



ORIGINAL RESEARCH PAPER

General Surgery

CLOSURE OF LARGE SIZED DUODENAL ULCER PERFORATION BY INTRALUMINAL OMENTAL INVAGINATION - A CLINICAL STUDY

KEY WORDS: Duodenal ulcer, different methods of closure large perforation.

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ABSTRACT

Background: Peptic ulcer disease is a function of derangements in intraluminal aggressive factors and defects in endogenous defense mechanisms. The modern management of peptic ulcer disease, based on the understanding of the vagal drive for acid secretion.

Aims and objectives: In this study we have adopted a different method for closure of large perforation (> 5 mm diameter) by "Omental invagination into the duodenum".

Materials and Methods: Study was carried out in the Upgraded Department of Surgery, Darbhanga Medical College & Hospital, during the period 2014-2017 on 50 patients who presented in the emergency with duodenal Ulcer perforation of more than five mm diameter as assessed intra-operatively.

Result: In our study, most common age group was 31-40 years. Majority of patients were male. Epigastric pain was the most common presenting symptom followed by absolute constipation and abdominal distension. 60.0% of patient had past history of alcohol intake while 50.0% had acid peptic disorder. Gastric outlet obstruction was more in the control group.

Conclusion: In the present study we have adopted a different method for closure of large perforation (> 5 mm diameter) by "Omental invagination into the duodenum" as advocated by M.D. Karanjia (Omental Plug Technique); therapy decreasing chances of omental ischaemia and postoperative leakage and comparing the results with various standard procedures followed in this hospital for closure of large sized duodenal perforation.

INTRODUCTION:

Peptic ulcer disease is a function of derangements in intraluminal aggressive factors and defects in endogenous defense mechanisms [1] Initial management for the complications of peptic ulcer disease was surgical in the 19th century, when Woelfler, Billroth and Van Rydigier described early gastric resection and gastro-enterostomies. The modern management of peptic ulcer disease, based on the understanding of the vagal drive for acid secretion, was started by Dragstedt with his description of vagotomy and its use in peptic ulcer disease [2] The understanding of upper gut – physiology led to the production and wide use of various anti-ulcer treatment such as antacids, H₂ antagonist, proton pump inhibitors. These innovations in the pharmacological management of this disease have vastly overshadowed surgical treatment in the 80's and 90's [3]

Etiopathogenesis of duodenal ulceration is multifactorial, however, acid pepsin and gastric contents attacking the mucosa are considered to be the main culprits. The degree of acid secretions varies with the disease state, still the corner stone of therapy for most routine duodenal ulcers is the diminution of acid which appears to adequately treat 80-90% of patients. [4]

The total amount of acid secretion is not as important as the time that the acid is secreted, especially when patients have high secretory volumes in a basal state. Basal secretion in ulcer patients, is usually high and eradication of basal secretion, particularly, night time secretion can be sufficient to treat most peptic ulcers [5] Duodenal ulcer patients are known to have larger parietal cell mass (Lee et al, 1993), which may be genetically determined. A family history of a parent or sibling with duodenal ulcer renders a person three times more likely to develop the condition.

Perforation, bleeding and stenosis are the usual complications of a chronic peptic ulcer, each one of them can occur without any previous history. Perforation and bleeding may be present de novo from acute ulceration. These complications add considerably to the morbidity and mortality of duodenal ulcer disease.

After perforation, duodenal contents escape through the perforation into the general peritoneal cavity resulting into peritoneal irritation or peritonism. Peritoneum reacts to this chemical irritation by exudation of copious amounts of watery

fluid, which gives the patient some relief of pain. This stage lasts for approximately 3-6 hours and if not treated will lead to diffuse bacterial peritonitis, after which, if not treated properly, the course is fatal. Patient dies due to septicemia and peripheral vascular failure. If treatment is started early with intravenous fluids, antibiotics, surgical closure of perforation with a thorough peritoneal lavage, the mortality gave to a significantly lower level [5] Various methods have been proposed from time to time for surgical closure of perforated duodenal ulcer with variable results such as : Simple ,Cellan Jones Closure Closure by Falciiform ligament ,Graham's Closure Laparoscopic omental patch repair, Combined laparoscopic endoscopic method using an omental plug However those techniques are not without their drawbacks, especially while managing large perforations (> 5 mm size). Mortality rates of upto 18% have been reported while managing large sized duodenal perforations by the standard techniques. Thus there is a need to find, evaluate and apply methods of managing these catastrophes [6]. In the present study we have adopted a different method for closure of large perforation (> 5 mm diameter) by "Omental invagination into the duodenum" as advocated by M.D. Karanjia (Omental Plug Technique); therapy decreasing chances of omental ischaemia and postoperative leakage and comparing the results with various standard procedures followed in this hospital for closure of large sized duodenal perforation. [6]

