

## Comparative Study on the Incidence of Gastric and Duodenal Perforation Presenting at DMCH Darbhanga Bihar

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### Abstract

**Background and Objectives:** Peptic ulcer disease affects more than 4 million people around the world every year. Complications are encountered in 10%-20% of peptic ulcers of which 2%-10% perforate. To evaluate and compare the incidence of gastric and duodenal perforation. To evaluate and compare the causes of gastric and duodenal perforation.

**Material and Method:** This was a retrospective comparative study which included 153 patients who were admitted and treated in the Upgraded Department of General Surgery at Darbhanga Medical College, Darbhanga for the management of peptic perforation peritonitis between July 2019 to December 2020.

**Conclusion:** Gastric and duodenal perforations are common presentations of peptic ulcer disease but each one of them have their own individuality. The incidence of peptic ulcer perforation had decreased in the past decade but the burden of disease still remains in our society. The incidence of gastric ulcer perforation has increased in the past few years and much still is needed to evaluate the cause and management of the disease.

**Keywords:** Gastric and Duodenal Perforation, Peptic Ulcer, Biopsy.

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### Introduction

Peptic ulcer disease affects more than 4 million people around the world every year [1]. Complications are encountered in 10%-20% of peptic ulcers of which 2%-10% perforate [2,3]. Peptic ulcer perforations are one of the commonest complications of peptic ulcer and they constitute a vast majority of acute abdomen coming to the surgical emergency in a hospital. It is indeed one of the most dreadful catastrophes of peptic ulcer. As Lord Moynihan had said, peptic ulcer perforation is one of the most common and overwhelming catastrophes that creates acute surgical emergency [4]. Out of all the complications of peptic ulcer, acute perforation appears to be the commonest in our country. According to Littre, the earliest case of peptic ulcer perforation on record dates back to the early 1670. The patient was daughter of Charles I of England, Henrietta Anne. She had suffered from dyspepsia. She had a sudden agonizing pain in the abdomen, which was uncontrolled along with vomiting. She died after 9 hours. She was thought to be a case of poisoning but on autopsy it was proven to be a case of peptic ulcer perforation<sup>2</sup>. Peptic ulcers are caused by increased aggressive factors, decreased defensive factors or both. This in turn leads to mucosal damage and subsequent ulceration. Persistent ulcer leads to peptic ulcer perforations. Protective (or defensive) factors include mucosal

bicarbonate secretion, mucus production, blood flow, growth factors, cell renewal and endogenous prostaglandins [5]. Clinical features of perforation are dramatic. Majority of the patients give previous history of vague pain in the upper abdomen, dyspepsia. Once the perforation has set in, the gastric and the duodenal contents escape through the perforation into the peritoneal cavity. This provokes a widespread peritoneal irritation called "peritonism". At the initial stage the victim cries in agony. The peritoneum reacts to this peritoneal irritation by secreting peritoneal fluid copiously. This outpouring of the fluid relieves the pain. This is the stage of reaction following which is diffuse bacterial peritonitis. Peptic ulcer disease constitutes of Gastric ulcer and Duodenal ulcer. These are considered as a single entity but when looked upon individually, both have different identity. Most of the features are common but variations are present.

The incidence of duodenal ulcer perforation is higher than gastric ulcer perforation. Mortality rate is greater in gastric ulcer perforation than duodenal ulcer perforation (10% - 40%) [6]. Time lapse between perforation and treatment is of crucial importance. Surgical intervention within 6 hours of perforation carries a very low mortality which rises steeply as duration extends beyond 12 hours. Many

small perforations, on favourable occasions get sealed off spontaneously while perforations of large size do not often seal itself. Multiple perforations although rare carry a grave prognosis. 90% of duodenal perforations are situated anteriorly on the first part of duodenum, 5%-10% on the posterior wall whereas 60% of gastric ulcer perforations are situated anteriorly on lesser curvature while 39% are all over the stomach, rest 1% on the posterior wall. Gastric perforations carries Worse prognosis than duodenal perforation [5].The association of haemorrhage, haematemesis or malena with perforation is not so common but greatly increases the mortality and morbidity rate of this disease. Despite recent improvements in the management of peptic ulcers and its perforations, duodenal perforation and gastric perforations carries a definite morbidity and mortality rates which vary from 3%-10% as determined by different workers.

