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Third degree heart block in acromegaly

Sir,

Intraventricular conduction defects and arrhythmias are common in acromegaly.¹ However, third degree heart block in acromegaly has not to our knowledge previously been reported.

A 57 year old male presented with an episode of syncope. The attack lasted a few seconds and was not associated with seizure, palpitation or chest pain. He had had hypertension for 10 years, impotence for 3 years and diabetes mellitus for a year. Physical examination revealed a large, coarse-featured man with obvious features of acromegaly. His pulse rate was 56/min, regular in rhythm, and did not change with posture. His blood pressure was 180/85 mmHg supine and 150/80 mmHg on standing. Examination of the jugular venous pulse revealed irregular 'cannon' a waves. The first heart sound was variable in intensity.

Electrocardiograph showed the presence of third degree heart block with wide QRS complexes and a ventricular rate of 56/min. Serum electrolytes, calcium and magnesium levels were normal. Chest X-ray showed cardiomegaly. The skull X-ray demonstrated a large pituitary fossa with a thin floor and computed tomographic (CT) scan of the brain revealed a pituitary tumour with suprasellar extension and erosion of the sphenoid sinus. Basal growth hormone level was elevated at 23.7 mIU/l (normal < 7 mIU/l). This failed to suppress during an oral glucose test using 75 g glucose. Temporary

pacing was instituted shortly following admission.

Further tests performed showed no significant heart rate response to exercise, intravenous atropine or isoprenaline infusion. An echo-Doppler examination of the heart showed minimal left ventricular hypertrophy, and normal mitral, aortic and tricuspid valves. There was no myocardial calcification or echocardiographic pattern suggestive of amyloidosis. Failure to respond to exercise, atropine and isoprenaline and the wide QRS of the escape pacemaker suggest the block is infraHisian. A permanent ventricular demand pacemaker (VVI) was implanted and he subsequently had a normal paced rhythm. He then underwent an uneventful transphenoidal hypophysectomy.

It is unlikely that the third degree heart block in this patient represents a problem unrelated to his acromegaly. There was no suggestion of digitalis intoxication, myocardial infarction, calcific aortic stenosis, cardiac amyloidosis or ventricular septal defect, all of which can cause third degree heart block. Idiopathic sclerosis of the conduction system and myocarditis, known causes of third degree heart block,² are more difficult to exclude. Myocardial biopsy was not performed. Diffuse interstitial myocardial fibrosis and myocarditis are common pathological findings in acromegalics with cardiac disease.^{3,4} In this patient the third degree heart block is considered secondary to diffuse interstitial fibrosis involving the conduction system.

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Nesidioblastosis in adults

Sir,

Nesidioblastosis is a rare disorder which is usually considered only as a cause of neonatal hypoglycaemia. It is characterized histologically by diffuse hyperplasia of pancreatic islets of Langerhans, with apparent budding of

new islets from the epithelium of exocrine ducts. The condition has been described in adults, in isolation and in association with other disorders: chronic pancreatitis, pancreatic duct obstruction, cystic fibrosis, gastrinoma, multiple endocrine neoplasia (MEN) and sulphonylurea therapy. The association of nesidioblastosis in an adult with an insulinoma has been reported on three occasions.¹⁻³ We describe here a fourth.

A 60 year old Caucasian male was admitted as an emergency with only a 5 day history of episodic drowsiness, slurred speech and confusion. Although he was obese, there was no history of recent weight gain. On examination he was drowsy, but rational and cooperative. History and examination were otherwise unremarkable and he was on no medication. Blood glucose was 1.7 mmol/l, with inappropriate elevation of serum insulin (80 mU/l) and C-peptide (2.7 mmol/l). Recurrent hypoglycaemia in ensuing days was only prevented by infusion of 4 litres of 10% dextrose each 24 hours, reducing to 1 litre of 10% dextrose in combination with oral diazoxide 250 mg twice daily. Abdominal computed tomography (CT) and coeliac axis angiography were unhelpful but a 1 cm tumour was found in the tail of the pancreas at exploratory laparotomy. 50% distal pancreatectomy and splenectomy was followed by an uncomplicated recovery. Apart from pneumococcal prophylaxis, he has received no further treatment and remains free from symptoms or other evidence of hypoglycaemia after two and a half years. Histological examination identified three discrete endocrine tumours, the largest 1 cm in diameter. Each showed a predominantly alveolar pattern, with copious amyloid. There was positive immunohistochemical staining for insulin and glucagon, but not for somatostatin. There was nesidioblastosis throughout the remaining pancreatic tissue and extending to the limit of the resection. The hyperplastic islets showed positive staining for the neuroendocrine marker PGP 9.5, as well as for insulin glucagon and somatostatin. The distribution of D-cells appeared normal.

This is the fourth reported case of nesidioblastosis occurring in association with an insulinoma. We believe the hypoglycaemia leading to presentation was the result of insulin secretion by the insulinomas, rather than by the surrounding tissue; nesidioblastosis must have been present throughout the unresected pancreas, and yet there has been no recurrence of hypoglycaemia. The pathogenesis of nesidioblastosis is unknown. It has been suggested that islet proliferation may result from reduced somatostatin secretion by pancreatic D-cells because somatostatin normally exerts a negative paracrine effect on islet cell function.⁴ Alternatively, it has been suggested that islet cell hyperplasia may occur as a result of islet stimulating immunoglobulins.⁵ In this case, the multiple insulinomas may have developed as a result of field change in pre-existing hyperplastic tissue, but it is equally likely that the insulinomas may have arisen *de novo*, and caused the nesidioblastosis by a paracrine effect. Such an effect could have been caused by insulin itself or, conceivably, by excessive secretion of amylin (IAPP) – as suggested by the presence of copious amyloid. If so, the absence of somatostatin in the insulinomas may have facilitated the development of nesidioblastosis by allowing proliferation of adjacent pancreatic tissue to take place unopposed.

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Masked faecal peritonitis in Cushing's syndrome

Sir,

We wish to report the case of a 71 year old woman who was admitted with a 3 week history of diarrhoea, anorexia, weight loss and abdominal pain. The past medical history revealed diverticular disease diagnosed 7 years previously. On examination, she was hirsute with generalized muscle weakness. She was in atrial fibrillation with mild left ventricular failure and ankle oedema. Investigations revealed a marked hypokalaemia (plasma potassium 1.2 mmol/l). Sigmoidoscopy was normal and a barium enema confirmed uncomplicated diverticular disease. The results of endocrinological investigations led to the presumptive diagnosis of ectopic Cushing's syndrome. A chest radiograph was normal. A limited abdominal computed tomographic (CT) scan showed bilateral adrenal hyperplasia. The patient was treated with metyrapone and over the subsequent 3 weeks plasma cortisol levels fell with an improvement in her clinical condition. A repeat abdominal CT scan was performed to locate a possible source of ectopic ACTH. Alarming extensive intra- and extra-peritoneal gas was demonstrated! The source of the ACTH was not.

At urgent laparotomy faecal peritonitis was found originating from a perforated diverticular abscess of the sigmoid colon. A sigmoid colectomy and ileostomy was performed. Post-operatively her condition deteriorated despite intense supportive therapy and she died of cardiorespiratory failure 6 days later. The autopsy showed that death had occurred as a direct result of perforated diverticular disease and peritonitis. Microscopic examination of the pancreas revealed a benign ACTH-secreting tumour which was not macroscopically evident.

The striking feature of this case was the lack of symptoms or signs of faecal peritonitis. In particular the patient was never pyrexial and the white cell count was