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A Clinical Study of Duodenal Ulcer Perforation

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Abstract:
Background*: Perforation of the duodenal ulcer is one of the commonest and lethal complications of chronic duodenal ulcer. 2-10% of peptic ulcer may perforate. Unless prompt diagnosis is made and early active surgical management is done the mortality rate is very high upto10%. It is the commonest cause of death resulting from surgical abdominal emergency next to intestinal obstruction only. Despite this and recent advances in both diagnosis and management of peptic ulcer disease, namely the improvement in endoscopic facilities, eradication of H. pylori and the introduction of the proton pump inhibitors, complications such as peptic ulcer perforation remain a substantial healthcare problem. This may be due to an increase in the risk factors for peptic ulcer complications. With this statistics , a study for clinical presentations, complications and incidence of peptic ulcer perforation was carried out to know the trends in population of the region .*
Materials and Methods*: Prospective study was conducted for period of 2 years from September 2015 to August 2017 on patients admitted in the Surgical Wards of Regional Institute of Medical Sciences, Imphal, with the diagnosis of duodenal ulcer perforation. Data was collected on various variables like age and sex incidence, occupation, clinical features, association with smoking and alcohol, investigations, size and site of perforation, post-operative complications and condition at follow-up.*
Results*: Of the 110 patients studied 106(96.3%) were males and 41-50 years was the most common age group. Majority (80%) belong to laborious workers commonly associated with alcohol intake and smoking. Pain was the most consistent symptom while guarding (89.1%), tenderness (81.8%) and obliteration of liver dullness(76.4%)were the most important signs present. Gas under the diaphragm was present in 97.3% of patients. Pre-operative shock, old age, longer duration of perforation, concurrent medical illness and higher grade of peritoneal contamination are the main factors affecting the morbidity and mortality in duodenal ulcer perforation. Mortality rate was 6.4% in this study. Simple closure with Graham's omentopexy followed by proton pump inhibitor drugs is an effective treatment procedure for duodenal ulcer perforation. H. pylori eradication after simple closure may be necessary to prevent recurrence of ulcer.*
Key Word*: Duodenal ulcer, Duodenal ulcer perforation, Pneumoperitoneum, Graham's omentopexy, H.pylori*

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I. Introduction

Perforation of the duodenal ulcer is one of the commonest and lethal complications of chronic duodenal ulcer. Unless prompt diagnosis is made and early active surgical management is done the mortality rate is very high. It is the commonest cause of death resulting from surgical abdominal emergency next to intestinal obstruction only.[1]

After Mikulicz first sutured a perforated duodenal ulcer in 1887, Hansen achieved the first successful operation. The sudden release of gastric or duodenal content into the peritoneal cavity through a perforation can lead to a sequence of events which, if not properly managed, is likely to cause death. In spite of development in both diagnosis and treatment of peptic ulcer disease, the incidence of perforation seems to be unchanged and even increased in some reports in older age groups.[1]

Mortality is influenced by patient's age, site of the ulcer, treatment delay, concurrent disease, preoperative shock and type of anaesthesia used. A majority of factors are interrelated, for instance, treatment delay seems to increase the mortality. Peptic ulcer is one of the commonest structural disorders of gastrointestinal tract. Though predominantly occurring in the stomach and duodenum, they are reported to occur elsewhere in the gastrointestinal tract, too. In order to call the defect chronic peptic ulcer, it must involve the full thickness of the mucosa reaching muscularis mucosa.[2]

Before the 19th century, peptic ulceration was uncommon, be it in the East or West. The first gastric ulcer described in human history probably belongs to a Chinese man who died 2000 years ago in the Western Han Dynasty from perforated gastric ulcer and whose well-preserved body was recently discovered in Ginzhou.

In the English literature, the pathology of gastric ulcer was first described in 1935 by Jean Cruveilhier. In those days, gastric ulcers were occasionally seen and duodenal ulcers were rare. At the turn of the 20th century, peptic ulcer, in particular duodenal ulcer rose to become one of the commonest medical conditions in Western countries, affecting 10% of men in their lifetime. In the East, its occurrence is equally common, and its prevalence has also been documented to be 10-11%. Midway through the 20th century, however, the incidence of peptic ulcer started to fall in Western and developed countries, while that in Asian countries such as Hong Kong and Singapore continued to rise, so that the frequency of perforated peptic ulcer in Hong Kong, for example, had been estimated to be five times that in New South Wales. However, in the past decade, although there has been no documented formal report in Asian countries, it is generally noted that the incidence of peptic ulcer has also been falling. At the same time in the West and especially in USA, the documentation of non-H. pylori, non-NSAID peptic ulcer appears to be on the increase, having been described as being in the region of 30% of all ulcers seen. Such ulcers appear to be much less common in Asia.[3]

However, in the recent years, globally, the incidence of peptic ulcer disease has fallen. Despite this and recent advances in both diagnosis and management of peptic ulcer disease, namely the improvement in endoscopic facilities, eradication of H. pylori and the introduction of the proton pump inhibitors, complications such as peptic ulcer perforation remain a substantial healthcare problem. This may be due to an increase in the risk factors for peptic ulcer complications.[4]

Peptic ulcer perforation affects almost 2-10% of peptic ulcer patients on the average. It presents with an overall mortality of 10% although some authors have reported between 1.3% and 20%. Being a life threatening complication, it needs special attention with prompt resuscitation and appropriate surgical management if morbidity and mortality are to be minimised. The pattern of perforated Peptic Ulcer Disease has been reported to vary from one geographical area to another depending on the prevailing socio-demographic and environmental factors. In the developing world, the patient population is substantial healthcare problem. This may be due to an increase in the risk factors for peptic ulcer viz. young male patients, late presentation and smoking. In the west the patients tend to be elderly and there is a high incidence of ulcerogenic drug ingestion. The diagnosis of perforated Peptic Ulcer Disease poses a diagnostic challenge in most of cases. The spillage of duodenal or gastric contents into peritoneal cavity causing abdominal pain, shock, peritonitis, marked tenderness and decreased liver dullness offers little difficulty in diagnosis of perforations. The presence of gas under the diaphragm on plain abdominal erect X-ray is diagnostic in 75% of the cases. Delay in diagnosis and initiation of surgical treatment of perforated Peptic Ulcer Disease has been reported to be associated with high morbidity and mortality after surgery for perforated Peptic Ulcer Disease. A successful outcome is obtained by prompt recognition of the diagnosis, aggressive resuscitation and early institution of surgical management.[4]

Chronic gastric and duodenal ulcers may be indistinguishable. The common complications of peptic ulcers are perforation, bleeding and fibrosis which may lead to stenosis in case of duodenal ulcer. The treatment of the perforated peptic ulcer is primarily surgical, some patients may be managed conservatively.[5]

Although chronic duodenal ulcer is so prevalent in local population, no remarkable work has been done so far to find out the incidence and clinical features in the population of this region. Lots of literatures are available about the disease but all the studies have been done in populations which are quite different from the people of this region in their life styles and environment.

Hence the present study was taken up to get some firsthand knowledge of chronic duodenal ulcer in general and duodenal ulcer perforation in particular. We also tried to get some idea, during the course of study about the outcomes of treatment and factors influencing it.

II. Material And Methods

This prospective study was carried out on patients admitted in the Surgical Wards of Regional Institute of Medical Sciences, Imphal, with the diagnosis of duodenal ulcer perforation**.**

Study Design: Prospective cohort study.

Study Location: This was a tertiary care teaching hospital based study done in Department of General Surgery, RIMS, Imphal, Manipur.

Study Duration: September 2015 to August 2017.

Sample size: 110 patients.

Sample size calculation: Prevalence of duodenal perforation as per previous studies is 10.[3,4,6]

$$N = \frac{4\,P(100-P)}{e^2}$$

N=samplesize, P= prevalence, e= standard error

$$N = \frac{4x10(100-10)}{5^2}$$

N=144 patients

Although the calculated sample size was 144 patients, 110 cases of duodenal ulcer perforation fulfilled the inclusion criteria and were studied during the period of study.

Subjects & selection method: A convenience sampling of all patients diagnosed with duodenal ulcer perforation admitted in Surgical Wards of RIMS, Imphal, during the period of study was done.

Inclusion criteria:

1. Patients presented with duodenal ulcer perforation were included, irrespective of age, sex or ethnicity.

Exclusion criteria:

1. Perforation in the gastrointestinal tract other than duodenum.
2. Traumatic perforation.
3. Pregnant patients.
4. Patients not willing to participate in the study.

