose are associated with increased renal excretion of calcium, potassium, and magnesium.[4] Hypokalaemia and its associated alkalosis lead to decreased plasma ionised calcium concentrations. Hypomagnesaemia, which is often associated with hypokalaemia, as in our patient, may impair both the secretion of parathyroid hormone and its peripheral action.[5] Our patient's symptoms were not controlled until magnesium was given parenterally with both calcium gluconate and a rapidly acting vitamin D preparation.

Loop diuretics can be hazardous in patients with asymptomatic hypoparathyroidism after thyroidectomy. Calcium and magnesium concentrations should be monitored closely during such treatment to avoid depletion of electrolytes and the associated morbidity and mortality.

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(Accepted 6 August 1987)

Department of Respiratory Medicine, City Hospital, Edinburgh EH10 5SB
ASAD BASHEY, BMSC, MRCP, senior house officer
WILLIAM MACNEE, MD, MRCP, senior lecturer

Correspondence to: Dr A Bashey, Department of Haematology, Hammersmith Hospital, London W12 0HS.

Effect of salmon calcitonin on symptoms and urinary excretion of 5 hydroxyindoleacetic acid in the carcinoid syndrome

Native somatostatin and its long acting analogue SMS 201-995 inhibit symptoms and urinary excretion of 5 hydroxyindoleacetic acid in patients with the carcinoid syndrome.[1] Calcitonin exerts many effects similar to those of somatostatin on the gastrointestinal tract and pancreas; we studied calcitonin's effect on symptoms and urinary excretion of 5 hydroxyindoleacetic acid in a patient with the carcinoid syndrome and compared its effect with that of somatostatin.

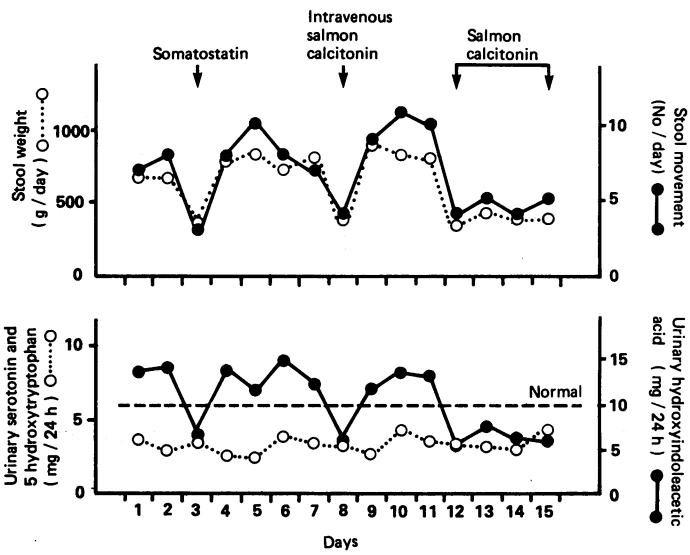
Case report

A 67 year old woman was admitted with an 18 month history of watery diarrhoea (7-10 bowel movements each day, 600-800 g stool weight), flushing (two or three times each day), and loss of weight. Examination showed hepatomegaly and a left epigastric mass. Her 24 hour urinary excretion of 5 hydroxyindoleacetic acid was raised (mean 14·5 mg/day, normal 2-10); urinary excretion of serotonin and 5 hydroxytryptophan was normal (figure). Echotomography and computed tomography showed a mass in the pancreatic tail infiltrating the surrounding structures. No hepatic metastases were detected. At operation the mass could not be removed; a biopsy showed "differentiated carcinoid tumour" (confirmed by the Grimelius technique).

For this study the patient was given a 6·3 MJ (1500 kcal) diet (20% protein, 40% fat, 40% carbohydrate), and drugs and food rich in serotonin were avoided. On day 3 she received an intravenous infusion of somatostatin 2·5 μg/minute for 24 hours. On day 8 she received an intravenous infusion of salmon calcitonin, 8 MRC units/hour for 24 hours. Salmon calcitonin was preferred to human calcitonin because of its greater potency. On days 12, 13, 14, and 15 salmon calcitonin was given subcutaneously, 100 MRC units every eight hours. Urinary excretion of 5 hydroxyindoleacetic acid and total urinary serotonin and 5 hydroxytryptophan were measured by a chromatographic colorimetric method. The patient gave written, informed consent to the protocol.

With somatostatin the patient's condition improved, with fewer bowel movements (three daily, weight 280 g/day), relief of cramping abdominal pain, and disappearance of flushes. Her urinary excretion of 5 hydroxyindoleacetic acid fell to 6·6 mg/day; her urinary excretion of serotonin and 5 hydroxytryptophan was unchanged. Mild fasting (7·9 mmol/l) and postprandial (10·5 mmol/l) hyperglycaemia was observed without glycosuria. Steatorrhoea developed (20 g total faecal fat, normal <10). When somatostatin was stopped her symptoms and raised urinary concentrations of 5 hydroxyindoleacetic acid recurred promptly. With infusion of salmon calcitonin the patient improved dramatically: bowel movements became less frequent (four daily) and stool weight was less (300 g/day), and her flushing disappeared. She experienced relief of her abdominal pain and a sense of wellbeing not experienced with somatostatin. Her urinary excretion of 5 hydroxyindoleacetic acid fell to 6·1 mg/day; urinary excretion of serotonin and 5 hydroxytryptophan was changed. Neither hyperglycaemia nor steatorrhoea developed. Serum calcium concentration was not affected. After the infusion there was a rebound in symptoms and increased excretion of 5 hydroxyindoleacetic acid. The effects of salmon calcitonin given subcutaneously were similar to those obtained with intravenous infusion.

The patient was still alive one year after the diagnosis and was doing well with treatment with calcitonin.



Effect of somatostatin and salmon calcitonin (continuous infusion and subcutaneous injection) on symptoms and biochemical abnormalities in patient with the carcinoid syndrome.

Comment

These data show that calcitonin mimics somatostatin in reducing clinical symptoms and urinary excretion of 5 hydroxyindoleacetic acid in the carcinoid syndrome. By inhibiting the release of serotonin from carcinoid cells calcitonin may account for some of the marked reductions in her symptoms. Calcitonin had similar effects on symptoms to somatostatin, but it also produced an independent analgesic effect, possibly due to a central neuromodulatory action.[2] Hyperglycaemia and transient steatorrhoea, commonly associated with somatostatin,[3] were not observed with calcitonin.

Our data suggest that calcitonin may be an alternative to long acting somatostatin analogues in the treatment of the carcinoid syndrome.

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(Accepted 30 July 1987)

Cattedra di Clinica Medica II, University of Pisa, 67-56 100 Pisa, Italy
ALESSANDRO ANTONELLI, MD, research fellow
PAOLO DEL GUERRA, MD, research fellow
ALESSANDRA FIERABRACCI, MD, research fellow
EUGENIO GORI, MD, research fellow
LIDIO BASCHIERI, MD, professor of clinical medicine

Correspondence to: Professor Baschieri.

Correction

Controlled trial of long term oral potassium supplements in patients with mild hypertension

An authors' error occurred in this paper by Alfonso Siani and others (6 June, p 1453). In table II the 95% confidence intervals, which read Supine: systolic −8·5 to −19·5, diastolic −5·9 to −14·1; Standing: systolic −4·9 to −17·1, diastolic −3·6 to −11·4, should have read Supine: systolic −6·2 to −21·8, diastolic −4·6 to −16·4; Standing: systolic −2·6 to −19·5, diastolic −2·7 to −12·1, respectively.