# **Original Research Article**

DOI: http://dx.doi.org/10.18203/2349-2902.isj20173209

# Clinical study and management of peritonitis secondary to perforated peptic ulcer

Dushyant Kumar Rohit<sup>1\*</sup>, R. S. Verma<sup>1</sup>, Grishmraj Pandey<sup>2</sup>

<sup>1</sup>Department of Surgery, Bundelkhand Medical College, Sagar, Madhya Pradesh, India

Received: 20 June 2017 Accepted: 13 July 2017

# \*Correspondence:

Dr. Dushyant Kumar Rohit, E-mail: rythmmakers@gmail.com

**Copyright:** © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

# **ABSTRACT**

**Background:** Perforated peptic ulcer is the most common cause among all causes of gastrointestinal tract perforation which is an emergency condition of the abdomen that requires early recognition and timely surgical management. Peptic ulcer perforation is associated significant morbidity and mortality. The aim of study is to evaluate the incidence, clinical presentation, management and outcomes of the patient with peptic ulcer perforation undergoing emergency laparotomy.

**Methods:** This retrospective study includes 45 patients who were operated for perforated peptic ulcer peritonitis at Bundelkhand Medical College and Associated Hospital, Sagar from March 2015 to April 2017. Paediatric patients of age less than 14 years, patients presenting as recurrent perforation were excluded from the study. A detailed history, clinical presentation and routine investigations were done in all cases.

**Results:** In the present study, most of the patients were male. Most of these patients presents with clinical signs of peritoritis between 24-48 hours after onset of the pain. Among the patients of peptic ulcer perforation, duodenal perforation (93.3%) is more common and which is the most common cause of perforation peritoritis. The diagnosis is made clinically and confirmed by presence of gas under diaphragm on radiograph. Exploratory laparotomy with simple closure of perforation with omental patch was done in all cases. The most common post-operative complication was wound infection (57.5%). The overall mortality was 11.1%.

**Conclusions:** Late presentation of peptic ulcer perforation is common with high morbidity and mortality. Surgical intervention with Graham's omentopexy with broad spectrum antibiotics is still commonly practiced.

Keywords: Duodenal ulcer, Laparotomy, Peritonitis, Peptic perforation, Smoking

#### INTRODUCTION

Perforation is defined as a hole and break in the containing wall or membrane of an organ or structure of body. Perforation occurs when erosion, infection or other factors create a weak spot in the organ and internal pressure causes a rupture which results in peritonitis which is defined as an inflammation of the membrane which lines the inside of the abdomen and all of the internal organs; this membrane is called peritoneum. Perforated peptic ulcer is the most common cause among

all causes of gastrointestinal tract perforations which is an emergency condition of the abdomen that requires early recognition and timely surgical management.<sup>1</sup> Perforated peptic ulcer allows entry of gastric and duodenal contents into the peritoneal cavity resulting in chemical peritonitis and further bacterial contamination which leads to suppurative peritonitis. Perforated peptic ulcer is a surgical emergency and is associated with short term mortality and morbidity in up to 30% and 50% of the total patients' respectively.<sup>2</sup> The incidence of perforated peptic ulcer is approximately 7-10 per 10000 populations

<sup>&</sup>lt;sup>2</sup>Intern, Bundelkhand Medical College, Sagar, Madhya Pradesh, India