Procedure methodology

1. The initial diagnosis of the duodenal ulcer perforation was made from the history, clinical findings supported by radiological findings and it was confirmed at laparotomy. Ultrasound and CT-Scan were used for inconclusive suspected cases and observation of progress and complications.
2. The following data were collected on admission to correlate the clinical parameters before surgery.
   a. Age, sex and socio-economic status of the patient.
   b. Antecedant history of ulcer of less than those of 3 months was classified as acute ulcer and more than 3 months prior to perforation were classified as chronic duodenal ulcer.
   c. Presence of ulcerogenic factors like consumption of alcohol, NSAIDS, Steroids and smoking.
   d. Presence of co-existing illness.
   e. Duration of symptom of perforation from onset of acute pain abdomen to operative intervention.
   f. Pre – operative shock (systolic blood pressure of less than 90mmHg).
3. Routine investigations included complete haemogram, blood grouping, serum urea, serum creatinine, electrolytes (Na+ and K+), urine and blood sugar random.
4. Pre-operavtive management included nasogastric suction, resuscitation with adequate intravenous fluids (crystalloids and colloids if required), intravenous anti ulcer drugs (proton pump inhibitors), analgesics and appropriate antibiotics parenterally. Adequate hydration was indicated by an hourly urine output of more than 30ml/hour.
5. All cases were managed surgically. Exploratory laparotomy through an upper midline incision was made. Site, size, induration in and around the perforation and peritoneal contamination were noted.
6. Simple closure of perforation by placing through and through stitches using 2-0 atraumatic polyglycolate suture and reinforcement with pedicled omental patch (Graham's omentopexy) was done. Peritoneal lavage with copious amount (3 to 4 litres) of normal saline was done. A tube drain (intraperitoneal) at the right flank was kept in all patients regardless of the size of perforation and degree of peritoneal contamination. The operations were performed either by consultants or senior residents under the supervision of consultants.
7. Peritoneal contamination was graded from -0 to IV. (Horowitz et al).[53]

Grade 0: No evidence of contamination
Grade I: Cloudy fluid.
Grade II: Fibrinous exudate.
Grade III: Free pus or major contamination.
Grade IV: Abscess.

8. Post- operatively the patients were kept nil by mouth until the return of their bowel activity, till then they were given intravenous fluids, injectable antibiotics with broad spectrum coverage and injectable analgesics along with injectable pantoprazole 40mg once a day. As and when required patients were given blood transfusion.

9. Nasogastric drains removed routinely on fourth postoperative day when bowel sound returned and intraperitoneal tube drains removed on fourth to fifth postoperative day routinely or when the output was less than 50mL per day.
10. Post operative morbidities, complications either systemic or local were recorded.
11. Mortality-postoperative hospitals deaths were recorded.
12. As facilities to study H. pylori are not available in our institute, H. pylori assay was not done.
13. On discharge, all patients were given anti ulcer drugs, proton pump inhibitors, for faster healing and prevention of recurrence. Patients with persistent peptic ulcer symptoms were given empirically anti-H. pylori triple drugs regime. Patients were followed up after discharge on 2 weeks, 1 month and thereafter monthly for a minimum period of 3 months.

Statistical analysis

The observations of the study were recorded in data base programme SPSS (IBM) version 21. The descriptive statistics like mean, percentages, proportions were used to analyse the association between duodenal ulcer perforation and socio demographic variables like age, sex, occupation etc. using pearson's chi-square test.(p value of < 0.05 was taken as significant).

III. Result

Age:

Altogether 110 patients were studied. The age ranged between 18 to 80 years with the mean age of 48.86 years. The highest number of patients belonged to the age group of 41 to 50 years with 38(34.5%) patients, followed by the age group of 51 to 60 years with 26(23.6%) patients. The youngest patient was 18 years and oldest was 80 years.

Sex:

Out of 110 patients there were 106 males and only 4 females. Majority of females belonged to older age groups. Table1. Shows the incidence of female were more in the age group of 71-80 years. In all other age groups there was male preponderance.

Table1. Age and sex wise distribution.

N=110

Age Groups (in years)	Sex		Total	Percentage
	Male	Female		
11-20	2	-	2	1.8%
21-30	9	-	9	8.2%
31-40	18	-	18	16.5%
41-50	37	1	38	34.5%
51-60	26	-	26	23.6%
61-70	11	1	12	10.9%
71-80	3	2	5	4.5%
Total	106	4	110	100%

Table1. Shows the incidence of female were more in the age group of 71-80 years. In all other age groups there was male preponderance.

Religion :

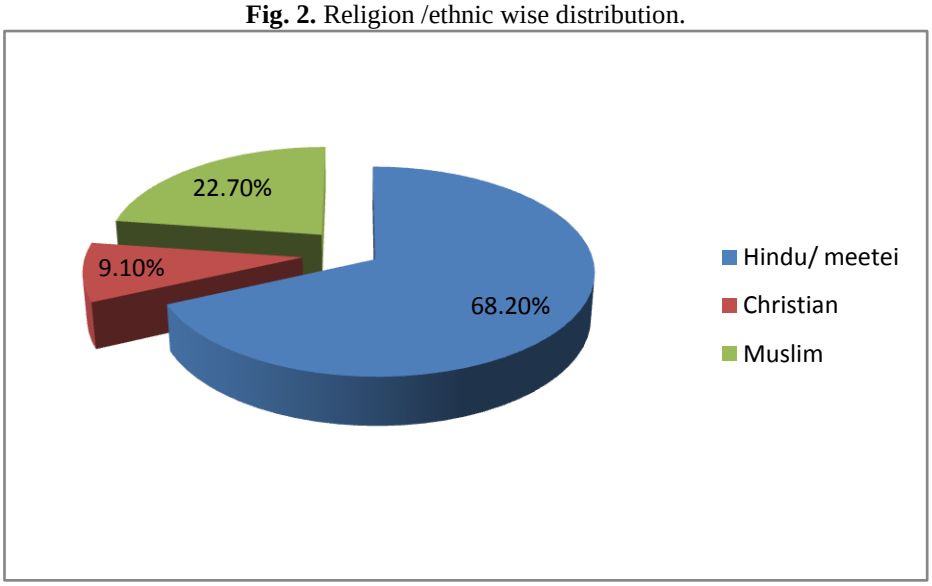
Table 2. Religion wise distribution of duodenal ulcer perforation.

N =110

Religion	No. of cases	Percentages
Hindu/ meetei	75	68.2%
Christian	10	9.1%
Muslim	25	22.7%
Total	110	100%

Table 2, shows that perforated duodenal ulcer was commonest amongst the Hindu/Meetei with 75 patients accounting for 68.2 % followed by Muslim 22.7(%) and Christian 9.1(%).

Fig. 2. Religion /ethnic wise distribution.



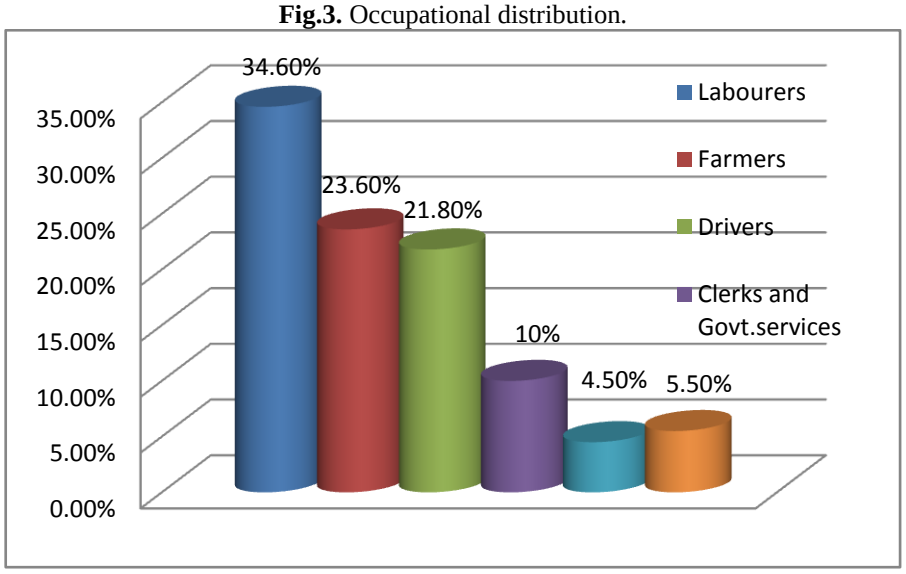
Occupation:
Pereforation of duodenal ulcer was more in people engaged in active physical works (Group A) than in those engaged in skilled labours or sedentary works (Group B).

Table 3. Incidence of perforation with different occupations.
N=110

	Occupation	**No. of cases**	**Percentage**
A. Labourious worker (unskilled+semiskilled)	Labourers	38	34.6%
	Farmers	26	23.6%
	Drivers	24	21.8%
B. Desk-worker(skilled workers)	Clerks and Govt. services	11	10%
	Students	5	4.5%
	Others	6	5.5%
Total		110	100%

According to the present study the highest incidence was among patients involved in active physical works (80%).

Fig.3. Occupational distribution.



