ACHALASIA CARDIA AND GASTRIC OUTLET STENOSIS IN A POSTMENOPAUSAL WOMAN: CASE REPORT

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SUMMARY

The orderly contractility of the oesophagus and the regulated ability of the pyloric sphincter allow the influx and efflux of gastric contents. When these physiological processes are impaired, gastric luminal transit is altered as expected in achalasia cardia and gastric outlet obstruction. Movement across the inlet and outlet of the stomach is therefore altered. A case of a 58-year-old woman diagnosed with simultaneous occurrence of achalasia cardia and gastric outlet stenosis resulting from chronic duodenal ulcer is presented. The diagnosis was based on clinical, radiological and intraoperative findings. This patient has remained well after a simultaneous anterior cardiomyotomy and H-M pyloroplasty. To my knowledge this is the first time that such an association causing gastric “inlet” and “outlet” obstruction has been reported.

INTRODUCTION

The aetiology of achalasia is unknown, but it has been suggested that it is congenital in origin and associated with such disorders as infantile pyloric stenosis or Hirschprung’s disease. The primary problem is neuronal in origin. Bolus transfer into the oesophagus is normal but the oesophageal musculature lacks the ability to propel its content down the oesophagus and into the stomach. Outlet obstruction secondary to chronic duodenal ulceration is usually due to a combination of fibrosis from repeated cycles of ulceration and healing, oedema, inflammation and pylorospasm associated with current activity of the ulcer.

A 58-year-old woman with gastric inlet and outlet stenosis due to achalasia cardia and chronic duodenal ulcer with fibrosis is presented. A combined restorative surgery will adequately ease these problems if done correctly.

CASE REPORT

H.E was a 58-year-old postmenopausal housewife with five years history of episodic pain and twenty six months history of difficulty in swallowing. The epigastric pain was dull, radiating to the back occasionally. It was aggravated by hunger and relieved by food ingestion and antacids. The patient had no chest pain but had associated cough for six months before presentation to the hospital. The cough was productive of whitish sputum and she choked occasionally at night. No history of haematemesis and no melena stool. The difficulty in swallowing was for both liquids and solids but more severe with liquid diets. The dysphagia was intermittent with regurgitation and chest pain. There was no associated painful swallowing. She vomited on average of 4-6 times per week, which contained previously eaten food that was foul smelling. She gave history of significant weight loss at presentation. No history of similar problems in the family was reported. She was Para 6+0 all alive. Menarche was attained at the age of fourteen and menopause at 45 years of age. She had no formal hospital based treatment before presenting to our hospital. No history of diabetes and hypertension was reported.

Examination revealed a chronically ill patient who was pale, wasted and had a body temperature of 37.8°C. She was dehydrated. The respiratory rate was 24 cycles per minute. The chest was clinically clear save for the poor air entry and fine crepitations at both lung bases. Pulse rate was 98 beats per minute, full volume and regular. The blood pressure was 120/75 mmHg. Abdomen was full, no visible peristalsis noted, no area of tenderness and no organ palpable. Succession splash was positive ten hours after the last ingested feed. A diagnosis of achalasia cardia was entertained and she had the following investigations.

Chest X-ray showed convex shadow to the right, with mega oesophagus, no clear air fluid level but with evidence of pneumonitis (Figure 1). Barium swallow showed oesophageal body dilatation, tapering of the oesophagogastric junction. Duodenal cap was deformed and very little flow of contrast medium into the small bowel (Figure 2). Sputum culture examination grew no organism. AFB stain was negative. She was non reactive to HIV II and I. Haemogram showed haemoglobin of 7g/dl with neutrophilia. The urea and electrolyte were essentially normal. The total serum protein level was 55g/dl with albumin 35g/dl. The patient was admitted and had antibiotic and two units of blood transfused. She had adequate rehydration and permunineR (a protein based infusion) infused. She had exploratory laparotomy after resuscitation and the following findings were noted. Indurated duodenum with dilated stomach. The oesophagus was dilated with constriction at oesophagogastric junction. Other abdominal organs were normal. The patient had combined Heller’s operation with H-M pyloromyotomy. The postoperative period was uneventful. The patient was able to swallow without difficulty and had remained well two years after discharge.
DISCUSSION

The synchronized ability of sphincters to relax allows sufficient control of digestive transit. Relaxation of lower oesophageal sphincter is essential for oesophageal emptying and, similarly, pyloric relaxation permits gastric emptying. When the relaxation response of these sphincters is impaired, luminal transit is altered, as is the case of achalasia and chronic duodenal ulceration with healing by fibrosis. In achalasia, there is a disturbance of oesophageal motility. There is absence of peristalsis in the body of the oesophagus and failure of the inferior oesophageal sphincter to relax in response to swallowing. The problem is easily diagnosed with typical complaints and radiological signs. There is difficulty in swallowing especially of liquid and cold food with regurgitations of food at a stage. The radiological features include a dilated oesophagus with a peak-like lower end. There is little or no barium in the stomach. The oesophagus may even reach a great proportion that it can even be seen on plain chest radiograph. Oesophagoscopy is invaluable in the diagnosis. Heller’s operation with the different modifications is usually adequate for its treatment. The outcome is good for early cases of achalasia.

Pyloric obstruction occurs after a long history of duodenal ulcer distress almost always resulting from severe cicatrical duodenal deformity and there is little expectation from conservative management. The cicatrisation results in the impairment of the propulsive motility of the antrum and the active relaxation of the pyloric sphincter. Such patients present with features of gastric outlet obstructions consequence upon failure of relaxation response of the pyloric sphincter. Barium meal examination is helpful in the diagnosis. Treatment should relieve the obstruction and control the ulcer diathesis.

We present a case of a 58-year-old woman diagnosed with achalasia and duodenal obstruction due to chronic fibrosis of duodenal ulceration. Extremely few cases of simultaneous occurrence of gastric inlet and gastric outlet obstruction have been reported in the literature. The reported ones have been between achalasia and hypertrophic pyloric stenosis; achalasia and mucosal diaphragm(1-3). In most of the reported cases achalasia was always present as the cause of inlet obstruction and with other outlet problems(4) The diagnosis was based on the clinical features and typical radiological features as presented above (Figures 1 and 2). Barium swallow confirmed the clinical suspicion. Manometric tests and oesophagoscopy could not be done due to some logistic problems.

These two problems caused distress symptoms, which affected the nutritional state and social life of the patient. Each of these problems is usually associated with significant morbidity from complications. The concomitant corrections of these problems were successful. This rare case is presented to alert surgeons of possible coexistence of these problems and that these problems can be treated simultaneously in well selected patients. To our knowledge, this is the first time such an association has been reported. The patient has remained well for two years since the conditions were diagnosed and treated simultaneously.

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REFERENCES