SPECTRUM OF SECONDARY PERITONITIS IN NORTH COASTAL ANDHRA PRADESH, INDIA

Janardhana Rao Konkena[1], Manmadha Rao Vayalapalli[2], Naveen Kumar Podili[3], Pruthvi Raj Karumuri[4], Ram Prakash Gurram[5]

[1]Assistant Professor, Department of General Surgery, King George Hospital.
[2]Professor, Department of General Surgery, King George Hospital.
[3]Postgraduate, Department of General Surgery, King George Hospital.
[4]Postgraduate, Department of General Surgery, King George Hospital.
[5]Postgraduate, Department of General Surgery, King George Hospital.

ABSTRACT

BACKGROUND

Secondary peritonitis due to perforation of Gastrointestinal Tract (GIT) is one of the most common surgical emergencies worldwide. The spectrum of aetiology of secondary peritonitis in tropical countries differs from western countries. This study was conducted in King George Hospital, Visakhapatnam, in the state of Andhra Pradesh, India. The purpose of this study is to highlight the salient causes of secondary peritonitis and its outcome.

METHODS

A retrospective analysis of 603 patients of secondary peritonitis was done from January 2013 to December 2015 at King George Hospital, Visakhapatnam, Andhra Pradesh, India. All cases which were found to have peritonitis as a result of perforation of any part of GIT at the time of surgery were included in the study. All cases with either primary peritonitis or that due to anastomotic dehiscence were excluded.

RESULTS

A total of 603 patients were studied. Among them, 493 (81.7%) were males and 110 (18.3%) were females. Most common cause of secondary peritonitis in our study was acid peptic disease(52.40%) followed by appendicular pathology(11.2%), gastrointestinal perforation due to injury of abdomen (9.62%), small bowel perforation due to typhoid aetiology (7.63%), gangrenous bowel (6.30%), ruptured liver abscess (2.98%), biliary peritonitis (1.99%) and some miscellaneous rare causes. Overall, mortality in this study was found to be 8.95%.

CONCLUSION

The spectrum of secondary peritonitis in North Coastal Andhra Pradesh differs from other areas in India and western countries. Highest incidence of secondary peritonitis is due to perforation noted in gastroduodenal region, which is in contrast to western hemisphere where the predominant cause is lower gastrointestinal tract. Considering the relatively higher rate of gastroduodenal perforation quoted in this study, it is vital that awareness about the role of Helicobacter pylori ought to be considered. Special focus must also be made to increase awareness regarding limited use of NSAIDS, which have been incriminated as a major cause of such pathologies and are also widely abused.

KEYWORDS

Secondary peritonitis, Acid peptic disease, Appendicular perforation, Typhoid ileal perforation.

HOW TO CITE THIS ARTICLE: Konkena JR, Vayalapalli MR, Podili NK, et al. Spectrum of secondary peritonitis in north coastal Andhra Pradesh, India. J. Evid. Based Med. Healthc. 2016; 3(65), 3536-3541. DOI: 10.18410/jebmh/2016/759

INTRODUCTION: Generalised peritonitis is a common surgical emergency in developing countries.[1] Despite advances in surgical techniques, good antimicrobial therapy, and intensive care support, it carries high morbidity and mortality while its management remains difficult and complex.[2] The spectrum of secondary peritonitis in tropical countries differs from western counterparts.[2]

Financial or Other, Competing Interest: None.
Submission 14-07-2016, Peer Review 27-07-2016,
Acceptance 03-08-2016, Published 13-08-2016.
Corresponding Author:
Dr. Naveen Kumar Podlii,
Room No. 73, PG Men's Hostel,
King George Hospital, Maharanipetta,
Visakhapatnam-530002.
E-mail: naveenkumarpodili@gmail.com
DOI: 10.18410/jebmh/2016/759

The management approach requires both knowledge of the signs and symptoms of peritonitis to aid diagnosis and an understanding of common causes to assist the surgeon in appropriate surgical care. Peritonitis can be classified as primary, secondary, or tertiary depending upon the source of microbial contamination. Primary peritonitis is secondary to extraperitoneal sources. The infection spreading mainly through haematogenous dissemination without visceral perforation. Secondary peritonitis on the other hand is caused by resident flora of the gastrointestinal or urogenital tracts. The organisms reaching peritoneum secondary to a mechanical breach, tertiary peritonitis maybe defined as a severe recurrent or persistent intraabdominal infection after apparently successful and adequate surgical source control of secondary peritonitis.[3]

AIMS AND OBJECTIVES: The purpose of this study is to highlight the salient causes of secondary peritonitis and its outcome in terms of morbidity and mortality.