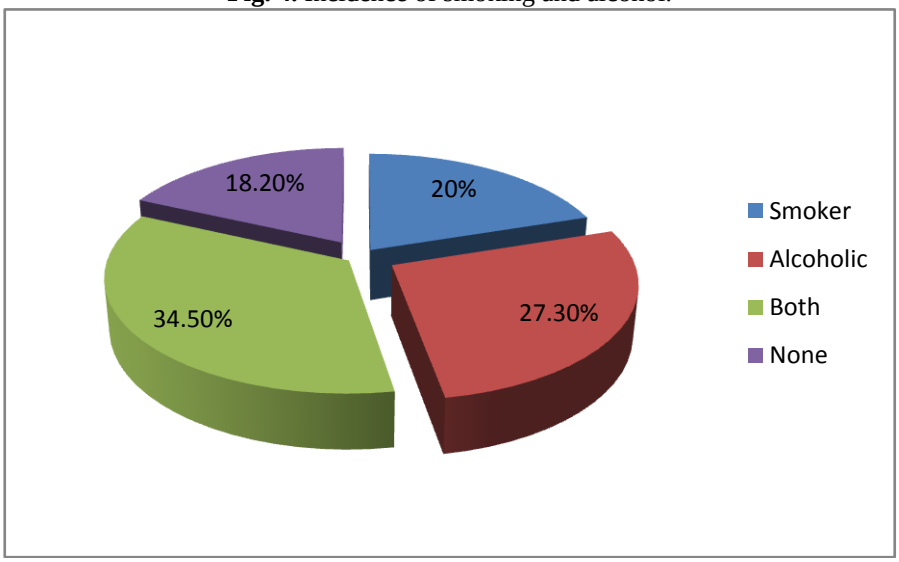
Smoking and Alcohol:

Table 4. Association with alcohol and smoking.
N=110

Habits	No. of patients	Percentages
Smoker	22	20%
Alcoholic	30	27.3%
Both	38	34.5%
No addiction to smoking and alcohol	20	18.2%

Table4. Shows the prevalence of duodenal ulcer perforation was high in both smokers and alcoholics.

Fig. 4. Incidence of smoking and alcohol.



Dietary habits:

Table 5: Dietary habits of all patients studied.

Dietary habit	Female n=4	Male n=106	Total n=110
Vegetarian	-	10 (9.4%)	10 (9.1%)
Non-vegetarian	4 (100%)	96 (90.6%)	100 (90.9%)
Spicy Food	4 (100%)	80 (75.5%)	84 (76.4%)
Bland Food	-	26 (24.5%)	26 (23.6%)
Irregular dietary habit	4 (100%)	100 (94.3%)	104 (94.5%)
Regular dietary habit	-	6 (5.6%)	6 (5.5%)

The above table 5 shows that all maximum patients were non vegetarian 90.9%, and perforation was high among those who take spicy foods, 76.4% and 94.5% of patients gave history of irregular dietary habit.

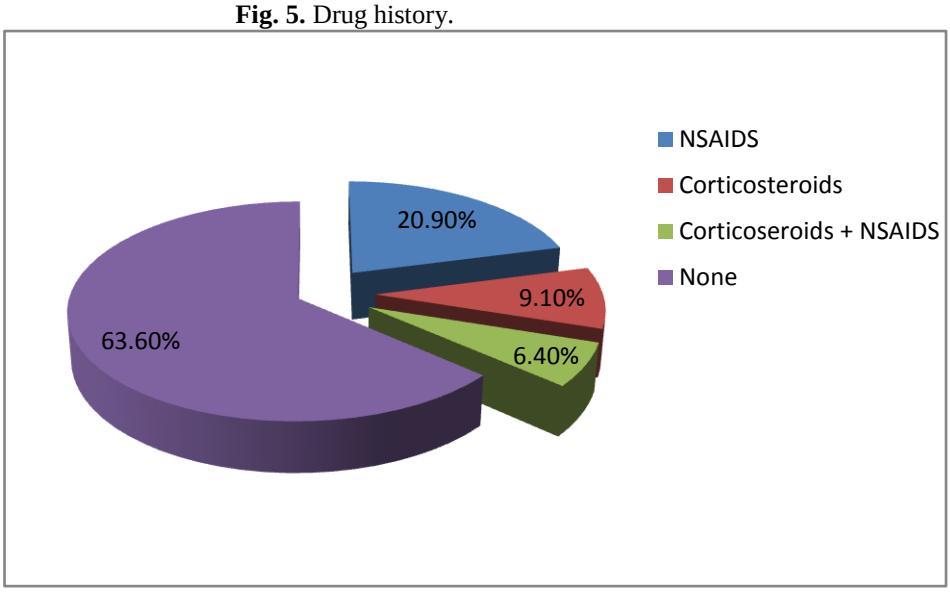
Drug history:

Table 6. Drug history of all patients with duodenal ulcer perforation.
N=110

Nature of drug	No. of cases	Percentage
NSAIDS	23	20.9%
Corticosteroids	10	9.1%
Corticoseroids + NSAIDS	7	6.4%
No History of drug intake	70	63.6%
Total	**110**	**100%**

In this study history of drug ingestion in form of NSAIDS or corticosteroids was seen in upto 36.4% of patients for various conditions like toothache, arthritis, or as antipyretics few days before perforation.

Fig. 5. Drug history.



Seasonal variation:

Table 7. Seasonal incidence of duodenal ulcer perforation.

N=110

Periods /months	No. of patients	Percentage
February to May	32	29%
June to September	28	25.5%
October to January	50	45.5%
Total	**110**	**100%**

In the present study the highest incidence of duodenal ulcer perforation occurred during October to January (45.5%), followed by February to May (29%).

Diurnal variation:

Table 8. Perforations occurring in different timings of day.

N =110

	No. of patients	Percentages
Early morning	30	27.3%
Noon	8	7.3%
Evening	20	18.2%
Night	52	47.2%
Total	**110**	**100%**

In this study maximum patients had onset of pain at night (47.2%), followed by early morning (27.3%).

Blood group:

Table 9. Blood groups of patients.

N=110

Blood group	No. of cases	Percentages
A	20	18.2%
B	25	22.7%
AB	11	10%
O	54	49.1%
Total	**110**	**100%**

Maximum incidence of duodenal ulcer perforations was seen in patients with blood group O (49.1%). The "O" group happens to be the group where duodenal ulcer is very common.[17]

Family history:

Only 8(7.3%) patients gave history of peptic ulcer disease but no patient gave history of duodenal perforation in immediate relatives.

CLINICAL FEATURES:

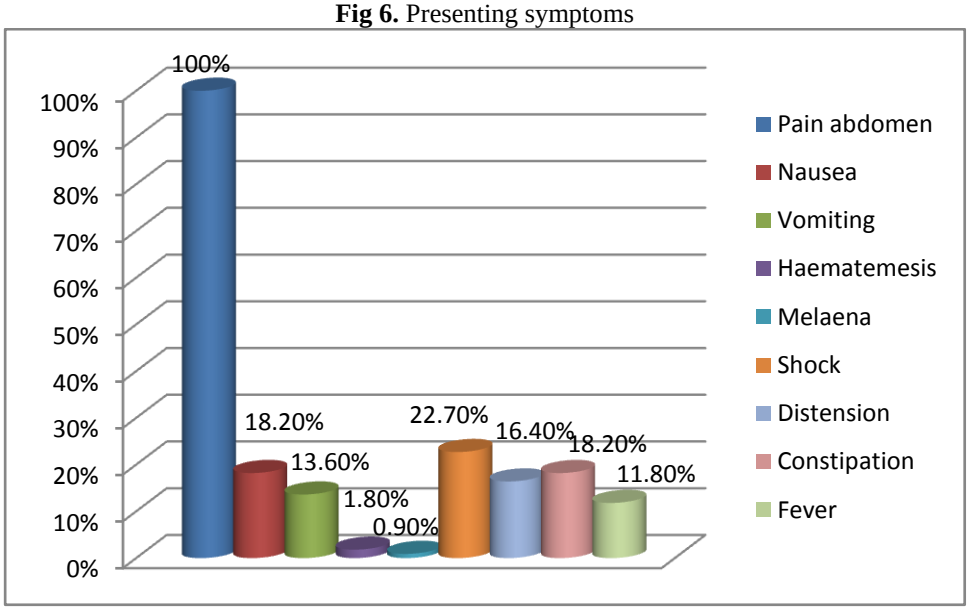
Symptoms of presentation:

Pain abdomen was the most consistent symptom present in all 110 cases. Nausea, vomiting, constipation and fever were the other symptoms. 22.7% of patients presented with features of shock symptoms, cold clammy skin, sweating and fainting attack.

