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Research Article

CLINICO-PATHOLOGICAL STUDY OF NON-TRAUMATIC HOLLOW VISCUS PERFORATION IN TERTIARY CARE HOSPITAL

[1]Dr. Subhash Sharma, [2]Dr. Kunal Malhotra, [3]Dr. Ashok Kumar Kaundal and [4]Dr. Anil Malhotra

[1,2]Resident Doctor, Department of Surgery, Indira Gandhi Medical College & Hospital , Shimla (Himachal Pradesh)
[3]Assoc.Professor, Department of Surgery, Indira Gandhi Medical College & Hospital , Shimla (Himachal Pradesh)
[4]Prof.& Head, Department of Surgery, Indira Gandhi Medical College & Hospital , Shimla (Himachal Pradesh)

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A B S T R A C T

Peritonitis due to perforation of viscus, either traumatic or non-traumatic is an important cause of morbidity and mortality in an emergency. Remarkably, however, only within the last century has significant progress been made in the successful treatment of the disease. The reduction in mortality from 90% at the turn of the century to the estimated 10-15% also includes support of improved and effective antibiotics along with our understanding of inflammatory respo. Nse & there is also a need to know the spectrum of presentation as well as the most frequent among them. Peritonitis as such has such diverse aetiology and thus there is a need to enlist the different a etiologies leading to the disease.

Objectives: The purpose of this study was to evaluate the relative incidence of various causes of perforation and also to find its relation to age, group and sex of the patient. Importance has also been given to the clinical presentation, relevant investigations and various modalities of treatment.

Materials and methods: This study has been based on the analysis of 200 cases of gastrointestinal perforation admitted toin single surgical unit of deptt. of surgery, I.G.M.C Shimla from1st Aug, 2013 to 31st July2018.

Observation: Out of 200 patients presented to emergency department with features of perforation peritonitis,132 patients (66%) of them were found to have peptic ulcer perforation. This was followed by appendicular perforation (9%). Tubercular perforation is relatively rare. Mortality rate was found to be12%, the cause of which was diagnosed as septicaemia.

Conclusion: Surgery is the line of management of perforation peritonitis. Early diagnosis with appropriate investigations and treatment with antibiotics, fluid and electrolyte balance and exploratory laparotomy is always advocated for better patient compliance and relatively low mortality.

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INTRODUCTION

Gastrointestinal perforation is a common emergency enc countered in a surgeon's practice with high morbidity and mortality. The initial credit of describing the signs and symptoms of perforated gastric ulcer goes to Ravlinson in1727. Henser (1891) was the first to close perforated gastric ulcer [1]. Causative factors and site of perforation vary enormously. Perforations of stomach and small intestine are on increase.

An increasing proportion of elderlypatients in Western society and availability of powerful NSAIDS continue to provide a fertile ground for upper gastro-intestinal tract perforations usually in3rd-4th decades, with a male preponderance, while the epidemiological trendis not the same worldwide &there is decrease in incidence in the West.

Peptic ulcers are chronic, often single that occurs due to acid peptic juices. About 98% of ulcersoccur either in the duodenum or in the stomach in the ratio of about 4:1 & 90% of duodenalulcers occur inanterior wall of first part of duodenum. Typhoid fever is caused by Salmonella typhi which has a seasonal incidence, it produces hyperplasia of reticuloendothelial system with necrosis and ulceration of gut leading to perforation, generally limited to the Peyer's patches in the terminalileum. These perforations are generally single; appear as punched out holes on the anti-mesenteric borders of the bowel [2]. Tuberculous ulcers affect most often ileum, proximal colon and peritoneum. Chronic inflammation causes thickening of the intestinal wall and narrowing of the lumen

Corresponding author:* **Dr. Kunal Malhotra
Resident Doctor, Department of Surgery, Indira Gandhi Medical College & Hospital , Shimla (Himachal Pradesh)

usually in the ileocaecal region.Ulcers are multiple and circumferential[3].

Acute appendicitis is classified into nonobstructive and obstructive type. In non-obstructive type, organ becomes turgid, dusky, red and haemorrhages occur intomucosa. The vascularityat the distal part is often jeopardized leading to gangrene of the tip of the appendix. In obstructive type, products of the inflammation become pent up& the inflammation proceeds more rapidly leading to gangrene orperforation. Perforation occurs more often at the site of an impacted faecolith [4].

