Medicine in the Elderly

The successful medical management of gastric outflow obstruction associated with the use of non-steroidal anti-inflammatory drugs in the elderly

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Summary: One hundred and nineteen consecutive elderly patients with endoscopically diagnosed peptic ulceration were reviewed. Associated gastric outflow obstruction was present in 10.1%. The presenting clinical features differed significantly from typical younger patients and most (11/12) were taking non-steroidal anti-inflammatory drugs, suggesting a possible role for these agents in the pathogenesis of gastric outflow obstruction. These elderly patients have been successfully managed by medical therapy alone.

Introduction

Patients with benign gastric outflow obstruction complicating peptic ulcer disease are usually male, have a long ulcer history and require surgery even after initial successful medical treatment.^{1,2} More recently endoscopic balloon dilatation has been suggested as an alternative therapeutic approach.³ The clinical features and management of gastric outflow obstruction in elderly subjects have not been previously studied in detail. Our report also includes details of the successful medical treatment of 12 elderly patients.

Patients, methods and results

All upper gastrointestinal endoscopies performed in two medical units for the elderly between October 1987 and December 1989 were reviewed. Of 119 patients (73 females) aged over 75 years with peptic ulceration there were 12 cases (10.1%) of gastric outflow obstruction. The mean age of these patients was 82 years (range 77–89 years), 10 were female.

The following endoscopic criteria were required to make the diagnosis: (1) an overnight fasting gastric residue greater than 200 ml; (2) a narrowed pylorus or first part of the duodenum impassable

by a standard 11 mm endoscope; (3) hyperaemic, oedematous, friable or scarred mucosa at the site of the stricture; (4) evidence of acute or chronic pyloric or duodenal ulceration.

The site of the obstruction was the pylorus in 10 subjects and the first part of the duodenum in the remainder. Benign inflammatory biopsies were universally present with no cases of malignancy. Severe oesophagitis, another recognized feature of gastric outflow obstruction, was also present in all cases. The diagnosis was further confirmed by barium study (9 subjects) and ultrasound (1 subject).

Presenting symptoms were of recent onset (mean 46 days, range 2–217 days). Vomiting (75%), weight loss (58%) and abdominal discomfort (67%) were the most common (Table I); severe ulcer pain was not a feature. Only 25% gave a past history of proven peptic ulceration and of these one had been previously successfully treated for gastric outflow obstruction by medical therapy alone and was already taking ranitidine (Table I). Eleven of the 12 had been taking non-steroidal anti-inflammatory drugs (NSAIDs) for a mean of 6 months (range 2 months–2 years).

Relevant physical signs (present in only 5 subjects) were epigastric tenderness and/or a succussion splash.

All responded rapidly to medical treatment alone with complete resolution of presenting symptoms after a mean of 8.3 days (range 2–14 days). NSAIDs were discontinued in all cases. Intravenous fluids and anti-emetics were given as appropriate. H₂-antagonists were prescribed orally in all

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Accepted: 8 May 1991.

This work was presented in part at the Spring 1990 meeting of the British Geriatrics Society in Belfast.

Table 1 Presenting clinical features

Case	Age	Sex	Presenting symptoms	Physical signs	Ulcer history	NSAID/H ₂ -antagonists
1	79	F	4 days abdominal discomfort & vomiting.	Nil	Nil	Azapropazone
2	84	F	2 days vomiting. No pain.	Mild epigastric tenderness.	Nil	
3	84	F	1 month vomiting, weight loss. No pain.	Succession splash, abdom. distension, visible peristalsis.	Nil	Ibuprofen
4	89	F	5 days vomiting, abdominal discomfort, weight loss.	Succession splash, epigastric tenderness.	Nil	Diclofenac Benorylate
5	80	M	3 days abdominal pain, vomiting, weight loss.	Epigastric tenderness.	Nil	Diclofenac
6	76	M	1 month nausea, vomiting	Nil	Nil	Azapropazone
7	79	F	abdominal discomfort. 8 weeks vomiting & weight loss. No pain.	Nil	Nil	Diclofenac
8	87	F	2 weeks vomiting, epigastric discomfort.	Nil	Nil	Ibuprofen Aspirin
9	86	F	3 months 'heartburn'.	Nil	DU x 2 1962/65	Diclofenac
10	80	F	1 week vomiting, weight loss, abdominal discomfort.	Epigastric tenderness.	DU x 2 1986/88	Piroxicam Ranitidine
11	82	F	6 months weight loss, abdominal discomfort.	Nil	Nil	Indomethacin
12	82	F	3 months weight loss. No pain.	Nil	GU 1988	Ibuprofen

