Review

Acute perforated peptic ulcer: On clinical experience in an urban tertiary hospital in south east Nigeria

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A B S T R A C T

Background: Acute perforated peptic ulcer is a leading cause of generalized peritonitis and its management has continued to be a challenging task in our environment.

Objective: There is a paucity of published reports on acute perforated peptic ulcers in our environment. This study was conducted to evaluate the different pattern of risk factors clinical presentations, management and clinical outcome of patients with acute perforated peptic ulcer in our setting and to highlight the factors that continue to account for the high mortality and morbidity as seen here.

Method: A retrospective study where data of seventy-six (76) patients managed for generalized peritonitis due to acute peptic ulcer perforation over a five year period (January 2006–December 2010) were retrieved from medical records of Enugu State University of Science and Technology Hospital (ESUTH). The patients’ biodata, clinical and operative findings and treatment outcome were extracted and analysed, after institutional ethical approval was secured. All other cases of generalized peritonitis not traceable to acute peptic ulcer perforation were excluded from the study.

Results: There were 76 patients; 58 males and 18 females (M:F = 3.2:1). Their ages ranged from 20 to 80 years with a mean of 39.5 yr and SD ± 13.10 years. Majority of the patients 49(64.4%) were 40 years of age and below and only 24 (31.6%) had a previous history suggestive of chronic peptic ulcer disease. Twenty five (32.9%) patients presented within 24 h of onset of symptoms of perforation with a mortality of 8.0%. Slightly more than half of our patients 39(51.3%) presented between 24 and 48 h with mortality of 17.9%. Twelve patients (15.8%) presented between 48 and 72 h and the mortality in this group was 58.3%. The latter two groups accounted for most of the mortality in our series.

All perforations were anterior perforations within the first 2.5 cm of the duodenum and all had simple closure with pedicled omental patch and peritoneal toilet with copious volumes of warm normal saline. Postoperatively all received Helicobacter pylori eradication therapy and proton pump inhibitors for at least two months.

Conclusion: Patient groups who presented early had low mortality rates, but patient groups who presented late had higher mortality rates. Overall mortality was 21%.

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1. Introduction

Peptic ulcer disease (PUD) is a worldwide health challenge. Globally the incidence of peptic ulcer disease is said to have fallen in recent years. Also recent advances have taken place in both diagnosis and management of peptic ulcer disease, namely improvements in endoscopic diagnostic and therapeutic facilities, the increased use of proton pump inhibitors and Helicobacter pylori eradication therapies. In spite of all these peptic ulcer perforation rates have remained unchanged and therefore remains a major health challenge.

The pattern of perforated PUD is said to vary from one geographical area to another, depending on some socio-demographic and perhaps environmental factors. In a developing country such as ours, the patients presenting with perforated PUD are young with a dominant male preponderance. This is in contrast to the developed countries were the patient population with perforated...
PUD are mainly the elderly with less pronounced incidence differences between sexes. It is probable that the very strong association with smoking and alcohol among the young male population may account for the high incidence in developing countries. Certainly in the West the high incidence is due to ulcerogenic drug ingestion amongst the elderly population. It is also noted that in the developing countries, the patients with perforated PUD, presented late to definitive management centres. Many patients first sought medical assistance from traditional healers and unauthorized medical personnel prevalent in developing countries.

Noteworthy in our environment is the fact that many patients gave no previous history of peptic ulcer disease before their perforation. The diagnosis of perforated PUD could pose a diagnostic challenge in most cases especially in patients with no previous history of PUD. However, sudden onset of severe abdominal pain, vomiting, shock, and classical signs of peritonitis all secondary to perforated PUD in a patient with a pervious history of PUD offers little difficulty in diagnosis of perforation. Other ancillary support like sub dia-

phragmatic gas on plain erect chest x-ray and observations on abdominal ultrasonography are of help in some cases.

The operative management of perforated PUD have hitherto been varied, but recently there is a definite shift from the traditional definitive peptic ulcer surgery to simple closure of the perforations with omental (Graham’s patch). This is followed up postoperatively with H. pylori eradication and administration of proton pump inhibitors therapies. This approach is even more pertinent here, where patients present late with gross and fulminant peptic ulcer disease and therefore not suitable for definitive peptic ulcer surgery.

Delay in diagnosis and prompt initiation of surgical management of perforated PUD have clearly been shown to be associated with high morbidity and mortality after surgery for perforated peptic ulcer disease. Early recognition, prompt diagnosis and aggressive resuscitation and early surgical intervention will clearly aid in keeping the morbidity and mortality low.

There is a paucity of reports on perforated peptic ulcer disease from our local environment, despite a fairly high number of cases seen. Our aim is to describe our experience in the management of perforated PUD in our environment, outlining the incidence, varied clinical presentations, management outcome and to highlight our limitations in keeping the morbidity and mortality low.

