FACTORS INFLUENCING MORTALITY IN PERFORATED DUODENAL ULCER FOLLOWING EMERGENCY SURGICAL REPAIR

Subedi S K[*], Afaq A[*], Adhikary S[*], Niraula S R[*], Agrawal C S[*]

* BP Koirala Institute of Health Sciences, Dharan, Nepal.

ABSTRACT

Duodenal perforation is a common abdominal catastrophe with excellent outcome if prompt resuscitation and surgical repair of perforation are done. The aim of this study was to identify factors associated with death after surgery in patients with duodenal perforation.

One hundred and forty-five patients who underwent Graham's patch repair for perforated duodenal ulcer between 14 April 2002 and 31 December 2004 were studied.

The mean age was 45.99 years and 61 patients (42.07 %) were referrals. There were 124 (85.52 %) males and 21 (14.48 %) females. There were 10 deaths (6.9 %). The mean time delay was 2.46 days. It was 2.37 days in survivors, 3.7 days in non-survivors. The time delay was 3.25 days for females and 3.13 days for patients referred from another hospital. The mortality was significantly associated with time delay between perforation and operation ($p<0.01$), presence of co-morbid conditions ($P<0.04$), respiratory rate ($p<0.02$), raised blood urea ($p<0.01$) and serum creatinine ($p<0.001$), size of perforation ($p<0.005$), amount of peritoneal fluid ($p=0.003$) and requirement of postoperative intensive care unit support ($p=0.003$).

Time delay between perforation and operation, preoperative blood urea and serum creatinine, size of perforation and amount of peritoneal fluid, presence of co-morbid conditions and need for post operative ICU support are the important predictors of outcome after emergency surgery for duodenal perforation.

Key Words: Acid peptic disease, duodenal perforation, Graham's patch, mortality, prognostic factors.

Address for correspondence :
Dr. Shree Krishna Subedi
BP Koirala Institute of Health Sciences, Dharan, Nepal
Email: sksubedi@yahoo.com

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INTRODUCTION

Duodenal perforation is the commonest clinical condition with abdominal sepsis in tropical countries.[1] It has an excellent outcome with early resuscitation and surgical repair of perforation. Delay in surgery leads to various complications and even death. Surgery for perforated peptic ulcer is associated with a mortality rate usually around 5-20 per cent,[1-7] but ranging from as low as 2.3 per cent to as high as 66.7 per cent according to the absence or presence of risk factors.[3] A number of factors are associated with increased mortality, including advanced age,[1,9] delayed surgery,[1,9] shock at presentation[1,3,7] and presence of co-morbid conditions.[1,2,4,8,9] The aim of the present study was to identify factors associated with death in a series of patients operated for duodenal perforation.

PATIENTS AND METHOD

Study design

One hundred and forty-five consecutive patients of perforated duodenal ulcer, who had undergone emergency laparotomy and repair of perforation with omental patch in B. P. Koirala Institute of Health Sciences between 14 April 2002 and 31 December 2004, were reviewed from medical record. The various factors studied were age, sex, etiology (smoking, alcohol intake, and history of use of nonsteroidal anti-inflammatory drugs, past history of acid peptic disorder), time delay between perforation and operation, presence of shock and dehydration at presentation, pulse rate, respiratory rate and blood pressure at the time of triage. The preoperative laboratory investigation included total leukocyte count, hemoglobin, blood urea, serum creatinine, random blood sugar, abdominal and chest x-ray and blood grouping. Serum amylase, arterial blood gas analysis and culture sensitivity of peritoneal fluid were done only in selected patients. The operative findings studied were size of perforation, amount and character of fluid in the peritoneal cavity. Presence of co-morbid condition and need for postoperative intensive care unit (ICU) support were also noted.

Management of patients

All patients were initially resuscitated with intravenous (IV) fluids and IV antibiotics were administered in the absence of proven peritoneal contamination. The patients were operated as soon as they were fit for general anesthesia. Twenty one (14.5 %) patients required post operative intensive care unit support; other patients were managed postoperatively in surgical wards.

