

Acute Perforated Duodenal Ulcer In Maiduguri

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Citation

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Abstract

Background: Effective medical management of peptic ulcer disease (PUD) has reduced the incidence of gastric outlet obstruction (GOO) as a complication, but the frequency of perforation, especially in the elderly, remains unchanged and is in fact on the increase. There is a changing trend in emergency surgery for perforated duodenal ulcer (PDU) from definitive anti-ulcer surgery to simple closure followed by *Helicobacter pylori* eradication. **Aims/Objective:** To present our experience in managing PUD with simple closure followed by *Helicobacter pylori* eradication. **Patients and Methods:** This is a retrospective study where hospital records of 55 patients managed for PUD over a 9 year period (Jan. 1999-Dec. 2007) were obtained from ward admission register, theatre operation registers and patient's case files from the medical records department. The patient's biodata, clinical and operative findings and treatment outcome were extracted and analysed. **Results:** There were 55 patients; 44 males and 11 females (M:F=4:1). Their ages ranged between 18 and 65 years with a mean \pm SD of 39.9 \pm 13.5 years. Most of the patients (34; 61.8%) were below 40 years of age and the majority (39; 71.0%) had a history suggestive of chronic peptic ulcer disease. Twenty-six (47.3%) of the patients presented within 24 hours of perforation, while 9 (16.4%) presented more than 72 hours afterwards. The latter group accounted for most (5; 55.6%) of the mortality. All the perforations were anterior pyloroduodenal and all except one had simple closure with omental patch followed by a course of a proton pump inhibitor and *Helicobacter pylori* eradication therapy. **Conclusion:** Simple closure with omental patch followed by *Helicobacter pylori* eradication was effective with low morbidity and mortality despite patients' late presentation in our center.

INTRODUCTION

Acute PUD is a frequent cause of generalized peritonitis in our environment. Effective medical management of PUD with proton pump inhibitors and therapy to eradicate *Helicobacter pylori* has limited the operative management of this disease to its complications^{1,2,3}. The operative management of PDU is varied^{4,5}. There is a general shift from the traditional definitive peptic ulcer surgery to simple closure with pedicled omentum according to Graham⁶, followed by a course of proton pump inhibitors and therapy to eradicate *Helicobacter pylori*^{7,8}. Definitive peptic ulcer surgery increases operative time and exposes the patient to prolonged anaesthesia. It also increases the risk of postoperative complications. Because of these problems, the acceptable management principle seems to be avoidance of definitive acid-reducing surgery in the presence of generalized peritonitis or peritoneal irritation. This is especially true in African patients who present late⁹. We have managed patients with acute perforated duodenal ulcer by simple closure of the perforation with adequate peritoneal lavage followed by a postoperative course of proton pump inhibitors and antibiotics. This study aims to review the

results of such a treatment modality in our patients.

PATIENTS AND METHODS

Patients managed for acute perforated duodenal at the UMTH in Maiduguri, between January 1999 and December 2007 were studied. Their clinical records were obtained from the admission and discharge registers, theatre operation records and the medical records central library. Data extracted from such records included bio-data, time of onset of symptoms, presentation, use of NSAIDs, history of peptic ulcer disease, resuscitative measures, operative findings and treatment, outcome of treatment and postoperative complications. Patients with other causes of perforation were excluded from the study.

Diagnosis was made from history, plain abdominal and chest radiographs, and confirmed at laparotomy; which was done after resuscitation with intravenous fluids and peri-operative antibiotics. Other investigations included haematological profile, serum urea and electrolytes and urinalysis, adequate hydration was indicated by an hourly urine output of 30ml/hour. A naso-gastric tube was passed to decompress the stomach and maintained till bowel sounds return.

Laparotomy was via a long mid-line incision, where an exploration was carried out to identify the site of the perforation and an estimate of its size. The peritoneal cavity was copiously lavaged with warm normal saline and the perforation closed with deeply placed 2/0 chromic catgut sutures tied over a piece of pedicled or free omentum. Where the perforation was sealed, its integrity was ensured and the peritoneal cavity toileted as required. All the patients were on parenteral antibiotics for 3 days which continued for 5 days in those with purulent peritonitis. The abdomen was closed en mass with 1 nylon (polyamine) suture. Patients were discharged on oral omeprazole, metronidazole and amoxicillin for 1 month.