Table 10. Presenting symptoms.
N=110

Symptoms	No. of cases	Percentage
Pain abdomen	110	100%
Nausea	20	18.2%
Vomiting	15	13.6%
Haematemesis	2	1.8%
Melaena	1	0.9%
Shock	25	22.7%
Distension	18	16.4%
Constipation	20	18.2%
Fever	13	11.8%

Fig 6. Presenting symptoms



Duration of Perforation:

Period of time from the onset of symptoms of perforation to the time of reporting to hospital was recorded. Every patient was prepared for operation as soon as the diagnosis was made and taken to the operating room after resuscitation.

Table 11. Time of reporting at hospital after perforation.
N =110

Time of reporting (in hours)	No. of cases	Percentage
0-6	12	10.9%
6-12	36	32.7%
12-24	42	38.2%
24 hrs & after	20	18.2%
Total	**110**	**100%**

Large group of patient presented late. Thirty six (32.7%) patients were operated upon with duration of perforation from 6-12 hours, 42(38.2%) patients were operated upon with duration of 12-24 hours and 20 (18.2%) patients were operated upon with duration of perforation more than 24 hours, increased mortality and morbidities were seen in this group.

Antecedant Ulcer History:

Table 12. History of peptic ulcer recorded in patients.

N=110

Previous history of peptic ulcer	**No. of patients**	**Percentages**
Antecedent ulcer history >3months	48	43.6%
Antecedent ulcer history <3months	20	18.2%
Absent	42	38.2%
Total	**110**	**100%**

Table12. Shows that 61.8% of patients in the present study had previous history of peptic ulcer in the form of epigastric pain, melaena, vomiting and treatment history. 38.2% of patients had no dyspepsia and ulcer history prior to perforation.

CLINICAL EXAMINATION:

General Physical Examination

Table 13. Shows the various findings in patients of duodenal ulcer perforation on clinical examinations, after onset of generalized pain and admission to the hospital.

N=110

Signs	No. of patients with percentages			
	Within 6 hrs.	6-12hrs.	12-24hrs.	After 24 hrs.
Pulse				
<100/min	11 (10%)	30(27.3%)	14(12.7%)	2(1.8%)
>100/min	1 (0.9%)	6(5.4%)	28(25.5%)	18(16.4%)
Blood pressure				
>90mmHg	12 (10.9%)	31(28.2%)	35(31.8%)	8(7.3%)
<90mmHg	-	5(4.5%)	7(6.4%)	12(10.9%)
Temperature				
Febrile	-	-	5 (4.5%)	8(7.2%)
Afebrile	12(10.9%)	36(32.7%)	37(33.6%)	12(10.9%)
Resp. Rate				
<30/min	12(10.9%)	35(31.8%)	40(36.4%)	16(14.5%)
>30/min	-	1(0.9%)	2(1.8%)	4(3.6%)
Hydration				
Fair	12(10.9%)	34(30.9%)	24(21.8%)	4(3.6%)
Dehydration	-	2(1.8%)	8(7.3%)	16(14.5%)

Tachycardia with pulse rate more than 100/min was noted in 41.9% of cases presented after 12 hours after onset of pain. Hypotension was said to be present if systolic blood pressure was less than 90mmHg and was recorded in 24 cases. Patient was regarded to have fever when body temperature was more than 100^0F and was noted in 12.7% patients.

Respiratory rate of more than 30/min was seen in 7(6.3%) of cases.

State of hydration was assessed and dehydration was seen in 26(23.6%) of cases.

Findings suggest that incidence of tachycardia, hypotension, tachypnea, fever and dehydration were increased in patients with longer duration of presentation to the hospital.

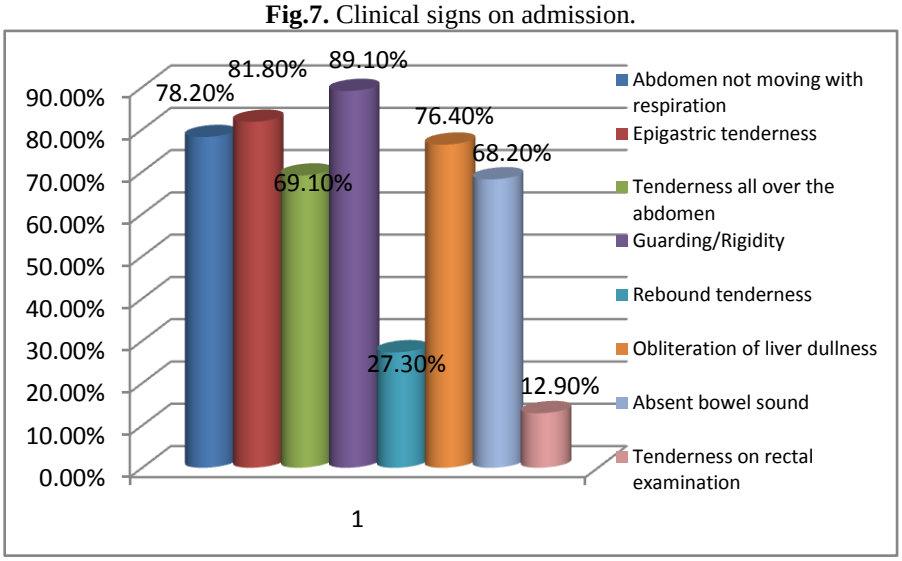
Signs:

Table 14. Clinical signs recorded on examination of abdomen.

N=110

Signs	**No. of patients**	**Percentage**
Abdomen not moving with respiration	86	78.2%
Epigastric tenderness	90	81.8%
Tenderness all over the abdomen	76	69.1%
Guarding/Rigidity	98	89.1%
Rebound tenderness	30	27.3%
Obliteration of liver dullness	84	76.4%
Absent bowel sound	75	68.2%
Tenderness on rectal examination	14	12.9%

Fig.7. Clinical signs on admission.



LABORATORY INVESTIGATIONS:

Routine laboratory investigations required to assess the general conditions of the patients and to know whether the patient was fit to undergo surgery or not were recorded.

Table15. Routine laboratory reports of patients.

Investigations	No. Of patients				Total
	1-6hrs	6-12hrs	12-24hrs	>24hrs	
Haemoglobin >10gm% <10gm%	 12 -	 33 3	 14 2	 17 3	 102(92.7%) 8(7.3)
Blood urea <40mg% >40gm%	 12 -	 35 1	 38 4	 12 8	 97(88.2%) 13(11.8%)
TLC 4000-11000/cummm >11000/cumm	 12 -	 35 3	 33 7	 13 7	 93(84.5%) 17(15.5%)
Serum Na+ Normal Abnormal	 12 -	 36 -	 38 4	 12 8	 89(80.9%) 12(10.9%)
Serum K+ Normal Abnormal	 12 -	 36 -	 40 2	 14 6	 102(92.7%) 8(7.3%)
	12	36	42	20	100%

Haemoglobin level in blood was below 10gm% in 8(7.3%) of patients.

Blood urea level more than 40mg% were recorded in 13(11.8%) patients and most of them belonged to the patients in shock with systolic blood pressure less than 90mmHg.

TLC, more than 11,000/cumm was recorded in 17(15.5%) patients and most of them belonged to the group who presented late at the hospital after 12 hours.

Electrolyte imbalances were seen in upto 10.4% of patients, significantly in those who presented late after 24 hours.

Table 16. Blood sugar of patients were recorded and shown in table.

Random blood sugar	**No. of cases**	**Percentages**
79-140mg% >140mg% <79mg%	99 9 2	90%% 8.2% 1.8%
Total	**110**	**100%**

9(8.2%) patients had random blood sugar above normal range 140mg% and only 2(1.8%) patients had lower random blood sugar below 79mg%, remaining patients had normal random blood sugar.

RADIOLOGY:
Plain X-ray abdomen in erect posture, AP view were taken for every patients and pneumoperitoneum indicated by the presence of free gas under the right dome of the diaphragm, could be demonstrated in 107(97.3%) cases. Only 3(2.7%) cases required CT scan abdomen for further confirmation of diagnosis.

OPERATIVE FINDINGS:

Table 17. Sites of perforation.

N=110

Site of perforation	**No. of cases**	**Percentages**
D1- anterior surface DI-posterior surface	110 -	100% -
Total	**110**	**100**

Anterior wall of first part of duodenum was the most commonly involved site of perforation in this study seen in all 110(100%) cases.

SIZE OF PERFORATION

Table 18. Size of perforations recorded.

N=110

Size of perforation	**No. of cases**	**Percentage**
0.5cm or less	28	25.5%
0.5cm-1cm	65	59.1%
1cm-2cm	15	13.6%
>2cm	2	1.8%
Total	**110**	**100%**

Perforations in this study ranged from 0.5cm to 2.5cm in diameter. Largest one measured 2.5cm in diameter, seen in only one case. 59.1% of cases had perforation of size 0.5cm to 1cm in diameter. 25.5% of cases had small perforation of size less than 0.5cm and 13.6% of cases had larger perforation of size 1cm to 2cm in diameter.