MATERIAL AND METHODS: This study was conducted at King George Hospital (KGH) in Visakhapatnam, the capital of North Coastal Andhra Pradesh, during the calendar years 2013-2015. KGH is a 1085 bedded tertiary care hospital rendering services to the people of North Coastal Andhra Pradesh and adjacent districts of Orissa and Chhattisgarh. The hospital has a 24 hours casualty department, 20-bedded surgical intensive care unit, several open wards with capacity for around 250 surgical patients, and equipped with two emergency operating rooms.

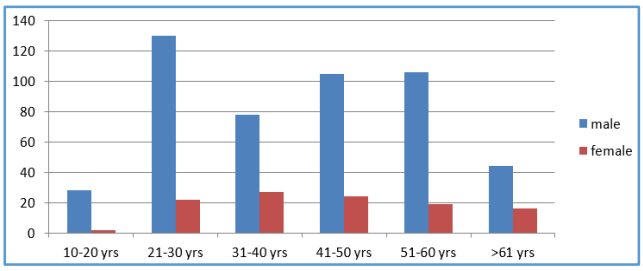
A retrospective study of 603 patients of secondary peritonitis was done over a period of last two years (January 2013-December 2015).

Inclusion Criteria: All cases found to have peritonitis as a result of mechanical breach in gastrointestinal and urogenital tract were included in study.

Exclusion Criteria: All the cases with either primary peritonitis or that due to anastomotic leak postsurgically were excluded.

All cases were studied in terms of clinical presentation, radiological evaluation, operative findings, and postoperative course. Data was collected from outpatient department, casualty records, emergency operation theatre, postoperative ward records, and death records. Data includes gender, age, date of admission, date of surgery, date of discharge or death, date of onset, and type of symptoms, presence of guarding, rebound tenderness, or rigidity, abdominal quadrant (s) affected, vital signs on presentation including heart rate, blood pressure, and respiratory rate, operative diagnosis, and surgical procedure. The results of initial complete blood counts, results of abdominal ultrasound, x-ray erect abdomen, biochemical values (creatinine and potassium values) were also taken into consideration.

RESULTS: Of the 603 cases that were studied, mean age of presentation was 43.58 years (range from 13 to 80 years) with majority of patients being males 493 (81.7%) and the remaining 110 (18.3%) being females. The male predominance over female (male/female-5:1) is similar to other studies.[1,2] Majority of the patients 152/603 were in 21-30 years age group (25.20%) followed by 129/603 in 41-50 years age group (21.39%) and 60/603 (9.95%) were in age group >60 years. Some patients had pre-existing illness 69 (11.47%) (Bar diagram-1).



Bar Diagram 1

Majority of the patients (591/603) presented with history of pain abdomen, 353 patients with vomiting, fever in 318, abdominal distension in 321, altered bowel habits in 233, and 58 patients presented with shock due to septicaemia. 22% of patients gave past history of NSAIDS use and 5% of patients gave history of preceding fever followed by onset of peritonitis symptom suggesting of typhoid aetiology. Among 527 patients who presented with guarding and rigidity, only 111 patients had localised signs while 416 had generalised guarding and rigidity. (Table-1).

Age group	
>61 yrs.	60/603(9.95%)
<60 yrs.	543/603
Pre-existing illness	**69/603(11.47%)**
Respiratory disease	19/69
Renal disease	17/69
Cardiovascular	13/69
Malignancy	16/69
Others	04/69
Clinical features	
Pain	591/603
Vomiting	353/603
Fever	318/603
Abdominal distension	321/603
Altered bowel habits	233/603
Guarding and rigidity	527/603
Localised	111/527
Generalised	416/527
Shock	058/603
Investigations	
Hypokalaemia (3.0 mg/dL)	59/603
S. creatinine (>1.7 mg/dL)	92/603
X-ray erect abdomen showing pneumoperitoneum	397/603

Table 1: Preoperative Data

Clinical presentation of the patients varied according to site and cause of perforation.[4,5] The patients of perforated peptic ulcer (316) usually had a short history of pain starting in epigastrium or upper abdomen along with generalised tenderness and guarding.[4,5] Among them, 22% of patients gave the history of NSAIDS use.