RESULTS

Fifty-five patients had emergency surgery for acute perforated duodenal ulcer during the study period. This consisted of 44 males (80.0%) and 11 females (20.0%). The male/female ratio was 4:1; the patients were aged 18-65 years, with a mean age of 39.94 \pm 13.5 years. The peak incidence was in the 4th decade (31-40 years; Fig. 1). Duration of symptoms before presentation was a few hours to 5 days (mean 2.8 days). Twenty-six patients (47.3%) presented within twenty-four hours of onset of symptoms, 14 between 24 and 48 hours and 9 over three days afterwards (Table 2). None of the perforations was associated with recent ingestion of non-steroidal anti-inflammatory drugs (NSAIDs). There was a history of chronic peptic ulcer disease in 39 (71.0%) patients. The commonest presenting symptoms were sudden onset of severe epigastric pain in 51 (92.7%), vomiting in 19 (34.5%), abdominal distention in 32 (58.2%) and fever in 16 (29.1%). Abdominal tenderness and classical signs of peritonitis were demonstrable in 49 (89.1%) and 46 (83.6%) patients, respectively, with 3 (5.4%) of the patients presenting in shock (systolic blood pressure \leq 80mmHg). Forty-nine (89.1%) of the patients had plain abdominal and chest radiographs done, with free gas under the diaphragm demonstrated in 32 (65.3%) of them. All the patients had anterior pyloroduodenal perforations with massive serous peritoneal fluid in 26, massive cloudy/opalescent peritoneal fluid in 12 and frank peritoneal pus with fibrinoid adhesions in 17 patients. Six of the perforations were found to be sealed. Three of the perforations were of minimal size (\leq 5mm) and 43 were massive ($>$ 10mm). Forty-eight (87.3%) of the patients had Graham's omental patch of the perforations. The simple closure was effected with either a pedicled omental patch (42 patients) or a free graft of omentum (6 patients). Those with sealed perforations had peritoneal lavage with warm

saline and mass closure of the abdomen. One patient had truncal vagotomy and Roux-en-Y gastrojejunostomy in addition to simple closure. The main postoperative complications were wound infection in 12 (21.8%), postoperative fever 20 (26.4%), chest infection 11 (20.0%) and incisional hernia in 3 (5.4%). There were 4 cases of leakage from the closure site that necessitated a re-exploration and closure. Three of these patients developed intra-abdominal abscesses that required open drainage. One of these patients developed peritonitis with septic shock that lead to his demise. The mean duration of hospital stay was 10 days (range 7-21 days). The mortality rate was 9 (16.4%). Causes of death included severe electrolyte derangement in 3 patients, septicaemia in 4 and acute renal failure in 2 others. There was no record of ulcer recurrence and the mean duration of follow-up was 5.2 month (range 2-13 weeks).

Figure 1

Fig. 1. Age and Sex distribution of the patients

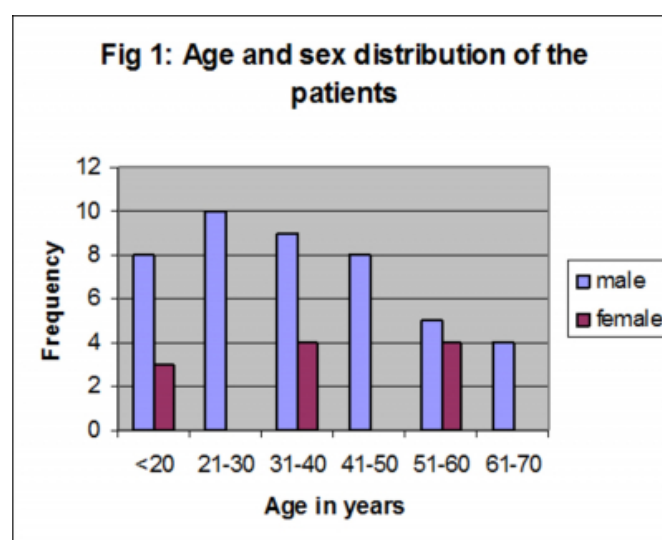


Figure 2

Table 1. Relationship between time of presentation and mortality

Time of presentation (hours)	Frequency (%)	Mortality (%)
0 – 24	26 (47.3)	1 (3.8)
24 – 48	14 (25.5)	2 (14.3)
48 – 72	6 (11.0)	1 (16.7)
>72	9 (16.4)	5 (55.6)
Total	55 (100)	9 (16.4)

The overall mortality rate was 16.4%.

Figure 3

Table: 2 Clinical presentation of the patients

Clinical presentation	Frequency (%)
Severe abdominal pain	49 (89.1)
Abdominal distention	32 (58.2)
Vomiting	14 (25.5)
Severe dyspepsia	32 (58.2)
Nausea	17 (30.9)
Fever	26 (47.3)
Constipation	3 (5.4)
Shock	3 (5.4)
Abdominal tenderness	49 (89.1)
Classical signs of peritonitis	46 (83.6)

Figure 4

Table: 3 Operative procedures done for the patients

Operative procedure	Frequency (%)
Simple pedicled omental patch	48 (87.3)
Simple free omental patch	6 (10.9)
Truncal vagotomy and pyloroplasty	-
Simple closure, truncal vagotomy and Roux-en-Y gastrojejunostomy	1 (1.8)
Total	55 (100)

Figure 5

Table 4. Postoperative complications

Complications	Frequency (%)
Wound infection	12 (21.8)
Postoperative fever	20 (36.4)
Chest infection	11 (20.0)
Intra-abdominal abscess	3 (5.4)
Incisional hernia	3 (5.4)
Wound dehiscence	3 (5.4)
Septicaemia	4 (7.2)