Chronic ulcer history of more than 3months was associated with larger perforations. 33(30%) in 0.5cm -1cm size group and 13(11.8%) in 1cm to 2cm group and 2(1.8%) of small perforation group 0.5cm and less.

Antecedant acute ulcer history of less than 3 months duration was seen in 17(15.5%) patients with perforation sized 0.5cm to 1cm, and 2(1.8%) cases in large 1cm to 2cm perforation group and in only 1(0.9%) case of small perforation less than 0.5cm group. Rest of the 42(38.2%) patients gave no ulcer history.

PERITONEAL CONTAMINATION:
The peritoneal contaminations found at time of operation were recorded in all patients and a scale was used for grading the contaminations from Grade 0 to grade IV.
Grade 0: No evidence of contamination.
GradeI : Presence of cloudy fluid.
Grade II: Fibrinous exudate
Grade III: Free pus or major contamination.
Grade IV: Abscess

Table 19(a). Correlation of peritoneal contaminations with size of perforations.

N-110

SIZE	**GRADE 0**	**GRADE I**	**GRADE II**	**GRADE III**	**GRADE IV**	**TOTAL**	**P value**
<0.5cm	2 (1.8%)	8 (7.3%)	16 (14.5%)	2 (1.8%)	-	28 (25.5%)	0.200
0.5-1cm	8 (7.3%)	5 (4.5%)	44 (40%)	8 (7.3%)	2 (1.8%)	67 (60.9%)	
>1cm	-	-	9 (8.2%)	4 (3.6%)	2 (1.8%)	15 (13.6%)	
Total	10 (9.1%)	13 (11.8%)	69 (62.7%)	14 (12.7%)	4 (3.6%)	110	

Table 19(b). Correlation of peritoneal contaminations with duration of perforations.

Duration	GRADE 0	GRADE I	GRADE II	GRADE III	GRADE IV	TOTAL	P value
6HRS	2 (1.8%)	3 (2.7%)	7 (6.4%)	-	-	12 (10.9%)	0.005
12HRS	8 (7.3%)	10 (9.1%)	16 (14.5%)	2 (1.8%)	-	36 (32.7 %)	
24HRS	-	-	38 (34.5%)	4 (3.6%)	-	42 (38.2%)	
>24HRS	-	-	8 (7.3%)	8 (7.3%)	4 (3.6%)	20 (18.2%)	
Total	10 (9.1%)	13 (11.8%)	69 (62.7%)	14 (12.7%)	4 (3.6%)	110	

Table 20. Correlation of peritoneal contaminations with pre-operative shock.

BP	GRADE 0	GRADE I	GRADE II	GRADE III	GRADE IV	TOTAL	P value
> 90mmHg	10 (9.1%)	13 (11.8%)	55 (50%)	8 (7.3%)	-	86 (78.2%)	0.050
< 90mmHg	-	-	14 (12.7 %)	6 (5.5%)	4 (3.6%)	24(22.8%)	
Total	10 (9.1%)	13 (11.8%)	69 (62.7%)	14 (12.7%)	4 (3.6%)	110	

Table 19 & 20 show that peritoneal contaminations were more when size of perforation were larger, the duration of perforation to operative treatment was longer and in presence of preoperative shock. In this study 18(16.4%) patients had gross peritoneal contamination of grade III and IV.

MORBIDITY AND MORTALITY:

MORBIDITY: Post operative complications.

Most of the cases (73.6%) had smooth recovery after operation. The operative morbidities were recorded in all patients and the findings were recorded.

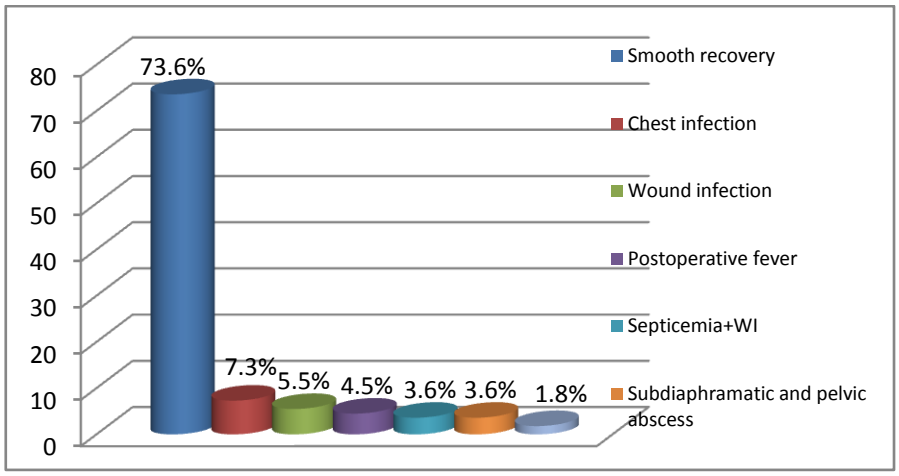
Table 21. Post operative complications.

N=110

Morbidity	No. of cases	Average days of Hospitalisation	Percentage
Smooth recovery	81	10 days	73.6%
Chest infection	8	14 days	7.3%
Wound infection	6	16 days	5.5%
Postoperative fever	5	12 days	4.5%
Septicemia	4	21 days	3.6%
Subdiaphramatic and pelvic abscess	4	21 days	3.6%
Wound dehiscence	2	40 days	1.8%
Total	**110**		**100%**

The operative morbidity rate was 26.3% in this study. Pulmonary complications noted in 7.3% of patients, lower lobe pneumonia and atelectasis, were the common complications, usually in older patients. Wound infections occurred in 5.5% of patients. Those were usually collection of pus subcutaneously and were drained in the early post operative days. Wound dehiscence occurred in 2(1.8%) of cases and they required secondary suturing. Two cases of subphrenic abscess and 2 cases of pelvic abscess occurred. Fever and mucous diarrhoea occurred in late postoperative days in these cases, confirmed with ultrasonography. Most of them resolved with antibiotics, only in one patient pus was drained, he died of pulmonary complications.

Fig. 8. Postoperative complications.



MORTALITY:

Patients who died of duodenal ulcer perforations were considered of the 110 patients in the study. Seven patients died in the series with an overall mortality of 6.4%.

Table 22. Mortality table.

Expired patients	**Age /sex**	**Duration of perforation**	**Size of perforation**	**Cause of death**
Patient 1	63/M	4 days	1cm	ARDS
Patient2	80/M	2days	1cm	Acute renal failure
Patient3	55/M	5days	1.5cm	Septic shock
Patient4	67/M	4days	0.8cm	ARDS
Patient 5	40/M	5days	2cm	Septic shock
Patient6	51/M	3days	1.5cm	ARDS
Patient 7	74/M	3days	1cm	Acute renal failure

The first case of duodenal ulcer perforation death was a 63 years male. He presented 4 days after perforation, had shock symptoms, peritonitis, had large perforation of 1cm. After operation he developed pulmonary complications, pleural effusion, and Adult respiratory distress syndrome and died after 5 days of operation.

The second patient was a 80 years male, reported 2 days after perforation. On presentation patient was severely dehydrated, developed renal failure and died after 3 days of operation.

The third case was brought from remote place after a lapse of 5 days and was in shock at time of admission. Septic shock symptoms, peritonitis with high TLC was present. Resuscitation, inotropic support and operation was done but died after 2 days of operation.

The fourth patient was a 67 years male with diabetes, reported after 4 days of perforation. Dehydration and peritonitis were present at time of admission and he died 5 days after operation due to pulmonary complications.

The fifth patient was 40 years male who presented late after a lapse of 5 days of perforation. He was in shock, high TLC count. Large perforation of 2cm was present with gross peritoneal contamination. He could not be recovered from anaesthesia and died on the day of perforation.

The sixth patient died of duodenal ulcer perforation in the study was a 51 years old male diabetes with heart disease of dilated cardiomyopathy. He presented late after 3 days of perforation was in shock. Large perforation of 1.5cm size was present with Grade IV peritoneal contamination. After operation he developed subphrenic abscess and pulmonary complications. He developed ARDS with renal failure and died after 6 days of surgery.

The seventh case was a 72 years old diabetic male patient, who was referred from another hospital after 3 days of perforation. He was in shock at presentation with peritonitis and died after 2 days of surgery due to pulmonary complications.

The overall morbidity rate was 26.3% and mortality was 6.4% in this study.

Table 23. Factors affecting mortality.

