



A study to assess the association of clinical predictors with leak after simple closure of duodenal ulcer perforation

General Surgery

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ABSTRACT

Peptic ulcer is one of the commonest diseases of GIT. Two main types are gastric and duodenal. In India ratio of duodenal to gastric ulcer is 30:1 (Rains & Mann – 1984). Duodenal ulcer is predominantly a disease of adult males. The aim was to find out the risk factors for the development of the leakage after simple closure of duodenal ulcer and to assess the association of clinical predictors associated with higher number of complications. The materials for the present work comprised of patients with perforated duodenal ulcer admitted in my department through surgical emergency of Patna Medical College and Hospital between the periods from August 2014 to July 2017.

KEYWORDS:

Peptic ulcer, perforation, simple closure, Duodenal ulcer, gastric ulcer

INTRODUCTION-

Acute perforation of a duodenal ulcer is one of the most common complications of duodenal ulcer. Ulcer perforation occurs in 15% of patients with a proved duodenal ulcer over a period of 5 yrs (Thomson et al 1960). Among perforations duodenal ulcer perforation outnumbers gastric ulcer perforation in a ratio 7.7:1 (watkins et al, 1984).

Acute free perforation of a duodenal ulcer can occur only when it perforates a free serosal surface usually interiorly or laterally following acute on chronic transmural ulceration. The size of perforations is usually proportional to the duration of ulcer. Following perforation: spillage of gastric juice, bile, pancreatic juice and intestinal juice into peritoneal cavity results in chemical burn of peritoneum, which has surface area nearly equal to that of body surface. Extent and intensity of it are directly proportional to character, amount and duration of spillage. Effects of spillage include an acute reaction reactive effusion of modestly dilute plasma into peritoneal cavity and juxta-peritoneal areolar tissues. Further consequence of inhibitive ileus is associated with pooling of intraluminal and mural enteric fluids. All these contribute to production of hypotension which reflects severity of the process. Effects of these are tremendous especially in aged who has reduced capacity to resist and type of metabolic on slaughter patients having associated medical illness. The condition complicates further when secondary bacterial infection which usually occurs late, supervenes.

Emergency management depends on stoppage of this peritoneal spillage, though there are flurries of interest in non operative management (Mukker, 1939 Taylor et al. Al 1952, sheely et al 1956) where spillage is controlled by continuous nasogastric suction, the general consensus of surgeons is that this catastrophic event should be treated by simple surgical closure. Later on a new era of performing definitive surgery in case of perforation in the form of partial gastrectomy (Harberer 1919, Judin 1937, cldey et. at 1955 etc) or truncal vagotomy with drainage procedure (Pierondoiri et al, 1952 Jorden et al 1966 etc), still later proximal gastric vagotomy (Johnston et al 1973. Sawyers et al – 1977, boey et al 1987 etc) have come in vogue.

Despite modern development of surgical management, overall mortality varies from 2.9% to 13.2%. This disparity reflects varying proportion of risk factor in individual series. Boey et al (1987) described associated medical illness, preoperative shock and perforation more than 24 hours duration as independent risk factors

which determined progress of cases of perforated duodenal ulcer, while Gogers F (1960) reported in addition to above three risk factors, advanced age, female sex, large size of perforation, excessive peritoneal contamination were other factors significantly increasing mortality and morbidity.

In the present work, factors possibly related to mortality and morbidity were examined prospectively in each patient. In order to assess risk factors when surgical policy was observed, the above factors were evaluated in the entire consecutive series of patients with regard to the simple closure of perforation. It offers further scope of improving surgical management of cases of perforated duodenal ulcers by identifying risk groups so that they can be managed accordingly to improve their outcome.

MATERIAL AND METHODS-

The materials for the present work comprised of patients with perforated duodenal ulcer admitted in my department through surgical emergency of Patna Medical College and Hospital between the periods from August 2014 to July 2017.

All the patients will be studied prospectively and evaluation of different risk factors will be done by taking detailed history, clinical assessment including radiological investigations as per indications and need.

RESULTS-

Table 1- Showing the age incidence of the patients (84 Cases)

Age in Years	No. of cases	Percentage
10-20	Nil	Nil
21-30	9	11%
31-40	18	22%
41-50	21	25%
51-60	26	31%
61-70	7	8%
71-80	3	3%

Table 1 shows that largest group falls in the 6th decade of life closely followed by 5th and 4th decades and majority of cases (78%) are in the age group of (31-60) years.

Table -2 Showing the sex incidence of the patients:

Sex	No. of cases	Percentage
Male	78	93%
Female	6	7%

Out of 84 cases 93% was male and 7% was female, male is to female ratio being 13.4:1.

Table – 3 Showing the symptoms of the patients at the time of admission

Symptoms	No. of cases	Percentage
Pain	84	100%
Nausea, vomiting	60	72%
Obstipation	57	68%

This tables shows that pain was the presenting symptom in all cases. Nausea and/or vomiting was present in 72% cases, while obstipation was present in 68% of cases

Table -4 Clinical signs at the time of admission.