Statistical analysis

Patients were grouped as hospital survivors and non-survivors to identify the factors associated with mortality. Non parametric tests; Chi-square (y^2) and Fisher's exact probability tests were used as measures of independence for categorical data and the Mann-Whitney U test was used for ordinal data. Probability value less than 0.05 was considered statistically significant.

RESULTS

There were 145 patients with the age ranging from 4 years to 76 years. The mean (± standard deviation) age was 46.0 (±13.4) years. Around 86% of patients were male. Sixty-one (42.1 %) patients were referred from another hospital. There were ten deaths resulting in an overall mortality rate of 6.9 %. Seven patients died due to septicaemic shock, two died due to disseminated intravascular coagulation and one died due to cardio-respiratory arrest secondary to pneumonia. Table I shows different variables that were analyzed for difference between survivors and non-survivors.

The mean age (45.6 years) in survivors was comparable with that in non-survivors (50.5 years) (p=0.403). The overall mean time from onset of symptoms to surgery was 2.45 days; it was significantly longer in those who died (3.7 days) than in those who survived (2.4 days) (p=0.008). Although females were at higher risk for death (p=0.031), they had significantly longer time delay (3.2 days) between perforation and operation as compared to males (2.3 days) (p=0.019). Eight of 61 referrals from another hospital died compared to 2 of 84 patients who were admitted directly to this hospital (p=0.018). The mean (±SD) time delay in referrals was 3.1 (±2.2) days and that in patients admitted directly was1.98 (±1.69) days (p=<0.001). Raised blood urea and serum creatinine were strongly associated with mortality (p=0.007 and 0.001 respectively). The mean (±SD) size of perforation

Table I: Clinical, laboratory and operative findings in patients who survived and patients who died (n=145)

	Survivors (n=135)	Non-survivors (n=10)	Total (n=145)	P
Mean age (years)	45.6 ±(13.5)	50.5 ±(11.8)	46.0 ±(13.4)	0.403
Sex ratio (M:F)	118:17	6:4	124:21	0.031
Referred from another hospital	53	8	61	0.018
Admitted directly	82	2	84	
Mean time delay (days)	2.4 ± (2.0)	3.7±(1.4)	2.5 ±(2.0)	0.008
Delay >24 hours	80	10	90	<0.001
Alcohol intake	58	2	60	0.196
Smoking	78	8	86	0.196
Past history of				0.601
APD	79	7	86	
Yes	44	3	47	
No	12	0	12	
Unknown				
History of NSAID intake	11	1	12	0.591
Shock at presentation	25	4	29	0.113
Dehydration at presentation	93	9	102	0.282
Co-morbid diseases	42	7	49	0.031
Mean pulse rate	95 ±(17)	105 ±(24)	96 ±(18)	0.201
Mean respiratory rate	24 ±(6)	28 ±(5)	24 ±(6)	0.015
Mean TLC (per mm^3)	12393 ±(4113)	11130 ±(6007)	12306 ±(4093)	0.522
Urea (mg%)	55.8±(40.5)	101.9 ±(57.7)	59.0 ±(43.3)	0.007
Urea >50	57	9	66	0.006
Creatinine (mg%)	1.1 ± (0.6)	2.1 ±(0.1)	1.2 ±(0.7)	0.001
Creatinine >1.4	30	8	38	<0.001
Haemoglobin (gram%)	13.8±(2.5)	13.1 ±(2.8)	13.8 ±(2.5)	0.369
RBS (mg%)	103.8 ±(30.5)	90.5 ±(41.3)	102.9 ±(31.4)	0.219
Size (mm)	7.6 ±(5.6)	10.8 ±(4.3)	7.8 ±(5.6)	0.004
Size >10 mm	39	7	46	0.012
Fluid (litres)	1.34 ±(1.0)	2.25 ±(1.13)	1.42 ±(1.05)	0.003
Fluid >2 litres	50	9	59	0.001
Postoperative ICU support	17	4	21	0.031