per year.<sup>3</sup> It presents as an acute abdominal condition with localised or generalized peritonitis and high risk for developing sepsis and death. Early diagnosis is essential but clinical sign can be obscured in elderly or immuno compromised patients and thus delay diagnosis. Well known precipitating factors are Helicobacter pylori infection, use of steroids, anti-inflammatory drugs, smoking, heavy and chronic alcoholic intake, trauma and gastric malignancies. The aetiology of the majority of the ulcer perforation is not known. Current use of nonsteroidal anti-inflammatory drugs has been shown to increase the risk for ulcer perforation 6-8 times and seems to account for about a quarter of the events.<sup>4,5</sup> Peptic ulcer disease considered as mucosal functional derangements due to intra luminal aggressive factors and defect in endogenous defense mechanism affecting the mucosa, these functional defects may be caused by H. pylori infection, a gram-negative helix shaped organism. A few strains of H. pylori infection were seen more frequently in patients with gastric and duodenal ulcer because it lives in an acidic environment. The role of the H. pylori infection in ulcer perforation is uncertain. In a study of patients with acute perforated duodenal ulcer the infection was as common among patients as among hospital control.<sup>6</sup> Smoking increases acid secretions, reduce prostaglandins and bicarbonate production, decreases mucosal blood flow and delay the healing of the gastric and duodenal ulcer. However smoking prevalence of 84% and 86% have been reported among patients with duodenal ulcer perforation and smoker have three-fold higher mortality from peptic ulcer perforation than non-smoker.<sup>7</sup> There is a changing trend in the occurrence of complication in peptic ulcer disease from bleeding, gastric outlet obstruction and lethal perforation of peptic ulcer which is the major life-threatening complication. These complications can occur in patients with peptic ulcers of any aetiology. Perforation occurs in about 5% and 10% of patients with active ulcer disease8. Duodenal, antral and gastric body ulcers accounts for 60%, 20% and 20% of the perforations respectively. The mainstay of management of perforated peptic ulcer peritonitis is surgery.8 Open and laparoscopic abdominal exploration is always indicated in perforated peptic ulcers, endoscopic, laparoscopic and laparoscopic assisted procedure are now increasingly being performed instead of conventional laparotomy and simple closure of perforation with omental patch.<sup>9,10</sup> Hemodynamic instability and severe signs of peritonitis make the decision for operation more urgent. With the introduction of H2 receptor antagonist, proton pump inhibitors and helicobacter pylori eradication in the management of chronic peptic ulcer disease has reduced the rate of definitive surgery for this disease though the rate of admission for acute perforation has changed little. 11 The incidence of peptic ulcer disease in normal population has declined over the past few years following a more streamlined pharmacological intervention. This can be contributed to efficiency of H2 receptor blockers and proton pump inhibitors.<sup>12</sup> Additionally, the recent advances in both diagnosis and management of peptic ulcer disease namely the improvement of endoscopic facilities, eradication of H. pylori and introduction of proton pump inhibitors eliminate the role of surgery in the elective management of peptic ulcer disease however the complications such as perforation remains a health care problem. The pattern of perforated peptic ulcer has been reported varying from one geographic area to another socio demographic and environmental factor in the developing world. The entity of peptic ulceration has changed as H. pylori infection have decreased and use of NSAIDs and cyclo-oxygenase-2 inhibitors has increased and several risk factors remain strongly associated with perforation including smoking and use of NSAIDs. Patients presenting with an acute abdomen suggestive of a perforated peptic ulcer are generally between 40 and 60 years of age. However, the number of patients over the age of 60 years has been gradually increasing. Approximately 50-60 percent of these patients have a history of peptic ulcer disease while smaller numbers have a history of use of NSAIDs. The frequency of perforated peptic ulcer is decreasing among the overall population but it is become more frequent among old people.<sup>13</sup> The spectrum of this disease in India is different from that of the western world. 14 Lower gastrointestinal perforation is more common in western countries while in India gastro duodenal perforation is the most common site for perforation peritonitis. Hence the study was undertaken to find the cause and contributing risk factor in India which affect prognosis in term of morbidity and mortality of patients.