Colonic perforation secondary to amoebic colitis is very rare (0-4%). Mucosal ulceration is a constant feature of amoebic colitis. Sigmoid colon and caecum are commonest sites of infection. In Crohn's disease, ileal disease is the most common accounting for 60% of cases, forming irregular serpentine ulcers in the long axis of the bowel. Penetration of ulcer may produce adhesions, fistulas, communicating or localised walled-off abscess. Ulcerative colitis starts in rectum in about 95% of cases and spreads proximally[5].

The Peak incidence of peptic ulcer perforation is between 46 decades and perforation due to gastric ulcer or carcinoma occurs in older age groups. Appendicular perforation occursin 2-3rd decades. While perforation due to ulcerative colitis is seen more in 3rd, 4th and 2nd decades in that order. Carcinoma colon, ischemic colitis and sigmoid volvulus are seen in older age group [6]. Tubercular perforations are common in age group of 2030yrs. Males are affected more in all except in ulcerative colitis where females arecommonly affected and with equal share in diverticulitis and appendicular perforations. In pepticulcer perforation, male to female ratio is 4:1[7].

Symptoms & signs perforation can be described in 3 stages(a) Stage of peritonism: causing chemical peritonitis lasting for about 6hrs.(b) Stag e of reactionary peritonitis: Theirritant fluid becomes diluted with peritoneal exudates.(c) Stage of diffuse peritonitis with "Facies hippocratica"- pinched and anxious looking face, sunken eyes and hollow cheeks[7].

In typhoid perforation, patient gives history of typhoid fever. Symptoms start almost 2nd to 4thweek of fever. Sudden collapse, fastthready pulse, subnormal temperature are the features of perforation with obliteration of liver dullness & shifting dullness. In tubercular perforation acute agonising pain with distension of abdomen and symptoms like dyspepsia, loss of weight and appetite, diarrhoea with blood and mucus in stools are the presenting complaints. Past history of tuberculosis may be obtained. Tenderness in the right iliac fossa and doughy abdomen are the signs [3].

Amoebic ulcer perforation presents either as a localised abscess, paracolic abscess or abdominal distension because of generalised peritonitis but without much pain or rigidity.

Investigations

A number of laboratory studies are considered routine in the evaluation of apatient with an acute abdomen. They helps to confirm that inflammation orinfection. A complete blood count with differential is valuable. Measurement of serum electrolytes, bloodurea nitrogen and creatinine; assists in evaluating the effect of such factors as vomiting or third-space fluid losses. Serum amylase and lipase can be elevated in disorders such as small bowel infarction or duodenal ulcer perforation.

The selection of radiologic examinations is determined by the clinical presentation. The acute abdominal x-ray series includes an upright chest, supine abdominal and upright or left lateral decubitus abdominal films. Free air indicating a perforated viscus is present in 70% of patients with perforated peptic ulcer [7]and therefore, its absence do not rule out perforation. In late cases, X-ray films may show ground glassappearance or multiple fluid levelsin addition to free gas because of paralytic ileus [8]. Ultrasound abdomen is particularly useful in appendicular perforation.

Abdominal paracentesis is well established in cases of acute ad omen& sheer anchor of the diagnosis is gross and microscopic examination and is accurate [9].

Treatment

Aspect of gastrointestinal perforation is still controversial. The treatment depends upon the age of the patient, pre-operative condition of the patient, time of presentation to hospital, type of pathology and experience of the surgeon.

Perforated peptic ulcer can be managed either conservatively or by surgical approach depending upon the patient general condition, time of presentation. Most patients with a perforated ulcer are treated by a simple suturing of the perforation. Closure with a definitive surgery and procedures are being done in certain patients.

The main option in a case of perforated gastric ulcer includes simple closureafter 4 quadrant biopsy, excision and primary closure or gastric resection [6]. In laparoscopic closure, perforation is closed with intracorporeal suturing with a omental patch which is secured with additional sutures. Following closure of the perforation, the abdomen is irrigated and aspirated with special attention to the pelvis and subphrenic spaces. A proximal gastric vagotomy or Taylor's procedure (anterior seromyotomy and posterior truncal vagotomy) may be performed [10].

In typhoid perforations, surgery has been generally accepted as the treatment of the choice. Typhoid ileal perforation has been treated with surgery and ant biotics as closure of perforation eliminates contamination and lessens toxaemia. Surgery is necessary as peritonitis is poorly localised and there is no effort by the omentum to seal off the perforation[11].Types of surgical procedureare ,simple closure of perforation in 2 or simple closure and exteriorisation of the sutured loop when the entire bowel is inflamed and leak of the suture line is to be expected[82]. Alternate procedures are described which includes closure of perforation with ileo-transverse colostomy resection of the affected loop and end-to-end anastomosis & tube ileostomy [12]. In appendicular perforation treatment of choice is emergency appendicectomy [4]. Tubercular perforation is usually associated with strictures. Simple suture of the perforation is adequate after proper drainage of the peritoneal cavity. Other surgical options are resection of the perforated segment, simple closure with bypass of stricture either by ileo-

ileostomy or ileo-transverse colostomy or stricturoplasty. Anti-tubercular drugs are given post operatively [3].

