

## PEPTIC ULCER PERFORATION: UNIVERSITY OF BENIN TEACHING HOSPITAL EXPERIENCE -BENIN CITY, NIGERIA.

NWASHILLI N.J<sup>1</sup>, IROWA O<sup>1</sup>

### Abstract

Peptic ulcer perforation is one of the surgical complications of peptic ulcer disease. Treatment can be operative or non-operative followed by proton pump inhibitor and eradication of *Helicobacter pylori*. The study was aimed at analyzing the clinical features, operative findings and outcome of patients who had operative management for peptic ulcer perforation. It was a retrospective review of 70 patients with intra-operative diagnosis of peptic ulcer perforation. The study was carried out at the University of Benin Teaching Hospital, Benin City, Edo state, Nigeria. The case files of 70 patients with intra-operative diagnosis of peptic ulcer perforation by open surgery over a 3-year period from September 2009 to August 2012 were retrieved from the medical records library. Clinical data obtained from the patients' case files were analyzed using SPSS 16. There were 59 (84%) males and 11 (16%) females with age range from 14-92 years. The male to female ratio was 5:1. The overall mean age of the patients was  $45 \pm 17$  years. Eighteen (26%) patients were in shock on admission. Simple closure and omental patch reinforcement was the operation done in all the patients. Sixty patients were well and discharged after surgery while 10 died. The overall mortality rate was 14%. There is a high incidence of perforated peptic ulcer in our centre. Simple closure with omental patch reinforcement, with proton pump inhibitor and eradication of *Helicobacter pylori* without definitive anti-ulcer surgery were the management approach adopted in our centre.

### Introduction

Peptic ulcer perforation is one of the surgical complications of peptic ulcer disease. Other complications include bleeding peptic ulcer and gastric outlet obstruction. Peptic ulcer perforation occurs in two to ten percent of peptic ulcer disease<sup>1</sup>. Following the introduction

of H<sub>2</sub>- receptor blockers and proton pump inhibitors, there has been a sharp decrease in elective peptic ulcer surgery. However, emergency operations for complications such as perforations are on the rise<sup>2</sup>.

Once the diagnosis of perforation has been made, it is generally agreed that emergency surgery should be performed as soon as the patient has been adequately resuscitated<sup>3</sup>. However, non-operative treatment in the form of intravenous fluids, nasogastric tube suction, intravenous antibiotics and anti-ulcer drugs has been shown to be safe and effective in selected patients as the perforation frequently gets

---

KEYWORDS: *Peptic ulcer perforation, simple closure with omental patch.*

<sup>1</sup>Department of Surgery, University of Benin Teaching Hospital, Benin City, Edo state, Nigeria

\*Correspondence

Nwashilli N.J

Department of Surgery, University of Benin Teaching Hospital, Benin, Edo state, Nigeria E-mail: namoforever@yahoo.com  
Mobile telephone: + 2348037214386

Conflict of Interest

No conflict of interest. The authors did not receive any grants or sponsorship for the study.

sealed spontaneously by omentum and adjacent organs<sup>4</sup> Accepted therapeutic options are either simple closure of the perforation followed by proton pump inhibitor immediately or simple closure with definitive anti-ulcer operation. Both options are followed by Helicobacter pylori eradication. There is a trend towards simple closure of perforation without definitive anti-ulcer surgery, followed by anti-secretory and anti-bacterial medication in recent years<sup>5</sup>.

This study was conducted to analyze the clinical features, operative findings and outcome of patients with peptic ulcer perforation.

#### Patients and Methods

This was a retrospective review of all patients with intra-operative diagnosis of peptic ulcer perforation at the University of Benin Teaching Hospital, Benin City, Edo state, Nigeria, over a 3-year period from September 2009 to August 2012. Seventy-five patients were diagnosed with peptic ulcer perforation by open surgery as noted from operation room register, however, 70 patients' case files were retrieved from the medical records library for analysis while five patients' case file were not seen; hence they were excluded from the study. Data obtained from the case files included demographic data, clinical presentation, chest radiograph and abdominal ultrasound findings, risk factors for peptic ulcer disease (alcohol consumption, smoking, use of non-steroidal anti-inflammatory drugs), type of surgery done, operative findings, complications, duration of hospital stay and outcome. Patients with systolic blood pressure of 90mmHg or less at presentation were recorded to be in shock. Descriptive statistical analysis was done using SPSS 16.