Rajendhar Singh et al studied 504 cases of gastrointestinal perforations and found that 54 (12%) patients developed secondary peritonitis secondary to perforated appendix.[4]

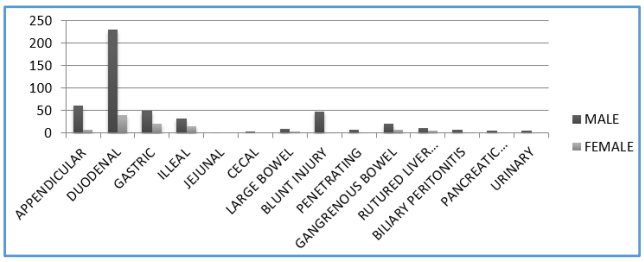
However, in our series, secondary peritonitis due to appendicular perforations was the underlying cause in 68 (11.2% of patients). The patients of appendicular pathology usually presented with initial pain abdomen around umbilicus and right iliac fossa then spreading to whole abdomen. Afridi SP from Pakistan had also reported that the patients who developed secondary peritonitis due to perforated appendix present with the typical history of pain starting in the periumbilical region then shifted to the right iliac fossa, or originated directly in the right iliac fossa and then spread to all over the abdomen.[5] In most of these patients, guarding and rigidity was localised to right lower quadrant of abdomen. The patients with perforated appendix belonged to younger age group.[4,5]

Patients with ileal perforation presented with central abdominal pain and with history of prolonged fever.[4,5] Patients with gangrenous bowel secondary to intestinal obstruction presented with classical features of intestinal obstruction like diffuse pain abdomen, vomiting, constipation, and distension of abdomen. These features were also associated with generalised guarding and rigidity suggestive of peritonitis.

Diagnosis of hollow viscous perforation was made in majority of patients with help of a simple plain x-ray of the erect abdomen. But, only 397 (65.83%) patients had air under diaphragm on x-ray of the erect abdomen.[4,5] However, in the remaining cases, the diagnosis was made with the help of ultrasonography or CECT of the abdomen. Electrolyte imbalance in form of hypokalaemia was seen in 59 patients and serum creatinine was raised in 92 patients.

All patients were resuscitated starting with insertion of two 16-gauge intravenous cannulas, nasogastric tube, and Foley's catheter. All patients received adequate fluid replacement, analgesic support, and adequate antibiotic coverage depending upon the local sensitivity of organisms. With the confirmation of the initial diagnosis of peritonitis, emergency laparotomy was performed in all 603 patients. Perforations in the GIT were treated either with primary double-layered closure, appendicectomy, segmental resection and anastomosis or loop ileostomy/colostomy depending upon the operative findings and general status of the patients. The peritoneal cavity was irrigated with an average of 3 litres of warm normal saline and drains were left in abdomen and wound was closed in layers. Patients were monitored postoperatively for recovery and early detection and management of complications.

Perforated duodenal ulcer, which was seen in 270 patients (44.77%) was the most common cause of secondary peritonitis followed by gastric perforation in 70 patients (11.6%), appendicular perforation in 68 patients (11.2%), blunt and penetrating injury to the abdomen leading to peritonitis in 58 patients (9.62%), small bowel perforation due to typhoid aetiology in 46 patients (7.63%), gangrenous bowel (including small and large bowel) in 38 patients (6.30%), ruptured liver abscess in 18 patients (2.98%), biliary peritonitis in 12 patients (1.99%), and other rare miscellaneous causes in the remaining patients (Table-2 and Bar diagram-2).