In perforation due to Crohn's disease, the disease segment must be removed. Anastomosis is either delayed or protected by a cutaneous stoma& the peritoneal cavity is irrigated and drain inserted. Exteriorisation and colostomy or subtotal or total colectomy has also been advised.

In diverticular perforation of colon, patient suffering from faecal peritonitis are in profound shock. Thus, initial aim is to institute resuscitative measures. Surgical procedures are (a). Operation in which the diseased segment is left in site at the initial procedure, is a 3-stage procedure. First stage: suture of perforation, peritoneal toilet and drainage of perforatedsite followed by transverse colostomy.

In second stage, resection of the involved segment is done and followed by third stage of closure of colostomy oroperation to remove the perforated segment by surgical excision, end colostomy and mucous fistula. Other modalities aresegmental excision, Hartman'sprocedure, primary resection and anastomosis mainly done in cases of complicated diverticular disease [13].

Perforation of colonic malignancy is often a result of obstruct ive causes of the colon. When acompetent ileo-caecal valve is present, a free perforation occurs in the dilated thin walled caecum. Perforation also may develop at the site proximal to the tumour. Free perforation is a life-threatening condition requiring emergency surgery. Resection of the perforated tumour bearing colon ispreferred.Proximal faecal diversion such as ileostomy and transverse colostomy should be done as the first stage [14].

The diagnosis and treatment of perforation of the G.I. tract remains a formidable problem; the mortality of which depends on early approach to the hospital, quick diagnosis, prompt surgical treatment, appropriate and adequate antibiotics. Thorough peritoneal lavage, adequate fluids and electrolyte replacement are the factors which improves the progress. However, there has been a reduction in morbidity and mortality due to better knowledge about the pathology, fluid and electrolyte imbalance and advances in anaesthesia and antibiotic therapy.

AIMS

The purpose of this study was to evaluate the relative incidence of various causes of perforation and also to find the incidence of G.I. perforation in relation to age, group and sex of the patient. Importance has also been given to the clinical presentation, relevant investigations and various modalities of treatment.

MATERIALS AND METHODS

This prospective study has been based on the analysis of 200 cases of gastro-intestinal perforations who were admitted on emergency basis in single surgical unit of deptt. of surgery, I.G.M.C Shimla from1st Aug, 2013 to 31st July 2018.

Cases were selected on the basis of clinical diagnosis and were confirmed by investigations in the age group more than eighteen years. Both the sexes were included.

Exclusion criteria were : age less than eighteen years, patients with blunt or penetrating injury to the abdomen with signs of hollow viscus perforation clinically or radiologically, iatrogenic perforation during laparotomy or laparoscopy or any other invasive procedure, perforations of genitourinary tract like U.B., ruptured ectopic pregnancy and gallbladder. In all the cases, baseline demographic data of all patients with general physical examination and systemic examination was done. monitoring of the vital signs with pre-operative correction of fluid and electrolyte imbalance and broad-spectrum antibiotics were started. The investigations done were complete hemogram, blood grouping and typing, renal function tests, serum electrolytes, widal test (in suspected cases), routine urine examination. Plain X- ray chest and abdomen (erect) to detect free gas under the diaphragm. Ultrasound abdomen was done to see the presence of freefluid in the peritoneal cavity and to rule out associated pathology in solid viscera. Paracentesis was done only in selected cases for confirmation in cases where X-ray showed no gas under the diaphragm.

Laparotomy was done in almost all the cases under general anaesthesia (8 appendicular perforations were done under spinal anaesthesia). Incision was taken depending upon the suspected site of pathology and when not confirmed, a rightparamedian or midline incision was taken. Viscera were inspected and site of perforation was identified. Appropriate surgical procedure was performed. In almost all cases of gastric perforation, tissue from the edge of the ulcer was sent for histopathological examination. Peritoneal lavage with saline was carried out and peritoneal cavity was drained using chest tube drain.