#### Results

There were a total of 70 patients with intra-operative diagnosis of peptic ulcer perforation. There were 59(84%) males and 11(16%) females with age range from 14 to 92 years. The male to female ratio was 5:1. The overall mean age was  $45 \pm 17$  years.

All the patients presented after 24 hours following perforation as suggested from the history of the time of onset of severe abdominal pain. Eight out of the 10 patients that died presented in shock on admission with large perforation (1-3cm in diameter). The mean time from presentation to the time of surgery was  $23.3 \pm 26.7$  hours.

Chest radiograph revealed air under the diaphragm in 50(94%) patients out of 53. There was no chest radiograph in the remaining seventeen patients. Abdominal ultrasound demonstrated free fluid with echogenic debris in the peritoneal cavity in 52(74%) patients.

Simple closure of the ulcer perforation with omental patch and warm saline lavage of the peritoneal cavity was done in 68(97%) patients while two patients with large ulcer perforation, in addition to simple closure and lavage had gastro-jejunostomy to divert gastric secretion away from the repaired perforation. None of the patients had a definitive anti-ulcer surgery in the form of vagotomy and a drainage procedure. All the patients had proton pump inhibitor therapy and Helicobacter Pylori treatment with antibiotics for two weeks. Five patients with gastric ulcer perforation had biopsy of the ulcer which showed features of chronic gastritis without evidence of malignancy.

**Table I. Age Distribution of the Patients with Peptic Ulcer Perforation**

Age (years)	Frequency	Percentage
11-20	2	2.86
21-30	15	21.43
31-40	15	21.43
41-50	19	27.14
51-60	4	5.71
61-70	6	8.57
71-80	8	11.43
81-90	-	-
91-100	1	1.43
	<b>70</b>	<b>100</b>

**Table II. Symptoms in Patients with Peptic Ulcer Perforation**

Symptoms	Frequency	Percentage
Mid-upper abdominal pain	28	40
Generalized abdominal pain	40	57
Right lower abdominal pain	2	3
Nausea	52	74
Vomiting	39	56
Anorexia	59	84
Fever	45	64
Abdominal distension	62	87
Constipation	36	51

**Table III. Signs in Patients with Peptic Ulcer Perforation**

Signs	Frequency	Percentage
Shock	18	26
Abdominal guarding	70	100
Generalized abdominal tenderness	70	100
Rebound abdominal tenderness	67	96
Abdominal rigidity	19	27
<b>Bowel sound:</b>		
Normal	2	3
Hypoactive	12	17
Absent	56	80

**Table IV. Features of Patients with Peptic Ulcer Perforation**

Features	Number	Percentage
<b>Risk factors:</b>		
Alcohol	49	70
Smoking	20	29
Use of NSAIDS	60	86
Previous peptic ulcer disease	70	100
<b>Site of perforation:</b>		
Anterior duodenum	62	89
Gastric	8	11
<b>Size of perforation (cm):</b>		
Small: < 1	38	54.3
Large: 1-3	31	44.3
Giant: > 3	1	1.4
<b>Post-operative complications:</b>		
Wound infection	12	17.1
Wound dehiscence	9	12.9
Leak from closed perforation	1	1.4
Burst abdomen	1	1.4

Sixty (86%) patients were well and discharged after surgery while 10(14%) patients died. The average duration of hospital stay was  $12 \pm 7.0$  days. There was no case of ulcer recurrence/re-perforation over a year of follow-up. Other features of the patients are shown on the Tables I-IV.

