



MANNHEIM PERITONITIS INDEX AS A PROGNOSTIC INDICATOR IN PATIENTS WITH PERITONITIS

Authors

* Adnan Rashid Mattoo¹, Rabia Sofi², Omar Rashid³

1. General & Minimal Access Surgeon, Department of General surgery, District Hospital Ganderbal (J&K), India
2. Post Graduate scholar, Department of Anesthesiology & Critical Care, Govt. Medical College, Srinagar, India
3. Senior Resident Department of Microbiology, SKIMS Medical College Bemina India

ABSTRACT

Background: Peritonitis is a common condition, faced by surgeons since centuries. It is a serious intra-abdominal infection (IAI), a frequently lethal condition, and continues to be one of the major problems that a surgeon has to face. Despite application of aggressive surgical techniques like irrigation with/without antibiotics, on demand reoperations, laparostomy, progress in antimicrobial agents and intensive care treatment, peritonitis continues to have a poor prognosis. **Objectives:** The aim of the present study was: To confirm the predictive value of MPI among the patients with intraoperative diagnosis of secondary peritonitis at the department of Surgery, S.M.H.S Hospital, Srinagar and to evaluate the severity of peritonitis on the basis of MPI. **Methods:** A prospective and observational study was carried out over a period of two years. A total of 172 patients were studied. **Results:** MPI score is strongly associated with outcome, and is an important index for predicting patient outcome in peritonitis. There was statistically significant increase in mortality with increase in MPI score, with survivors having a mean MPI of 18.0 and non-survivors having a mean score of 33.7. Peptic ulcer perforation was the most common etiology (30.81%), followed by appendicular perforation (27.33%). Small gut perforation was the next most common cause (9.88%) and gut gangrene cases amounted to 6.40%. Other etiological sources were genitourinary tract perforations (6.40%), postoperative peritonitis (5.23%), gall bladder perforation (2.33%), ruptured liver abscess (1.74%), Meckel's diverticulum perforation (1.74%) and gastric perforation (1.16%). Mortality in our study was 13.37%, with 23 patients dying out of the 172 operated patients. Highest mortality was seen in patients with colorectal perforation and in patients with gastric perforation,

followed by postoperative peritonitis. **Conclusions:** We conclude that the prognosis of peritonitis has improved due to application of modern surgical techniques; however a severity index is needed to be more objective. Mannheim Peritonitis Index is a useful method to predict outcome in these patients. All MPI adverse factors except for colonic origin behaved as expected. MPI is easy to calculate, does not need any laborious work, economically the cheapest for us and there is a marked difference between survivors and non-survivors.

KEYWORDS: Perforation, Mannheim Peritonitis Index (MPI), Morbidity, prognosis, secondary Peritonitis.

Introduction:

Peritonitis is a common condition, faced by surgeons since centuries. It is a serious intra-abdominal infection (IAI), a frequently lethal condition, and continues to be one of the major problems that a surgeon has to face. Despite application of aggressive surgical techniques like irrigation with/without antibiotics, on demand reoperations, laparostomy, progress in antimicrobial agents and intensive care treatment, peritonitis continues to have a poor prognosis.

Until the end of the last century, peritonitis was treated non-surgically with a mortality of about 90%.¹ In 1926, *Kirschner* in Germany showed that mortality of peritonitis could be reduced by strict implementation of surgical principles, and the mortality rate dropped to less than 50% during the period of 1890-1924.²

The high morbidity and mortality due to peritonitis is largely attributed to the large surface area of the peritoneum (2 square meter), which is almost equivalent to the skin surface area, its propensity to absorb bacterial toxins rapidly and to the presence of mixed infections following perforations of the gastrointestinal tract. Toxemia and hypovolemia are the two lethal factors which ultimately lead to multiple organ failure and must be promptly handled if a satisfactory end result is to be achieved. With the developments in various fields of the management of peritonitis, the high mortality associated with peritonitis has considerably reduced.^{3,4,5}

The commonest cause of secondary peritonitis is a hollow viscus perforation, which may be spontaneous (70-80%) or postoperative (20-30%).^{1,2} Gastrointestinal perforations account for about 25% of acute abdominal emergencies and are still associated with considerable mortality and morbidity.⁶

Perforation peritonitis is the most common surgical emergency in India and there is paucity of data from India regarding its etiology, prognostic evaluation, morbidity and mortality pattern.⁷