Post-operative patients were put in nasogastric tube with continuous aspiration, intravenous fluids, and appropriate antibiotics. Pantoprazole/ rabeprazole were given in cases of peptic ulcer perforation. Vital signs were monitored along with intake-output chart and biochemical parameters. Recovery was observed and complications which occurred were noted and treated accordingly. Regular follow-up of the patients was carried out for a month.

Statistical Analysis

All numerical data were expressed as mean with standard deviation (SD). All the statistical tests were done using Excel/SPSS software. Discrete and continuous variables were compared using Pearson's coefficient, Chi square test and Student t-test as appropriate. Multiple comparisons were made using ANOVA. A p value <0.05 was taken as statistically significant.

Observation and Analysis

The total200 cases of hollow viscus perforation were studied, 182 emergency laparotomies were done and in 18 cases B/L flank drainages were done. Out of all cases,61% were due to duodenal perforations. Our study revealed the sites of perforations in the gastro-intestinal tract as per Table .1.

The commonest site of perforation was in first part of duodenum 61% , followed by stomach 12% and appendix 9% and 18% of cases were due to other causes.

According to aetiology of the perforation, peptic ulcer perf oration (73%) was the major causative factor leading to peritonitis. This was followed by appendicular perforation while colonic perforations were least common (Fig .1).

The majority of patients (38%) were in the age group of 30-39 years for 38% of cases while the least were in group 18- 20 years accounting for 3% of cases. Male patients were predominant with male females ratio of 3.7:1and peptic ulcer perforation was heavily in favour of male patients with male to female ratio of 5.1:1(Table:2).

Clinical features

All the cases in our study complained of pain abdomen. Only 3 4% of allcases had Vomiting& was seen in 14%, 9%,1% 1% and 7% in appendicular, malignant, tubercular and idiopathic and B/l flank drainage cases respectively. It was watery in 26% of all cases, it contained food particles and some of them had blood or bile stained vomitus and it was non-projectile. Majority cases (90%) had abdominal distension, . Fever was noted only in 25% cases which was of moderate degree and was not associated with chills or rigors.

Past history: Chronic pain abdominal pain was seen in 44% case. Previous history of fever in the recent past was found in 14% cases, out of which 4% cases were found to be of typhoid fever, which was followed by pain abdomen. Previous history of drug intake (NSAID's) was found in 40% cases of peptic ulcer perforation. 2% patients had previous history of tuberculosis that had been treated with antituberculosis treatment.Habits:46% patients were chronic smokers and 31% of them were used to take alcohol& 16 patients were chronic alcoholics.

Signs in cases of gastro-intestinal perforation

General condition: Dehydration was seen in 54% cases while 72% cases exhibited tachycardia and12% cases presented with shock.

Tenderness along with guarding/ rigidity was the classical signs noted inpatients with perforation peritonitis. Apart from these signs, obliteration of liverdullness and absent bowel sounds was also noted. All the patient had tenderness while distension was seen in34% cases. Guarding/ rigidity was present in 94% cases. Bowel sounds were absent in 80% cases. Obliteration of liver dullness was noted in 56% cases. All of the cases were associated with tenderness and distension was present in 77% cases (except incases of appendicular and tubercular perforation where distension was not observed.

Obliteration of liver dullness was seen in majority of peptic ulcer perforation cases (66%) though it was inconstant finding in appendicular (2%) and tubercular perforation (3%).

Investigations:X-ray chest and erect x-ray abdomen was taken immediately after the clinicaldiagnosis of the perforation was made for free gas under the diaphragm which was seen in 69% cases. Few cases also showed dilated bowel loops and presence of free fluid. Presence of pneumoperitoneum in relation to aetiology was seen in 78%, 1.5%,1.5%,3%,2% and 14% in peptic ulcer, appendicular, typhoid, malignant, tubercular, idiopathic perforations & in bilateral flank drainage respectively.

Ultrasonography of the abdomen was done patients in whom perforation was suspected. Evidence of perforation was indirect and presence of free fluid with echogenicity was suggestive of perforation. In 86% of patients showed presence of free fluid. Widal test was positive in two cases of ileal perforation.

Abdominal paracentesis was done in only about 24 out of200 cases; indications being for the diagnosis in those patients whose clinical presentation were not suggestive ofperforation and with no clinical signs of peritonitis. The aspirated fluid was also sent for cytological, biochemical and microbiology laboratories for further evaluation.

As per bacteriological culture profile of peritoneal fluid obtained on abdominal paracentesis / laparotomy or by flank drainage, most common organism found in peritoneal fluid culture was E. coli (71%) than followed by klebsiella 14%, Enterobacter 6%, citrobactor 3%, staph. aureus 3%, bacteroidsand 2%, post streptococcal 1%.