DU – duodenal ulcer; GU – gastric ulcer

Table II Investigations, management and follow up

Case	Endoscopy findings*	Histology	Barium meal	Management† (all drugs per day)	Outcome	Follow up/ Maintenance therapy Weight (change)
1	DU with obstruction	Severe inflammatory changes	—	Cimetidine 800 mg	settled 14 days	No recurrence 9 months. wt. 64 kg (+ 2.8) Cimetidine 400 mg
2	Pyloric stenosis with canal ulcer & DU	Chronic inflammatory change of pyloric type mucosa with ulceration	Delayed gastric emptying. Pyloric stenosis & DU	i.v. fluids Cimetidine 1200 mg Metoclopramide 30 mg	settled 2 days	No recurrence 1 year. Cimetidine 800 mg
3	Pyloric stenosis with DU	Chronic inflammation pyloric type mucosa	Enormous stomach no barium flow to duodenum	i.v. fluids Cimetidine 1600 mg Metoclopramide 30 mg	settled 5 days	No recurrence 3 months. wt. 58.6 kg (+ 3.6) Cimetidine 400 mg
4	Pyloric ulceration with canal stenosis	Chronic inflammation & ulceration of pyloric type mucosa	Pyloric stenosis & DU	Cimetidine 800 mg Domperidone 30 mg	settled 10 days	No recurrence 2 years wt. 64 kg (+ 2.0) Cimetidine 400 mg
5	DU with obstruction	Ulcerated pyloric mucosa & granulation tissue	Features of pyloric stenosis	Cimetidine 1200 mg Metoclopramide 30 mg	settled 10 days	No recurrence 10 months wt. 73 kg (+ 4.0) Cimetidine 400 mg
6	Rigid pylorus with stenosis	Inflamed pyloric type mucosa	Distended stomach Marked narrowing of pyloric canal	Ranitidine 900 mg Metoclopramide 30 mg	settled 14 days	No recurrence 1 year wt. 91.6 kg (+ 13.6) Ranitidine 300 mg
7	Pyloric ulceration with stenosis	Inflamed mucosa & granulation tissue	Delayed gastric emptying with pyloric stenosis	Ranitidine 300 mg	settled 7 days	No recurrence 1 year wt. 45 kg (+ 3.3) Ranitidine 150 mg
8	Inflamed pylorus with tight stenosis	Active inflammation	Delayed gastric emptying + + Narrowed pylorus & chronic DU	Ranitidine 300 mg	settled 10 days	No recurrence 3 months Died 5 months later
9	Pyloric stenosis & canal ulcer	Active gastritis	Pyloric canal stenosis	Cimetidine 800 mg	settled 4 days	No recurrence 2 years wt. 43.9 kg (— 2.7) Cimetidine 400 mg
10	Pyloric stenosis & canal ulcer	Inflamed gastric & pyloric mucosa	Food residue + + pyloric canal stenosis	Ranitidine 300 mg Bismuth chelate 480 mg	settled 7 days	No recurrence 6 weeks wt. 57.6 kg (+ 1.0) Ranitidine 300 mg
11	Pyloric stenosis. Canal ulcer & DU	Inflammatory pyloric mucosa	Grossly dilated stomach, pyloric stenosis & DU	Ranitidine 300 mg	settled 6 days	No recurrence 7 months wt. 37.5 kg (+ 0.9) Ranitidine 150 mg
12	Pyloric stenosis & canal ulceration	Inflamed pyloric type mucosa	—	Ranitidine 300 mg	settled 10 days	No recurrence 5 months wt. 80 kg (— 3.0) Ranitidine 300 mg

cases. Doses used varied between patients (ranitidine 300–900 mg daily or cimetidine 800–1600 mg daily). In the one patient already taking ranitidine the dose was increased and bismuth chelate (De-Nol) added. No recurrence of symptoms was reported with H₂-antagonist maintenance therapy (ranitidine 150–300 mg daily or cimetidine 400–800 mg daily). Patients were followed up for a mean of 10 months (range 1.5–24 months). In the 10 subjects for whom records were available, weight gain followed treatment in 8, including all but one of those presenting with weight loss (Table II). One patient died of an unrelated cause.

Discussion

Our diagnostic criteria for gastric outflow obstruction are comparable with previous reports,^{2–4} although nuclear medicine studies were not available. In particular, failure to pass an 11 mm diameter endoscope through the stricture has been used to confirm the diagnosis.³

The overall incidence of benign gastric outflow obstruction is declining. This is attributed to the more successful medical treatment of peptic ulcers with the introduction of H₂-antagonists.⁴ We have found the incidence in the elderly comparable with younger subjects (12% of 738 with peptic ulceration),² but higher than in a previous retrospective surgical study of ulcer disease in the aged which does not provide clinical or diagnostic data for the sub-group with gastric outflow obstruction.⁵

In some respects the clinical features of our subjects differ considerably from typical younger patients who have well documented chronic peptic ulceration with long histories of intermittent ulcer pain. In such cases the development of gastric outflow obstruction has been heralded by persistent and worsening ulcer pain, weight loss and finally vomiting. In contrast, gastric outflow obstruction was the presenting feature of peptic ulceration in most of our patients. The absence of

physical signs, however, is not unusual.^{1,2} Although peptic ulceration was the underlying cause of gastric outflow obstruction in all our patients, ulcer pain is often absent in the elderly⁶ and may have been further masked by the use of NSAIDs thus delaying presentation.⁷

Our success with medical management alone contrasts with the findings in younger patients where it is reported that up to 94% with duodenal ulcer disease and outflow obstruction ultimately require surgery despite intensive medical therapy including cimetidine,² and with older patients in whom 87.5% are reported to require surgery.⁵ Our mean response time was 8.3 days. This is noteworthy as the standard criteria for surgery has been failure to respond to intensive medical therapy within 5 days.²

Diminished levels of prostaglandin E₂ have recently been implicated in the pathogenesis of gastric outflow obstruction associated with pyloric oedema and scarring in younger patients.⁸ This suggests a possible mechanism whereby NSAIDs might predispose to gastric outflow obstruction and we believe it should be added to the list of possible NSAID-induced gastrointestinal complications in the elderly.

Our success with medical treatment may be explained by a predominant element of pyloric spasm and oedema induced by acute ulceration and NSAIDs. However, even in this clinical context, over 60% of younger patients have required surgery.² We believe weight gain, as documented in most subjects, to be an important marker of the successful treatment of peptic ulcer disease.

This series suggests that, in contrast to typical younger patients with gastric outflow obstruction who are male, have longstanding and worsening ulcer symptoms¹ and require surgery,² the elderly are predominantly females taking NSAIDs who present acutely and ultimately respond to medical management alone although this may take longer than the conventional 5 days.

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