Serial no.	**Particulars of patients**	**Total no. of patients**	**Mortality**	**p- value**
1.	< 50 >50	67 43	1 6	0.010
2.	Male Female	106 4	7 0	0.050

3.	<24 hrs delay >24hrs delay	90 20	0 7	0.0005
4.	<1cm size >1cmsize	93 17	4 3	0.100
5.	Preoperative hypotension >90mmHg <90mmHg	 86 24	 0 7	0.0005
6.	Peritoneal contamination Grade0 GradeI GradeII GradeIII GradeIV	 10 13 69 14 4	 0 0 3 2 2	0.0005

Table 23 shows 7 patients died out of 180 duodenal ulcer perforated patients, which accounts for a death rate of 6.4%. Sixty seven patients were below 50 years and had mortality of 1; 43 patients were above 50 years, had a mortality of 6. Preoperative haemodynamic instability was seen in 24 patients and 7 of them died postoperatively. Twenty patients were operated upon late after 24 hours, mortality of 7 patients was seen among those who were operated after 24 hours after onset of symptoms. Mortality was higher in contaminated cases.

IV. Discussion

This study comprised of 110 cases of duodenal ulcer perforation came to Department of surgery, RIMS, Imphal, Manipur during the period from August 2015 to September 2017. The study excludes patients of traumatic, benign or malignant gastric or other perforation other than duodenal ulcer perforation. Appropriate epidemiological data, clinical parameters were correlated with operative findings, morbidity and mortality.

1. Age distribution:

Duodenal ulcer perforations is common in the age group 41-50 years.

Table 23. Comparison of age incidence with other studies.

Studies	**Year**	**Peak age in years**
Gupta BS et al[15]	2003	51-60
Goudar B et al[1]	2010	51-60
Chalya et al[4]	2011	21-30
Chaudhary V& Mathur R[11]	2012	18- 30
Kumar PV eta al[12]	2013	30-50
Present study	2017	41-50

Perforation is more common in older age groups as reported in previous studies. However study of Chaudhary V& Mathur R[11] found increased incidence in younger age. According to Kang JY et al[14] (2006) duodenal ulcer perforation in elderly is associated with higher mortality rate.

2. Sex incidence:

In the present series of 110 cases the majority of patients were male with incidence of 96.4%, only 4 patients were female. Many authors have reported that incidence is high in males when compared to females.

Table 24. Comparison of sex incidence.

Studies	Year	% in males
Chaudhary V & Mathur R[11]	**2012**	**81.2**
Kumar PV et al[12]	**2013**	**100**
Bansod A et al[13]	**2014**	**82.85**
Mohan VS & Siddarth O[17]	**2014**	**77.2**
Present series	**2017**	**96.4**

According to Chalaya L et al[4] (2011) male female ratio of peptic ulcer perforation was 1.3:1. Rigopoulos A et al[20] in their study of 256 cases of perforated gastric and duodenal ulcer found maximum incidence in male- sex ratio 6.31/1.

The high incidence of male can be explained on the basis of greater hardship, stress, anxiety, indulgence in alcohol and smoking.

3. Occupation:

It is believed that duodenal ulcer perforation occurs in those people who are engaged in heavy manual labour. PC Sood and RL Gupta[24] (1996) mentioned duodenal ulcer perforation is more common in manual labourers and lower socio economic groups.

Rao VS & Siddharth O[16] (2014) found labour class dominated the incidence of perforation.

Similar findings were seen in the present study with higher incidence upto 80% seen in unskilled manual labourers and semiskilled lower socioeconomic sections. This observation has an implication on accessibility to health care facilities and awareness of the disease.

4. Religion:

There is no evidence of particular religion affected by perforated peptic ulcer, however, Donderici et al[25] (1994) reported a higher incidence of perforation in Muslims during Ramdan.

Malhotra et al[26] (1964) reported Punjabis are immune to ulcer while South Indians are more prone to ulcer and complications, the high prevalence related to their pattern of diet and eating habit.

In the present study higher incidence seen amongst Meetei (Hindu) accounting for 68.2% followed by Muslim 22.7(%), which seems to correlate to the population ratio, also hypothetically can probably be attributed to higher intake of more oily and spicy foods.

5. Habits: Smoking and alcohol

According to Turkdogan et al[27] (1999) alcohol act as noxious agent causing gastric mucosal damage, stimulates acid secretion and increases serum gastrin level.

Nuhu A et al[28] (2000) found increased incidence of duodenal perforation among smokers. According to them smoking inhibits pancreatic bicarbonate secretion, resulting increased acidity in duodenal blub, also inhibits duodenal ulcer healing.

Mohan VS & Siddarth O[17] (2014) found in their study most of their patients of duodenal ulcer perforation was both alcoholics and smokers. Similar findings was shared by Kumar PV et al[12] (2013) in their study, with 66% of cases.

The present study found higher prevalence (34.5%) of duodenal ulcer perforation among those who has happened to be both alcohol consumers and smokers at same time.

6. Dietary Habits:

Thompson HL[29] (1937) did not find role of dietary habit in aetiology of peptic perforation.

Crohn BB et al[30] (1946) however stated that large meal, heavy drinking and exertion encouraged perforation.

Shepherd JA[31] (1975) stated that distended stomach after a meal might be a causative factor but perforation was found to occur in empty stomach too.

In the present study maximum patients of duodenal ulcer perforation were non vegetarian 90.9%, had spicy food76.4% and gave history of irregular dietary habit 94.5%.

7. Family History:

Kozoll et al[32] (1960) found that family history of peptic ulcer was positive in 1.5% of patients admitted for duodenal ulcer. However Boey J et al[33] (1982) found in 20.7% of patients with acute duodenal ulcer perforation had positive family history.

Mukerjee A & Naveen N[2] (2014) in their study reported 10% of duodenal ulcer patients had a positive family history while none of gastric ulcer patients had a family history.

In the present series family history of peptic ulcer is seen in only 8(7.3%) of patients but there is history of duodenal ulcer perforation in any of their immediate relatives.

8. Blood Group:

Horowitz et al[52] (1996) found higher incidence of duodenal ulcer perforation in blood group O and suggested the gene of blood group O determines the severity of duodenal ulcer probably by means of an effect on mucosa of gastrointestinal tract.

Kumar PV et al[12] (2013) demonstrated a high incidence of blood group O (50%) in duodenal ulcer perforation patients. Similar findings was seen in study of Mohan VS & Siddarth O[17] (2014) with higher incidence of blood group O (43.2%) among those patients of perforated duodenal ulcer.

In this study also shows maximum incidence of duodenal perforation is seen in patients with blood group O (49.1%).

9. Drug and Perforation:

Glanborry[42] in 1977 showed that more than 80% of his patients with perforated peptic ulcer were taking NSAIDS at time of operation.

Chalaya L et al[4] (2011) reported NSAIDS to be important cause of perforated peptic ulcer. NSAIDS inhibits prostaglandin synthesis so further reducing mucosal blood flow.

Chaudhary V & Mathur R[11] (2012) found 18.57% of their peptic ulcer perforated patients have history of NSAIDS use.

Mukherjee A et al[2] (2014) also reported 20% of duodenal ulcer patients with complications have NSAIDS history.

In this study history of drug ingestion was seen in upto 36.4% of patients in form of NSAIDS (20.9%), corticosteroids (9.2%) or both (6.4%) for various conditions like toothache, arthritis, or as antipyretics few days before perforation. From these studies it appears that antipyretics, NSAIDS, corticosteroids may have a role in peptic ulcer perforation probably by impairing mucosal barrier.

10. Diurnal Variation:

Jamieson RA (1947) and Mackay (1966), reported from Scotland that perforation occurs most on Friday and Saturday between 5 to 6 pm, least on Sunday.[21]

Pelmar ED and Newark NJ (1972) reported most perforations occurred between noon to 6pm.[22]

In this study maximum patients had onset of pain at night (47.2%), followed by Early morning (27.3%) which is similar to study of Svance C[23] (1993) who found perforation to be more common in night than in morning.

CLINICAL FEATURES:

Symptomatology

1. Pain:

Most authors mentioned sudden acute agonizing pain as the main chief complaint of duodenal ulcer perforation.

Schmitz et al[60] in their series found 89% of patients experienced sudden onset of pain, 28% of them having pain radiation to one or both shoulder. In the series of Chalya L et al[4] (2011) sudden onset of epigastric pain seen in 97.6% of patients.

Mohan VS & Siddharth O[17] (2014) series of 250 cases of duodenal perforation excruciating pain was seen in all cases. In majority of cases it started in epigastric region and gradually spread all over abdomen, few cases pain is reported first in right iliac fossae. Shoulder tip pain was seen in 10% of cases.

In the present study all patients 100% presented with generalized pain abdomen.

Table 25. Comparison of symptoms with other studies.

Study	Year	Epigastric pain	Generalised pain
Schmitz et al	1953	3.5%	87%
Kozoll et al	1961	54.3%	33%
Chalaya L et al	2011	97.6%	-
Mohan VS et al	2014	90%	100%
Present study	2017	-	100%

2. Vomiting and Nausea:

In the present study nausea and vomiting seen in 18.2% and 13.6% of cases respectively.