Treatment

Ryle's tube aspiration, intravenous fluids with Ringer lactate, dextrose saline,5% dextrose haemacel, appropriate antibiotics were given & out of hundred cases 264were subjected to laparotomy. General anaesthesia was given for all the patients after endotrachealintubation except for 4 casesof appendicular perforation where spinal anaesthesia was given.

Graham's technique of simple closure of the perforation was done followed by omental pedicle patch in 99%of all the patients of peptic ulcer perforation. Cellon Jones technique of closure of perforation with a free omental patch was done in 2 cases. Tissue for biopsy was taken in 12 cases of gastric perforation.2 case of gastric perforations were due to malignancy and for that gastric resection followed by gastro-jejunostomy was done (Fig:2). All cases of typhoid ulcer perforation were found to be in the ileum(Fig:3) and were treated by simple closure in 2 layers after trimming the edges in 8 cases and due to multiple perforations resection anastomosis was done in 4 cases.

The six cases of tubercular perforation were in the ileum. In4 patients, resection of the diseased segment followed by end-toend anastomosis was performed and in 2 cases sticturoplasty was done including the perforation. Anti-tubercular treatment was advised for 18 months. All the patients of appendicular perforation were treated with appendicectomy.

Before closing the abdomen, a through wash was given with saline and drains were kept in either one or both the flanks. Post- operatively all the vitals were monitored and necessary investigations done. Patients were treated with adequate fluids, antibiotics and blood transfusion in selected cases.

Post-operative complications

Post operatively 34% patients had complications. Wound sepsis12 %, chest infection5%,3%residual abscess, 2%burst abdomen, 12% death, 3%wound sepsis & chest infection. Burst abdomen was seen in 3 case and onepatient developed incisional hernia after 8 months of surgery. 12 %patients undergoing bilateral flank drainage eventually died due to comorbid conditions. 4% cases of wound sepsis were treated by secondary suturing after control of infection and other cases were allowed to heal by secondary intention.

Burst abdomen was treated by mass closure of the abdomen with tension sutures. 3% patients of appendicular perforation had residual pelvic abscess, 2 of which were drained per rectally and 4 patients left attended as the quantity was negligible. Majority of post-operative complications inrelation to aetiology were seen in patents of perforated peptic ulcer specially chest infections (5%).

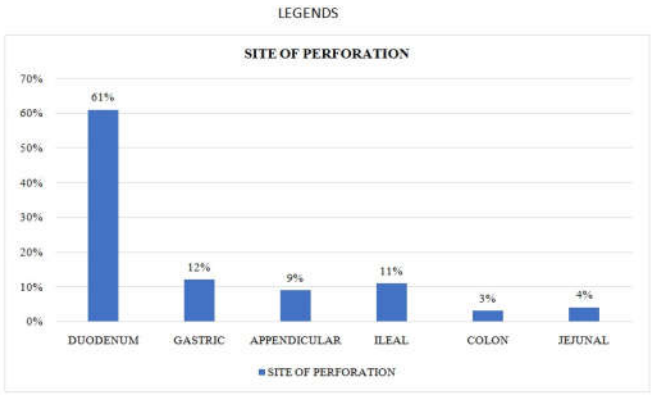

Table 1 Showing sites of perforations.