#### Discussion

In this study, a total of 70 patients were diagnosed with perforated peptic ulcer over a 3- year period giving an average of 23 cases per year which is a high incidence compared to six cases per year reported by Nuhu et al<sup>6</sup>, four cases per year as seen in Ile-Ife, Nigeria<sup>7</sup> and 15 cases per year as reported by Ugochukwu

et al<sup>8</sup> in South-East Nigeria. Chalya et al<sup>9</sup> reported an average of 17 cases annually in Tanzania which was similar to what Schein et al<sup>10</sup> reported in South Africa. These differences may reflect differences in the rate of exposure to risk factors for perforated peptic ulcer disease from one country to another.

Out of the 70 patients with perforated peptic ulcer noted in this study, 59 (84%) were males while 11 (16%) were females. This is comparable to other studies<sup>11, 12</sup> with male predominance. This is believed to be due to the lifestyle and also the risk factors that contribute to the formation of ulcer and later perforation which men are more prone to<sup>13</sup>.

The overall mean age of the patients was  $45 \pm 17$  years in this study. Lorand et al<sup>14</sup> reported a similar mean age of  $45 \pm 16$  years in their patients with perforated peptic ulcer. Ohene-Yeboah et al<sup>15</sup> in Ghana reported a higher mean age of  $52 \pm 18$  years in their patients while Nuhu et al<sup>16</sup> reported a lower mean age of  $39.9 \pm 13.6$  years in their study in Maiduguri, Nigeria. These variations in the mean age may be attributed to the age at which the patients are exposed to the various risk factors predisposing to peptic ulcer disease and subsequently perforation.

Alcohol consumption, smoking and intake of non-steroidal anti-inflammatory drugs were noted in many of the patients in this study as shown in Table IV. Alcohol, as a noxious agent causes gastric mucosal damage, stimulates acid secretion and increases serum gastrin levels<sup>17</sup>. Smoking on the other hand inhibits pancreatic bicarbonate secretion resulting in increased acidity in the duodenal bulb and inhibition of healing of duodenal ulcer<sup>18</sup>. Sixty patients out of 70 took non-steroidal anti-inflammatory drugs in this study. Horowitz et al<sup>19</sup> found that 50% of patients with perforated duodenal ulcer had a prior history of non-steroidal anti-inflammatory drugs intake. In another study by Lanas et al<sup>20</sup>, aspirin was associated with 70% of upper gastrointestinal perforations versus 26.9% of controls ( $P=0.001$ ). Non-steroidal anti-inflammatory users are at a substantial risk for symptomatic peptic ulcer disease including bleeding and perforation.

The commonest presenting symptom in all the patients in this study was abdominal pain. Mid-upper abdominal pain was present in 28 (40%) patients, generalized abdominal pain in 40 (57%) and right lower abdominal pain in 2 (3%) patients. Gujar et al<sup>21</sup> reported in their study of 186 patients with perforated

peptic ulcer that all had abdominal pain which is in agreement with this present study. Sondashi et al<sup>22</sup> reported abdominal pain in 62.8% of their patients with perforated peptic ulcer while Christensen et al<sup>23</sup> reported abdominal pain in 84% of their patients.

Eighteen patients were in shock on admission, out of which eight died giving a mortality rate of 44%. Gujar et al<sup>21</sup> reported shock on admission in 40 (21.5%) patients out of 186 patients. Seventeen out of the 40 died giving a mortality of 42.5%. In another study by Kocer et al<sup>24</sup>, shock on admission was present in 16 (5.9%) patients out of 269 patients and morbidity and mortality in these patients were 93.8% and 68.8% respectively. Shock on admission, confounding medical illness and prolonged perforation has been found to be a useful tool in predicting outcome<sup>25</sup>.

Abdominal signs noted in the patients in this study included generalized abdominal tenderness 70(100%), guarding 70(100%), rebound tenderness 67(96%), abdominal rigidity 19(27%) and absent bowel sound in 56(80%) patients. Elfatih et al<sup>26</sup> in Khartoum reported that in more than 80% of their patients with perforated peptic ulcer, the classical presentation of sudden onset of sharp epigastric pain which later became generalized was present. They also noted that the board-like abdominal rigidity was present in 45(77.6%) out of 52 of their patients. This was attributed to the younger age group in their study (25 patients out of 52 were below 30 years) unlike in this present study where most of the patients were older with mean age of  $45 \pm 17$  years. Elderly patients with acute peritonitis are much less likely to have the classic findings of rebound tenderness and abdominal rigidity<sup>27</sup>

