being evaluated for another current illness. This is most likely to occur in patients from areas where *B burgdorferi* is endemic. Misdiagnosis is by far the most common reason for an apparent lack of response to antibiotic treatment.²⁴ The American College of Rheumatologists and the Council of the Infectious Diseases Society of America have recommended that patients with non-specific symptoms such as chronic fatigue or myalgia without a history of classic features of Lyme disease but with positive serology do not receive parenteral antibiotic treatment: cost effectiveness analysis has shown that treating such patients results in many more cases of antibiotic toxicity and other complications than cures of atypically symptomatic Lyme disease.²⁹

I thank Dr E C Guy and Professor P J Watt for their constructive criticism and help in preparing this manuscript.

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A Difficult Case

Severe gastroparesis diabeticorum in a young patient with insulin dependent diabetes

"Gastroparesis diabeticorum is more often overlooked than diagnosed." C J Dowling and colleagues present a difficult case of a young woman with this condition who had failed to respond to treatment. Four experts not concerned with the case give their views on how she might be treated.

Case report

Clare J Dowling, Sudhesh Kumar,
Andrew J M Boulton

Abnormalities in autonomic function affecting the gastrointestinal tract can often be shown by quantitative testing but only a few diabetic patients experience symptomatic autonomic neuropathy of the gut. Occasionally patients have severe disabling symptoms that may be difficult to manage. We describe a 28 year old woman found to have insulin dependent diabetes mellitus in 1979 and who has currently been in hospital for almost a year with severe and resistant gastroparesis. In the years before her admission to this hospital she had been investigated elsewhere for nausea and vomiting and had been labelled as suffering from anorexia nervosa. Subclinical nausea had been present for several years, and the vomitus she produced was seen to contain food that she had consumed a few days earlier. The vomiting and nausea made it difficult to control her diabetes, and she was thought to have "brittle" diabetes because of the wild swings in her glycaemic control. She was diagnosed as having the

nephrotic syndrome in the year before her arrival here, and renal biopsy confirmed that this was due to diabetic nephropathy. At that time her serum creatinine concentration was 137 $\mu\text{mol/l}$. She was first admitted for her current symptoms almost a year ago with a three day history of a severe exacerbation of her nausea and retching, although milder symptoms had been present long before.

There was some deterioration in her renal function with a serum creatinine concentration of 204 $\mu\text{mol/l}$ and a 24 hour urinary protein concentration of 4.52 g/l. The possibility of gastroparesis diabeticorum was considered, and gastric emptying studies were performed with colloid porridge labelled with technetium-99m. This showed poor gastric contractions particularly at the gastric antrum, and transit of the radio-labelled meal into the pylorus was slowed. The half time of gastric emptying was over two hours, consistent with gastroparesis. A later repeated study of gastric emptying showed some improvement of peristalsis in the antral area but confirmed delayed gastric emptying consistent with gastroparesis. Oesophageal manometry studies showed normal motility over the distal section of the oesophagus with a reduction of contractions at the most proximal section, but this was not affecting propagation of contractions distally.

**Department of Medicine,
Manchester Royal
Infirmary, Manchester
M13 9WL**

Clare J Dowling, *senior house
officer*

Sudhesh Kumar, *clinical*

lecturer in medicine

Andrew J M Boulton, *reader
in medicine*

**Diabetic Department,
Kings College Hospital,
London SE5 9RS**

Michael Edmonds,
consultant physician

**Department of Surgery,
Genesee Hospital,
Rochester, NY 14607-1004,
USA**

Peter Fielding, *chief*

**Department of Nutritional
Sciences, Faculty of
Medicine, University of
Toronto and Clinical
Nutrition and Risk Factor
Modification Center, St
Michael's Hospital,
Toronto, Ontario, Canada
M5S 1A8**

David J A Jenkins, *professor*

**Department of
Neurosciences, Royal Free
Hospital School of
Medicine, London
NW3 2PF**

Gareth Llewelyn, *consultant
neurologist*

Gastroscopy showed grade 2 oesophagitis with a few petechiae around the cardia presumed to be caused by her vomiting and a large amount of residue in the stomach. Biopsies of the gastric antrum showed normal gastric mucosa with no evidence of metaplasia, *Helicobacter pylori*, dysplasia, or neoplasia.

The symptoms of nausea and vomiting continued unremittingly. Every recommended drug regimen was tried² (metoclopramide, erythromycin, domperidone, and cisapride), all without any notable benefit. Even some rather unusual combinations of drugs were tried, including a concoction anecdotally said to be effective, consisting of tincture of belladonna, co-magaldrox, and diphenhydramine. The latter suffered the fate of all previous attempts at medical control of the nausea with no real change from the state of continued nausea and vomiting. Her symptoms were noted to be worse during intercurrent illnesses such as infections and when she was hyperglycaemic. She also seemed to suffer a worsening of her symptoms when premenstrual.