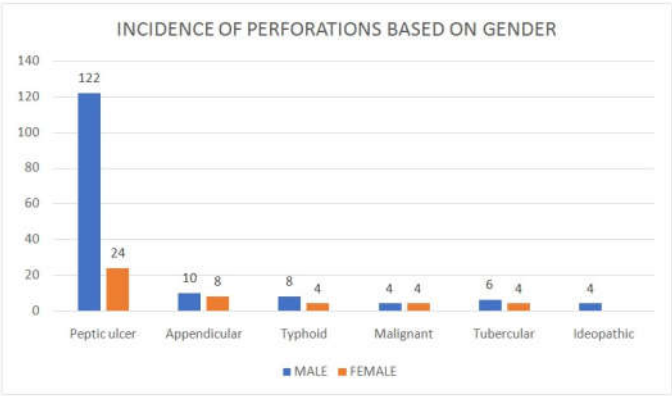

Table 2 Gender wise aetiology of perforation

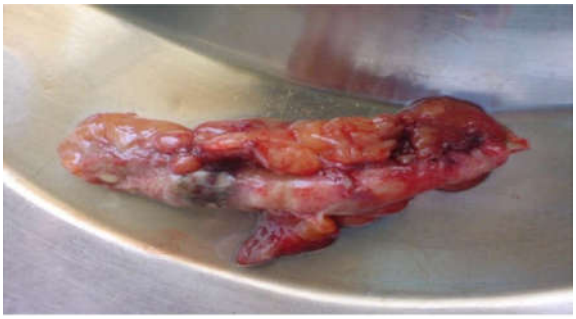

Fig 1 Showing Perforated appendix

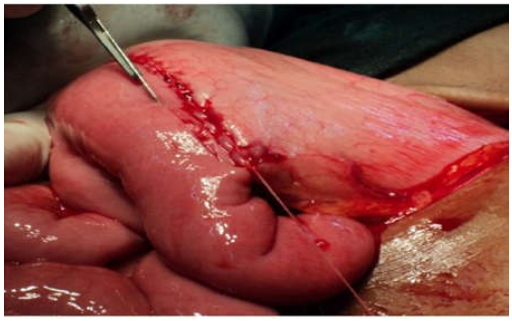

Fig 2 Showing gastro-jejunostomy following partial gastrectomy for malignant gastric perforation.

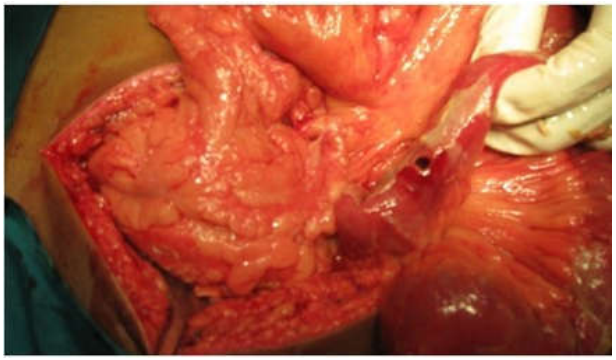

Fig 3 Showing ileal perforation due to typhoid

DISCUSSION

Gastrointestinal perforation constitutes 20 % of total emergency operationsin our hospital. In our institution, appendicitis ranked first in the abdominal emergencies followed by perforation and obstruction in that order. This pattern has been observed globally [6].

In our study of 200 cases, the incidence of peptic ulcer perforation was highest constituting 66 %. This was followed by appendicular (9%) and typhoid (4%) perforation. Tubercular perforation (3%) and malignant perforation (2%) constituted the rest. Marshall P *et al* (1999) [15] found an incidence of typhoid (4%) perforation. Tubercular perforation (3%) and malignant perforation59.12% of peptic ulcer perforation, 17% typhoid, 15.65% appendicular and 6.38%traumatic perforation in their analysis of 658 cases. In our study, the incidence of peptic ulcer perforation and appendicular perforation correlates with the study butvariation w as seen in incidence of typhoid perforation. The incidence of typhoid perforation has reduced mainly as a result of availability of highly effective antibiotics.

In our study, the commonest site of perforation is first part of duodenum followed by gastric, appendix, ileum, jejunum and colon similarly M Dandapat [6] (1991) and others [7] found that for gastrointestinal perforation the commonest site is duodenum, followed by ileum, stomach and appendix. Age wise, maximum incidence of perforation irrespective of path ology was seen between 30-39 years. Other studies observed similar incidences of age between 32-39 yearsd[6]As peptic ulcer is more common in younger age group up (3 4 decade) and asit is the cause of perforation in 73% of our cases, the incidence in 4 decade isunderstandable. S N Mathur [16] (1991) have reported similar incidences. Appendicular perforation was seen in younger age group in our study, which is similar to the observation by M.C. Dandapat *et al* [6] (1991). Malignant perforation was noted in older age group [14].

The ratio of men to women with all types of perforation, irrespective of pathology of perforation was 3.76: 1. M.C. Dandapat [6] (1991) reported a sex incidence of 8.4:1. In peptic ulcer perforation the sex incidence showed remarkable predominance in the ratio of 5.6:1. Peptic ulcer perforation is predominantly seen in male and it is seen inour study. Similar observation was seen by others [12]. In clinical features pain abdomen, vomiting, distension and fever were the predominant symptoms. Pain abdomen was seen in all cases and similar finding hasbeen reported by 1984) and J C Baid[2] (1988). In peptic ulcer perforationmost of our patients gave history of pain in the epigastric region, it has been reportedby S N Mathur [16] (1991).

History of fever in the recent past followed by pain abdomen w as a diagnostictool for typhoid perforation clinically. S K Nair [7] (1981) have observed similar history. Fever was also seen in few cases of appendicitis next to pain which was also found in a study conducted by Charles. N [17] (1992).