Chest radiograph revealed air under the diaphragm in 50(94%) patients out of 53 in this present study. This is consistent with a study<sup>28</sup> that showed that in over 70% of cases of perforated peptic ulcer, air is seen under the diaphragm on chest radiograph. Fraser and colleague<sup>29</sup> found that in perforated peptic ulcer, the shorter the duration (less than 12 hours) between perforation and radiological examination, the less the chance of positive free intra-peritoneal gas. All the patients in this study presented after 24 hours of perforation, hence the increase yield of air under the diaphragm. Abdominal ultrasound demonstrated significant free fluid in the peritoneal cavity in 52 (74%) patients. Abdominal ultrasound is useful in detecting free fluid in the peritoneal cavity, but abdominal computed tomography scan is more sensitive in detection of both fluid and minimal amount of free air which may not be detected by ultrasound<sup>30</sup>.

Sixty-two (89%) patients had duodenal perforation while 8 (11%) had gastric perforation in this study. This agrees with the report of Dakubo et al<sup>31</sup> and Bin-Taleb et al<sup>32</sup> but differs from the study of Sondashi et al<sup>22</sup> in Zambia which reported gastric perforation in 82.8% of their patients, followed by duodenal (14.3%) and only one pyloric perforation. The high numbers of gastric perforation was attributed to dietary or genetic factors.

Simple closure and omental patch without definitive anti-ulcer surgery has been the most commonly performed procedure for perforated peptic ulcer since its popularization by Graham in 1937<sup>33</sup>. Graham patch involves plugging the perforation with free omental plug. This is achieved by placing three sutures with a piece of free omentum laid over the sutures, which are then tied. No attempt

was made to close the perforation before applying the free omental plug in order not to narrow the duodenum. In this study, the perforations in all the patients were closed first with a suture; then a pedicled omentum was overlaid over it and sutured. This is different from the Graham's omental patch described above and the Cellan-Jones<sup>34</sup> technique of simple closure with pedicled omental patch. Nuhu et al<sup>16</sup> in a study of 55 patients with perforated peptic ulcer; 42 patients had simple closure with pedicled omental patch while six patients had simple closure with free graft of omentum. Four patients had leakage from closure site and no case of ulcer recurrence over 5.2 month follow-up period. Masao et al<sup>35</sup> performed laparoscopic pedicled omental patch repair in 11 patients and conventional open pedicled omental patch in four patients with perforated peptic ulcer. There was no case of leak and no ulcer recurrence over 11-month follow-up period. In this study, there was only one case of leak from the closure site which sealed on non-operative management and there was no case of ulcer recurrence/perforation over one year follow-up period. Closure of ulcer perforation with omental patch and use of proton pump inhibitors and antibiotics for eradication of *Helicobacter pylori* have virtually eliminated the need for traditional definitive anti-ulcer surgery of truncal vagotomy and drainage procedures<sup>5</sup>.

Post-operative complications noted in this study were wound infection in 12 (17%) patients, wound dehiscence in nine patients, leak from the closed perforation noticed from the abdominal drain in a patient which sealed without surgical intervention and burst abdomen in a patient. This is in agreement with a study by Gujar et al<sup>21</sup> that reported wound

infection as the commonest complication in 53 (28.49%) out of 98 patients but differs from a study by Khan et al<sup>36</sup> that reported chest infection as the commonest post-operative complication. Most of these complications occurred due to delayed presentation. The delay before surgical treatment is a strong determinant for increased complication rates and hospital costs<sup>37</sup>.

In this present study, 60 patients were well and discharged after surgery while 10 died giving a mortality rate of 14%. Mortality rates in perforated peptic ulcer vary between 4-30% in different studies<sup>38, 39</sup>. Mortality rate in this present study was similar to mortality rate reported by Jyrki et al<sup>40</sup> but differs from a higher mortality of 16.4% reported by Nuhu et al<sup>6</sup>. The higher mortality reported by Nuhu et al<sup>6</sup> was attributed to severe sepsis and electrolyte derangements. Late presentation and delayed intervention may have accounted for the mortality recorded in this present study. Also, the differences in mortality rates may also be explained by the differences in age composition of the patients and other risk factors of perforation.