#### **METHODS**

This retrospective study includes 45 patients who were operated for peptic ulcer perforation peritonitis in Unit III, Department of Surgery, Bundelkhand Medical College and Associated Hospital, Sagar from March 2015 to April 2017 over a period of about two years. The details of the patients were retrieved retrospectically from patient's case record kept in the Medical Record Department, Surgical Ward and Operation theatre register. The study was conducted on the basis of all the patients admitted through emergency or as an elective case from outpatient department. 45 patients with age greater than fourteen years admitted with perforated peptic ulcer peritonitis and underwent laparotomy as simple closure with omental patch were included in this study. Paediatric patients of age less than 14 years presenting as peptic ulcer perforation peritonitis, recurrent perforation, stomal ulcer perforation and who were undergo other than simple closure of perforation was excluded. The data of each patient was collected in a pro forma form designed for study and it includes the details of age, sex, duration of symptoms prior to admission, clinical presentation and investigations. A detailed history (education, occupation, socio economic status, stress, smoking, tobacco and alcohol intake, use of NSAIDs and previous history of peptic ulcer) and physical examination were carried out and routine investigations were done in all cases. Most of the patients had received no proper treatment for their illness and almost all patients had sought initial medical attention from untrained medical practitioner and only presented to us following a dramatic worsening of their symptoms of peritonitis. All patients were resuscitated with intravenous fluids, nasogastric decompression of the stomach and urethral catheterization for urinary output monitoring. Intravenous antibiotics consisting of third generation cephalosporin and metronidazole started immediately. Investigations includes complete blood picture, blood sugar, blood urea, serum electrolyte, HBsAg, HIV, chest and abdominal X-ray and abdominal pelvic ultrasound. Patients unfit for surgery were initially treated with abdominal drain under local anaesthesia as a temporary measure prior to definitive laparotomy. Upon adequate resuscitation as shown by blood pressure greater than or equal to 100mmhg systolic and urinary output more than 30ml per hour underwent exploratory laparotomy under spinal anaesthesia. A midline incision was employed. During surgery the site and size of perforation, amount and type of peritoneal contamination were noted. Perforation closed by simple closure with omental patch which later on become the Graham's patch. After the closure of the perforation, peritoneal lavage with copious volume of normal saline is done. All patients had mass closure of abdominal wall with proline number 1 suture with intra-abdominal drain left in situ (pelvic and paracolic gutter). Postoperatively all patients were put on broad spectrum antibiotics and oxygen through nasal prongs. Those patients requiring intensive care were shifted to surgical ICU. Patients were followed up every day with continued bedside monitoring of vitals in the immediate postoperative period. After satisfactory improvement, patients were discharged from the hospital with advice regarding diet, anti-ulcer drugs and quitting of smoking and alcohol. The results were analysed and compared with available published literature in the form of tables.

#### **RESULTS**

A total of 45 patients who presented with peritonitis due to perforated peptic ulcer underwent emergency laparotomy as simple closure with omental patch were studied. The patient consisted of 38 males (84.5%) and 07 females (15.6%). The frequency of peptic perforation is much greater in male as compared to female.

Table 1: Distribution of patients as per sex.

Sex	No. of cases	Percentage
Male	38	84.5%
Female	07	15.5%

The ages of the patients ranged from 16 to 75 years. The youngest patient in this study was 16 years old and oldest was 75 years old with peptic perforation. The peak incidence was in the 4th and 5th decade of life.

Table 2: Age distributions of patients.

Age (years)	No. of cases	Percentage
10-19	01	2.2%
20-29	04	8.8%
30-39	07	15.5%
40-49	12	26.6%
50-59	14	31.2%
≥60	07	15.5%

Table 3: Time interval between onset of symptoms and presentation (hours).

Time interval (hours)	No. of cases	Percentage
00-12	02	4.4%
12-24	12	26.6%
24-36	16	35.5%
36-48	06	13.3%
48-60	05	11.1%
60-72	04	8.8%

Eighty percent of the patients were from rural area and belongs to the lower socio-economic status. The duration of symptoms of perforation before presentation were few hours to 72 hours. 02 patients (4.4%) presented within 12 hours of onset of symptoms, 12 patients (26.6%) presented between 12-24 hours, 16 patients (35.5%) presented between 24-36 hours, 06 patients (13.3%) presented between 36-48 hours, 05 patients (11.1%) presented between 48-60 hours and 04 patients (8.8%) presented between 60-72 hours.

Table 4: Associated risk factors and their frequencies.

