Original Research Article

Study to determine etiological factors and surgical outcome of nontraumatic abdominal hollow viscus perforation

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INTRODUCTION

Hollow viscus perforation leading to peritonitis is a common emergency faced by a general surgeon.1 Late presentation, missed diagnosis and late interventions are frequent causes of morbidity and mortality.2 Now a day’s inadvertent use of NSAIDS and analgesics available over the counter forms one of the most common risk factors.3 Perforation of stomach, duodenum and small bowel forms considerable proportion of emergency than colonic perforations.4,5 Perforation of large bowel represents major surgical challenge to the clinician, because it is rapidly fatal condition, death being caused by sepsis from peritoneal contamination by various aerobic and anaerobic pathogens.6,7 Surgery is mainstay in management of hollow viscus perforations. High index of suspicion is essential to diagnose hollow viscus perforations, because diagnostic delay may result in significant morbidity and mortality.

Thus in an interest to find out etiological factors, to assess the common type of perforations with their clinical presentation, surgical outcome and finally prognosis and complications; forms the basis of present study.
Objectives

Objectives of current study were to study different etiological factors of hollow viscus perforation and to find out different treatment modalities and surgical outcome of hollow viscus perforation.

METHODS

Current study was conducted in our institution which included total 60 cases of acute abdomen with guarding presented to surgery OPD or emergency department and got admitted.

Sample size

Sample size was calculated using formula:

\[ \frac{4pq}{L^2} \]

Where confidence interval was taken as 90%, error was taken as 10% and absolute precision was taken as 5%. Considering the prevalence of most common etiology of hollow viscus perforation i.e. drug induced as 28% calculated minimum sample size was 54.8

Study design, study population and duration

Current study was conducted in tertiary care hospital which included all the patients with acute abdomen and guarding per abdominally. Current study was a prospective study conducted between 1 January 2018 to 31 December 2018 (12 months) with follow up period up to 30 June 2019 (6 months).

Inclusion criteria

Inclusion criterion for current study was all clinically suspected patients with features of hollow viscus perforation of age group 15-75 years.

Exclusion criteria

Exclusion criteria for current study were cases of esophageal and genitourinary tract perforation and patients who do not want to participate in study.

Procedure

Current study was carried out on total of 60 patients presenting with sudden onset pain in abdomen with guarding and rigidity per abdominally for etiology, surgical management and post-operative complications.

Data was collected from each patients which included age, sex, history (pain in abdomen with associated symptoms, history of long term drugs intake), addiction, clinical parameters (fever, duration of symptoms, vitals, guarding, rigidity, tenderness) lab parameters (pre-operative profile, kidney function test, Widal in selected cases), radiological investigation; X-ray abdomen standing, ultrasonography abdomen and pelvis and computerized tomography in selected cases, diagnostic peritoneal lavage in selected cases, surgical management and post-operative complication.

On admission every patient was started on ceftriaxone 1gm Intravenous 12 hourly and metronidazole 500 mg intravenous 8 hourly. Ryle’s tube and Foley’s catheterization was done and patient was posted for emergency laparotomy. If presence of pus noted intra operatively then sample was taken for pus culture sensitivity. After emergency laparotomy we changed antibiotics according to pus culture and sensitivity report. The samples were sent for histopathological examination depending on operation done. Post-operative course of patients and complications if at all was noted. Patients follow up was done after 1 month and 3 months.

Statistical analysis

Statistical analysis was done by using SPSS software version 16.

RESULTS

Age incidence

A total of 60 patients of hollow viscus perforations were studied from January 2018 to December 2018 at our hospital. The youngest patient was 16 years (appendix perforation) and oldest was 75 years (ileal perforation). The maximum incidence of perforation occurred in 30 to 49 years.

Table 1: Age incidence.

| Age (years) | N | %  |
|-------------|---|----|
| <20         | 01| 01.6|
| 20 to 39    | 31| 51.6|
| 40 to 59    | 23| 38.3|
| >60         | 05| 08.3|

Sex incidence

The majority of the patients were female with male to female ratio being 1:1.2.

Site of perforation

Current study revealed the different sites of perforation in the gastrointestinal tract as mentioned in Table 2.