Bar Diagram 2

Diagnosis	Male	Female	Total
Appendicular	60	8	68(11.2)
Gastroduodenal			
Duodenal	230	40	270(44.77)
Gastric	50	20	70(11.6)
	280	60	340(56.38)
Small bowel perforation			
Ileal	32	14	46(7.63)
Jejunal	2	-	2(0.33)
	34	14	48(7.96)
Large bowel	5	3	8(1.33)
Injury to abdomen			
Blunt	46	2	48(7.96)
Penetrating	7	1	8(1.33)
Pancreatic injury	2		2(0.33)
	55	3	58(9.62)
Gangrenous bowel			
Small	15	5	20(3.32)
Large	14	4	18(2.98)
	20	8	38(6.30)
Ruptured liver abscess	12	6	18(2.98)
Biliary peritonitis			
GB perforation	7	3	10(1.66)
Cystic duct leak	1	1	2(.33)
	8	4	12(1.99)
Necrotising pancreatitis	6	1	7(1.16)
Urinary			
Bladder rupture	4	1	5(0.83)
Iatrogenic	1	-	1(0.16)
	5	1	6(0.99)
Total	**493 (81.7%)**	**110 (18.3%)**	**603**

Table 2

Among the 343 cases of gastroduodenal perforation, 316 are due to acid peptic disease and 24 cases were due to malignancy and 3 cases were due to traumatic gastric perforation. Among the 316 perforations due to peptic ulcer disease, 270 were due to duodenal perforation, the remaining 46 were due to perforation of benign gastric ulcer. Duodenal to gastric perforation ratio is 29:1 compared to other studies conducted in India, which is 7:1[4] and 15:1.[2] Contrary to this, the ratio was 4:1 in United Kingdom[6] and 4:1 in United States.[7] Gastric ulcer rarely present with

perforation peritonitis compared to duodenal ulcer.[8] Gastric perforations are related to the wide spread use of NSAIDS,[8] as seen in our study where 22% patients had positive history of NSAIDS abuse. Gastric malignancy can present as perforated gastric ulcer, which accounts for 1% of its complications. Perforation due to malignant ulcer of the stomach has higher incidence compared to perforation due to malignancy of other parts of GIT,[9] as seen in our study where we encountered 24 malignant ulcer perforations out of 73 gastric perforations.

Among the 113 cases of small bowel perforations, 46 cases were due to typhoid pathology. Among these 46 cases of enteric fever, 2 cases presented with multiple perforations involving distal ileum. Of the remaining small bowel perforation cases, 20 were due to gangrenous bowel, 45 were due to trauma and 2 were due to ileocecal tuberculosis. Perforation due to ileocecal tuberculosis per se is rare. Most common presentation is hyperplastic ileocecal tuberculosis resulting in obstruction. Perforation usually occurs during the usage period of antitubercular regimen,[10] as seen in our study where two cases presented during antitubercular treatment period. Among the traumatic small bowel perforation cases, 15 were associated with mesenteric tear and 5 were associated with splenic lacerations resulting in splenectomy. Among 45 cases of traumatic small bowel perforations, 2 were jejunal of which 1 was jejunal transection, remaining 43 were ileal of which 5 were having ileal transections.

Among the 34 cases of large bowel perforations, 18 were due to gangrenous sigmoid volvulus, 8 were due to malignancy of rectosigmoid junction among which one patient had synchronous growth in ascending and transverse colon and 8 cases were due to trauma. Among these traumatic perforation cases of large bowel, 2 were in cecum, 3 were in transverse colon, and 3 were in rectosigmoid junction.

Among the 9 cases of peritonitis due to pancreatic pathology, 7 were due to necrotising pancreatitis and 2 were due to pancreatic injury. Of these 2, one was associated with splenic laceration. Pancreatic necrosis occurs in approximately 20% of patients with acute pancreatitis and is necessary for the development of secondary pancreatic infection. However, pancreatic necrosis by itself, even when accompanied by organ failure is not an absolute indication for surgery. A trial of medical treatment for all patients with sterile pancreatic necrosis is in order.[11]

Among the 12 cases of biliary peritonitis, 10 were due to gall bladder perforation and 2 were due to cystic duct leak, a complication of laparoscopic cholecystectomy. Gall bladder perforation has been reported to occur in 3 to 10% cases of acute cholecystitis in adults.[12] Risk factors for gall bladder perforation in adults include age greater than 60 years, immunosuppression, steroid use, and severe systemic disease.[12](Table-3).