Associated risk factors	No. of cases	Percentage
Previous history of PUD	15	33.3%
Alcohol use	30	66.6%
Cigarette smoking	28	62.2%
Use of NSAIDs	12	26.6%

Table 5: Presenting symptoms in patients with peptic perforation.

Symptoms	No. of cases	Percentage
Pain in abdomen	45	100%
Vomiting	34	75.5%
Fever	21	46.6%
Abdominal distension	26	57.7%
Constipation	16	35.5%

The patients presented to the hospital within 24 hours were stable. Postoperative morbidity and mortality was less in these cases. The data reflects that early presentation to the hospital and early treatment causes less mortality and morbidity. Patients who presented after 24 hours have associated with high morbidity and mortality. There was a positive past history of chronic peptic ulcer disease in only 15 patients (33.3%). 30

patients (66.6%) and 28 patients (62.2%) gave history of intake of heavy and chronic alcohol and smoking. 12 patients (26.6%) had a positive history of ingestion of NSAIDs.

Most of the patients presented with severe upper abdominal pain, abdominal distension, vomiting, fever and constipation.

The commonest presenting symptoms were severe abdominal pain in 45 patients (100%), vomiting in 34 patients (75.5%), abdominal distension in 26 patients (57.7%). On physical examination, guarding and rigidity was present in 38 patients (84.4%) followed by abdominal distension in 36 patients (80%), abdominal tenderness in 42 patients (93.3%), absent bowel sounds in 31 patients (68.8%), shocked state (systolic blood pressure  $\leq$  90mmhg) in 34 patients (75.5%) and pulse rate  $\geq$ 120 per minute in 36 patients (80%).

Table 6: Physical signs in patients with peptic perforation.

Findings	No. of cases	Percentage
Guarding and rigidity	38	84.4%
Abdominal distension	36	80%
Abdominal tenderness	42	93.3%
Absent bowel sounds	31	68.8%
Shocked state	34	75.5%
Pulse rate ≥120/min	36	80%

In patients with suspected perforation peritonitis, X- ray chest and erect abdominal X-ray were done. In majority of cases, free gas under diaphragm seen in 36 patients out of 45 cases accounting for about 80% of the cases. 22 patients (48.8%) had abdominal pelvic ultrasound showing free peritoneal fluid typical of peritonitis. All patients had HIV and HBsAg tests done routinely and none in our study was positive.

Table 7: Frequency of presence of free gas under diaphragm.

Free gas under diaphragm	No. of cases	Percentage
Present	36	80%
Absent	09	20%

All patients underwent exploratory laparotomy through an upper midline incision after adequate resuscitation with intravenous fluids, intravenous antibiotics, nasogastric tube suction and vital signs monitoring. The urinary output ≥30ml/hour was used to ascertain adequate resuscitation. On exploration, all the patients had generalised peritonitis with varying amount of bile and pus in the peritoneal cavity. There was no tendency towards wall of perforation by omentum. Operative findings show the nature of peritoneal exudates were cloudy bilious in 24 patients (53.3%), serosanguinous in

16 patients (35.5%) and frank pus with fibrinoid adhesions in 05 patients (11.1%). None of the perforation in our study was sealed. In 11 patients (24.4%), the size of perforation was ≤5mm in diameter and in 34 patients (75.5%) it was  $\geq$ 10mm in diameter. Most common site of perforation was in the first part of the duodenum in 42 patients (93.3%) where gastric perforation present in 03 patients (6.6%). The incidence of pyoperitonium is common with late presentation. All patients had the perforation closed with vicryl 2-0 suture over a pedicle omental patch and all had copious peritoneal lavage with normal saline. Intra-abdominal drain was feet in situ before mass closure of abdominal wound with left proline number 1 suture. In postoperative period, various complications were noted. Wound infection was found to be the most common complication in patients presenting with peptic perforation wound infection was found in 26 cases (57.7%) followed by pulmonary infection in 12 patients (26.6%), intra-abdominal abscess in 04 patients (8.8%), burst abdomen in 03 patients (6.6%) and septicaemic shock was recorded in 05 patients (11.1%).