Etiology of perforation

Peptic ulcer was the major causative factor leading to perforation peritonitis (46.4%). This was followed by appendicular (25%) and typhoid perforation (8.3%).
Tubercular perforation and malignancy were the least common causes. Out of 4 patients of sigmoidal perforation, two turned out to be malignant, one was iatrogenic during colonoscopy and the other one was diverticular.

**Habits**

25 patients were chronic smokers and 32 were alcoholics.

**Table 4: Symptoms.**

| Symptoms          | N | %  |
|-------------------|---|----|
| Pain Abdomen      | 60| 100|
| Vomiting          | 50| 83.3|
| Distension        | 45| 75 |
| Fever             | 42| 70 |
| Constipation      | 10| 16.6|

**Table 5: Signs in cases of gastrointestinal perforation.**

| Abdominal Signs               | N  | %  |
|-------------------------------|----|----|
| Distension                    | 45 | 75 |
| Tenderness                    | 60 | 100|
| Guarding/rigidity             | 58 | 96.6|
| Obliteration of liver dullness| 38 | 63.3|
| Absent bowel sounds           | 40 | 66.6|

**General conditions**

Dehydration was seen in 24 cases, tachycardia was seen in 56 cases and shock was observed in 5 cases.

**Signs in cases of gastrointestinal perforation**

Tenderness (100%) along with guarding/rigidity (96.6%) was the classical signs noted in patients with perforation peritonitis.

**Investigations**

X-ray; after clinical diagnosis of perforation, patients were admitted and shifted to radiology department. X-ray chest and erect x-ray abdomen was taken to look for free gas under the diaphragm. This was found in 41 cases (68.3%) in our study.

**Ultrasonography of the abdomen**

It was done in patients in whom clinical signs of perforation were inconsistent. Evidence of perforation was indirect and presence of free fluid with echogenicity was suggestive of perforation. In clinically suspected cases of appendicular perforation ultra-sonography was done to confirm the diagnosis.

**Widal test**

Widal test was found to be positive in 5 cases of ileal perforation.

**Abdominal paracentesis**

It was done in 2 out of 60 cases, where pneumoperitoneum was not evident on X-ray standing abdomen.

**Past history**

Chronic pain in abdomen was seen in 17 cases. History of fever in the recent past was found in 6 cases out of which 5 cases were found to be typhoid positive. History of drug intake (NSAID) was found in 15 cases of peptic ulcer perforation. One patient had history of tuberculosis that had been treated with anti Koch’s treatment.
and CT scan abdomen-pelvis was inconclusive. Abdominal paracentesis helped in taking decision of laparotomy in these cases. The aspirated fluid was also sent for cytological, biochemical (ADA, sugar, protein, LDH, CBNAAT) and microbiology laboratories for further evaluation.

**CT scan of abdomen and pelvis**

It was done in 6 cases where clinical suspicion of perforation peritonitis was present but air under diaphragm was not seen on X-ray.

**Treatment**

General; immediately after establishment of diagnosis and admission blood samples were sent for investigations. ECG was done. Ryle’s tube insertion and Foley’s catheterization was done. Adequate intravenous fluid administration, monitoring of urine output, correction of shock and electrolyte imbalance was done. Intravenous Injection ceftriaxone 1 gm 12 hourly and Injection metronidazole 100 ml 8 hourly were started. Antibiotics were changed in post-operative period as per culture report if surgical site infection is found or patient is in septicemia not responding to above mentioned antibiotics.

Anesthesia; after all preparations and informed and written consents patients were posted for exploratory laparotomy. General anesthesia was given for 26 patients after endotracheal intubation. In 34 cases spinal plus epidural anesthesia was given.

Specific treatment: Graham’s technique of simple closure of the perforation was done followed by omental patch in all 29 cases of the of peptic ulcer perforation. All the cases of appendicular perforation were treated with appendicectomy followed by peritoneal wash with normal saline. Abdominal drain was placed in 9 cases where gross peritoneal contamination was found.