### Objectives

To evaluate and compare the incidence of gastric and duodenal perforation. To evaluate and compare the causes of gastric and duodenal perforation.

### Material and Method

This was a retrospective comparative study which included 153 patients who were admitted and treated in the Upgraded Department of General Surgery at Darbhanga Medical College, Darbhanga for the management of peptic perforation peritonitis between July 2019 to December 2020.

### Inclusion Criteria

Patients presenting with features of perforation peritonitis having peptic perforation.

### Exclusion Criteria

Patients having sealed peptic perforation (no apparent perforation seen) detected during operation and patients having traumatic upper gastrointestinal perforations.

Diagnosed case of peptic perforation not giving consent.

Analysis was done using 1 way ANOVA with post hoc tukay's Software used was SPSS.

### Presenting Complaints at Time of Admission

- Pain abdomen
- Distention
- Nausea/vomiting
- Passage of flatus and faeces
- Haematemesis or malena

### History of Present Illness

- **Pain abdomen**
- Mode of onset: sudden or gradual.
- Time of onset.

- Duration of exacerbation of ulcer symptoms and conditions preceding the perforation.
- Site, Progress, Character, Radiation, Relation to food, aggravating factors, Relieving factors.

### Nausea/ vomiting

- Its relation with pain
- Frequency and quantity
- Quality: bilious / non-bilious / feculent/ food / blood
- Character and colour

### Distension

Abdominal girth measurement

### Bowels

For the passage of flatus, faeces, malena, blood or mucus in stool.

### Past history

History was taken for, Peptic ulcer disease with duration and treatment, Symptoms suggestive of peptic ulcer, Previous history of dyspepsia and treatment, Previous episodes of pain abdomen, History of haematemesis or malena, Chronic medical or surgical disease

### Drug history

For chronic medical and surgical illness, Drugs like aspirin, NSAIDs, anti-malarials etc

### Physical Examination

#### General Examination

Built, Nutrition / dehydration, Pulse, Temperature, Blood pressure, Respiration, Pallor, Oedema, Jaundice, Tongue, Face

#### Systemic Examination:

All the systems were examined especially the cardiovascular and the respiratory system. Special attention was given to detect diaphragmatic pleurisy and pneumonia.

#### Provisional Diagnosis

#### Investigations

Routine hemogram: TLC / DLC / Hb% / BT/ CT/ ESR

Others: blood sugar, serum electrolyte, serum amylase, blood grouping and cross matching, Renal function test, Liver function test

#### Final diagnosis

#### Intra Operative Findings

Site of perforation, Size of perforation, Procedure Biopsy taken or not

#### Post-operative Period

Complications if any, Discharged or death

**Results****Table 1: Annual Incidence**

Year	Percentage of peptic perforation
2015	57.60
2016	58.02
2017	46.06
2018	44.32
2019	39.10
2020	35.66

The annual incidence was 35.66% incidence. Incidence of peptic perforation peritonitis has decreased in recent past.

**Table 2: Incidence of gastric and duodenal perforation**

Site of Perforation	No. of cases (n = 153)	Percentage
Gastric Perforation	47	30.72
Duodeneal Perforation	106	69.28

Most of the perforations were duodenal perforations with ratio of duodenal perforation: gastric perforation 2.26:1.

**Table 3: Socio-economic status**

Socio- Economic Status	No. of Duodenal perforation (n=106)	Percentage of Duodenal Perforation	No. of Gastric Perforation (n=47)	Percentage of Gastric Perforation
Low	70	66.04	29	61.7
Middle	28	26.42	11	23.4
High	8	7.54	7	14.9

Low socio-economic status patients had a higher incidence rate (D.U.P 66.04%, G.U.P 61.7%) as compared to middle and high economic status respectively.