The prognosis and outcome of peritonitis patients depends on the interaction of many factors; which in turn depend upon the patient, the disease itself and, the diagnostic and therapeutic intervention carried out. General health of the patient, old age, concurrent medical illness, arterial hypotension at the time of admission, delay in surgical intervention, origin of sepsis and extent of peritoneal contamination are various factors contributing to a significant increase in morbidity and mortality in these patients.⁸⁻¹¹

Presently one of the most accepted scores is *APACHE-II*, which integrates various physiological variables during the first 24 hours within the intensive care unit (ICU) with age and chronic health status of the patient. It has been recommended by the surgical infection societies for risk stratification of patients with peritonitis.¹² However its calculation is both complex and time consuming and the definition of chronic health status is not clear cut. Also, it lacks specificity for peritonitis as there is no definite assessment of operative findings of peritonitis. Other scoring systems in use are *sepsis severity score (SSS)*, *peritonitis index Altona (PIA)*, *POSSUM (physiological and operative severity score for the enumeration of mortality & morbidity)* and the *Mannheim peritonitis index (MPI)*. Possum has so many variables that it's difficult to use clinically. In our hospitals we are required to deal with serious shortage of equipment and lack of staff. Most of our district and sub-district hospitals do not even have an ICU. Various studies have concluded that APACHE-II score as well as MPI correctly determine the severity of intra-abdominal infections and are strongly and independently associated with prognosis, but MPI has the advantage of simplicity and easy application.¹³

The aim of the present study was:

1. To confirm the predictive value of MPI among the patients with intraoperative diagnosis of secondary peritonitis at the department of Surgery, S.M.H.S Hospital, Srinagar.
2. To evaluate the severity of peritonitis on the basis of MPI.
3. To make a prognosis of survival-mortality, considering the risk factors analyzed in this index.

Methods:

At the Postgraduate Department of Surgery, SMHS Hospital Srinagar, a prospective and observational study was carried out over a period of two years. A total of 172 patients were studied.

Peritonitis was defined as intra-abdominal infectious disease, verified during surgery, caused by perforation or infection of a visceral organ or ischemia / necrosis of a part of the gastrointestinal tract due to strangulation, or postoperative peritoneal infection. Patients fulfilling the standard criteria for the study were included. All male and female patients aging 13 years or older, seen at the department of surgery, SMHS hospital, with intraoperative diagnosis of peritonitis, regardless of the etiology were enrolled in the study.

Following patients were excluded:

Patients ≤ 12 years, Patients operated upon and diagnosed at other hospitals, Patients without surgical confirmation of peritonitis, Patients with multiple trauma, Patients with primary peritonitis, pancreatitis, and those with intra-abdominal sepsis associated with peritoneal dialysis (CABG catheters).

All the patients were received in the surgical emergency ward of SMHS hospital and adequately resuscitated and catheterized under all aseptic precautions. In the resuscitative phase, we followed the surviving sepsis guidelines in order to deliver standard therapy to every patient, which included intravenous rehydration with isotonic crystalloid solution, oxygen support (if needed), broad spectrum antibiotic therapy, warming the patient and Ryles tube aspiration and blood transfusion when indicated.

All the patients were evaluated, a detailed history taken and thorough clinical examination was done. Various investigations were carried out as per proforma, viz. complete hemogram, KFT (blood urea, serum creatinine, blood glucose and serum Na⁺ and K⁺), arterial blood gas analysis (ABG), ECG. A plain x-ray chest in a standing position covering both domes of diaphragm with a purpose of demonstrating free gas under diaphragm was done. In incapacitated patients a lateral decubitus view was done instead. X-ray abdomen (erect and lying down) was done. USG abdomen was done in all cases. Needle paracentesis was done in some patients with evidence of free fluid in abdominal cavity, but clinically not consistent with peritonitis. Diagnosis of peritonitis was made on clinical history, examination and radiological investigations, but was confirmed only on exploration.

All the patients in the study group were subjected to emergency laparotomy, the operating decision being taken by the senior resident / consultant on duty. Informed consent was obtained from the patient or the legal representative when the patients were temporarily incapacitated due to severity of their illness. The study was undertaken under the tenets of medical ethics for good clinical practices.

The surgical procedure performed depended upon the operative findings and the surgeon choice, as no guidelines could be laid down, due to varied etiology. However, in general, following principles will be followed:

- Repair – source control
- Purge – peritoneal toilette
- Decompression – external drainage

Incision made depended on the suspected pathology. At surgery, the source of contamination was sought for and controlled. The peritoneal cavity was irrigated with 5-6 liters of warm normal saline (in cases of diffuse generalized peritonitis), and the decision to insert a drain was based upon the source and degree of peritoneal spillage and upon the decision of the operating surgeon. All specimens/organs removed were sent for HPE. The abdomen was closed with continuous number one vicryl sutures in a layered manner in most of the cases.