All the 5 cases of typhoid ulcer perforation were found to be in the ileum and 4 cases were treated by simple closure in two layers after trimming the edges and 1 case required resection anastomosis. In all 5 patients tissue was sent for histopathology. One case of tubercular perforation was in the ileum and had to undergo resection of the diseased segment followed by diversion ileostomy. One case had perforation at ileo-caecal region and was treated with Right hemicolecotomy with ileostomy. Anti-tubercular treatment was advised for 6 months. Out of 4 cases of idiopathic causes of perforation, one jejunal perforation was treated with simple closure in two layers with feeding jejunostomy. Two were ileal perforations. One patient was treated by simple closure in two layers, other patient required resection anastomosis. One was caecal perforation where Right hemi colectomy done with end to end anastomosis of ileum with transverse colon. Three patients found to have malignancy on histopathological examination. One was gastric where simple closure with Graham’s patch operation was done. Patient was lost in follow up. Other two patients had sigmoid colon growth. For which resection with adequate margins and anastomosis was done with diversion colostomy. Then patient was referred to oncologist for further management.

In an Iatrogenic perforation, closure in two layers was done with freshening of edges as there was very minimal faecal contamination. Diverticular perforation was found in sigmoid colon where resection and end to end anastomosis was done followed by diversion colostomy.

**Peritoneal lavage**

In all cases where laparotomy was done, peritoneal toilet was performed with normal saline.

**Drainage**

The laparotomies were accompanied by drainage of peritoneal cavity. Out of 60 patients, in 45 cases we kept two drains one in Morrison’s pouch and other in Left pelvis. In 9 patients of appendicular perforation abdominal drain was kept in right pelvis.

**Post operative**

Post operatively all the vitals were monitored and necessary investigations done. Patients were treated with adequate fluids, antibiotics and blood transfusion. Early ambulation, posturing and breathing exercises were practiced in all cases from the second day. Bowel sounds appeared on third or fourth day. Drain removal, Ryle’s tube removal and suture removal was done depending on clinical judgment in individual case.

**Table 6: Complications.**

| Complications        | N | %  |
|----------------------|---|----|
| Wound infection      | 9 | 15 |
| Chest infection      | 4 | 6.6|
| Residual abscess     | 0 | 0  |
| Faecal fistula       | 1 | 1.6|
| Septicemia           | 5 | 8  |
| Burst abdomen        | 1 | 1.6|
| Death                | 5 | 8  |

**DISCUSSION**

The present prospective study was carried out in our institute. 60 cases with diagnosis of hollow viscus perforation were studied. The results obtained in this present study were compared with the previously conducted similar studies.

**Age incidence**

In the present study, the youngest patient was 16 years and oldest was 75 years. The maximum age incidence in
this study was between 20 to 39 years. The Velappan et al observed an age trend between 20 to 39 years.\textsuperscript{7} In Sachin et al study maximum age incident was found in 40-59 years (53.4%).\textsuperscript{9}

### Table 7: Age incidence in different studies.

| Age (years) | Velappan et al\textsuperscript{7} (n=100), frequency (%) | Dinesh et al\textsuperscript{9} (n=60), frequency (%) | Present study (n=60), frequency (%) |
|-------------|----------------------------------------------------------|----------------------------------------------------------|-------------------------------------|
| <20         | 14 (14)                                                  | 05 (8.3)                                                 | 01 (1.6)                            |
| 20-39       | 45 (45)                                                  | 23 (38.3)                                                | 31 (51.6)                           |
| 40-59       | 31 (31)                                                  | 32 (53.4)                                                | 23 (38.3)                           |
| >60         | 10 (10)                                                  | -                                                        | 05 (8.3)                            |

### Table 8: Sex incidence in different studies.

| Sex        | Velappan et al\textsuperscript{7} (n=100), frequency (%) | Dinesh et al\textsuperscript{9} (n=60), frequency (%) | Present study (n=60), frequency (%) |
|------------|-----------------------------------------------------------|--------------------------------------------------------|-------------------------------------|
| Male       | 77 (77)                                                   | 48 (80)                                                 | 27 (45)                             |
| Female     | 13 (13)                                                   | 22 (20)                                                 | 33 (55)                             |