**Dietary Habit**

Dietary Habits	No. of Duodenal perforation (n=106)	Percentage of Duodenal Perforation	No. of Gastric Perforation (n=47)	Percentage of Gastric Perforation
Vegetarian	14	13.21	6	12.77
Non-Vegetarian	92	86.79	41	87.23

Non-Vegetarians have higher incidence as compared to vegetarians.

**Table 4: Size of perforation**

Size of Perforation	No. of Duodenal perforation (n=106)	Percentage of Duodenal Perforation	No. of Gastric Perforation (n=47)	Percentage of Gastric Perforation
< 2 mm	27	25.47	9	19.15
2 mm – 5 mm	67	63.21	11	23.4
6 mm – 10 mm	7	6.6	24	51.06
>10 mm	5	4.72	3	6.39

Most of the patients of gastric perforation had rent size between 6-10 mm (51.06%), where as in duodenal perforation maximum patients had smaller size of rent 2-5 mm (63.21%).

**Table 5: HPE report**

H.P.E Report	No. of DUP (n=106)	% of DUP	No. of GUP (n=47)	% of GUP	p-value
Acute Inflammatory Lesion	106	100	38	38.05	0.00
Malignancy (Adenocarcinoma)	0	0	4	8.51	0.02
Acute Inflammatory Lesion with fungal element (Candida albicans)	0	0	5	10.64	0.01

Incidence of malignancy was seen in 8.51% of gastric perforation on histo-pathological report of biopsy taken from the edge. Fungal element was seen in 10.64% of cases of gastric perforation.

**Table 6: Causes of death**

Cause	No. of Duodenal perforation (n=18)	Percentage of Duodenal Perforation	No. of Gastric Perforation (n=11)	Percentage of Gastric Perforation
Septic peritonitis	7	38.88	3	27.27
Shock	5	27.77	3	27.27
Leakage	1	5.56	2	18.18
Aspiration Pneumonia	2	11.11	0	0
Renal failure	1	5.56	2	18.18
Medical illness	2	11.11	1	9.1

Most common cause of death in duodenal perforation was due to septic peritonitis (38.88%) but in gastric perforation shock and peritonitis were seen to be the most common cause of death (27.27%).



**Gas under diaphragm**



**Duodenal perforation**



**Duodenal perforation**



**Gastric perforation**

**Table 7: Mortality rate**

Post-operative outcome	No. of Duodenal perforation (n=106)	Percentage of Duodenal Perforation	No. of Gastric Perforation (n=47)	Percentage of Gastric Perforation
Alive	88	83.02	36	76.6
Death	18	16.98	11	23.4

Mortality was more in gastric perforation (23.4%) as compared to duodenal perforation (16.98%).



**Gastric Perforation**

### Discussion

The total number of cases of peritonitis of different etiology admitted in our surgical emergency during the time period was 429. Out of which 153 cases were of peptic ulcer perforation peritonitis. Thus the incidence of peptic ulcer perforation in our study was 35.66%. Sharma et al (1981) found that the most common cause of peritonitis was peptic ulcer perforation [6]. Bhale Rao (1983) found 32.9% cases of peptic ulcer perforation [7]. Rajendra Singh Jhobta, et al (2006) reported that most common cause of surgical emergency in India is peptic perforation peritonitis. Rajandeep Singh Bali, et al (2014) reported 45% cases and commonest cause of peritonitis. Thus the findings in the present series are in fair agreement with findings of the above workers. As per our hospital records the cases of peptic ulcer perforation in 2015 was 57.6%, in 2016 was 58.02%, in 2017 was 46.6%, in 2018 was 44.32%, in 2019 was 39.1% and in 2020 was 35.66%. Thus it is seen that the annual incidence of peptic ulcer perforation has decreased. Svanes C (2000) reported a decrease in the incidence of peptic perforation by 4- 11% annually over the last decade. Hermansson M et al (2009) suggested that incidence of peptic perforation has fallen considerably in recent years [8]. Most common site of perforation was in 1<sup>st</sup> part of the duodenum (69.28%). Gastric perforation was 30.72%. All perforations were anterior in position. Duodenal perforation was more than gastric perforation but the incidence of gastric has increased. Cherian J V, et al (2010) reported that incidence of duodenal perforation is more common but there is rise in incidence of gastric perforation in Indian subcontinent [9]. Wysocki A, Budzynski P, Kulawik J et al (2011) reported that previously predominated duodenal perforation is now shifting