All the cases were monitored postoperatively in general ward or intensive care ward, as per the severity of peritonitis. Patients were kept on intravenous fluids and intravenous broad spectrum antibiotics (drug regimen was not uniform) with or without ryles tube aspiration. Orals were allowed depending upon the original pathology and return of bowel sounds.

Once the intraoperative diagnosis of secondary peritonitis was made and operative findings registered, the patient was accepted for the study. Using data recollection sheets, risk factors found in the MPI were classified according to the values in the index, and the individual variable scores added to establish the initial MPI score.

In addition to personal data such as name, age, sex, the following intra-hospital information was registered. File number, date of admission and discharge / death, days hospitalized, date of surgery and information related to illness (surgical findings, medical treatment and evolution of illness).

Patient evolution was followed, indicating presence of complications and discharge due to improvement or death. Outpatient follow up was continued for 30 days, to establish perioperative morbidity and mortality. Mortality was defined as death occurring postoperatively in the hospital before discharge. Follow up was done clinically; however, relevant investigations were done as desired. The postoperative complications, we specifically looked for, were wound infection, burst abdomen, enterocutaneous fistulas, intra-abdominal abscess, anastomotic leak, respiratory complications, sepsis and death.

MANNHEIM PERITONITIS INDEX

(Score assigned to each risk factor)

Risk Factor	Points	
Age > 50 years	5	
Female sex	5	
Organ failure	7	
Malignancy	4	
Evolution time (preoperative duration of peritonitis ≥ 24 hours)	4	
Origin of sepsis non-colonic	4	
Diffuse generalized peritonitis	6	
Character of exudate	Clear	0
	Purulent, cloudy	6
	Fecal	12
Total Score	47	

Adapted from Billing et al.¹⁴

The minimum possible score is zero, if no adverse factor is present, and the maximum is 47 if presence of all is confirmed. Patients were grouped into three categories according to MPI points as done by **Fugger et al.**¹⁵

- I – MPI \leq 20
- II – MPI 21-29
- III – MPI \geq 30

These three groups were related to mortality, and for survivors, in hospital stay before clinical discharge was used to assess morbidity.

The patients were also divided into survivors and non-survivors, and clinical data and scores were compared between the two groups.

A useful clinical reference value for MPI was determined, which can be regarded as a cutoff value or the threshold, above which the therapeutic approach has to be more aggressive. This has been kept as 26 in most studies (**Billing**).¹⁴

Each factor in the index was subjected to analysis to assess its significance (in terms of survivors and non-survivors). Frequency tables for cause of peritonitis and their MPI was made and these were related to mortality. Correlations between mortality and clinical factors were conducted by univariate analysis; their statistical significance was evaluated by means of chi-square test for categorical variables and by means of Student t-test for continuous variables. In order to identify those factors independently related to an increased risk of mortality, a logistic regression model was built. For statistical comparison, a p-value <0.05 was considered significant. The Statistical Package for the Social Sciences software (version 16.0) (SPSS™, Chicago, Illinois, USA) was used for statistical analysis.

Results:

The study population included 172 patients of peritonitis admitted in the department of General Surgery. Majority of the patients were males (117) and 55 patients were females, with a male: female ratio of 2.13:1 (Table 1).

Table 1: Demographic profile of the patients:

Age (yr)	Male		Female		Total		p value
	n	%	n	%	n	%	
≤ 20	31	26.5	10	18.2	41	23.8	>0.05
21 to 30	29	24.8	15	27.3	44	25.6	
31 to 40	22	18.8	10	18.2	32	18.6	
41 to 50	21	17.9	3	5.5	24	14.0	
51 to 60	9	7.7	12	21.8	21	12.2	
61 to 70	3	2.6	5	9.1	8	4.7	
> 70	2	1.7	0	0.0	2	1.2	
Total	117	68.0	55	32.0	172	100.0	
mean ± S D	33.2 ± 15.7 (13, 80)		37.9 ± 17.0 (13, 70)		34.7 ± 16.2 (13, 80)		

Preoperative duration of acute symptoms varied from a minimum 3 hours to a maximum of 6 days. Abdominal pain was complained by all the 172 patients (100%). Most of the patients had generalized abdominal pain (74%), while in others it was localized mostly to the right side initially and latter became generalized. It was sudden in onset in 46% of patients. The next common symptom was vomiting (69.77%) and was mostly projectile and bilious. Abdominal distension was complained by 76 patients and low grade to high grade fever by 75 patients. Constipation was present in 42 patients while as, 10 patients presented with loose motions. A total of 73 patients had loss of appetite, and 5 patients complained of bleeding per rectum.