In addition to gastroparesis diabeticorum, which she undoubtedly has, psychological factors may have contributed to the problem. The patient was seen on numerous occasions to be putting her fingers down her throat to assist retching. She claimed vomiting lessened her symptoms of nausea and abdominal bloating. After detailed psychiatric review, however, it was concluded that she did not suffer from a psychiatric eating disorder in addition to her gastroparesis.

Those for surgery . . .

Those in favour of surgery pointed out that this young patient has been in hospital for almost a year with no real change in her symptoms, and medical treatment seemed to have failed. They claimed that with her inability to eat any solid food and the persistent vomiting she has a poor prognosis. As radiological studies have shown normal function of the small bowel gastric emptying procedure may give her considerable relief of her symptoms and result in a reasonable quality of life.

Those against surgery . . .

Those opposing surgery pointed out that gastric emptying procedures may be associated with other problems, and the patient's overall quality of life may not be improved. They argued that there is no real evidence to indicate that bypass surgery would have much symptomatic benefit. The patient has now had this problem for at least a year and probably longer. Although current thinking is that most cases remit spontaneously in less time than this, she could still improve given more time.

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Establish enteral feeding

Mike Edmonds

Diabetic gastroparesis refractory to medical treatment is a rare but major management problem. Its previously poor prognosis, however, has been improved recently by the use of percutaneous endoscopic gastrojejunostomy with enteral feeding.¹

In patients presenting with intractable vomiting it is important in the planning of management that the

cause of vomiting is clearly established. Features in this case which point to a diagnosis of gastroparesis are the history of vomiting food that had been consumed a few days earlier, the large amount of residue in the stomach on gastroscopy in the absence of mechanical obstruction or mucosal abnormalities, delayed gastric emptying on scintigraphic studies, difficulties in diabetic control resulting from irregular eating and delayed gastric emptying, and, finally, long term diabetes with advanced complications including nephropathy and autonomic neuropathy. Autonomic neuropathy can usually be confirmed by grossly abnormal results on cardiovascular autonomic function tests.

The treatment plan should be percutaneous endoscopic gastrojejunostomy with enteral nutrition, optimal diabetic control, avoidance of drugs that slow down gastric emptying, and management of factors which exacerbate gastroparesis such as uraemia. Gastroparesis can remit but it may take several months and then the feeding tube can be removed.

Percutaneous endoscopic gastrojejunostomy is preferable to more invasive surgical intervention. It can be carried out under local anaesthesia, with insertion of a feeding tube into the jejunum through a gastrostomy.² It is wise to avoid periodic bolus feeding and to administer the feeds via a constant infusion pump in the early evening and overnight. The total energy in a balanced nutrient solution can be administered over 12-16 hours, leaving the daytime free from feeds.¹ Another percutaneous approach is the insertion, under fluoroscopic guidance, of two gastrostomy catheters, one to the duodenal-jejunal flexure for feeding and the second placed in the gastric fundus for decompression.³ Surgery should be tried as a last resort. Several operations have been carried out, including pyloroplasty, gastric resection, and gastric jejunostomy, but with limited success.⁴

With the establishment of enteral feeding, a return to reasonably stable diabetic control may be possible. It is important to avoid excessive hyperglycaemia, which is known to slow gastric emptying,⁵ and improvement in gastric motor function may accompany tighter blood glucose control.⁶ If the feed lasts 16 hours, between 6 pm and 10 am, then subcutaneous insulin may be given as a mixture of short acting soluble insulin and intermediate acting isophane insulin 30 minutes before the feed and as soluble insulin early the next morning.

Hypothyroidism should be excluded, and drugs known to exacerbate gastroparesis—such as tricyclic antidepressants, opiates, phenothiazines, and anticholinergic agents such as diphenoxylate hydrochloride with atropine sulphate—should be avoided.

This patient almost certainly has diabetic nephropathy and her serum creatinine concentration has risen from 137 $\mu\text{mol/l}$ to 204 $\mu\text{mol/l}$ over about one year. Gastric autonomic dysfunction is exaggerated by chronic uraemia, and patients become more symptomatic with increasing renal impairment. Thus, renal function should be monitored by serial measurements of creatinine clearance and glomerular infiltration rate with early treatment of hypertension, fluid overload, and electrolyte abnormalities, particularly abnormal potassium concentration. There may be improvement in the serum creatinine concentration if the vomiting ceases. Alternatively, there may well be a relentless deterioration in renal function, eventually needing kidney transplant, which is often associated with a substantial improvement in gastric emptying.⁷

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