to gastric perforation. In duodenal perforation, the maximum age incidence occurred between 31-40 years (36.79%) followed by 41-50 years (25.47%). The minimum age recorded was 11 years and maximum was 76 years. In gastric perforation, the maximum age incidence occurred between 41-50 years (36.17%) followed by 51-60 years (23.4%). The minimum age of presentation was 12 years and maximum was 74 years. Thorsen K, Soreide JA, et al (2013) reported in their series that most of the peptic ulcer perforations irrespective of location occurred in middle age group between 30-50 years [10]. mSujit M Chakma, et al (2014) reported in their series that most of the patients of peptic perforation peritonitis were in between middle age group in North East India. Next were patients in between age group 55-65 years with more of gastric perforation in comparison to duodenal perforation. Biopsy was taken from the margins of the perforation for both duodenal and gastric perforations and was sent for histopathological examination.

All the HPE reports of duodenal perforation showed acute inflammatory lesions. The reports of HPE of tissue from gastric perforations showed variations. 80.85% of reports showed acute inflammatory lesions. 8.51% had adenocarcinoma and 10.64% of reports showed fungal elements (hyphae). Stechnberg I, et al (1981) reported that malignancy in gastric perforation was up to 6%. Lehnert T, et al (2000) reported that 10-16% of all gastric perforations are caused by gastric carcinoma [11]. Our study shows near resemblance to the studies mentioned above. Fungal cause of perforation (*Candida albicans*) is a rare cause. *Candida albicans* is ubiquitous fungus present in healthy individuals and normal commensal of the gastro-intestinal tract.

Candida infections generally develop when the host's immune response is compromised with malignant tumours, diabetes, patients on steroids, extreme of ages, malnourishment, or immune-suppressant drugs. Ears P, et al (1972) reported 4.35% of Candidal infection in gastric perforation [12]. Tsukamoto H, et al (1986) reported 5.9% incidence of Candidal infection as a cause of gastric perforation. Our study had patients who were diabetic, old age and who were suffering from gastric perforation malnourished teenager due to Candida albicans. In our study, mortality rate for gastric perforation (23.4%) was higher than duodenal perforation (16.98%). Age related mortality was seen in elderly patients with maximum in age group 51-60 years. Mortality above age group 50 years was more in gastric perforation (74%) than duodenal perforation (55%). Most common cause of death was peritonitis and shock in post-operative patients for both duodenal and gastric perforation. Kocer B, et al (2007) reported a mortality rate of 37.7% in age above 65 years. mLau J Y, et al (2011) reported mortality in peptic perforation to be around 30%. [13] Buck D L, et al (2014) reported a mortality rate of 28% in elderly patients [14]. Sarcide K, Thorsen K, et al (2014) reported mortality rate from 20-30% as age advances. Rajshekher Patif, et al (2015) reported a mortality rate of 44% on age group above 60 years [15].

### Conclusion

Gastric and duodenal perforations are common presentations of peptic ulcer disease but each one of them have their own individuality. The incidence of peptic ulcer perforation had decreased in the past decade but the burden of disease still remains in our society. The incidence of gastric ulcer perforation has increased in the past few years and much still is needed to evaluate the cause and management of the disease. The major risk factors that remain at large are, due to ignorance and lack of awareness that the common people have. The elderly and the low socioeconomic status population are the ones at major risk even though no age group is free from the risk of the disease.

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