### Conclusion

This study has shown that there is a high incidence of perforated peptic ulcer in our centre. It also affirms that duodenal ulcer perforation was more common than gastric perforation. Simple closure and omental patch reinforcement was a safe surgical approach in patients with perforated peptic ulcer. Additional treatment with proton pump inhibitor and treatment with antibiotics for eradication of *Helicobacter pylori* was sufficient to

prevent recurrence in the short-term. Further follow-up of the patients that survived is required to validate the long-term outcome.

### References

1. Behrman SW. Management of complicated peptic ulcer disease. *Arch Surg* 2005; 140: 201-208.
2. Mohammed HT, Din M, Shoaib AQ. Outcome of omentopexy as primary repair in perforated duodenal ulcer. *J Coll Physicians Surg* 2007; 17: 731-735.
3. Tanphiphat C, Tanprayoon T, Na Thalang A. Surgical treatment of perforated duodenal ulcer. A prospective trial between simple closure and definitive surgery. *Br J Surg* 1985; 72: 370-372.
4. Crofts T, Park K, Steele R. A randomized trial of non-operative treatment for perforated peptic ulcer. *N Engl J Med* 1989; 320: 970-973.
5. Gurleyik E. Changing trends in emergency surgery for perforated duodenal ulcer. *J Coll Physicians Surg Pak* 2003; 13: 708-710.
6. Nuhu A, Madziga AG, Gali BM. Acute perforated duodenal ulcer in Maiduguri. *The internet Journal of Surgery* 2008; 21:1
7. Lawal OO, Fadiran OA, Oluwale SF, Campbell B. Clinical pattern of perforated prepyloric and duodenal ulcer at Ile-Ife, Nigeria. *Trop Doct* 1998; 28: 152-155.
8. Ugochukwu AI, Amu OC, Nzegwu MA, Delibe UC. Acute perforated peptic ulcer: on clinical experience in an urban tertiary hospital in South-East Nigeria. *Int J Surgery* 2013; 11: 223-227.
9. Chalya et al. Clinical profile and outcome of surgical treatment of perforated peptic ulcers in North-Western Tanzania: a tertiary hospital experience. *World Journal Emerg Surg* 2011; 6: 31.

