Successful medical treatment of peptic pyloric stenosis: Dr Sippy revisited

ABSTRACT - Background: Surgery and balloon dilatation are perceived by many as the principal treatments for peptic pyloric stenosis. We questioned whether, with the availability of modern acid suppressant treatment, this was still appropriate or whether patients could be managed with medical treatment alone.

Methods: Seventeen consecutive patients with peptic pyloric stenosis were treated with endoscopic gastric drainage, followed by oral omeprazole in 15 or cimetidine in two. Gastric emptying half times for solids and liquids were assessed in 11 of the 17 patients when they had become asymptomatic.

Results: Endoscopic drainage and medical treatment successfully relieved symptoms in all 17 patients, although the gastric emptying studies in 11 patients still showed prolongation in eight. Symptoms resolved completely after a mean of 28 days. Five patients relapsed when changed from omeprazole to cimetidine treatment, but all responded to re-starting omeprazole. Four patients remain well on cimetidine alone.

Conclusions: Medical treatment preceded by endoscopic gastric drainage was effective in all patients in this series and may be the preferred choice of treatment in patients with pyloric stenosis.

In 1915, Sippy reported that peptic pyloric stenosis would respond to intensive acid suppressant medical treatment. However, in recent years, surgical treatment and balloon dilatation have been accepted by many as the principal initial treatments for pyloric stenosis caused by benign peptic disease. The availability of effective acid suppressant therapy in the form of H₂ receptor antagonists and proton pump inhibitors led us to question this and to re-investigate the natural history of pyloric stenosis in patients receiving these drugs.

Patients and methods

We defined peptic pyloric stenosis as the finding at endoscopy of a residual gastric volume of >500ml after overnight fast, a narrowed pyloric canal that would not admit a standard 9.2mm or 10.5mm endoscope and no endoscopic or biopsy evidence of malignancy. Seventeen consecutive patients presenting to the department of gastroenterology at this hospital between 1983 and 1996 satisfied these criteria and constitute our study group.

Patients presented with symptoms of vomiting (14 patients), dyspepsia or abdominal pain (9 patients), weight loss (6 patients) and haematemesis or melaena from associated oesophagitis (3 patients) (see Table 1). The duration of symptoms ranged from 1–120 months (mean 15 months). In 10 patients, pyloric stenosis was suspected before endoscopy on the basis of symptoms and the clinical findings of a succussion splash. The mean age at presentation was 65.6 years (range 30–81 years). Two patients were already receiving an H₂ receptor antagonist at presentation (one patient for eight years, one for 21 months). Two patients were taking aspirin and one a non-steroidal anti-inflammatory drug.

Treatment

All patients were treated with gastric aspiration at the time of endoscopy. This aimed to achieve dryness, as near as possible. Fifteen patients were started on oral treatment with a proton pump inhibitor (omeprazole, 20mg daily) on the day of endoscopy and two on an H₂ receptor antagonist (cimetidine, 800mg twice daily). For the first 24 hours after gastric aspiration, patients were advised to take fluids only. Solid food was then introduced gradually if the patient’s symptoms improved.

Follow up

Patients were assessed every two weeks until their symptoms resolved, and were followed up for a median of 42 months (range 3–159 months). In 11 patients, the gastric emptying half-time (1/e) for solids and liquids was assessed separately using radioisotope methods previously described. Assessment took place between 1 and 58 months after starting treatment, at a time when the patient was asymptomatic. Results of rapid urease tests for Helicobacter pylori were available for 10 patients at presentation.

Results

Symptoms started to improve in all patients within 24 hours after endoscopic gastric drainage. The median time to complete resolution of symptoms was 21 days (range 5–90 days). Nine patients were managed as day cases, but the other eight spent between 5 and 13 days (mean 9 days) in hospital.