Cause of perforation	n-603	Mortality - 54/603(8.95%)
Gastroduodenal	**343**	
Acid peptic disease	316	31/316(9.80%)
Malignancy	24	
Trauma	3	
Small bowel	**113**	11/113(9.73%)
Typhoid	46	
Gangrenous bowel	20	
Trauma	45	
Tuberculosis	2	
Appendicular perforation (11.2%)	**68**	-
Large bowel	**34**	2/34(5.80%)
Gangrenous bowel	18	
Malignancy	8	
Trauma	8	
Ruptured liver abscess	**18 (2.98%)**	4/18(22.22%)
Biliary peritonitis	**12**	3/12(25.00%)
Gall bladder perforation	10	
Cystic duct leaks	2	
Necrotising pancreatitis	**9**	3/9(33.33%)
Trauma	7	
Pancreatitis	2	
Bladder rupture	**6**	-
Trauma	5	
Iatrogenic	1	

Table 3

All patients of gastroduodenal perforation (343) were treated with primary closure of perforation. Edge biopsy was performed from all the gastric perforation cases. However, the histopathological report was suggestive of malignancy in only 24 cases. Laparoscopic repair of gastroduodenal perforation by running sutures is an option.[13] All patients of appendicular perforation were managed with appendicectomy and local peritoneal lavage.[4,5]

All cases of typhoid ileal perforation were treated with simple primary closure of perforation. Primary repair of the typhoid perforation is a safe and effective treatment;[14] laparoscopic repair being another option.[15] Those with multiple ileal perforations involving distal ileum resulted in limited resection of the affected segment of ileum and cecum followed by ileo-ascending colon anastomosis. All cases of traumatic small bowel perforation were treated with primary closure while those of jejunal and ileal transections were treated with resection and anastomosis. Among the 20 cases of gangrenous small bowel leading to secondary peritonitis, 9 cases underwent ileoileal, 6 cases underwent ileojejunal, 2 cases underwent ileo-ascending, and 3 cases ileo-transverse anastomosis. Among the 34 cases of large bowel perforation, 18 were due to sigmoid volvulus for which sigmoidectomy was done. Of the 8 cases of malignant large bowel perforation, 7 were treated with diversion loop colostomies and one patient with synchronous growth in ascending and transverse colon was managed with an

ileostomy. Among the 2 cases of caecal perforation, one was treated with primary closure and the other with caecostomy.

All cases of secondary peritonitis due to ruptured liver abscess were treated with laparotomy and drainage. All cases of secondary peritonitis due to pancreatic pathology were treated with laparotomy and lavage.

All cases of biliary peritonitis due to ruptured gall bladder were treated with emergency open cholecystectomy and peritonitis due to cystic duct leaks were treated with laparotomy and ligation of cystic duct stump. All cases of peritonitis due to urinary bladder rupture were treated with laparotomy and primary closure.

Postoperative complications recorded were wound infection in 10.61%, electrolyte imbalance in 9.76%, septicaemia in 5.46%, pneumonia in 3.47%, intra-abdominal collection in 1.66%, and wound dehiscence in 0.83%. (Table-4).

Wound infection	64(10.61%)
Electrolyte imbalance	59(9.76%)
Septicaemia	33(5.46%)
Pneumonia	21(3.47%)
Abdominal collection	10(1.66%)
Wound dehiscence	5(0.83%)
Overall morbidity	192(31.78%)
Mortality	8.95(54/603)

Table 4

The overall mortality associated with peritonitis was 8.95% (54/603). The highest mortality was observed in necrotising pancreatitis leading to peritonitis in 33.33% (3/9) and in biliary peritonitis 25.00% (3/12). The mortality associated with ruptured liver abscess was 22.22% (4/18) and small bowel perforation was 9.73% (11/113). In spite of its high incidence perforation due to acid peptic disease had a mortality of 9.80% (31/316), which is very less compared to other studies.