Rigopoulos A et al[20] (2011) found that second most common complaint in peptic ulcer perforation was vomiting seen in 72.7% gastric perforation and 60.84% in duodenal perforation. Chaudhary V and Mathur R[11] also reported the 51% of their cases of peptic ulcer perforation has vomiting.

Table 26. Comparison of nausea & vomiting incidence with other studies.

Study	Year	Incidence of vomiting	Nausea
Rigopoulos A[21]	2011	60.84%	-
Kumar PV et al[12]	2013	90%	-
Chaudhary V & Mathur R[11]	2012	51%	-
Chalaya L[4]	2011	36.9%	36.9%
Present study	2017	18.2%	13.6%

3. Haematemesis and Melaena:

According to Stabile BE et al[54] (1979), bleeding in perforation occurs due to posterior kissing ulcer and recommended definitive surgery in such patients. Schmitz et al[60] (1953) found haematemesis or melaena in 27% of 186 patients in their series.

Mukherjee A and Naveen N[2] (2014) reported incidence of 10 % of bleeding associated with perforated duodenal ulcer in their study which according to them was associated with grave prognosis.

Table 27. Comparison of upper GI bleeding incidence with other studies.

Study	Year	Incidence of upper GI bleeding
Schmitz et al[60]	1953	Haematemesis & melaena 27.3%
Wangesteen[61]	1972	Haematemesis or melaena 6.1%
Mukherjee A et al[2]	2014	Haematemesis and melaena 10%
Present study	2017	Haematemesis1.8%and melaena0.9%

In the present study haematemeis and melaena are seen in 1.8% and 0.9% of patients with significant ulcer history of more than 3 months.

4. Activity at onset:

Leur[62] (1949) found that 53.2% of his patients were engaged in activity when perforation occurred.
Kozoll and Meyer[32] in 1961 reported that 33.4% of duodenal ulcer perforation occurred while patient were at rest or asleep, 53.9% while patient were at work or during an exercise and 12.7% while patient were eating or drinking.
Gear[63] (1968) reported perforated duodenal ulcer following injury and operation thus attributing trauma as possible predisposing factor for perforation.

Table 28. Comparing activity at onset with other study.

Activity	Kozoll et al[32] (1961)	Present study
Rest	46.1%	50.9%
Working	53.9%	49.1%

It appears that the onset of perforation was unrelated to the activity of the patient as in present study 50.1% of the perforation occurred while sleeping or relaxing.

5. Antecedent Ulcer history:

Most studies reported significant antecedant history of duodenal ulcer in majority of patients with perforated duodenal ulcer.

Table 29. Comparing antecedant ulcer history with other studies.

Study	Year	Significant antecedant ulcer history
Boey J et al[33]	1982	72%
V. Morurougayan	1994	76%
P.C. Sood & R.L. Gupta[24]	1996	78%
Mohan VS & Siddarth O[17]	2012	68%
Vinceet C et al[11]	2014	20.8%
Bansod A[13]	2014	32.85%
Present study	2017	43.6%(>3months) 18.2%(<3months)

In the present study 43.6% of patients gave significant ulcer history for a period of 3 months or more prior to perforation, while 18.2% gave history of dyspepsia prior to perforation while 38.2% of patient has no ulcer history prior to perforation which is similar to findings to previous studies.

GENERAL PHYSICAL EXAMINATION:
SIGNS

1. Pulse :

In the study tachycardia with pulse rate more than 100/min was noted in 53 (41.9%) cases presented after 12 hours after onset of pain. Out of the 53patients 7 patients died in postoperative period making the overall mortality of 13.2%.

De Bakley[8] (1940) reported normal pulse rate are found if patient presented early after perforation i.e. within 6 hours of onset of perforation.

Kozoll and Meyer[32] (1961) found pulse rates of over 110 per minute in 16.6% of their patients and more than 50% mortality in them. Chaudhary V and Mathur R[11] (2012) reported tachycardia in all their patients.

2. Blood Pressure:

Hypotension was said to be present if systolic blood pressure was less than 90mmHg and was recorded in 24 (21.8%) patients. 19 of them reported after 12 hours of onset of pain. There were 7 deaths in patients with shock with the high mortality rate of 29.2% in shock patients. Hence blood pressure at time of admission has important prognostic significance. The observations are found to be similar with the findings of other authors.

Chalaya L et al[4] (2011) reported overall mortality rate of upto 50% of patients presenting with shock, blood pressure less than 100mmHg, concluding blood pressure at time of presentation an important prognostic significance.

Mohan VS and Siddarth O[17] (2014) reported shock as grave prognosis in patients presented with shock with mortality rate of 69.2%.

3. Temperature:

Schmitz et al[60] (1953) found fever in 42% of patients. According to Kozoll and Meyer[32] (1961) patients of perforation with fever more than 102^0 F on admission has poor prognosis as a surgical candidate.

Mohan VS and Siddarth O[17] (2014) in their series of 100 cases of perforation 50% of perforated duodenum patients had fever.

Patient was regarded to have fever when body temperature was more than 100^0F and was noted in 23 (20.9%) patients. These patients have peritonitis and higher grade of peritoneal contaminations with poor outcomes.

4. Respiratory rate:

In Kozoll and Meyer[32] (1961) Series 80.8% of patients on admission had respiratory rate of less than 30 per minute with mortality rate of 17.6%. But in patients with respiratory rate of more than 30 per minute the mortality rate was 39.2%. According to them the mortality rate rises rather preciously with abnormally rapid respiration.

In this study normal respiratory rate was seen in most patients, higher respiratory rate of more than 30 per minute was seen in only 7(6.3%) of cases, these patients were febrile and 4 of them died accounting mortality of 57.1%.

5. Abdominal examination:

In this study most of the patients 86(78.2%) had an abdomen which not moving with respiration on examination. Tenderness all over the abdomen was found in 76(69%) of patients. Epigastric tenderness was seen in 90(81.8%) of cases, guarding all over the abdomen with rigidity were recorded in 98(81%) of the patients. Rebound tenderness was seen in 30(27.3%) of cases, and tenderness on rectal examination was seen in 14(12.9%) of the patients. Obliteration of liver dullness was seen in 84(76.4%) of cases. Bowel sound was absent in 75(68.2%) patients.

Hence examination of abdomen for presence of guarding, rigidity, rebound tenderness, masking of liver dullness, absent bowel sound and tenderness on per rectal examination are important signs of peritonitis and presence of these signs are reasonable indications for exploration.

The present study's findings are similar to the findings of other studies, Schmitz et al (1953), Chaudhary v and Mathur R[11] (2012) and Mohan VS and Siddarth O[17] (2014).

Schmitz et al[60] found extreme abdominal tenderness in 80%, rigidity in 73% and silent abdomen in 73% of patients.

Chaudhary V and Mathur R[11] reported abdominal tenderness in all patients, guarding rigidity in 95.6%, obliteration of liver dullness in 81.3% and silent abdomen in 36.5% patients.

Mohan VS and Siddarth O[17] in their series of 250 cases found abdominal rigidity in all cases, obliterated liver dullness in 68%, shifting dullness in 60% of cases, absent bowel sound in 70% of cases.

LABORATORY INVESTIGATIONS:

1. Haemoglobin:

In the present study patient with haemoglobin less than 10gm% was seen in 8(7.3%) patients. Four of the anemic patients with haemoglobin less than 10gm% were included among those who died in postoperative

period accounting for the mortality rate of 50% which indicates mortality increases with decrease in haemoglobin level.

The findings were in support with other studies, Kozoll and Meyeret al[60] (1962) found that 4% patients admitted with perforated gastroduodenal ulcer had haemoglobin less than 9gm% compared to other patients and they have higher mortality rate (70.6%). Mohan VS and Siddarth O[17] (2012) found 30% of their patient of peritonitis patients were anemic and mortality rates were higher in them.

2. Blood Urea:

In the present study 13(11.8%) of patients had blood urea level of more than 40mg/dl. Most of these patients reported late after 24 hours and had dehydration at time of presentation and were in shock. 6 patients died in the post operative patients making the mortality rate of 46.2%. This indicates that mortality increases with the increase in serum urea level > 4omg/dl. Similar result was found by Kozoll and Meyer[60] who found 27.2% of patients had serum urea level more than 40mg% with associated mortality rate of 61.9% in their study.

3. Total Leucocyte Count:

In the present study total leucocyte count above 11,000/cumm was found in 17 (15.5%) of patients and most of them belong to the group who presented late at the hospital after 12 hours. They had signs of peritonitis and signified the onset of bacterial infection. The findings were similar to Seeley et al[66] (1956) who reported that longer the interval since perforation the higher is the white cell count and is associated with increased mortality and morbidities.

Mohan VS and Siddarth O[17] (2014) found 14.3% of their perforated duodenal ulcer patients have leucocytosis and 18% have leucopenia.