2. Patients and methods

This is a retrospective study of patients managed for acute perforated duodenal ulcer at ESUT Teaching Hospital Parklane, Enugu, an urban tertiary and Teaching Hospital in Southeast Nigeria between Jan 2006 and Dec 2010 (a five year period). All data were retrieved from the medical records department of the hospital. Data extracted from these records included bio-data, time of onset of symptom of perforations, clinical presentations which included clinical history, age of and sex of patient, past history of peptic ulcer disease, alcohol intake, cigarette smoking, use of NSAIDS and other drugs such as steroids. Also extracted was the physical finding supporting generalized peritonitis, presence of shock, fever, conscious state, resuscitative measures, operative findings and treatment. Outcome of treatment, including length of hospital stay and post-operative complications (morbidity) and mortality were also noted.

The diagnosis of generalized peritonitis was made from history and physical examinations alone, but in some cases, plain abdominal, chest radiographs as well as ultrasound scans of abdomen and pelvis was used as ancillary support to clinical findings. In some cases diagnosis were confirmed only at laparotomy. Other investigations performed included blood urea, and serum electrolyte studies, haematological indices and urinalysis. Adequate resuscitation were achieved with intravenous fluids, intravenous antibiotics (third generation cephalosporin plus metronidazole) and naso-gastric tube suction to decompress the stomach. Urinary output of >30 ml/h indicated adequate hydration and resuscitation.

After adequate resuscitation, laparotomy was performed utilizing a middle through a midline incision. Exploration was carried out to identify the site of perforation, to estimate the size and also the volume and nature of peritoneal exudate. The duodenal perforation was closed with interrupted 2/0 vicryl sutures tied over pediced omentum (Graham omentopexy). Liberal peritoneal wash out was done with copious volumes of warm normal saline. Intra-abdominal vacuum drain was left insitu and abdomen closed with mass suture utilizing No 2 Nylon sutures. Most of the surgical operations were performed by Consultant surgeons, and others by senior Residents under the supervision of the consultant surgeons. All patients received intravenous fluids, continued nasogastric tube suction until bowel sounds returned and oral feeding commenced. In addition, all patients received intravenous antibiotics utilizing third generation cephalosporin and metronidazole infusion for a period ranging from four to six days postoperatively. Patients were discharged home on omeprazole, metronidazole and amoxicillin or augmentin for six weeks.

3. Results

81 Patients underwent emergency laparatomy for acute perforated peptic ulcers. Out of these, 5 patients were excluded from the study because of incomplete data, and therefore failed to meet the inclusion criteria. Thus data from 76 patients were analysed (an average of 15 cases annually). The patients consisted of 58 males (76.3%) and 18 females (23.7%) (M:F = 3.2:1). The ages of the patients ranged from 20 to 85 years, with a mean age 39.5 ± 13.1 years. The peak incidence was in the 4th decade (31–40yrs) (Fig. 1) Duration of symptoms of perforation before presentation were a few hours to 72 h (mean 41 h). 4 patients (5.3%) presented within 12 h of onset of symptoms; 21 patients (27.6%) presented between 12 and 24 h. 28 patients (36.8%) presented between 24 and 36 h 11 patients (14.5%) presented between 36 and 48 h, whilst 7 patients (9.2%) presented between 48 and 60 h and 5 patients (6.6%) presented between 60 and 72 h (Table 1).

There was a positive past history of chronic peptic ulcer disease in only 24 patients (31.6%). Only 7 patients (9.2%) had a positive history of ingestion of non-steroidal anti-inflammatory drugs (NSAIDS). 55 patients (72.4%) and 42 patients (55.3%) gave history of alcohol abuse and cigarette smoking respectively. Most patients who smoked also abused alcohol (Table 2).