APD, acid peptic disease; NSAID, non steroidal anti-inflammatory drugs; RBS, random blood sugar, TLC, total leukocyte count.

was 7.8(±5.56) mm. It was larger (10.8 mm) in patients who died than in patients who survived (7.58mm) (p=0.004). Seven of 10 non survivors had size more than 10 mm as compared with 39 of 135 survivors (p=0.012). The mean amount of peritoneal fluid was 1.34 liters in survivors and 2.25 liters in patients who died (p=0.003). Fifty of 135 survivors and 9 of 10 patients who died had fluid more than 2 liters (p=0.001). Purulent contamination of peritoneal cavity and /or adhesion was present in 17 survivors and two non-survivors. Postoperative ICU support was required by 17 of 135 survivors and 4 of 10 who died (p=0.031).

History of smoking was present in 86 (59.31 %) patients and that of alcohol intake was present in 60 (41.38 %) patients. Twenty nine (20 %) patients had shock and 102 (70.34 %) patients had dehydration at the time of presentation. Alcohol intake, smoking, NSAID intake, past history ofAPD, shock and dehydration at presentation, pulse rate, total leukocyte count, hemoglobin level, random blood sugar and serum amylase had no influence in mortality. Serum electrolytes (Na”” and K””) were obtained from 89 patients and they were not important for mortality.

DISCUSSION

With the availability of effective drugs for peptic ulcer disease the incidence of peptic ulcer perforation is decreasing in many parts of the world.[10] Simple closure

of perforation is effective[11,12] and has been the traditional therapy for perforated ulcer.[13] In highly selected patients (in whom the perforation seems to have sealed), non-operative therapy may be appropriate.[14] In recent years laparoscopic repair of the perforation is becoming more popular. This is partly because morbidity has been reported to be less in laparoscopic closure due to minimal access required.[15,16] But in our setup laparatomy with peritoneal lavage and closure of perforation with omental patch is the usual mode of treatment for duodenal perforation till date.

There is a wide variation in outcome after surgery for perforated peptic ulcer depending upon the type of patient as well as the type of surgery (laparoscopic or open).[5,17-19] The mortality rate in our study was 6.9%. Previous studies have described age to be the important predictor of mortality. But we did not find any significant association between age and mortality. One recent study[20] in F344 rats demonstrate that aged small bowel mucosa exhibits a proliferative and adaptive capacity in response to small bowel resection that was similar to that of the young animals. It is possible that increased mortality associated with age due to various factors associated with age than age itself. Time delay for surgery has been shown to affect outcome.[1-9] In a prospective study, S Robinson Smile et al.[3] showed that there was a 1.75 fold increase in the risk of death in those who underwent late (>24 hours) surgery. This has been supported with prospective[1,9] as well as retrospective[7] studies. Wakayama et al.[2] found that time delay more than 12 hours to be important. In the present study the mean time to surgery was significantly longer in non-survivors. The mortality associated with female gender and referrals was largely because they has significantly longer time delay between perforation and operation.

In contrast to various studies,[1-3,7] our study did not find preoperative shock to be important prognostic factor. We agree with previous studies[1,2,4,8,9] that comorbid conditions are important predictors of mortality.

Three factors namely systolic blood pressure, heart rate and hemoglobin level used in Jabalpur prognostic scoring system were not found to be important prognostic indicators whereas respiratory rate was. Surprisingly, some previous studies assessing surgical outcomes from duodenal perforation have not specifically addressed serum creatinine.[3,8] but our study supports the sutdies[1,21] showing serum creatinine to be a predictor of outcome. Wakayama et al[2] found blood urea to be associated with poor outcome whereas S Robinson Smile et al.[3] found blood urea not significant. In our study blood urea was found to be significantly associated with mortality. Need for postoperative ICU support and its association with mortality has not been reported in previous studies. We found that there was a greater need for postoperative ICU support in patients who died. In conclusion, patients with duodenal perforation who died after surgical repair were operated late, had higher blood urea and serum creatinine level. They had larger size of perforation and more fluid in peritoneal cavity .There was a greater need for postoperative ICU support for them.

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