Table 8: Postoperative complication and their frequency rates.

Postoperative complication	No. of patients	Percentage
Wound infection	26	57.7%
Pulmonary infection	12	26.6%
Intra-abdominal abscess	04	8.8%
Burst abdomen	03	6.6%
Septicaemic shock	05	11.1%

The mean duration of hospital stays, in those that survived was 10 days (range 9-25 days). The patient is discharged with tablet pantoprazole, metronidazole, and amoxyclav for six weeks and to avoid alcohol drinking and smoking. The overall mortality rate in our study was in 05 patients (11.1%). The cause of death is septicaemia due to severe peritonitis.

#### DISCUSSION

Peritonitis due to perforation in the hollow viscera is commonly encountered in surgical practice. Peritonitis is caused by introduction of bile and infection into sterile peritoneum through perforation of hollow viscera. Peptic ulcer perforation is the most common cause for perforation peritonitis. 15,16 Most of the patients in our study i.e. 38 patients (84.5%) were male which is similar to other studies.<sup>8,17</sup> But most of the western studies do not find any significant sex distribution for perforated peptic ulcer. 18 Cases of perforated peptic ulcer peritonitis are on rise in females due to smoking and alcohol drinking.<sup>19</sup> Commonest age group of presentation was in 4th and 5th decade of life. This study is similar to other studies in developing countries but differ from the demographic profile from developed countries where majority of the patient are above 60 years of age.<sup>20</sup> In this study only 26.6% patients had a history of ingested NSAIDs. The

high incidence of perforated peptic ulcer occur in male in our study may be due to smoking and excessive alcohol consumption. Most patients who smoked also abused alcohol. Alcohol predispose to gastric ulceration, stimulates gastric acid secretions as well as enhancing gastrin release.21 It has been shown that the mean prevalence of H. pylori infection in patients with peptic perforation ranges from 65-70% and is significant risk factor for peptic perforation. However, study were unable to determine the presence of H. pylori infection in our study because of unavailability of reagent. Only 33.3% of patients in our study had positive past history of chronic PUD which is similar to previous studies.<sup>22</sup> It has also been shown that in many developing countries that in most cases the diagnosis of PUD is first made following perforation. The present study confirms the existence of silent PUD in majority of our patients. The lack of previous symptoms of PUD and therefore no treatment exposes most patients to a higher risk of PUD perforation. It has been reported that the time from onset of symptom of perforation to definitive treatment is a good indicator of outcomes. In the present study, most of our patients presented late more than 24 hours from the onset. Late presentation to the hospital, patient's poor general condition, old age and co-morbid conditions are factors responsible for higher mortality morbidity.<sup>23,24</sup> Pain in abdomen, vomiting, abdominal distension, fever and constipation were the predominant symptoms in our study. Pain in abdomen was seen in all the cases similar to the findings noted by Jobta RS. In the present study, majority of cases had guarding and rigidity (84.4%) at presentation. Abdominal distension in 80% patients and absent bowel sounds in 68.8%. In most of the studies conducted worldwide tenderness was present in all cases JB Baid and TC Jain found guarding rigidity in 85% cases, abdominal distension in 56% cases.<sup>25</sup> The diagnosis of peritonitis is made clinically and confirmed by presence of free gas under diaphragm which is diagnostic of perforation peritonitis but absence does not exclude the presence of perforation. This sign is visualised in 80% cases in our study. Dandaput MC and colleagues noticed gas under diaphragm in 72.35%.<sup>18</sup> Willium N and Evensen NW have reported 60-70% cases showing gas under diaphragm.<sup>26</sup> This study correlated well with the above-mentioned studies. The success of proton pump inhibitor virtually eliminated need for elective surgery. Peptic perforation becoming common in older patients associated with higher incidence of recent consumption of NSAIDs.<sup>17</sup> Significant association noted between alcohol, cigarette smoking and concomitant use of NSAIDs. All patients of peptic perforation were treated as an emergency laparotomy. At laparotomy 93.3% patients had anterior wall duodenal perforation.<sup>27</sup> The amount of peritoneal contamination was determined by size of perforation. In our study 75.5% patients had massive perforation with size ≥10mm. The nature of peritoneal exudates is also a determinant of the duration of perforation before surgical intervention. Cloudy bilious or serous exudate was seen in patients that presented earlier. Patient with intra peritoneal frank pus