Tenderness was present in all the patients, where as rebound tenderness was elicited in 157 patients. Guarding was present in 138 patients and varying degrees of abdominal distention was observed in 76 patients. Liver dullness was obliterated in 42 patients. Bowel sounds were normally present in 41 patients, sluggish in 115 patients and absent in 16 patients (Fig 1).

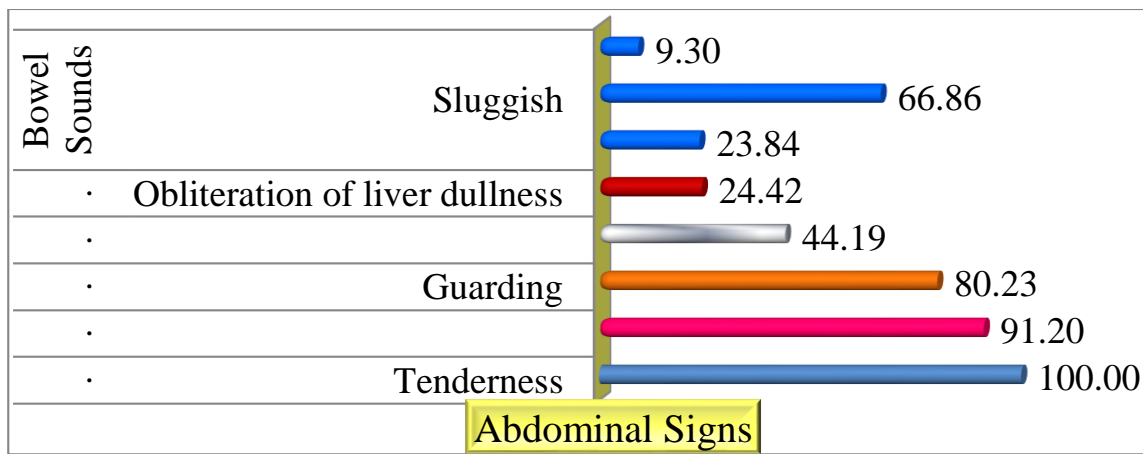


Fig.1

Using the previously mentioned factors, individual scores of each patient were added up to get the individual *MPI* score. *MPI* scores ranged from 4 to 43. Survivors had a minimum *MPI* of 4 and a maximum score of 33 (a mean of 18.03). Non survivors had a minimum *MPI* score of 10 and maximum score of 43, with a mean *MPI* score of 33.70 (Table 2).

Table: 2:- Distribution of *MPI* Scores among various etiological groups:

ETIOLOGY	MPI		P value
	SURVIVORS (n=149)	NONSURVIVORS (n=23)	
	Mean MPI +SD	Mean MPI +SD	
PEPTIC ULCER PERFORATION	16.15+7.47	29.60+11.24	0.001
APPENDICULAR PERFORATION	17.65+5.80	37.00+0.00	0.002
SMALL INTESTINAL PERFORATION	19.64+6.06	34.00+2.65	0.001
GANGRENE GUT	20.33+6.50	32.00+7.07	0.049
POST OPERATIVE PERITONITIS	19.50+7.15	34.00+2.65	0.013
COLORECTAL PERFORATION	13.67+2.34	34.50+5.54	<0.0001
GENITO-URINARY PERFORATION	20.00+4.36	-	-
GB PERFORATION	23.67+4.16	37.00+0.00	0.109
OTHERS	18.67+5.13	39.00+2.83	0.002
TOTAL	18.03+6.46	33.70+6.46	<0.0001

The Etiology of peritonitis in the study population is given in the (table 3).

