Acute gastric dilatation – a delayed complication of percutaneous endoscopic gastrostomy

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Abstract

Acute gastric dilatation presenting 15 months after a percutaneous endoscopic gastrostomy is reported. The gastric dilatation was associated with local sepsis around the gastrostomy and resolved after removal of the gastrostomy tube.

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Although a number of minor and serious complications have been associated with percutaneous endoscopic gastrostomy,¹ the morbidity of the procedure is low² and percutaneous endoscopic gastrostomy feeding is widely used.³ With increasing usage new complications related to percutaneous endoscopic gastrostomy are likely to be recognised and we report, for the first time,

the development of acute gastric dilatation as a delayed complication of percutaneous endoscopic gastrostomy feeding.

Case report

An 83 year old woman presented with a two year history of intermittent but progressively worsening dysphagia and weight loss. A barium swallow showed a smooth gradual tapering of the distal oesophagus with considerable delay in emptying into the stomach and no gastro-oesophageal reflux. The proximal two thirds of the oesophagus was dilated with appreciable tertiary contractions. Endoscopy excluded a mechanical stricture but showed bizarre contractions and a dilated oesophagus. A diagnosis of achalasia was made although the patient refused manometric confirmation. A Rigidflex Microvasive balloon dilatation was complicated by an oesophageal tear with mediastinal and surgical emphysema despite a modest pressure of 5 psi being used for pneumatic dilatation. She responded to conservative treatment, intravenous fluids, antibiotics, and nil by mouth. There was a subjective improvement of swallowing when oral intake started again.

Within three months she became almost completely dysphagic; treatment with sublingual nifedipine and amitriptyline was ineffective. Further pneumatic dilatation was considered to be hazardous and the patient was reluctant to consider surgery. Percutaneous endoscopic gastrostomy was discussed with the patient and considered an acceptable option because of her continuing weight loss.

In January 1991 a Bower percutaneous endoscopic gastrostomy (E Merck, Four Marks, Alton, Hants) was inserted without immediate complications. She tolerated percutaneous endoscopic gastrostomy feeding very well and gained 5 kg in weight. She was on 1500 ml of Fresubin (Fresenius) per day provided by the dietitians and the percutaneous endoscopic gastrostomy site was regularly inspected by the District Nurse. In September 1991 she sustained a right hemiparesis from which she made a slow but adequate recovery. Her swallowing fluctuated from complete dysphagia to partial ability to swallow some solids. She was under regular follow up in the outpatient clinic every six weeks.

In March 1992 she presented urgently with a 24 hour history of abdominal distension, nausea, abdominal discomfort, and regurgitation of oral intake. The percutaneous endoscopic gastrostomy site was purulent with serosanguinous discharge and bacteriological culture grew *Staphylococcus aureus* and *Escherichia coli*. She was not pyrexial and blood cultures were negative. The most dramatic finding was a very

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Figure 1: X ray of abdomen showing massive gastric dilatation with percutaneous endoscopic gastrostomy in situ.

Figure 2: Gastrograffin meal showing the percutaneous endoscopic gastrostomy balloon (arrowed) within the stomach and contrast in the small intestine, excluding mechanical obstruction.

distended upper abdomen with succussion splash. An abdominal radiograph showed massive gastric dilatation (Fig 1).

Nasogastric suction yielded over 1500 ml of fluid for each of three consecutive days despite cessation of enteral feeding. She was treated with Co-amoxiclav 3 g/day to which both isolated organisms were sensitive. A gastrograffin contrast study did not show mechanical obstruction and the balloon of the percutaneous endoscopic gastrostomy tube could be seen in the stomach near the antrum and was not displaced (Fig 2). As the gastric dilatation was not appreciably improved after five days the percutaneous endoscopic gastrostomy tube was removed. Plasma urea and electrolytes, creatinine, calcium, blood glucose, and thyroid tests were normal. She was not anaemic and her white cell count was normal throughout her admission. After removal of the percutaneous endoscopic gastrostomy she improved rapidly and gastric distension disappeared on radiological examination within 48 hours. The gastrostomy site also healed within four days. She was discharged as she was managing adequate intake of solids and liquids slowly with a proposal to reintroduce percutaneous endoscopic gastrostomy feeding when her swallowing became worse.

Discussion

Wound infection is a frequent complication of percutaneous endoscopic gastrostomy. Although usually easily treatable, it may lead to serious complications such as peritonitis¹ and necrotising fasciitis.^{4,5} Over an 18 month period we have encountered four gastrostomy site infections in 24 procedures. Apart from the reported case, the other three infective episodes occurred within a week of the procedure and resolved with local or systemic antibiotics, or both. Diabetes mellitus, steroid treatment, malnutrition, and H₂ receptor antagonists have been cited as risk factors for infective complications^{6,7} but the reported case had none of these risk factors.

The late development of gastric dilatation in the patient makes it unlikely that the percutaneous endoscopic gastrostomy was directly interfering with gastric motility. Although formal gastric emptying studies were not done, contrast studies before insertion of the percutaneous endoscopic gastrostomy tube failed to show delayed gastric emptying, suggesting that a generalised motility disorder of the upper gastrointestinal tract was unlikely. Rapid improvement in gastric dilatation after removal of the percutaneous endoscopic gastrostomy tube suggests that the gastrostomy itself was implicated in the process. This was the first episode of infection at the gastrostomy site in this patient and the temporal sequence of events suggests that infection might have been an important predisposing factor in precipitating acute gastric dilatation.

No metabolic abnormalities were present in this patient and her only medication was isosorbide mononitrate and frusemide at the time of the complication. Ileus has been reported as a complication during the postprocedure period¹ but not as a delayed complication. The complication was not associated with any change in the rate of feeding through the gastrostomy tube (100 ml/hour).

This complication highlights the continuing need for follow up of longterm percutaneous endoscopic gastrostomy patients in a specialised centre.

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