AIMS AND OBJECTIVES:

In the present study we have adopted a different method for closure of large perforation (> 5 mm diameter) by "Omental invagination into the duodenum" as advocated by M.D. Karanjia (Omental Plug Technique).

MATERIALS AND METHODS:

This work entitled "CLOSURE OF LARGE SIZED DUODENAL ULCER PERFORATION BY INTRA-LUMINAL OMENTAL INVAGINATION – A CLINICAL STUDY" was carried out in the Upgraded Department of Surgery, Darbhanga Medical College & Hospital, during the period 2014-2017 on 50 patients who presented in the emergency with duodenal Ulcer perforation of more than five mm diameter as assessed intra-operatively. All patients of both sexes, with perforation in the anterior wall of 1st part of duodenum, size varying from 0.5 cm to 2.5 cm were studied under following headings after dividing them randomly into study group (omental invagination) and control group (omental patching) consisting of 25 patients each.

These patients were assessed clinically with particularly emphasis on Age, Sex, Address, Occupation, Duration of acute pain, vomiting, absolute constipation, distension of abdomen and fever.

History of – Pulmonary Tuberculosis, Hypertension, Diabetes Mellitus, Ischaemic heart disease, regular intake of NSAIDS, steroids, alcohol.

Appearance of patients, pulse, respiratory rate, temperature, blood pressure were noted with special attention to the extent of toxemia and fluid and electrolyte imbalance.

The operations were conducted under General Anaesthesia with the patients in supine posture, and same suture materials were used in all the patients.

All observations were carefully recorded on a specially designed case sheet and the results were compared using

statistical methods. We have compared this technique of closure with one of the standard techniques (omental patching) of closure for large sized perforation (>5 mm) post operative parameters of comparison are (1) Haemorrhagic Ryles tube aspirate, (2) Leakage rate from closed perforation, (3) Development of postoperative gastric outlet obstruction as assessed clinically, (4) Total stay in hospital (days).

RESULT:

in our study Predominant age group was 31-40 years, Majority of patients were male, lower socio-economic status. Epigastric pain was the most common presenting symptom followed by absolute constipation and abdominal distension [Table 1]. Long standing perforation was the most common risk factor present in majority of patients [Table 2] Majority of perforations were in the range of 5-8 mm size and round in shape [Table 3] Absence of bowel sound was the most common clinical sign followed by rigidity and tenderness [Table 4]

TABLE : 1

S.No.	Symptoms	Study Group		Control Group		Total	
		Number	(%)	Number	(%)	Number	(%)
1	Epigastric Pain	25	100.0	25	100.0	50	100.0
2	Abdominal Distension	20	80.0	22	88.0	42	84.0
3	Absolute Constipation	21	84.0	22	88.0	43	86.0
4	Vomiting	10	40.0	8	32.0	18	36.0
5	Fever	6	24.0	5	20.0	11	22.0

TABLE – 2

S. No.	Past History	Study Group	Control Group	Total
(A) Past illness :				
1.	Pulmonary TB	2	1	3
2.	Hypertension	2	3	5
3.	Diabetes Mellitus	1	2	3
4.	IHD	1	2	3
5.	Acid Peptic disorder	13	12	25
(B) Intake of :				
1.	NSAIDS	7	5	12
2.	Steroids	1	2	3
3.	Alcohol	16	14	30

TABLE – 3

S. No.	Investigation	Study Group	Control Group	Total	
				No.	%.
1	X-ray Abdomen (Erect) showing gas under Diaphragm	20	21	41	82.0
2	Ultrasound Abdomen	5	4	9	18.0
	• Gas	5	4	9	18.0
	• Free fluid	5	4	9	18.0