Table:- 3. Etiology of peritonitis in the study population:

ETIOLOGY	NUMBER OF PATIENTS	PERCENTAGE
Peptic Ulcer Perforation	53	30.81
Appendix Perforation	47	27.33
Small Intestinal Perforation	17	9.88
Colorectal Perforation	12	6.98
Gangrene Gut	11	6.40
Genito-Urinary Perforation	11	6.40
Post Operative Peritonitis	9	5.23
Gall bladder Perforation	4	2.33
Ruptured Liver Abscess	3	1.74
Perforated Meckel's Diverticulum	3	1.74
Gastric Perforation	2	1.16
Total	172	100

Out of the total of 172 patients studied, 23 patients died post operatively – a mortality rate of 13.37%. Among the 23 deaths, 9 were males-7.69% mortality and, 14 were females -25.45% mortality. Among the various etiological groups, peptic ulcer perforation had a 9.43% mortality rate, where as there was 1 death in the perforated appendix group (2.13%). There were 17 patients with small intestinal perforation, out of which 3 died-17.65% mortality. Six patients out of the 12 colorectal perforations died, a mortality of 50%. Gangrene gut had a mortality of 18.18%, where as no patient died among the genitourinary tract perforation groups. Post operative peritonitis had a mortality of 33.33%. We had 1 death among the four gall bladder perforations, 1 death among the 3 patients with ruptured liver abscess and, among the two patients with non peptic ulcer gastric perforation, 1 died. There was no death within the 3 patients with Meckel's diverticulum perforation (Fig 2).

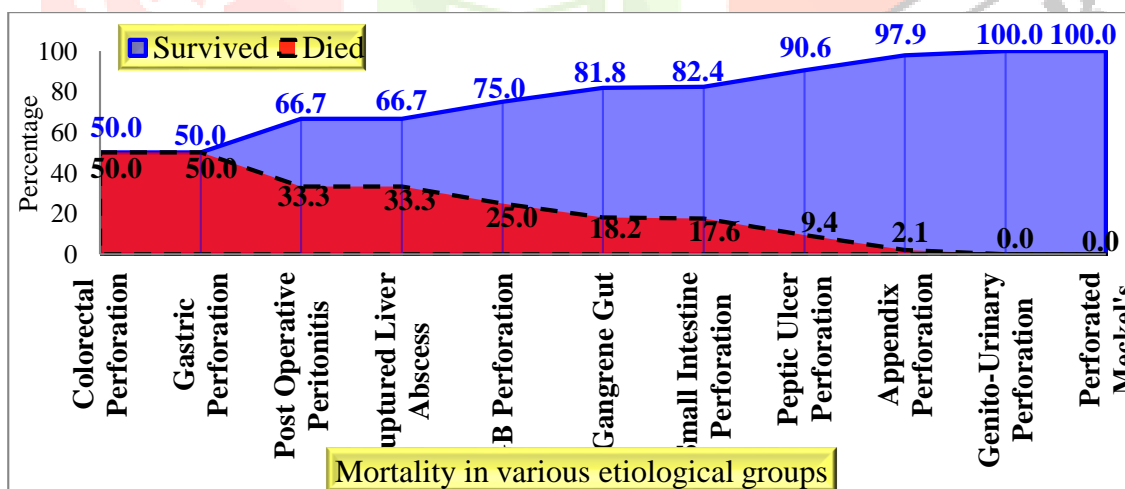


Fig.2

Discussion:

The prognosis of perforative peritonitis is still severe and the sepsis which often develops in these cases is responsible for mortality rates ranging between 19% and 26% in recent series, despite progress in surgery, antibiotic therapy and intensive care.^{16,17}

Just like the experience of others, there was a preponderance of males in our study. There were 117 males (68%) and 55 females (32%) with a male: female ratio of 2.13:1. Similar observations have been made by **S. K. Bhansali (1967)**⁶, who observed only 8 females out of 96 cases of peritonitis. **Rodolfo et al (2002)**¹⁸ had a sample of 174 patients, 84 were females (48%) and 90 were males (52%).

Regarding the etiology of peritonitis in our population, the most common cause of peritonitis was **peptic ulcer perforation**. We had 53 cases (30.81%) of peptic ulcer perforation, out of which, 52 had duodenal ulcer perforation, while one patient had a gastric ulcer perforation. The age in this group ranged from 16 years to 70 years, with a male to female ratio of 5.6:1 (45 males and 8 females). **Edward Crawford (1985)**¹⁹ had also found peptic ulcer perforations to be the most common cause of peritonitis. **Dawson (1963)**²⁰ reported on incidence of 45%. **Appendicular perforation** is the second most common cause in our study with 47 patients (27.33%), followed by **small gut perforation** in 17 patients (9.88%) and **colorectal perforation** in 12 patients (6.98%). **Gut gangrene** was the cause of peritonitis in 11 patients (6.40%), **genitourinary tract perforation** in 11 patients (6.40%) and **postoperative peritonitis** in 9 patients (5.23%). We also had 4 patients of **gall bladder perforation**, 3 patients with **ruptured liver abscess** and 3 patients with a **perforation of Meckel's diverticulum**.