Non-steroidal anti-inflammatory drugs are known to precipitate peptic ulcerdisease and even give rise to complications like perforation, bleeding etc; Mechanism of action being mediated through prostaglandin synthesis blockade. 40 of 66 cases of peptic ulcer perforation revealed the history of NSAIDS injection.

Dehydration was the common cause after gastric perforation and was most consistent physical sign in our patients occurring in about 54% of cases; a feature also observed by S K Nair [7] (1981). Dehydration occurs mainly as a result of accumulation of fluid in the peritoneal cavity, intestine and due to vomiting apart from other causes.

Tachycardia was commonly seen in cases who presented with intestinal and appendicular perforation (due to shrinkage of circulation fluid volume). In our study, tachycardia was noted in 72% cases. J C Baid [2] noted it in 77% of cases in his study. On examination of abdomen, tenderness was recorded in all the cases, distension in 68 cases, guarding/ rigidity in 188 cases, obliteration of liver dullness in 112 cases, absent bowel sounds in160 cases. Distension was not found in majority of appendicular perforation as there i s only little spillage and localisation of peritonitis.

In most of the study conducted worldwide, tenderness was pre sent in all the cases of gastrointestinal perforation. In a study conducted by J C Baid and T C Jain [2] (1988) of 54 cases, found distension in 46 cases, guarding/ rigidity in 5 4 cases, obliteration ofliver dullness in 28 cases and absent bowel sounds in 29 cases. Our study correlates almost with the above-mentioned study with regard to signs of perforation.

In investigations, even though presence of gas under the was noted. This may be due to confinement of the perforation as well as absent air in the lumen. Ultrasound abdomen is readily available, non-invasive, easily repeatable investigation to find out the free fluid in the peritoneum diaphragm is a hallmark of hollow viscus perforation, absence of this does not exclude the possibility of perforation. This sign is visualised only in about 75% of perforation cases. In our study, we found it in69% of cases. N William and N W Everson[8] (199)h ave quotes “in 60-70% of cases the free gas under can be detected”. M C Dandapat and colleague s[6] (1991) notices gas under the diaphragm in 72.35%. Our study correlates well with the above-mentioned study. In only 0.5% of appendicular perforation, gas under the diaphragm due to gastro-intestinal perforation and more importantly in the diagnosis of injury to the solid organs in thetraumatic cases associated with hollow viscus perforation. William N& N. W. Everson [8] 1997in a study found peritoneal free fluid in all the cases. In our study, wefound free fluid in almost all the cases in which we did ultra sound. This was confirmed by laparotomy.

Abdominal paracentesis was done in 24 cases where Xray showed no free air. S P S Rao *et al* [9] (1997) obtained positive results in 96% ofcases of gastro-intestinal perforation. So, paracentesis should carry out more diligently in all cases of perforation and not only it will show the peristalsis but alsomay help to detect site of perforation and associated visceral injuries in cases of trauma.

Widal test was positive in 25% cases in our study while different studies have reported the positivity rate of 70% to 75% in cases of typhoid perforations [11,18]. As per bacteriological culture profile, most common organism found in peritoneal fluid culture was E. coli (71%) than followed by klebsiella 14%, Enterobacter 6%, citrobactor 3%, staph. aureus 3%, bacteroids and 2%, peptostrptococci 1%. This finding is similar to results of other studies [9].

Treatment: Out of 122 peptic ulcer perforations, cases of 24 gastric and108 cases of duodenal perforation, none of the cases were taken for definitive surgery. The decision was based upon the operative finding of contamination of the peritoneal cavity. Almost all of the patients presented after 810hrs, frank peritonitis was expected and thus definitive surgery was not performed in presence of gross contamination. Thus, simple closure of the perforation was performed with omental patch. Worldwide literature is in agreement with the same. Malignant gastric perforation was managed by partial gastrectomy followed by gastrojejunostomy. M C Dandapat [6] (1991) in his study did the same. Malignant colonic perforation was managed by Hartman’s procedure (permanent colostomy) after closure of perforation. Ileal growth perforation was managed by resection and anastomosis.

For typhoid perforation, after trimming the edges, simple closure of theperforation was done in 8 cases, 4 cases had multiple perforations and thus resectionand anastomosis was

done. Eggleston F. C [11] (1979) and. Chowhan M. K; S. K. Pandey [18] 1982 have reported the operation of choice as simple closure of perforation in 2 layers. For all the cases of appendicular perforation, appendicectomy was done and most of the literatures suggest the same [4].