represented those with a prolonged delay between onset of symptoms and surgical intervention. This was seen in 11.1% of our patients. Operative management consists of simple closure of perforation followed by omentopexy as described by Graham's.<sup>28</sup> Drain is usually removed on 3rd to 5th postoperative day or when the drainage is less than 50ml. nasogastric tube usually removed on 3rd or 4th postoperative day. Enteral feeding started on 4th postoperative day depending upon bowel sounds. All patients were given chest physiotherapy and nebulisation. Wound infection was the most common postoperative complication in our study; the reason for this was due to heavy contamination of the wound due to severe bacterial peritonitis. Other complications include pulmonary infection, intra-abdominal abscess, septicaemia and burst abdomen. The reason for these complications was 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 septicaemia in many patients and gross peritoneal soilage due to delayed presentation. The mortality in our study was 11.1% (05 patients). Jobta R et al reported mortality of 10% that is comparable with this study. 14 Worldwide literature showed that decrease in mortality of perforation peritonitis which ranges from 25% in 1940 as reported by Bakey D, Jagdish AS et al in their study reported mortality of 8.5%.<sup>29,30</sup> The cause of death was septicaemia, all of which occurred few hours-days postoperatively.

#### **CONCLUSION**

Peritonitis due to peptic ulcer perforation is more common in India in contrast to western countries were lower gastrointestinal perforation is more common. Late presentation of peptic ulcer perforation is common with high morbidity and mortality. Surgical intervention with Graham's omentopexy with broad spectrum antibiotics is still commonly practiced. Outcome is significantly affected by delayed presentation, presence of pus in the peritoneal cavity and presence of shock. Early presentation of patients with avoidance of NSAIDs, smoking and alcohol consumption and life style modification may reduce morbidity and mortality in patients with peptic perforation.

## **ACKNOWLEDGMENTS**

Authors express sincere thank to Department of Surgery, Bundelkhand Medical College, Sagar, Madhya Pradesh, India for providing the best facilities for the research work.

Funding: No funding sources Conflict of interest: None declared

Ethical approval: The study was approved by the

institutional ethics committee

### REFERENCES

- 1. Yeung KW, Chang MS, Hisiao CP. CT evaluation of gastrointestinal tract perforation. Clin Imag. 2004;28:329-33.
- 2. Moller MH, Adamsen S, Thromsen RW. Multicentric trial of a perioperative protocol to reduce mortality in patients with peptic ulcer perforation. Br J Surg. 2011;98(6):802-10.
- 3. Zinner MJ, Schwartz SI, Ellis H, Ashley SW. Maingot's Abdominal Operations 10th edition: Appleton and Lange; 1997:983-984.
- 4. Rudriguez LAG, Jick H. Risk of upper gastrointestinal bleeding and perforation associated with individual NSAIDs. Lancet. 1994;343:769-72.
- 5. Henry D, Dobson A, Turner C. Variability in the risk of major gastrointestinal complication from NSAIDs. Gastroenterol. 1993;105:1078-88.
- Reinbach DH, Cruickshank G, MC loll KEL. Acute perforated duodenal ulcer is not associated with helicobacter pylori infection. Gut. 1993;34:1344-7.
- 7. Smedley F, Hickish T, Taube M, Yale C. Perforated duodenal ulcer and cigarette smoking. JR Soc Med. 1988;81:92-4.
- 8. Goenka MK, Kochhar R, Ghosh P, Mehta SK. Changing pattern of peptic ulcer in India. J Clin Gastroenterol. 1991;13(5):57-9.
- 9. Siu WT, Chau CH, Law BK, Tang CN. Routine use of laparoscopic repair for perforated peptic ulcer. Br J Surg. 2004;91:484-4.
- Hainaux M, Agncessen SE, Berlinotti R, DC Maertelaer. Accuracy of MDCT in predicting site of gastrointestinal tract perforation. AJR An J Roentgenol. 2006;187:1179-83.
- 11. Russell RCG, Williams NS, Bulstrode CJK. Bailey and Love's Short Practice of Surgery, 23rd edition. Arnold; 2000:903-914.
- Niyaz A. 23 years of the discovery of the helicobacter pylori: Is the debate over? Ann Clin Microb Antimicrob. 2005;4:17.
- Dunn BE, Cohen H, Blaser MJ. Helicobacter pylori. Clin microbial Rev. 1997;10:720-41.
- 14. Jobta RS, Attri AK, Kaushik R. Spectrum of perforation peritonitis in India-Review of 504 consecutive cases. World J Emerg Surg. 2006;1;26.
- 15. Batra P, Gupta D, Narag R, Rao S, Batra R. Spectrum of gastro duodenal perforation peritonitis in rural central India. J MGIMS. 2013;18:44.
- Bali RS, Verma S, Agarwal PN, Singh R, Talwar N. Perforation peritonitis in the developing world. ISRN Surg. 2014:Article ID 105492:4.
- 17. Dandaput MC. Gastrointestinal perforation: Review of 340 cases. Indian J Surg. 1991;53(5):189-91.