TABLE – 4

S. No.	Investigation	Study Group	Control Group	Total	
				No.	%.
1	Bowel sounds absent	24	24	48	96.0
2	Rigidity	22	23	45	90.0
3	Tenderness/Rebound Tenderness	20	23	43	86.0
4	Masking of liver dullness	21	19	40	80.0
5	Distension of Abdomen	20	22	42	84.0
6	Presence of free fluid (Clinically)	9	7	16	32.0

DISCUSSION:

50 patients of all age groups and both sex who presented in the central emergency of Darbhanga Medical College & Hospital with perforated duodenal ulcers of more than 5 mm diameter were taken. Of the total 50 patients half were in the 4th decade of their life, while 28% were in the fifth decade. The sex ratio (Male : Female) in the present study was 7.1 : 1. These

findings were in agreement with that of Hug and Love and Bailey. Both of them mentioned peak incidence in middle aged. Goenka (1991) also reported that half of his cases were more than 40 years of age, however he mentioned Male : Female ratio of 4.2 : 1 contrary to our findings. Sabiston (1997) and Schwartz (7th edition) reported a changing trend in peptic ulcer disease with more number of elderly and female patients. the reasons for this may be the socio-cultural difference between India and the western world. [7]

Two third (64%) of our patients were from the lower socio-economic status with majority of them from rural background. Middle and upper socioeconomic status patients contributed to only 16% in the present study.

This may be due to illiteracy and ignorance as well as high rate of alcohol abuse in rural and lower socio-economic strata. Psychological stress to meet out the day to day needs of the life might also be playing an important role in causation of peptic ulceration.

The single most important presenting symptom in this study was acute severe Epigastric pain present in all of the patients. Sabiston (1997), Bailey and Love (2000) also mentioned epigastric pain as the most common presenting symptom in duodenal ulcer perforation. In two thirds of the patients, duration of epigastric pain was between 24-48 hours in total 84% of the patients presented after 24 hours of starting of pain. The reason for this late presentation was that most of the patients were from rural areas, which are not easily accessible, secondly ignorance on the part of patient, their families and local quacks further delays the transfer of the patients to a higher center, thirdly our center being a tertiary referral center receives mostly delayed and complicated cases refused by other practitioners. [9]

Other presenting symptoms in order of decreasing frequency were absolute constipation (86%) abdominal distension (84% patients), vomiting (36%) and fever (22% patients). These findings support the views of other authors.

During general examination (Table 6) 84.0% (42 patients) of patients were found to have tachycardia, 90.0% (45 patients) were found to have tachypnoea, 78.0% (39 patients) were having hypotension, while pallor was present in 62.0% (31

patients). 64.0% (32 patients) of patients were found to have normal temperature. These findings of tachycardia, sudden onset severe generalized abdominal pain and shock in a patient of peptic ulcer perforation are in agreement with that of Love and Bailey.[11]

14 patients had one or the other concurrent medical illness as diabetes, hypertension or Tuberculosis. These conditions do not directly play a role in etiology and causation of peptic ulceration but contribute to the seriousness of the illness by weakening the defense system of the patient and making them more vulnerable to other diseases.

30% (15 patients) of our patients had given a history of intake of either NSAIDs or NSAIDs and oral steroids both some of whom were suffering from arthritis. Thus making a substantial contribution towards the causation of peptic ulceration and later on perforation. This supports the view of Johnsson (1979) who proposed that NSAIDs being a Prostaglandin inhibitor disrupt the Prostaglandin driven support of the mucosal barrier, thus predisposing it to the injury due to gastric acids. Alcohol abuse, another important factor in causation of peptic ulceration was positive in 60% (30 patients) of our patients. It is known both to stimulate parietal cell secretion of acid by increasing histamine release and to be a cause of acute gastritis. [12]

A previous history of Acid peptic symptoms were present is only 50% (25 patients) of our patients. This is far less than 90% incidence of APD as reported by Hug (1990).

On abdominal examination, Bowel sounds were absent in 96% (48 patients) of the patients while rigidity and tenderness were present in about 90% of patients. Other important clinical signs were masking of liver dullness 80% (40 patients), distension of abdomen in 84% (42 patients) and presence of free fluid in abdomen clinically in 32% (16 patients) of the patients. These findings are in agreement with that of Sabiston, Bailey and Love and other authors.