The overall morbidity in our study was 31.78% and mortality was 8.95% comparable with other studies[6,7] despite delay in presentation to the emergency department. This was probably attributed to lower mean age of patients in our study. Morbidity and mortality was more in patients who presented late to emergency department and in elderly patients who were more than 60 years old and in patients with pre-existing co-morbid illness.[4,5]

DISCUSSION: This study outlines the aetiology, associated presenting clinical features, and outcomes of surgically managed peritonitis in a tertiary care centre in Visakhapatnam. As per studies done in tropical countries, secondary peritonitis was most commonly encountered in the younger age group with mean age of 36.8 years.[2] The mean age of perforation peritonitis in our study was 43.58 years, which is in contrast to most of the studies of western countries where the mean age is more than 60 years.[6,16] Our study of secondary peritonitis showed a male preponderance, which is similar to studies conducted in other areas of Indian subcontinent.[2,4,5]

Gastroduodenal perforation was commonly seen in the 4th and 5th decade in our study. The mean age of presentation of perforation of duodenal ulcer is 43.81 years and the mean age of gastric perforation is 48.57 years, which is very less compared to the study in the West where the perforation was commonly seen in 6th and 7th decades.[6,7] This is probably the reason why mortality due to peptic ulcer perforation in our study was less despite its higher incidence.

Perforation of proximal part of the GIT was more common in our study, which is similar to another study conducted in India.[2] Duodenal ulcer perforation was the most common perforation noticed in our study, which is comparable to other studies conducted in Indian subcontinent like studies by Afridi SP et al[5] and study by Gupta S and Kaushik R.[17] However, this is in contrast to studies conducted in other parts of world like Japan,[18] America,[19] and Greece[20] where perforation was common in the distal part of GIT. This is also in contradiction to other studies in India as well like study by Quereshi AM[21] who reported that majority of perforations involved distal GIT such as ileum.

Not only the site of GIT perforation, but the aetiological factors also show a wide geographical variation. Khanna et al[22] from Varanasi, India studied 204 cases of GIT perforation. Among them, 108 cases were due to small bowel perforation, which was attributed to typhoid aetiology, which is predominant in some parts of tropical countries. Noon et al[23] from Texas studied 430 patients of GIT perforation and found 210 cases to be due to penetrating trauma and 68 due to peptic ulcer perforation. In our study conducted on 603 cases, 316 perforations were due to acid peptic disease. This data shows the importance of trauma as aetiological factor in developed countries whereas infections (Typhoid, Helicobacter pylori) and analgesics abuse are the predominant aetiological factors in developing countries.

It is noticed in our study that proper hydration, good antibiotic cover, and simple closure of perforation significantly reduced mortality rate.[2,4,5] Early diagnosis and treatment lead to improved results in terms of mortality and morbidity. Majority of patients in our series presented late with the time interval between the onset of symptoms and admission varying from 6 hrs. hours to 10 days with an average of 3.4 days. Delay in seeking treatment associated with other factors such as pre-existing illness was one of the major reasons for high mortality and morbidity in our series. Kaur N et al in their study also attribute delay in seeking surgical treatment as an important cause for high morbidity.[24] Interestingly, we also found positive correlation between the duration of symptoms and mortality, while Kotiso et al noted 7.6% mortality rate in patients with symptoms of 2 days or less compared with 25% among those with symptoms over 2 days in duration.[25]

Surgical treatment of secondary peritonitis is highly demanding. Some authors have adopted laparoscopy as preferred surgical approach for the management of secondary peritonitis.[14] Laparoscopy is an emerging facility and in emergency setup it is still in its infancy being performed in only a few medical institutions of India. Due to the non-availability of laparoscopy in our emergency setup during the study period, no patient was treated laparoscopically.

CONCLUSION: The presentation of secondary peritonitis in India continues to be different from its western counterpart. In majority of cases, the presentation to the hospital was late with established generalised peritonitis with purulent/faecal contamination and varying degree of septicaemia. Good preoperative assessment and early management will decrease the morbidity, mortality, and complications of secondary peritonitis.