- 18. Thorsen K, Soreide JA, Kvaloy JT, Glomsaker T. Epidemiology of perforated peptic ulcer age and Gender adjusted analysis of incidence and mortality. World J Gastroenterol. 2013;19(3):347-5.
- 19. Ng EK, Lam YH, Sung JJ, Yung MY. Eradication of helicobacter pylori prevents recurrence of ulcer after simple closure of duodenal ulcer perforation: randomized controlled trial. Ann Surg. 2000;23(12):153-8.
- Chaya LP, Mabula JBM, Mehenbe MD, Jalec HM. Clinical profiles and outcomes of surgical treatment of perforated peptic ulcer in north western Tanzania- a tertiary hospital experience. World J Surg. 2011;6:31.
- 21. Tusedogan MK, Hekim H, Aksoy H. The epidemiological and endoscopic aspects of peptic ulcer disease in van region. East J Med. 1999;4(1):6-9.
- 22. Lawal OO, Fadiran OA, Oluwale SF, Campell B. Clinical pattern of perforated pre pyloric and duodenal ulcer. Trop Doct. 1998;28:152-5.
- 23. Kocer B. Surmeli S, Solak C, Unal B. Factors affecting mortality and morbidity in patients with gastro duodenal perforation. J Gastroenterol Hepatol. 2007;22(4):565-70.
- 24. Baloch I, Bhatti Y, Shah A, Loshari AA. Our experience in 250 cases of gastro duodenal perforation. Rawal Med J. 2009;36(3):316-8.
- 25. Baid JC, Jain TC. Intestinal perforation. Indian J Surg. 1988;50:353-7.
- William N, Everson NW. Radiological confirmation of intra peritoneal free gas. Ann Royal College Surg. 1997;79(1):8-12.
- 27. Mahvi DM, Krantz SB. Stomach In; Townsend CM, Mattox KL: Sabiston textbook of surgery 19th edition Gurgaon: Elsevier: 2013:1182-1226.
- 28. Richter JE, Folk GW, Vaezi MF. Helicobacter pylori and gastroesophageal reflux disease: the bug may not be all bad. Am J Gastroenterol. 1999;93:1800-2.
- 29. Bakey D, Jordan GL. Surgical management acute gastrointestinal perforation. Am J Surg. 1961;101:317-23
- 30. Arveen S, Jagdish S, Kadambari D. Perforated peptic ulcer in South India: an institutional perspective. World J Surg. 2009;33(8):1600-4.

**Cite this article as:** Rohit DK, Verma RS, Pandey G. Clinical study and management of peritonitis secondary to perforated peptic ulcer. Int Surg J 2017;4:2721-6.