The commonest presenting symptoms were severe upper abdominal pain in 69 patients (90.8%), severe nausea in 34 patients (44.7%) vomiting in 22 patients (28.9%). Abdominal distension in 66 patients (86.8%), abdominal tenderness in 70 patients (92.1%), shocked state (systolic blood pressure ≤90 mm Hg in 57 patients (75%). Classical signs of generalized peritonitis were elicited in 68 patients (89.5%) (Table 3). 42 patients (55.3%) had plain abdominal and chest radiographs performed with free subphrenic gas demonstrated in 28 (66.7%) of them. 30 patients (39.5%) had abdomino pelvic ultrasound studies which was positive for free peritoneal fluid and features suggestive of peritonitis is 28 (93.3%) of them.
All patients had laparotomy through an upper midline incision after adequate resuscitation with intravenous fluids, 1V antibiotics, nasogastric tube suction and vital signs monitoring. Urinary output of ≥30 mls per hour was used to ascertain adequate resuscitation. At laparotomy all patients had anterior duodenal perforations. Most perforations showed attempt at omental sealing. The nature of peritoneal exudates were cloudy bilious in 40 patients (52.6%) Serosanguineous in 25 patients (32.9%) and frank pus with fibrinoid adhesions in 11 patients (14.5%). None of the perforations in our series was sealed. In 17 patients (22.4%) the size of perforation was ≤5 mm diameter and in 59 patients (77.6%) it was >10 mm diameter. All patients had the perforation closed with 2/0 vicryl sutures over a pedicled omental patch and all had copious peritoneal lavage with warm normal saline. Intra abdominal drains were left in situ before mass closure of abdominal wound with No 2 Nylon sutures.

Post-operative complications were recorded in 48 patients (63.2%) (Table 4). The most frequent complication was surgical site infection in 30 patients (39.5%), pulmonary infection in 10 patients (13.2%), continuing peritonitis in 8 patients (10.5%). Re-perforation in 5 patients (6.6%) necessitated re-exploration and re-closure, 2 of these patients further developed intra abdominal abscess and one a duodenal fistula. Overall 7 patients (9.2%) developed post-operative intra abdominal abscess. Cardiopulmonary arrest was recorded in 6 patients (7.9%) all of whom died. The cardiopulmonary arrest occurred a few minutes to a few hours after surgery either in the recovery room or Critical Care Unit. Continuing septic shock was recorded in 4 patients (7.9%), 4 of these developed acute renal shutdown and electrolyte imbalance which led to their demise. Prolonged paralytic ileus was recorded in 3 patients (3.9%) one of whom developed wound dehiscence. Overall 4 patients (5.3%) developed wound dehiscence or burst abdomen. Incision hernia occurred in 3 patients (3.9%) at follow up.

The mean duration of hospital stay in those that survived was 10 days (range 7–25 days). The overall mortality rate in our series was 21.1%.

Causes of death included cardiopulmonary arrest in 6 patients, acute renal shutdown in 4 patients, septicaemia in 3 patients, severe electrolyte imbalance with prolonged paralytic ileus and duodenal fistula in one patient respectively. The mean duration of follow up was 4.4 months (range 4–16 weeks).

### 4. Discussion

In this review a total of 76 patients were enrolled over a five year period giving an average of 15 cases annually. This figure is similar to that reported from Tanzania12 nd by Schein et al.13 Low (4 per year) incidence of perforated PUD is reported in North-East region Nigeria.14 The difference between our figures and those of the North East region of Nigeria reflect differences in rate of risk factors for perforated peptic ulcer disease from one region and another. The non-alcohol intake in North-East Nigeria region on account of the Islamic religion may be a factor for the low incidence.

Most the patients 58 (76.3%) were males (male: female ratio 3.2:1). This is similar to other studies14,15. In this study, the commonest age group of presentation was in the 4th decade (21–40 yrs of age) with a mean of 39.5 ± 13.1 years. Our study is similar to other studies in developing countries,12,16 but differs from demographic profile from developed countries where the majority of the patients are above 60years of age and the incidence of perforated PUD found to be higher in females taking ulcerogenic drugs.17,18 In our series only 9.2% had a history of ingestion of NSAIDS. The high incidence of perforated PUD amongst young males in our environment may be due to smoking and excessive alcohol consumption prevalent amongst this age group. Most patients who smoked also abused alcohol. It is known that smoking inhibits pancreatic bicarbonate secretions, which tend to neutralize acid secretion, thus predisposing to increased acidity in the duodenal bulb. It also causes delay in duodenal ulcer healing.19 Alcohol on the other hand predisposes to gastric ulceration, stimulates gastric acid secretion as well as enhancing gastrin release.20

It has been shown that the mean prevalence of H. pylori infection in patients with a perforated PUD range from 65 to 70% and is a significant risk factor for perforated PUD especially in young patients, which constitute majority of our patients.21 However we were unable to determine the presence or otherwise of H. pylori infection in our series because of unavailability of reagents.

Only 31.6% of patients in our series had a positive past history of chronic PUD. This is in agreement with previous studies22,23 but in...
the sharp contrast to study by Nuhu et al. in North-East Nigeria.14 It has also been shown that in many developing countries that in most cases the diagnosis of PUD is first made following a perforation.23 The present study confirms the existence of silent PUD in majority of our patients (69.4%). The lack of previous symptoms of PUD and therefore no treatment exposes most patients to a higher risk of PUD perforations.