Post-operative complications: 34% patients developed post-operative complications in our study where wound sepsis was the commonest (12%). This is may be due to the fact thatcontamination of surgical incision occurs and also patients being anaemic ormalnourished. M C Dandapat [6] (1991) reported wound sepsis in 13.5% of gastrointestinal perforation. Most of the appendicular perforation did not have much complication. This is a result of less contamination and younger age patients who can withstand surgery.

Many patients had chest infection as a complication (5%). This m ay be dueto prolonged immobilisation and associated COPD in old patients. One patient of typhoid ulcer perforation had burst abdomen which was operated and treated. After 8months, same patient presented with incisional hernia and underwent mesh repair.

Overall mortality in our study was 12%, all of them had bilateral flank drainage done due to comorbid conditions making them unfit for general anaesthesia.

Worldwide literature shows a decrease in mortality of gastro-intestinal perforation. This decrease in mortality may be attributed to the use of appropriate antibiotics, adequate resuscitation and advanced surgical techniques. Recent studies suggest a mortality rate of less than 5%[19].

CONCLUSION

As majority of the perforation was due to acid peptic disease, appropriate treatment of ulcer disease may reduce this dreaded complication. This has been achieved with the concomitant use of proton-pump inhibitors and anti H-pylori treatment. Early recognition and treatment of appendicitis will further reduce the incidence. Surgery is the main modality of treatment in case of perforation peritonitis and is advised after adequate resuscitation. This results in low mortality.

Bibliography

1. Krukowaki Z. H; Mathsen N. A *et al*.1984.Emergency Surgery for Diverticular Disease complicated by generalised peritonitis. Br. J. Surg. Vol 7; 921-922.
2. Baid, J. C; T. C. Jain. 1988. Intestinal perforation especially due to blunt injury abdomen. Ind J Surg; 50; 335-337.
3. Minhas, S S; Merwaha D.C; Gupta R.R.1992.Tubercular Perforation"; Ind J Surg; 54; 263.
4. Yamn, D; Vorger, H.1998.Perforated Appendicitis. Am Surg, 63(10), 970-975.
5. Kahandelal, C; V. R. Sharma. 1990.Fulminating Amoebic Colitis. Ind J Surg;52(10-11), 530-536.
6. Dandapat M; C. Mukerjee. 1991.Gastro-intestinal perforation. Ind J Surg; 53(5); 188-193.
7. Nair, S. K; V. J. Sinha; Sudhir, Kumari. 1981.Nontraumatic intestinal Perforation. Ind J Surg; 43; 371377.
8. William, N; N. W. Everson. 1997. Radiological Confirmation of intraperitoneal free gas. Ann of Royal coll Surg Edg; 79(1), 8-12.
9. Rao, S. P. S; B. R. Prakash. 1977.Evaluation of diagnostic abdominal paracentesis in acute surgical condition of abdomen. Ind. J. Surg; 39; 285-289.
10. Takeuchi, H; Kauana, T. 1998.Laparoscopic treatment of perforated duodenal ulcer and omental patch. surglaparosc-endosc. 8(2); 153-156.
11. Eggleston, F. C; Santosh, B; Singh, C. M. 1979.Typhoid perforation of the Bowel. Ann of Sur; 190; 21-34.
12. Mc Donough, J. M; Mathsen, N. A. 1972. Factors affecting prognosis in perforated peptic ulcer.Am. Jr. Sur 123; 411-415.
13. Krukowaki, Z. H; Mathsen, N.A.1984.Emergency Surgery for Diverticular Disease complicated by generalised peritonitis. Br. J. Surg; 7; 921-923.
14. Fielding, L. D; Wells, B.1974. Survival after primary and after staged resection for large bowel obstruction caused by cancer. Bri J Surg; 61; 16-18.
15. Marshall, P; P. Ramaswamy.1999. Evaluation of a Protocol for a non-operative management of perforated peptic ulcer. Bri J Surg; 86; 131-136.
16. Mathur, S. N; P. Khandelwal. 1991.Peptic perforation. Ind J Surg; 53(6); 251-153.
17. Charles, S; N. Mock;M. D Joseph. 1992.Improvement in survival from typhoid ileal perforation. Ann of Sur; 215;244-249.
18. Chowhan, M. K; S. K. Pandey. 1982.Typhoid enteric perforation. Br J Surg; 69; 173-175.
19. C. Palanivelu. 2018.Art of Laparoscopy Surgery. Vol 1; Ed 1; ch.55. J.P.brother's medical publisher (P) Ltd. India.

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