### Table 9: Etiological incidence in different studies.

| Etiology        | Velappan et al\textsuperscript{7} (n=100), frequency (%) | Rao et al\textsuperscript{10} (n=31), frequency (%) | Kumar et al\textsuperscript{8} (n=60), frequency (%) | Present study (n=60), frequency (%) |
|-----------------|----------------------------------------------------------|------------------------------------------------------|------------------------------------------------------|-------------------------------------|
| Peptic ulcer    | 62 (62)                                                  | 21 (68.7)                                             | 13 (41.6)                                            | 29 (46.4)                           |
| Appendicular    | 16 (16)                                                  | 16 (51.6)                                             | 05 (16.2)                                            | 15 (25)                             |
| Typhoid         | 09 (09)                                                  | 07 (22.6)                                             | --                                                   | 05 (8.3)                            |
| Tubercular      | 05 (05)                                                  | 10 (32.3)                                             | 01 (3.4)                                             | 02 (3.3)                            |
| Idiopathic      | 06 (06)                                                  | --                                                   | 10 (32.3)                                            | 04 (6.4)                            |
| Malignant       | --                                                       | --                                                   | 02 (06.4)                                            | 03 (4.8)                            |
| Iatrogenic      | --                                                       | 01 (3.2)                                             | --                                                   | 01 (1.6)                            |
| Diverticular    | 04 (04)                                                  | --                                                   | --                                                   | 01 (1.6)                            |

### Table 10: Percentage of clinical features in different studies.

| Symptoms        | Velappan et al\textsuperscript{7} frequency (%) | Rao et al\textsuperscript{10} frequency (%) | Present study frequency (%) |
|-----------------|-------------------------------------------------|---------------------------------------------|----------------------------|
| Pain abdomen    | 98 (98)                                         | 65 (100)                                    | 60 (100)                   |
| Vomiting        | 86 (86)                                         | 24 (36.9)                                   | 50 (83.3)                  |
| Distension      | 95 (95)                                         | 31 (47.7)                                   | 45 (75)                    |
| Fever           | 95 (95)                                         | 41 (63.1)                                   | 42 (70)                    |
| Constipation    | 7 (7)                                           | --                                           | 10 (16.6)                  |

### Table 11: Percentage of abdominal signs in different studies.

| Abdominal Signs          | Velappan et al\textsuperscript{7} frequency (%) | Rao et al\textsuperscript{10} frequency (%) | Present study frequency (%) |
|--------------------------|-------------------------------------------------|---------------------------------------------|----------------------------|
| Distension               | 95 (95)                                         | 31 (47)                                     | 45 (75)                    |
| Tenderness               | 100 (100)                                       | 65 (100)                                    | 60 (100)                   |
| Guarding/rigidity        | 100 (100)                                       | 52 (80)                                     | 58 (96.6)                  |
| Obliteration of liver dullness | 68 (68)                              | -                                           | 38 (63.3)                  |
| Absent bowel sounds      | 70 (70)                                         | 46 (70)                                     | 40 (66.6)                  |

**Sex incidence**

The ratio of men to women with all types of perforation irrespective of etiology of perforation is 5.9:1. In Velappan et al study whereas 4:1 in Sachin et al and 1.7:1 in Rao et al study.\textsuperscript{7,9,10} But in current study we found slight female preponderance, with female: male ratio of 1.2:1.
Etiological incidence

Peptic ulcer perforation was the major cause of gastrointestinal perforation in our study constituting about 46.4% followed by appendicular (25%), typhoid(8.3%) and tubercular (3.3%). Idiopathic cause constituting for 6.4%. Similar findings of peptic ulcer as most common etiology were reported in Velappan et al and Rao et al study.7,10 Incidence of typhoid perforation was somewhat less in our study compared to other studies. The incidence has reduced mainly as a result of availability of highly effective antibiotics. Incidence of tubercular perforation has also decreased significantly. Incidence of appendicular perforation is slightly higher in our study compared to other studies because of the increased number of patients with low socio-economic status, thus delay in seeking health care.