Gastric emptying studies, carried out in 11 patients a mean of 17 months after diagnosis, showed normal emptying in three, and prolonged emptying in the remainder (see
Table 1. Clinical details of patient and gastric emptying half times (t/2) for solids and liquids∗.

| Patient no. | Age at presentation (y) | Symptoms† | Symptom duration (mth) | Residual volume on endoscopy (ml) | Initial treatment | Time from diagnosis to gastric emptying study (mth) | Liquid emptying (min) | Solid emptying (min) | H. pylori status | Time to complete symptom resolution (mth) | Follow up (mth) |
|-------------|--------------------------|-----------|------------------------|---------------------------------|------------------|---------------------------------------------|---------------------|-------------------|----------------|----------------------------------------|----------------|
| 1           | 49.6                     | Vo, Wt    | 12                     | 1,250                           | Omeprazole       | 15.4                                        | 312                 | 207               | –              | –                                      | 11             |
| 2           | 71.4                     | Vo, Hm    | 2                      | 550                             | Omeprazole       | 31.8                                        | 41                  | 42                | +ve            | 90                                     | 63             |
| 3           | 30.0                     | Vo, P     | 120                    | 750                             | Omeprazole       | 57.8                                        | 44                  | 108               | –              | –                                      | 21             |
| 4           | 62.7                     | Vo, P     | 1                      | 1,000                           | Omeprazole       | –                                            | –                   | –                 | –              | 14                                     | 65             |
| 5           | 85.9                     | Vo, Wt, P | 4                      | 600                             | Omeprazole       | 8.6                                          | 112                 | 171               | +ve            | 14                                     | 42             |
| 6           | 77.6                     | Vo, P     | 1                      | 350                             | Omeprazole       | 5.6                                          | 30                  | 22                | –              | –                                      | 5              |
| 7           | 45.1                     | Vo, P     | 2                      | 500                             | Cimetidine        | –                                            | –                   | –                 | –              | 30                                     | 69             |
| 8           | 57.4                     | Vo, P     | 350                    | Omeprazole                      | 13.7             | 25                                           | 54                  | +ve               | 21             | 45                                     |
| 9           | 58.5                     | Vo, Wt    | 3                      | 1,000                           | Omeprazole       | 38.7                                        | 286                 | 182               | +ve            | 10                                     | 69             |
| 10          | 81.0                     | Vo, P     | 4                      | 500                             | Omeprazole       | –                                            | –                   | –                 | 90             | 67                                     |
| 11          | 71.9                     | Vo        | 24                     | 600                             | Omeprazole       | 2.2                                          | 1290                | 183               | –              | 60                                     | 3              |
| 12          | 79.2                     | Vo, P     | 12                     | 2,000                           | Omeprazole       | 2.6                                          | 29                  | 77                | –              | –                                      | 28             |
| 13          | 71.6                     | Hm        | 2                      | 600                             | Cimetidine        | –                                            | –                   | –                 | –              | 7                                      | 159            |
| 14          | 73.5                     | Vo, Wt    | 1                      | 700                             | Omeprazole       | 4.2                                          | 84                  | –                 | –              | 28                                     | 29             |
| 15          | 48.4                     | Vo, Hm    | 500                    | Omeprazole                      | 0.8              | 73                                           | 83                  | +ve               | 7              | 22                                     |
| 16          | 78.9                     | Pt, P     | 2                      | 800                             | Omeprazole       | –                                            | –                   | –                 | +ve            | –                                      | 5              |
| 17          | 71.4                     | P         | 3                      | 650                             | Omeprazole       | –                                            | –                   | –                 | +ve            | 40                                     | 5              |

∗ Normal range liquid 42–55 minutes, solid 44–64 minutes.
†Vo = vomiting, Wt = weight loss, P = pain, Hm = haematemesis or melaena.

There was no relation between the duration of treatment and the gastric emptying half-time (Table 1). In one patient, liquid emptying was very prolonged (t/2 = 1,290 minutes), but emptying was much shorter for solids (t/2 = 183 minutes). Despite this prolonged emptying time, the patient was asymptomatic and ate normally.

