Letters to the Editor

Massive gastric dilatation and acute pancreatitis – a case of the ‘Ramadan syndrome’?

Sir,

The association of acute pancreatitis and massive gastric dilatation has previously been reported in this journal1,2 and elsewhere.3 A further case is described here.

A 14 year old Muslim boy presented with a 24 hour history of epigastric pain, vomiting and progressive abdominal distension developing 5 hours after breaking the traditional Ramadan fast. On examination he was dehydrated and tachycardic (120 per minute) with marked abdominal distension but no signs of peritonism. A supine abdominal X-ray showed gross gastric dilatation. Routine blood tests revealed an amylase of 751 IU/l, an elevated urea 14.2 mmol/l and hypokalaemia (2.8 mmol/l). Despite initial management with nasogastric aspiration and intravenous fluids, the patient’s condition deteriorated with development of hypovolaemic shock. His abdomen became rigid with absent bowel sounds and a repeat film showed progression of gastric dilatation. Amylase had risen to 2,428 IU/l.

Following resuscitation, laparotomy was performed as a perforated viscus could not be excluded. An enormously dilated stomach was confirmed, but in addition oedema of the pancreas and retroperitoneal tissues was evident. A gastrostomy was fashioned. The patient required total parenteral nutrition postoperatively but otherwise made a complete and uneventful recovery.

Although the association of acute pancreatitis with massive gastric dilatation is well recognized, the aetiological mechanism remains obscure. The case reported here is similar to others1,2,3 and the primary diagnosis appears to be acute gastric dilatation with secondary pancreatitis. The former may mask diagnosis of the latter, which of itself requires vigorous early management to avoid complications. Fluid sequestration and reduction in plasma volume is common in acute pancreatitis and, when this condition co-exists with gastric dilatation, such potential hypovolaemia is exacerbated by fluid sequestration in the dilated stomach.

Whatever the precise physiological mechanism(s) involved, reflux of duodenal contents up the pancreatic duct is the most likely mechanism for initiation of pancreatitis secondary to gastric dilatation associated with a rise in intraduodenal pressure.4 Such a rise could result from gastric dilatation with duodenal ileus. Alternatively pancreatitis per se could result from direct stimulation of the pancreas by the presence of food in the stomach via release of cholecystokinin–pancreozymin (CCK–Pz).5

Two of these recorded cases have been described in patients with anorexia nervosa and refeeding appears to trigger the condition. A parallel may therefore be drawn between anorexia nervosa and the food restriction which is customary in Ramadan (variable period of food deprivation – from dawn to dusk over a month in a lunar calendar). Gastric dilatation may occur as a response of the stomach to food intake following a period of deprivation, perhaps mediated via a gastrointestinal hormone. This association is important clinically as fluid replace-ment and efficient nasogastric aspiration must be initiated early if laparotomy is to be avoided.

The simultaneous occurrence of acute pancreatitis and massive gastric dilatation under circumstances of food restriction followed by refeeding could perhaps appropriately be called ‘Ramadan syndrome’, a term familiar to clinicians in Muslim societies but scantily documented in the literature.

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References


Urinary bladder carcinoma initially manifested as brain metastases

Sir,

Angulo et al. have recently reported two cases of bladder carcinoma, the clinical presentation of which was due to brain metastases.1 We describe a further patient with transitional cell carcinoma of the bladder who presented with ophthalmoplegia and diabetes insipidus due to pituitary fossa involvement by secondary tumour. This brings to four the number of reported cases of primary bladder carcinoma presenting with neurological symptoms caused by brain metastases.1,2

A 45 year old woman presented with a 4 week history of headache and right-sided facial numbness and a 2 day history of polyuria, polydypsia and dysuria. Multiple right-sided cranial nerve palsies (III, V, VI, VII and XII) and a left pelvic mass were detected on clinical examination. Examination of the urine revealed red blood cells and a significant growth of Escherichia coli. Cranial computed tomography demonstrated a lesion in the sella turcica with erosion of the right sphenoid bone. Ultrasound of the pelvis indicated a mass in the region of the right ovary. The patient was deemed unfit for