It has been elaborately reported that time from onset of symptoms of perforation to definitive treatment is a good indicator of outcome. In the present study most of our patients presented late, more than 24 h from the onset of symptoms. Our findings are in agreement with previous studies in developing countries.19,23–25 Late presentation in our series may be attributed to the fact that most patients first sought medical assistance from unauthorized medical personnel and/or traditional healers. This practice is common in our environment and it is attributed to ignorance, religious beliefs, compounded by lack of easy accessibility to health care facilities and high cost implication of hospital treatment. Hospital care is only sought when pain becomes unbearable and the patient’s condition is deteriorating. Studies have shown that a lower mean period between perforation and surgical intervention was associated with lower mortality rates.26 Our series had a mean period of 41 h between onset of symptoms and definitive treatment and consequently a fairly high mortality rate at 21.1%, as compared with Bin-Taleb et al.26 who had a mean period of 22.15 h with a low mortality rate of 3.9%

The diagnosis of perforated PUD is usually a clinical diagnosis.22,27

At laparotomy all patients had anterior duodenal perforations with no gastric perforation. Also no gastric perforation was reported from a study in Maiduguri Nigeria.14 A high duodenal to gastric ulcer ratio of 25:1 was found in Sudan in North Africa.28 These ratios are in sharp contrast to low ratios 3.1 to 4.1 of duodenal to gastric ulcers reported from the Western world.29 The amount of peritoneal contamination is determined by the size of perforation.24 In our series the majority (77.6%) of patients had massive perforations with a size of more than 10 mm in approximate diameter. The nature of the peritoneal exudates is also a determinant of the duration of perforation before surgical intervention24. Cloudy bilateral or sero-sanguineous exudates were seen in patients that presented earlier. Patients with intra-peritoneal frank pus represented those with a prolonged delay between onset of symptoms and surgical intervention. This was seen in 11 (14.5%) of our patients. In this group of very late presenters, the mortality rate was very high. None of the perforations in our series was found to be sealed.

No patient had definitive antitulcer surgery like vagotomy plus drainage. The reasons were firstly that most of the patients had moderate to severe peritoneal sojage that precludes any type of definitive antitulcer surgery.30 Secondly simple closure of perforated gastric or duodenal ulcer has now generally accepted as a standard procedure and is been shown to be quick and simple to perform, safe with acceptable morbidity and mortality.15,30 Following the simple closure of the perforation, all patients had copious peritoneal lavage with warm normal saline and mass closure of the laparotomy wound with No. 2 Nylon sutures with intra abdominal suction drain left in situ because of the possibility of reperforation. A six weeks course of Helico Pylori eradication therapy and proton pump inhibitors were administered to all our patients postoperatively.

The overall complication rates in our series was 63.2% (Table 4) which is higher than reports from elsewhere.14,13 However some other studies reported the same high complication rates as observed in our series. Surgical site infection was the most common complication in our series and is in agreement with other studies.12 The reason for the high rates of surgical site infection was due to heavy contamination of the wound due to the severe bacterial peritonitis. Other complications included pulmonary infections, continuing peritonitis, intra abdominal abscesses, re-perforation and duodenal fistula, continuing septic shock, prolonged paralytic ileus and wound dehiscence. The reasons for these complications were multifactorial viz: delay between onset of symptoms and presentation. Critically ill patients at presentation necessitating prolonged resuscitation and therefore further delay before surgical intervention, shocked state and septicemia in many patients and gross peritoneal sojage due to delayed presentation.

Mortality rate in our series was 21.1% and is similar to other reports in developing countries.14,33 In our study, we however found high mortality in patients who were >40 years of age, delayed presentation (>24 h), shock (systolic Bp ≤ 90 mm Hg) on admission. The causes of death were cardiopulmonary arrest all of which occurred few minutes to few hours post-operatively, continuing septic shock with acute renal shut down and electrolyte imbalance.

5. Conclusion

Perforated peptic ulcer disease continues to be encountered frequently in our environment especially occurring predominantly in young males, most of whom are not known to suffer from PUD previously. Compounding this is the late presentation of most of our cases, which resulted invariably in high mortality rates recorded in our series and in most areas of the developing world.

It would be appropriate for government agencies to put a stop to the activities of untrained medical personnel, who are the first attendants to see these acutely ill patients so that these patients can present earlier to definitive care centres for prompt diagnosis and management of this surgical emergency.

Encouragement on stopping smoking of cigarettes and advocating only moderate amounts of alcoholic intake may help change the demography of patients in this environment with PUD and its complications.

Ethical approval

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Author contribution

Dr Ugochukwu surgeon originator and contributor, Dr Nzegwu pathologist coordinator and proof reader, Dr Amu contributing author, Dr Dilibe Surgeon and contributor.

Conflicts of interest

None declared.

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