REFERENCES

1. Adesunkanmi ARK, Oseni SA, Adejuyigbe O, et al. Acute generalised peritonitis in African children: assessment of severity of illness using modified APACHE II score. ANZ J Surg 2003;73(5):275-279.
2. Dorairajan LN, Gupta S, Deo SV, et al. Peritonitis in India-a decade's experience. Tropical Gastroenterology 1995;16(1):33-38.
3. Ordonez, CA, Puyana JC. Management of peritonitis in the critically ill patient. Surgical Clinics of North America 2006;86(6):1323-1349.
4. Jhobta, RS, Attri AK, Kaushik R, et al. Spectrum of perforation peritonitis in India-review of 504 consecutive cases. World J Emerg Surg 2006;1(1):26.
5. Afridi SP, Malik F, Ur-Rahman S, et al. Spectrum of perforation peritonitis in Pakistan: 300 cases eastern experience. World J Emerg Surg 2008;3(1):31.
6. Crawfurd E, Ellis H. Generalised peritonitis-the changing spectrum. A report of 100 consecutive cases. Br J Clin Pract 1985;39(5):177-178.
7. People JB. Candida with perforated peptic ulcer. World J Surg 1986;100(4):758-764.
8. Ohene-Yeboah M, Togbe B. Perforated gastric and duodenal ulcers in an urban African population. West African Journal of Medicine 2007;25(3):205-211.
9. Franco R, Simone R, Daniele M, et al. Perforated gastric carcinoma: a report of 10 cases and review of the literature. World J Surg Oncol 2006;4:19.
10. Kasahara K, Fukuoka A, Murakawa K, et al. Tuberculous peritonitis developing during chemotherapy for pulmonary and intestinal tuberculosis: a case report. Respirology 2005;10(2):257-260.
11. Bradley EL, Allen K. A prospective longitudinal study of observation versus surgical intervention in the management of necrotising pancreatitis. The American Journal of Surgery 1991;161(1):19-25.
12. Petrozza JC, Mastrobattista JM, Monga M. Gall bladder perforation in pregnancy. Amer J Perinatol 1995;12(5):339-341.
13. Koninger J, Bottinger P, Redecke J, et al. Laparoscopic repair of perforated gastroduodenal ulcer by running suture. Langenbeck's Archives of Surgery 2004;389(1):11-16.
14. Ramachandran CS, Agarwal S, Dip D Goel NB, et al. Laparoscopic surgical management of perforative peritonitis in enteric fever. A preliminary study. Surgical Laparoscopy, Endoscopy, and Percutaneous Techniques 2004;14(3):122-124.
15. Saxe JM, Cropsey R. Is operative management effective in treatment of perforated typhoid? The American Journal of Surgery 2005;189(3):342-344.
16. Svanes C, Salvesen H, Espehaug B, et al. A multifactorial analysis of factors related to lethality after treatment of perforated gastroduodenal ulcer. 1935-1985. Annals of Surgery 1989;209(4):418-423.
17. Gupta S, Kaushik R. Peritonitis-the eastern experience. World J Emerg Surg 2006;1(1):13.
18. Shinagawa N, Muramoto M, Sakurai S, et al. A bacteriological study of perforated duodenal ulcer. Jap J Surg 1991;21(1):1-7.
19. Washington BC, Villalba MR, Lauter CB, et al. Cefamandole-erythromycin-heparin peritoneal irrigation. An adjunct to the surgical treatment of diffuse bacterial peritonitis. World Journal of Surgery 1983;94(4):576-581.
20. Nomikos IN, Katsouyanni K, Papaioannou AN. Washing with or without chloramphenicol in the treatment of peritonitis. A prospective clinical trial. World Journal of Surgery 1986;99(1):20-25.
21. Samuel JC, Qureshi JS, Mulima G, et al. An observational study of the aetiology, clinical presentation, and outcomes associated with peritonitis in Lilongwe, Malawi. World J Emerg Surg 2011;6(1):37.
22. Khanna AK, Misra MK. Typhoid perforation of the gut. Postgraduate Medical Journal 1984;60:523-525.
23. Noon GP, Beall AC, Jorden GL. Clinical evaluation of peritoneal irrigation with antibiotic solution. World J of Surg 1967;67:73.
24. Kaur N, Gupta MK, Minocha VR. Early enteral feeding by nasoenteric tubes in patients with perforation peritonitis. World J Surg 2005;29(8):1023-1027.
25. Kotiso B, Abdurahman Z. Pattern of acute abdomen in adult patients in Tikur Anbessa Teaching Hospital, Addis Ababa, Ethiopia. East and Central African Journal of Surgery 2007;12(1):47-52.