On investigating the patients, two patients were found to be severely anaemic while mild to moderate degree of anaemia was present in half of the patients. Total Leucocyte count showed a rise in 94% (47 patients) of the patients, with Neutrophilia in 88% (44 patients) of the cases and lymphocytosis in 6% (3 patients) of the cases. Blood urea was raised marginally in four patients while in two patients who were in a state of shock it was raised markedly (80 mg/dl).

On X-ray examination of abdomen in erect posture, gas under diaphragm was seen in 82% (41 patients) of the patients. Sabiston reported that gas under diaphragm was seen in 70% of cases. Schwartz (23rd edition) reported Pneumoperitoneum in 75% of cases. 9 patients in whom gas under diaphragm could not be detected on abdominal X-ray in erect posture were subjected to ultrasonic examination of abdomen which revealed presence of free fluid and gas in all the patients thus giving a sensitivity index of 100% In the present study, five major risk factors were identified. More the number of risk factors in a patients, poorer the prognosis.

Boey-Wong and Ong (1987) identified three risk factors namely:

1. Presence of serious concurrent medical illness.
2. Preoperative shock
3. Perforation of more than 24 hours duration.

They reported a mortality rate of 10% with any one of the risk factor, 45.5% with two and 100% with all three risk factors. Sawyers et al mentioned two more risk factors.

1. Gross suppurative peritonitis
2. Age > 70 years with ulcer history of several months. All these five risk factors were considered in the present study.

In the present study patients with perforation in anterior wall of 1st part of duodenum were taken 70% (35 patients) of the patients were having perforation between 5-8 mm in diameter and remaining 30% (15 patients) were having perforations of more than 8 mm in diameter majority of the perforations were round in shape; with ten patients having indurated margins.

Various post-operative complications that developed in Study Group (Intraluminal omental invagination) and control group (Omental patching) patients showed:

Haemorrhagic Ryles Tube aspirate in 8% (2 patients) patients of study group and 4% (1 patient) of control group. All these patients were managed conservatively with blood transfusions and monitoring of vital signs and all of them survived.

While there was no re-leakage from perforation site in study group postoperatively, 12% (3 patients) patients in control group developed this complication. Several factors may have played a role, which appear to be perforation of long standing duration (>24 hours), pre-operative shock, anaemia hypoproteinaemia complex, indurated margin of perforations and large size of perforation (all three patients having > 8 mm size of perforation). These results are in agreement with those reported by Kalpesh Jani and A.K. Saxena et al who used same technique for closure of large sized duodenal perforation.

Post operative mortality was 8% (2 patients) in study group and 20% (5 patients) in control group.

One of the patients of study group who died had active T.B of chest, anaemia (Hb-4.9 gm%), perforation of long duration and eventually succumbed to persisting septicaemic shock and chest infection on 3rd post operative day. Another patient of study group had history of acid peptic disorder, hypertension, alcohol intake, preoperative shock, perforating of long duration (72 hours) and eventually died due to gross suppurative peritonitis and continuing septicaemic shock on 4th day. Among control group 3 of the patients who developed re-leakage from perforation site eventually died due to septicaemia. One patient who died had history of ischaemic heart disease, diabetes mellitus, and alcohol while other had anaemia (4.9 gm%), long standing perforation (24 hours) and presented with shock. (Non palpable pulse and non recordable B.P.) thus taking mortality to 20% (5 patients) in control group.

Average hospital stay in study group was 11 days and in control group was 13 days. It was a bit longer in control group due to greater number of patients of control group developing serous and purulent discharge from wound and pelvic abscess which was drained per-rectally resulting in improvement in patients condition. Clinical follow up of all patients was advised at 4 weeks and 3 months.

CONCLUSION:

Maximum incidence of perforation of duodenal ulcers occurred during 4th decade of life Disease are common in male and poor socio-economic status and of rural background. Alcohol consumption and Positive history of NSAID and/or steroid was are most common Risk factor. Five major risk factors were identified which can affect the prognosis of the patients. These are Age > 70 years, Associated serious medical illness, Preoperative shock, Long standing perforation.

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