DISCUSSION

A total of 55 patients were treated for acute perforated duodenal ulcer in our hospital over a 9-year period; giving an average of 6 cases per year which is a low incidence. This is similar to 4 cases per year seen in Ile Ife, Nigeria¹⁰. Most of the patients (44; 80%) were males (male:female ratio: 4:1), similar to other studies where the male:female ratio ranged from 3.3:1 to 9:1^{11,12}. The commonest age at presentation was between 21 and 40 years with a mean of 39.9 +/-13.5 years which differs significantly from other reviews from Africa that had an average of 64.8 +/-11.4 years¹¹. In the Caucasian series, the majority of the patients

were above 60 years and the incidence is higher in elderly females¹³. Most of our patients 39 (71.0%) had past history of chronic peptic ulcer disease; this is in sharp contrast to 47% reported by Lawal and colleagues¹⁰ in southwestern Nigeria. The reason for this difference is not quite apparent. The diagnosis of acute PUD was mainly clinical in our series, with typical symptoms of perforation peritonitis manifesting especially in those with a past history of chronic peptic ulcer disease. However, a high index of suspicion supported by an abdominal paracentesis and the demonstration of free air under the diaphragm on a plain chest radiograph was needed to make a diagnosis in those with atypical features. In some, the final diagnosis was only made intraoperatively. There are well known risk factors for PDU such as corticosteroid ingestion, immunosuppression, alcohol, smoking and chronic ingestion of NSAIDs especially in elderly patients, but none was a reason for perforation in our series. A report from Ghana showed that 47.7% of perforations seen were associated with the use of NSAIDs¹¹. This may be related to the age composition of the patients in the Ghanaian study where the mean age was above 60 years compared to our patients whose mean age was 39.9 years (and 61.8% were below 40 years of age).

The time lapse between an episode of acute duodenal ulcer perforation and surgical intervention is a critical determinant of survival¹⁴. Although 29 (55.7%) of our patients had surgical intervention within 24 hours of presentation, and 23 (44.2%) had theirs more than 24 hours afterwards, when this time is added to delays these patients had before presenting to hospital, it will be seen that the majority of these patients actually had surgical intervention much more than 24 hours from time of perforation. A mean period of 22.15 hours between perforation and surgical intervention was reported in 156 patients studied by Bin-Taleb and colleagues¹⁵. A corresponding low overall mortality of 3.9% was reported in the same study compared to 16.4% in this study.

The findings at laparotomy vary depending on the site, size and duration of the perforation. All the patients in this study had anterior pyloroduodenal perforations; similar to other reported series¹⁶. The size of the perforation determines the amount of peritoneal contamination¹⁴. Forty-three (82.7%) of our patients had massive perforations, more than 10mm in approximate diameter. The degree of peritoneal soilage is crucial in patients with peritonitis due to acute duodenal ulcer perforation and early surgical intervention prevents further contamination of the peritoneal cavity and removes the source of infection.

Sealed perforations are a possibility at laparoscopy or laparotomy; the former was reported by Fujii and colleagues¹⁷ where it was managed conservatively with serial ultrasonography. This was seen in 6 (11.5%) of our patients. In our cases the peritoneum was thoroughly lavaged with 3 to 5 liters of warm normal saline, the site of the sealed perforation was inspected and its integrity ascertained, then the laparotomy wound was closed in-mass. The patients were subsequently managed on a 4-week postoperative course of a proton pump inhibitors and therapy to eradicate *Helicobacter pylori* similar to those that had simple closure. Simple closure of perforated duodenal ulcer is generally accepted^{12,18}, and particularly so when there is delay in presentation and severe peritonitis has set in. This was the case in 29 (52.9%) of our patients. The patients that presented within 24 hours of perforation were all treated by simple closure except one that had a definitive anti-ulcer surgery. These patients would have had one form of definitive anti-ulcer surgery or another to achieve a permanent cure for the ulcer diathesis; but currently with effective medical therapy using a course of proton pump inhibitors complimented with *Helicobacter pylori* eradication, this has become unnecessary. Simple closure has the added advantage of being easy, quick, and safe and can be applied in most situations by most surgeons.

The major postoperative complications were postoperative fever, wound and chest infection (Table 4). The causes of these complications were multifactorial, viz.: delay in presentation, delay in surgical intervention, gross peritoneal soilage, septicaemia and shock. The delay in surgical intervention, after the patient presents to hospital, is usually due to the time taken to resuscitate these very ill patients. The mortality rate of 16.4% in our series is similar to the report by Barut and colleagues¹⁹ but low when compared to similar studies^{9,11} in the sub-region. This may be explained by the differences in age composition of the patients and other risk factors of perforation. The deaths were due to septicaemia and electrolyte derangements.

We conclude that simple closure with omental patch and proton pump inhibitors in combination with antibiotics to eradicate *Helicobacter pylori* was the most common approach to treatment of acute perforated duodenal ulcer in Maiduguri. Definitive ulcer surgery and treatment for *Helicobacter pylori* may be advocated in selected cases in the developing world where there may be doubt about

compliance to drug therapy, follow-up and high chances of re-infection. Most of the mortality in this study was associated with late presentation.

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