Signs	No. of cases	Percentage
Abdominal distension	63	75%
Abdominal Tenderness	64	76%
Abdominal Rigidity	74	88%
Absent Bowel sound	74	88%
Dehydration	76	90%
Hypotension	24	28%

Table 4 shows that important clinical signs were abdominal distension (75%), abdominal tenderness (76%), abdominal rigidity (88%) and absent bowel sound in (88%) of cases. Dehydration was significant in 90% of cases while severe hypotension was present in 28% of cases.

Table – 5 Showing the analysis of Personal History

Type of Habits	No. of cases	Percentage
Alcoholic	15	33%
Smoker	30	63%
Irregular Bowel habit	13	28%

Out of 84 cases 47 cases (56%) had personal history regarding alcoholism, smoking and irregular bowel habits. Among these 47 cases smoking was very common. It was found in 63% of cases 33% were addicted to alcohol, while 28% had irregular bowel habits.

Table- 6 Showing interval between perforation and operation.

Time in hours	No. of cases	Percentage
0-12 hrs	12	14%
13-24 hrs	37	44%
25-48 hrs	21	25%
More than 48	14	20%

This table shows that 37 cases (44%) had operation done within 13-24 hrs after perforation while 21 cases (25%) within 25-48 hrs, 12 cases (14%) within 0-12 hrs and 14 cases (20%) after 48 hours.

Table – 7 Showing the Incidence of Leak in relation to Sex

Sex	No. of cases	No. of Leak	Percentage
Male	78	3	4%
Female	6	2	34%

This table show that incidence of leak among female was (34%) more than male (4%).

77% of cases had a previous history of peptic ulcer symptoms. Associated relevant medical illness were 6 cases (7%) having diabetes, 3 cases (4%) had ischemic heart disease; 7 cases (8%) had hypertension 6 cases (7%) had chronic Lung disease, 2 cases (3%) had tuberculosis.

Incidence of Leak in cases of duodenal ulcer perforation is mostly seen in 6th decades of age.

37 cases (44%) had operation done within 13-24 hrs after perforation while 21 cases (25%) within 25-48 hrs, 12 cases (14%) within 0-12 hrs

and 14 cases (20%) after 48 hours.

The study shows that Leak was more if operations performed after 24 hours of perforation. shows that hypotension significantly affects the chance of Leak.

The commonest immediate postoperative complication was wound infection in 16 cases (19.4%), following by respiratory complications in 11 cases (13%), where as reperforation or leak was found in 5 cases (6%) while wound dehiscence, renal failure, burst abdomen and death were found in 10%, 4% and 10% cases respectively.

It shows that when the no. of complications were 1 to 3 in an individual the interval of operation from perforation were > 24 hrs in about 50% of cases and it raised sharply to 100% when no. of complications was 4 in individual.

DISCUSSION-

Cases of perforated duodenal ulcer admitted in my department through surgical emergency of Patna Medical College and Hospital, Patna between August 2014 to July 2016. All the cases have been studied as thoroughly as possible. It was mainly a clinical study to evaluate risk factors associated with development of complications after simple closure of duodenal ulcer perforation. All the cases were operatively proved cases of perforated duodenal ulcer. As far as possible, different aspects have been dealt with, to obtain a clear clinical profile. As it is a grave emergency, minimum reliance was kept on ancillary aids. Nevertheless certain routine investigations were done and studied in order to come to a conclusion.

In this series majority of cases of perforated duodenal ulcer were found between the age of 31 to 60 years The peak age incidence has been reported to be 40-49 years by Graham RR (1937); 44.6 years (mean age) by Booth et al (1971); 51.3 + 17.7 years by Boey et al (1982); 44 years (mean) by Tamphiphat et al (1985) etc..

The diagnosis of perforation was substantiated and correlated by presence of history of peptic ulcer symptoms in majority (78%) of cases. The duration of symptoms beign between 2 to 5 years in majority (53%) of them (Table 6 and 7). This was comparable with Mitra (1982) who reported 72% cases of duodenal perforation with History suggestive of peptic ulcer. Tonphipha et al (1985) reported 66% of cased with history of ulcer symptoms.

In analysis of personal history (Table – 8) history of alcoholism smoking or irregular meal habits were found in 47 cases (56%). Among these smoking was present in 63% cases, alcoholism in 33% of cases and irregular meal habits in 28% of cases. This association indicates that these factors have some definitive role in pathogenesis of duodenal ulcer. Rains and Mann (1988) described that smoking conclusively delayed ulcer healing and irregular meal habits, alcoholism and accessory role in etiological factors.

Out of 84 cases, immediate postoperative complications were encounter in 28 cases (33%) and total no.of complicatins was 57. Out of these wound infection was being the commonest complication (19%) next in the series were cases of respiratory complication which occurred in 11 cases (13%) wound dehiscence, Burst abdomen, leak and renal failure were present in 8 (10%) of cases (Table – 25). Wongensteen el at (1972) described in their series post operative complications of 14% intra abdominal abscess and pneumonia being commonest complications. Boey et al (1982) reported wound infection and pulmonary complications as the commonest complication. Kohli et al (1988) reported 26% cases of post operative complications, the commonest being wound infection and chest infections.