Repeat endoscopy was carried out in seven patients between two months and five years after presentation. After an overnight fast, the maximum gastric residue was 100ml, and in all but one patient the endoscope could be passed into the duodenum. This patient had a prolonged emptying time for solids (t/2 = 108 minutes) but normal emptying for liquids (t/2 = 44 minutes). Paired barium studies were available in a further three patients. The first study had been done within a week of presentation and the second when the patient was asymptomatic, between two and four months after starting treatment. All three showed improvement in gastric emptying and a reduction in the gastric size at rest (see Fig 1).

In seven of the 15 patients given omeprazole, an H₂ receptor antagonist was substituted 3–6 months after starting treatment. Five of these patients had a return of their initial symptom of vomiting between 3 and 24 months (mean 16.2 months) after switching from omeprazole to cimetidine. All responded to re-starting omeprazole. One patient, who was negative for H. pylori, was changed from omeprazole to cimetidine on two occasions. He relapsed both times, but responded to re-starting omeprazole. Four of the 17 patients (including the two initially started on cimetidine) have remained well on cimetidine alone. Seven of the 11 patients tested for H. pylori were positive.

Sixteen of the 17 patients remain asymptomatic, a mean of 54 months after diagnosis (range 5–159 months). One patient died from an unrelated intracerebral haemorrhage three months after diagnosis.

Discussion

The incidence of pyloric stenosis, which has been reported to complicate duodenal ulcer disease in 7–15% of patients, is declining in western countries. It took us more than 10 years to gather our group of 17 cases, and they represent less than 1% of new patients with duodenal ulcer seen during this time. In contrast, Mabogunje and Lawrie in northern Nigeria saw 106 patients requiring surgery for benign duodenal obstruction over a 12 year period. Although our group is small by these standards, it constitutes the largest consecutive series of patients with endoscopically diagnosed pyloric stenosis managed with modern acid suppressant treatment.

One of the criteria for surgery used to be failure to respond to medical treatment within five days. All our patients improved sufficiently to eat a normal diet within a week of starting treatment, although 13 weeks elapsed before one patient had complete remission of symptoms. Other workers have also noted that a recovery period greater than five days is needed.

The relapse of five patients when switched from omeprazole to an H₂ receptor antagonist (one on two occasions) points to acid suppression as the prime factor in reducing oedema and stenosis. Intravenous omeprazole has been advocated for relief of gastric outflow obstruction.
ever, our study suggests that after gastric aspiration, gastric emptying is sufficient to allow orally administered drugs to be effective.

Elimination of *H. pylori* is important in the long term management of patients with duodenal ulcer, and might have allowed the withdrawal of acid suppressing drugs in some of our patients. This is of particular importance in developing countries where long term acid suppression treatment might not be affordable. Of the 10 patients in our series for whom data were available, six were positive and four negative for the organism. However, this may be an underestimate of the prevalence because patients were tested at presentation, when the large gastric residual volume might have inhibited detection of the infecting organism.

The rate of gastric emptying, using a single measure of $t_{1/2}$, remained prolonged in 8 out of 11 patients, despite complete absence of symptoms. However, surgical drainage for peptic pyloric stenosis may also result in delayed gastric emptying, especially if truncal vagotomy is also performed, and prolonged gastric emptying *per se* would not be an argument for surgical drainage.

Even though there was no appreciable residual gastric volume in any of our patients who underwent follow up endoscopy, this possibility after overnight fasting, even in the absence of symptoms, is an important consideration for the anaesthetist who has to manage these patients pre-operatively.

Our study suggests that pyloric stenosis caused by peptic ulcer disease is more likely to be a result of reversible inflammatory oedema than fixed cicatrization of the pyloric canal. Patients should be treated medically with gastric aspiration and a proton pump inhibitor. Complete resolution of symptoms may take up to three months and gastric emptying may still be prolonged, but patients – many of whom are elderly – will be free of symptoms without the need for surgery. We concur with Dr. Sippy, who commented in 1915 that: 'unbelievable as it may seem, cases of duodenal ulcer recurrent for years, that have finally developed a high grade pyloric obstruction due to actual anatomic narrowing from indurated, infiltrated and edematous tissue have yielded completely to the [medical] management'.

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