Clinical features

Pain in abdomen, vomiting, distension and Fever were the predominant symptoms in current study. Pain abdomen was seen in all cases. Similar findings have been reported in Velappan et al and Rao et al study.7,10

Signs

Abdominal distension and guarding/rigidity are two common signs observed in our study. These findings are similarly observed in Velappan et al and Rao et al study.7,10

Investigations

Presence of gas under the diaphragm has been a trademark of hollow viscus perforation but absence of this does not exclude the possibility of the perforation. This sign is visualized in about 41 cases (68.3%) in our study. In Velappan et al study air under diaphragm is seen in 75% of cases and in Dinesh et al study it is seen in 86.7%.7,9

Ultra sound abdomen is readily available, noninvasive investigation but it gives only indirect evidence of perforation through presence of free fluid with echogenicity highly suggestive of perforation. Widal test was positive in 5 (8.3%) cases in our study, which is comparable with Velappan et al study where positive titer was found in 9% patients.7

Treatment

In peptic ulcer perforation simple closure of the perforation was performed with omental patch in all the cases. Most of the literatures are in agreement with the same. For all cases of appendicular perforation appendicectomy was done and most of the literatures suggest the same. For other perforations simple closure of perforation in two layers after trimming of edges, Resection of involved segment with end to end anastomosis are the treatment options depending on intra operative findings. These are comparable other studies and standard literature.

Post-operative complications

Most common post-operative complication we observed is wound infection which is seen in 15% of the patient. It is comparable with Rao et al study where it was 16.9%.10 Chest infection is seen in 6.6% of the patients, which is less than Dinesh et al (33.33%) and Mohan Rao et al (16.9%) study. This is probably due to chest physiotherapy, spirometry, early mobilization and use of nebulization.10

Mortality

Current study reported mortality of 8% (5 patients). The cause of death was mainly due to septicemia with multi-organ dysfunction. Dinesh et al reported mortality of 3.33% in his study and Rao et al 3.1%.9,10 Late presentation, older age group, and co morbidities increases mortality.

CONCLUSION

We studied a total of 60 patients of non-traumatic abdominal hollow viscus perforation, and we concluded that hollow viscus perforation leading to peritonitis is one of the most common emergencies faced by a general surgeon. Most common age group involved is 30-39 years. Peptic ulcer perforation is the most common cause of hollow viscus perforation. Abdominal pain and vomiting were the most common chief complaints and tenderness with guarding rigidity being the most commonly observed sign. Gas under diaphragm on X-ray standing abdomen is suggestive of hollow viscus perforation but, it is not obligatory. Surgery is the main modality of treatment. Wound infection is the most commonly observed post-operative complication. Early presentation, proper diagnosis and timely interventions decrease morbidity and mortality.

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REFERENCES

1. Langell JT, Mulvihill SJ. Gastrointestinal perforation and acute abdomen. Med Clin Am. 2008;92:599-625.
2. Dhika V, Singhal S, Pande S, Chowla A, Anand KS. Non-Steroidal drug induced gastrointestinal toxicity: mechanism and management. JIACM. 2003;4:315-22.
3. Inderbir S. Oesophagus, stomach and intestine, textbook of human histology, 2nd ed. New Delhi; Jaypee Brothers Medical Publisher; 1994:212-25.
4. Janikalpesh A, Saxena AK. Management of large sized duodenal ulcer perforation by omental plugging, a new technique, a prospective study of 100 patients. Ind J Surg. 2000;62(2):134-8.

5. Jhobta RS, Attri AK, Kaushik R, Sharma R. A spectrum of perforation peritonitis in India- review of 504 consecutive cases. World J Em Surg. 2006;1: 1146-9.

6. Kellog LC. A treatise on peptic ulcer perforations. Surgery. 1939;6:524-30.

7. Dhanapal V, Selvam K. Clinical study and management of hollow viscus perforation of abdomen. Int Surg J. 2017;4(5):1773.

8. Kumar VB, Mathew AS. Clinical study of abdominal hollow visceral perforation, non traumatic. J Evol Med Dent Sci. 2014;3(30):8366-71.

9. Dinesh HN, Sachin V, Sreekanth, et al. Clinical evaluation and management of peritonitis secondary to hollow viscus perforation. J Evol Med Dent Sci. 2016;5(19):950-3.

10. Rao M, Samee AA, Khan SM. Hollow viscus perforation: A retro spectrum study. Int J Recent Sci Res. 2015;6(3):3250-4.

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