10. Schien M, Saadia R, Decker GA. Perforated peptic ulcer at the J.G. Strijdom Hospital. A retrospective study of 99 patients. *S Afr Med J* 1986; 70: 21-23.
11. Ahmed W, Qureshi H, Alam SE, Zuberi JS. Perforated duodenal ulcer; a long term follow-up. *J Pak Med Assoc* 1990; 40: 158-159.
12. Plummer JM, Melfarlene ME, Newmhan. Surgical management of perforated duodenal ulcer, the changing scene. *West Indian Med J* 2004; 53: 378-381.
13. Murty OP, Fan LY, Siang TL, binti Hasbullah NE, binti Mohd Ismail NE. Fatal gastrointestinal perforations in sudden death cases in the last 10 years at UMMC-Malaysia. *Internet Journal of Medical Update* 2007; 2: 31-36.
14. Lorand I, Mllinier N, Sales JP, Douchez F, Gayral F. Results of laparoscopic treatment of perforated ulcers. *Chirurgie* 1999; 124: 149-153.
15. Ohene-Yeboah M, Togbe B. Perforated gastric and duodenal ulcers in an urban African population. *West Afr J Med* 2006; 25: 205-211.
16. Nuhu A, Madziga AG, Gali BM. Acute perforated duodenal ulcer in Maiduguri: experience with simple closure and *Helicobacter Pylori* eradication. *West Afr J Med* 2009; 28: 384-387.
17. Turkdogan MK, Hekim H, Tuncer I, Aksoy H. The epidemiological and endoscopic aspect of peptic ulcer disease in Van region. *Eastern Journal of Medicine* 1999; 4: 6-9.
18. Stabile BE, Passaro EP. Duodenal ulcer: a disease in evolution. *Curr Probl Surg* 1984; 21: 1-79.
19. Horowitz J, Kukora JS, Ritchie WP Jr. All perforated ulcers are not alike. *Ann Surg* 1989; 209: 693-696.
20. Lanas A, Serrano P, Bajador E, Esteva F, Benito R, Sainze R. Evidence of aspirin use in both upper and lower gastrointestinal perforation. *Gastroenterology* 1997; 112: 683-689.
21. Gujar N et al. Immediate results of omentopexy in perforated duodenal ulcer: a study of 186 cases. *Al Ameen J Med Sci* 2012; 5: 29-38.
22. Sondashi KJ, Odimba BFK, Kelly P. A cross-sectional study on factors associated with perforated peptic ulcer disease in adults presenting to UTH, Lusaka. *Medical Journal of Zambia* 2011; 38: 15-22.
23. Christensen S et al. Short-term mortality after perforated or bleeding peptic ulcers among elderly patients. *BMC geriatrics* 2007; 7: 1471-1473.
24. Kocer B, Surmeli S, Solak C, Unal B, Bozkurt B, Yildirim O et al. Factors affecting morbidity and mortality in patients with peptic ulcer perforation. *J Gastroenterol Hepatol* 2007; 22: 565-570.
25. Boey J, Choi KY, Alagaratnam TT, POO A. Risk stratification in perforated duodenal ulcers. A prospective validation of predictive factors. *Ann Surg* 1986; 205: 22-26.
26. Elnagib E, Mahadi SE, Mohammed E, Ahmed ME. Perforated peptic ulcer in Khartoum. *Khartoum Medical Journal* 2008; 1: 62-64.
27. Laurell H, Hasson LE, Gunnarsson U. Acute abdominal pain among elderly patients. *Gerontology* 2005; 52: 339-344.
28. Stuart C. Perforated peptic ulcer: radiological review of 50 consecutive cases. *Journal of Medical Imaging and Radiation Oncology* 1960; 4: 32-38.
29. Frazer GM, Frazer ID. Gastrograffin in perforated duodenal ulcer and acute pancreatitis. *Clin Radiol* 1974; 25: 397-402.
30. Sofic A, Beslic S, Linceder L. Early radiological diagnostics of gastrointestinal perforation. *Radiol Oncol* 2006; 40: 67-72.
31. Dakubo JC, Naaeder SB, Clegg-Lampsey JN. Gastro-duodenal peptic ulcer perforation.

- East Afr Med J 2009; 83: 100-109.
32. Bin-Taleb AK, Razzaq RA, Al-Kathiri ZO. Management of perforated peptic ulcer in patients at a Teaching Hospital. Saudi Med J 2008; 29: 245-250.
  33. Graham RR. The treatment of perforated duodenal ulcers. Surg Gynecol Obstet 1937; 64: 235-238.
  34. Cellan-Jones CJ. A rapid method of treatment in perforated duodenal ulcer. BMJ 1929; 36: 1076-1077.
  35. Masao M, Motoharu N, Tsunekazu H, Satomi S. Laparoscopic omental patch repair for perforated peptic ulcer. Annals of Surgery 1995; 221: 236-240.
  36. Khan JS, Bhopal FG, Mehmood N, Yusuf A, Iqbal M. Perforated duodenal ulcer. A ten-year experience. Park J Surg 2001; 17: 25-28.
  37. Khursheed M, Faud, Safar H, Dashti H, Behbehani A. Laparoscopic closure of perforation duodenal ulcer. Surg Endosc 2000; 14: 56-58.
  38. Paimela H, Oksala NKJ, Kivilaakso E. Surgery for peptic ulcer today. Dig Surg 2004; 21: 185-191.
  39. Chou NH, Mok KT, Chang HT et al. Risk factors of mortality in perforated peptic ulcer. Eur J Surg 2000; 166: 149-153.
  40. Jyrki TM, Heikki K, Pasi O, Seppo OL. Factors that predict morbidity and mortality in patients with perforated peptic ulcer. Eur J Surg 2002; 168: 446-451.