In the present series the overall mortality was 10% (Table – 25) nearby similar overall mortality was found by many author such as 9.5% by Mastings and Machida (1961); 10% by MC Conough et al (1972); 8% by Kohli et al (188).

CONCLUSION-

Cases of duodenal ulcer perforation present as acute abdominal catastrophe with high mortality rate which is 10% in this series. There were 5 cases (6%) that had developed leakage after simple closure Out of 5 cases 3 cases expired in post operative period due to septicemia. Rest two cases were managing one by operative procedure and other by conservative method. Factors affecting chances of survival from duodenal perforation are multiple and interrelated. Advanced age, female sex, perforation in full stomach, severe dehydration,

perforation > 24 hours, chronicity of ulcer, large size of perforation, associated medical illness and large amount of peritoneal spillage are factors found to increase rate of leak and mortality. Among this severe dehydration, preoperative hypotension, associated medical illness, status of stomach at the time of perforation and size of perforation are factors which dramatically increase the rate of leak. Risk of an individual patients increases with increase in number of these factors present in the case. So it is evident that rate of development of leak can be lowered down only if all the cases are strictly evaluated for risk factors and managed accordingly

REFERENCES-

1. Au chincloss, M. 1952 : Ann. Surg. , 135., 134-138
2. Benjamin Travers 1816 : Ouoted by Watson, J, Ho, B.M.J., 2: 169, 1930.
3. Bennett, W.H. 1896 : Quoted by Ingram et al Am. J. Surge., 84:30, 1952
4. Berne, C. J. et al 1958 : Surgery, 44:591-603
5. Boey, J. et al 1982 : Ann. Surg. 195:265.
6. Boey, J. et al 1982 : Am. J. surg. 143:635-639.
7. Boey, J. et al 1987 : Ann. surg. 205:22.
8. Boneti, T. 1679 Quoted by colllos D.C. Am.J.surg. 77:684.1949.
9. Booth R. A & 1671 : Br.J.surg 58:42-44.
10. Braun.W 1897 : Quoted by Colllos D.C. Am.J.surg. 77:329.1949.
11. Cassel p. 1969 : Gut. 10:572-574.
12. Ceneviava et al 1989 : Br. J. Surg. 73:427-430.
13. Chatterjee B.D. et al 1981 : Ind. J. surg. 43 (1):87
14. Collins D.C. 1949 : Am. J. surg. 77:684-694
15. Colley et al 1955 : Ann. surg. 141:840.
16. Fean.H.P. 1894 : Quoted by colllos D.C. Am.J.surg. 71:684.1949.
17. Dean ACB et al 1862 : Gut. 3:60-64.
18. Debakey.M.E. 1940 : Surgery, 8:852.
19. Downalson.G.A. & 1970 : Am. J. Surg. 120:306-311. Jarrett f
20. Emmett. J.M & 1953 : Ann. Surg. 138:320.
21. Fellolano. D.V et al 1984 : Am. J. Surg 148: 764-767.
22. Gee.S.M. 1892 : Quoted by Watson.J.H. B.M.J.2: 169.
23. Gllmour.J. 1953 : Lancet. 1:870-873.
24. Graham.R.R. 1937 : Surg. Gyn.obs. 64:235-238.
25. Gray et al 1974 : Am. J. sueg. 127:109-104.
26. Hinshaw, D.W. et al 1968 : AM. J. Surg., 115:173.
27. Hofkin, G.A. 1966 : AM. J. Surg. 111:193-196.
28. Illingworth, CPW.Et.al 1946 : Br. Med. J., 1: 787
29. Ingram, I.N & Ervin, J.R 1952 : A.J. Surg. 123:406
30. Jacopo Pendada 1793 : Quoted by wtson, J.H. B.H.J. 2: 169, 1930
31. Jarret, F & Don 1972 : Am. J. Surg. 123:406
32. Jennings, D. 1940 : Quoted by Hardy, J.D. & walker, J.R. Am J Surg, 153:911, 1961
33. Hohn Abererombe 1824 : Quoted by Collins, D.C. Am. J. Surg. 77, 684, 1949.
34. Johnston, D et al 1973 : Br. J. Surg., 60: 790-797
35. Johnston, P.H. & Koromapai, F.L. 1976 : Surg. Gyn. obs. 142:391-395
36. Jordon, G.L. et al 1966 : Surg. Arch., 105:395
37. Judin, S.S. 1937 : Surg. Gyn. obs. 64:64
38. Kay, P.H. et al 1978 : Br. J. surg. 65:801-803.
39. Kao, I.C. et al 1964 : Surgery, 56:487.
40. Keetly, C.B. 1902 : Quoted by Hastings et al Am. J. surg, 102:136, 1961
41. Kohli, V. et al 1988 : Ind, J. surg. 50:184-186.
42. Kozoll, D.D. & Meyer, K.A 1960 : Surg. Gyn. Obs. 111:607
43. Kohli, D.D. & Meyer, K.A. 1961 : Arch. Srug. , 84:646-661
44. Kozoll, D.D. & Mayer, K.A. 1962 : Arch. Surg., 84:646-661
45. Lowdon A.G.R. 1952 : Lancet, 1:1270