RADIOLOGICAL INVESTIGATIONS:

Plain X-ray abdomen in erect, left lateral posture and X-ray chest PA posture were taken in all patients and pneumoperitoneum with free gas under right dome of diaphragm was detected in 107(97.3%) of cases. 3(2.7%) cases with no free gas under right dome of diaphragm were confirmed perforation with CT scan.

This finding was similar to the findings with other authors.

Table 30. Comparison of X- ray abdomen erect for gas under diaphragm with other studies.

Study	**Year**	**Percentage of cases**
Kozoll and Meyer[60]	1961	84%
Chalya et al[4]	2011	75%
Chaudhary V et al[11]	2012	92.7%
Bansod A[13]	2014	95.7%
Mohan VS et al[17]	2014	96%
Present study	2017	97.3%

PREOPERATIVE FINDINGS:

1. Site of Perforation:

In the present study perforation at D1 anterior wall was found in all 110 cases.

Table 31. Findings of the present study is similar with the other authors

Study	**Year**	**Percentage of D1 perforation**
Kozoll et al[60]	1961	75%
William H et al[50]	1961	95%
Rigopoulous A[20]	2011	90%
Kumar PVet al[12]	2013	98%
Present study	2017	100%

2. Size of Perforation:

Perforations in this study ranged from 0.5cm to 2cm in diameter. 60.9% of cases had perforation of size 0.5cm to 1cm in diameter. 25.5% had small perforation of size less than 0.5cm and 13.6% of cases had larger perforation of size 1cm to 2cm in diameter. The mean size of perforation was 7mm.

The findings in the study were similar to the other authors. Aubrede L et al[46] (1961) reported that perforation sizes ranged from few mm to almost 2cm in diameter in their series. Howritz et al[52] (1989) had reported a mean ulcer size of 4-8mm in duodenal ulcer perforation, 1-13mm in prepyloric ulcer perforation, 7.8-12mm in pyloroduodenal ulcer perforation. . Rigopoulos A et al[20] (2014) classify duodenal perforation less

than 3mm as small, from 3 to 6mm are average, between 7-10mm large and more than 10mm as very large. In their study over 80% of cases had small perforations.

In the study it was found that chronic ulcer history of more than 3months was associated with larger perforation 33(30%) in 0.5cm -1cm size group and 13(11.8%) in 1cm to 2cm group and 2(1.8%) of small perforation group 0.5cm and less.

Antecedant acute ulcer history of less than 3 months duration was seen in 17(15.5%) of patients of perforation size 0.5cm to 1cm, and 2(1.8%) of large 1cm to 2cm perforation patients and in only 1(0.9%) of small perforation less than 0.5cm patients. Rest of the 42(38.2%) patients gave no ulcer history.

Similar findings were reported in other studies. Gilmour J[51] (1953) suggested that perforations smaller than 5mm are usually acute and those larger than 5mm are chronic.

Baker RJ[53] (1997) reported large, chronic , indurated ulcers especially the ones with perforations greater than 5mm in diameter, had a high incidence of perforation and stenosis and required an adjunctive ulcer operation in addition to or in lieu of the patch closure.

Gupta BS et al[15] (2003) classified duodenal large perforations in their study as small less than 1cm, large less than 3cm and giant exceeding 3cm. According to them large perforations had significantly increased hospital stay, leak rates and morbidity.

3. Peritoneal Contamination:

Goudar B et al[1] (2011) reported in their study found intra abdominal contamination related to the size and duration of perforation. They also found higher mortality rate with higher grade of peritoneal contaminations.

Boey J et al[33] (1982) found 6.7 % of their patients had gross contaminations such gross contaminations were seen in patients with long standing perforation.

The findings in the study were similar to the previous studies, 18(16.4%) of patients in the series had gross peritoneal contamination of grade III and IV. 12 (10.9%) patients with gross peritoneal contamination presented late after 12 hours, larger size perforation of 1-2cm were found in 6(5.5%) of the patients with gross contaminated peritoneal cavity. 10(0.9%) patients with gross peritoneal contamination were in shock preoperatively at time of presentation and 7(6.4%) of them died in postoperative period.

Hence the incidence of gross peritoneal contamination was more associated with patients with long standing perforation i.e. more than 12 hours, those with larger size of perforation and had shock pre-operatively, also the mortality is associated more in such patients.

OPERATIVE MORTALITY, MORBIDITY AND FACTORS AFFECTING

In the present study the overall mortality rate was 6.4% and morbidity was 26.3%, out of 110 cases of duodenal ulcer perforation treated with simple closure and omental patch. The morbidity and mortality are associated with age of the patients, haemodynamic instability, treatment delay more than 24 hours, peritoneal contaminations.

The findings in this series were similar with other authors.

De Bakley (1939) reviewed the literature on surgical mortality of perforated peptic ulcer and found the mortality rate of 23.4%.

Boey J et al[33] (1982) examined the operative risk factors for patients with perforated duodenal ulcer. The operative mortality was 4.2% and complication rate was 12.7%.

Goudar B et al[1] (2011) in their retrospective study of perforated duodenal ulcer found the overall mortality rate was 13%.

Chalaya L et al[4] (2011) in a combined retrospective and prospective study of patients operated for perforated peptic ulcer reported the overall mortality was 5%-25%, rising as high as 50% with age.

In recent study conducted by Mohan VS and Siddarth O[17] (2014) on 250 cases of duodenal perforation they found mortality rate was 3.6%. Higher mortality was found in old age, treatment delay of 48 hours between onset and surgery and preoperative shock.

Helicobacter pylori:

Ng et al[98] (2000) noted that 81% of patients with perforated DU were infected with H. pylori. Kate V et al[99] (2001) reported 73% prevalence of H. pylori in perforated peptic ulcer. In the present study, association of H. pylori infection in peptic ulcer perforation could not be proved due to lack of facilities in our institute. However 'triple regime for anti H. pylori" was prescribed to 19(17.6%) patients who showed persistent peptic ulcer symptoms during follow up inspite of getting anti ulcer drugs. This compares well with the studies of Gupta BS et al[15] and Nuhu A et al[28] which have successfully used simple closure followed by eradication of H. pylori as a treatment for perforated peptic ulcer. This was however in contrast to the studies of Jordan GL[100] and

Gray JG[101] which reported emergency definitive surgery as a means to prevent recurrence and re-operation rates. However in developing countries delay in presentation often prevents any attempt at definitive surgery.[4]

Follow Up:
All patients 103(93.6%) who survived were followed up for a minimum period of three months, and all received anti ulcer drugs, proton pump inhibitors to prevent peptic ulcer recurrence. In 19(17.3%) patients, peptic ulcer symptoms persisted. Anti H. pylori triple drug regime with anti ulcer drugs prescribed empirically for them and all of them responded well to it. Mohan VS and Siddarth O[17] reported recurrence rate of 56% out of 190 patients followed, 40 patients had H. pylori infection and 24 were having ulcer symptoms even after H. pylori eradication regime and were subjected to definitive surgery.

V. Conclusion

In this study 110 patients, of perforated duodenal ulcer treated operatively in the Department of surgery RIMS, between August 2015 to September 2017, were studied. Correlation of epidemiological profiles, clinical parameters, operative findings and post operative follow up for complications were studied and analysed.

Duodenal ulcer perforation is more common in males (96.4%) in the age group of 41 to 50 years with 38(34.5%) patients, followed by the age group of 51 to 60 years with 26(23.6%) patients.
Perforation is more common in Meetei/Hindu –valley people.
People engaged in manual works with poor socio economic status are more prone to have perforation.
Ingestion of NSAIDS and steroids, Smoking and alcohol consumption may increase the risk of duodenal ulcer perforation.
Dietary habit also seems to play a role in the pathogenesis of peptic ulcer and complications. Spicy foods and irregular diet may increase the risk of duodenal ulcer and perforation.
The maximum incidence of perforation occurs in the months of October to January.
The incidence of perforation is more common in patients with 'O' blood group.
Sudden onset of abdominal pain, in the epigastrium is a constant symptom (100%). Vomiting, constipation and fever are not so common.
Abdominal tenderness, rigidity and obliteration of liver dullness are important signs and absence of bowel sound is one of the early signs of perforation peritonitis.
Plain X-ray abdomen (erect), including both domes of diaphragm is useful in detecting pneumopeitoneum.
The commonest site of perforation is the anterior D1.
Early diagnosis, resuscitation and preoperative management are as important as surgical procedure.
Simple closure with Grahm's omentoplasty followed by proton pump inhibitor drugs is an effective treatment procedure of duodenal ulcer perforation.
H. pylori eradication after simple closure may be necessary to prevent recurrence of ulcer.
Pre-operative shock, old age, longer duration of perforation, concurrent medical illness and higher grade of peritoneal contamination are the main factors affecting the morbidity and mortality in duodenal ulcer perforation. Mortality rate was 6.4% in this study.

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