serum immunoglobulins and IgG led us to suspect the auto-immune nature of the problem.

The immune-mediated hearing loss can present as a solitary event or as a part of a more generalised auto-immune disorder. It is usually bilateral and symmetrical; vertigo and tinnitus may or may not be present. Since there is no characteristic presentation, such a diagnosis should be considered in a patient presenting with any combination of these symptoms. Certain clinical features should raise the alarm (box).⁷

Steroids and immunosuppressant therapy are the mainstay of treatment. We used azathioprine and obtained a good response. Fluctuations in hearing thresholds were observed during the initial adjustment of the steroid dose. Subsequently, the hearing remained improved to a useful level, despite gradual tapering of the steroids (figure 2). In conclusion, immune-mediated hearing loss is one of the few forms of sensorineural deafness that are treatable by medical means.⁹ The diagnosis can sometimes be difficult due to a wide variation

Features suggestive of immune-mediated hearing loss

- sensorineural hearing loss in early and middle aged adults] [for whom no other cause can be found
- slowly progressive sensorineural hearing loss (weeks or months rather than hours or days)
- sensorineural hearing loss in a person with an already existing auto-immune condition

in presentation and unavailability of a specific diagnostic test. However, a thorough clinical examination and non-specific laboratory tests are needed in all suspicious cases.

Final diagnosis

Immune-mediated sensorineural hearing loss.

Keywords: auto-immune disease; sensorineural hearing loss; ulcerative colitis

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Hypercalcaemia and abdominal pain

David Scott-Coombes, Andrew Williams

A previously fit 75-year-old woman presented to Accident and Emergency with a 12-hour history of constant upper abdominal pain. Clinical examination revealed pallor but no jaundice and guarding in the epigastrium. Her biochemical results were serum amylase 2878 IU/l (normal <90), bilirubin 33 µmol/l (3-20), alkaline phosphatase 42 IU/l (30-150), aspartate transaminase 354 IU/l (10-50), albumin 38 g/l (35-50), calcium 2.85 mmol/l (2.2-2.6).

Questions

- 1 What is your differential diagnosis for the abdominal pain?
- 2 Comment on the relationship between the serum calcium and amylase levels.
- 3 What further investigations would you request?
- 4 How would you further investigate and manage the hypercalcaemia if it were due to primary hyperparathyroidism?

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Answers

QUESTION 1

The serum amylase is markedly elevated, favouring a diagnosis of acute pancreatitis. Elevation of the bilirubin and alkaline phosphatase are suggestive of obstructive jaundice and should lead you to consider an obstruction of the common bile duct leading to pancreatitis. Whilst hypercalcaemia is a recognised cause of non-specific abdominal pain and even peptic ulceration (bones, stones, psychic moans and abdominal groans), the magnitude of the serum amylase indicates acute pancreatitis.

QUESTION 2

Hypercalcaemia has been proposed by many authors to be a cause of acute pancreatitis and this relationship is now quoted in many established textbooks. Historical literature reported a higher incidence of acute pancreatitis amongst patients diagnosed with primary hyperparathyroidism compared with a normal hospital population,¹ but more recent literature has not observed this increased incidence casting doubts on any association.² Acute pancreatitis is a recognised complication following coronary artery by-pass surgery and the administration of calcium chloride has been observed to be the single most reliable predictor for the development of pancreatitis in a dose-related manner.³ However, this may just be an epiphenomenon, perhaps related to hypotension-induced pancreatic injury. At present, the association between hyperparathyroidism and acute pancreatitis is unproved. Hypocalcaemia may also be associated with acute pancreatitis. Favoured mechanisms of true hypocalcaemia include calcium deposition in necrotic adipose tissue, cleavage of parathyroid hormone by pancreatic enzymes and a shift in extracellular and intracellular calcium concentration.⁴

QUESTION 3

Recommended further investigations are listed in box 1.

QUESTION 4

Adhere to the general principles of endocrine surgery (box 2).

A diagnosis of acute pancreatitis was confirmed by computed tomography scan of the abdomen. Abdominal ultrasound scan did not reveal gallstones, although the distal common bile duct was mildly dilated to 9.5 mm. The patient was administered analgesia and intravenous fluids and the pancreatitis resolved. A subsequent endoscopic retrograde cholangiopancreatography showed a normal papilla but cannulation of the duct failed. Primary hyperparathyroidism was confirmed by

Further investigations

- measurement of prognostic indicators (eg, Glasgow Score⁵)
on admission: white cell count $>18 \times 10^9/l$; blood glucose $>12 \text{ mmol/l}$, lactate dehydrogenase $>400 \text{ IU/l}$, aspartate transaminase $>240 \text{ IU/l}$
during first 48 h: PaO_2 8.0 kPa, albumin $<32 \text{ g/l}$, white cell count $>15 \times 10^9/l$, aspartate transaminase $>100 \text{ IU/l}$, lactate dehydrogenase $>600 \text{ IU/l}$, blood glucose $>10 \text{ mmol/l}$, plasma urea $>16 \text{ mmol/l}$
- chest X-ray, particularly if the arterial blood gas results are deranged
- abdominal ultrasound scan to look for gallbladder stones and choledocholithiasis, to examine the appearance of the pancreas and liver, and to measure the diameter of the extrahepatic bile ducts
- measurement of parathyroid hormone to determine the cause of hypercalcaemia

Box 1

Investigations and management

- confirm the diagnosis: simultaneous hypercalcaemia and inappropriate level of parathyroid hormone
- render the patient safe: intravenous fluids when calcium exceeds 3.0 mmol/l , and bisphosphonates when calcium exceeds 3.5 mmol/l
- consider localisation: not indicated when primary neck exploration performed by an experienced endocrine surgeon
- is surgery indicated? While there is debate as to whether peptic ulcer and acute pancreatitis are indications for parathyroidectomy, a calcium in excess of 2.95 mmol/l is an indication
- what operation? Excise adenomas and discuss with the patient the merits of total or subtotal parathyroidectomy in the presence of multiglandular disease (hyperplasia)

Box 2

simultaneous hypercalcaemia and an elevated parathyroid hormone. On neck exploration, a solitary 800-mg adenoma was excised from the right superior parathyroid gland. The postoperative calcium levels immediately returned to normal. Six months later the patient remains well with a normal serum calcium and no further attacks of acute pancreatitis.

Final diagnosis

Primary hyperparathyroidism, caused by a solitary adenoma, leading to pancreatitis.

Keywords: adenoma; pancreatitis; hypercalcaemia; hyperparathyroidism

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