In **Edward's series (1985)**¹⁹, incidence of appendicular perforation was 20%, postoperative peritonitis 8% and small gut perforation was 8%. **Nicholas et al (1993)**²¹ has reported colonic perforation as the most common cause, followed by peptic ulcer perforation and appendicular perforation. Higher incidence of colonic perforations in his series is because of high prevalence of diverticulitis in that population, which is very low in our setup. **Nwigwe et al (2007)**²² had 12 patients of perforated appendicitis, 12 patients of small intestinal perforation, 10 patients of intestinal obstruction, 6 cases of perforated peptic ulcer and 6 cases of malignant colonic perforation in their series of 67 patients.

Pain abdomen was the presenting symptom, complained by all of our 172 patients. Vomiting was the next common feature (120 patients) followed by abdominal distension (76 patients) and fever (75 patients). Constipation was complained at presentation by 42 patients. Tenderness was present in 100% of the patients, rebound tenderness in 91.20% guarding in 80.23% patients and abdominal distention in 44.19% of our patients.

Nicholas et al (1993)²¹ has reported a similar picture with abdominal pain in 100% patients, tenderness in 79% and guarding and distention in 50% of his patients.

On chest radiograph, free gas under the domes of diaphragm was present in 63 patients (36.63%). 81% of patients with peptic ulcer perforation had free gas under diaphragm. **Dandapat and Mukherjee (1991)**²³ found free gas under diaphragm in 72% of his series of peptic ulcer perforation patients.

After resuscitation of the patients, operative management was done. All patients were given intraoperative high volume peritoneal lavage with warm normal saline. A conservative surgical approach was attempted in every patient as long as it was possible. Appendectomy was done in the cases of perforated appendicitis with peritonitis, while closure over an omental patch was done in cases of all peptic ulcer perforations. In patients, where the source of sepsis was small or large intestine, simple closure was done wherever possible with or without exteriorization or resection anastomosis. **Whitman (1991)**² recommends similar operations with high volume peritoneal lavage.

In our series of 172 patients operated for secondary peritonitis, we had 23 deaths – a mortality of 13.37% in our population. Older publications have reported a mortality reaching 90%, when the treatment was non-surgical¹, which dropped to less than 50% in the beginning of 20th century due to strict implementation of surgical principles.² Our study has resemblance with that of **Edward Crawford (1985)**¹⁹. He had similar types of patients as we had and his overall mortality was 16%. **Desa and Mehta (1983)**²⁴ had a mortality of 24.8%, **Angelo Nespoli (1993)**²⁵ had a mortality rate of 20.5% in his series.

In the appendicular perforation group, we had one death (2.13% mortality). **Desa and Mehta (1983)**²⁴ have reported a mortality of 3.4%, while **Nicholas et al (1993)**²¹ has reported one death in patients with appendicular perforation in his series – a mortality of 5%. **Malik et al (2010)**²⁶ had no death among 20 patients of appendicular perforation in his series.

We had a 33.33% mortality in the postoperative peritonitis group, which is comparable to other studies. **Edward Crawford (1985)**¹⁹ had 37% mortality in this group, while **Nicholas et al (1993)**²¹ reported a 50% mortality. **Malik et al**²⁶ also had 33.3% mortality in this etiological group, which is similar to our observation.

Fifty nine (34.30%) of our patients developed complications. Mostly the complications were medical, but some were surgical. Wound infection was the most common complication (15.70%). 13 patients developed postoperative shock, out of which 10 died. Septicemia was documented in 15 patients out of which 5 survived. 14 patients (8.14%) developed postoperative renal failure (creatinine ≥ 2 mg/dl) out of which 11 died. 11 patients developed respiratory tract infection, out of which 4 died.

Among the surgical complications, 9 patients (5.23%) developed burst abdomen, 8 patients (4.65%) developed paralytic ileus; and 7 patients (4.07%) developed anastomotic leak. 6 patients (3.49%) developed postoperative intra-abdominal abscess and 2 patients (1.16%) developed bile leak.

MPI score of each patient has been calculated and postoperative mortality and morbidity has been analyzed with reference to various factors included in the MPI, namely female sex, age >50 years, presence of organ failure, malignancy, preoperative duration of peritonitis ≥ 24 hours, non-colonic origin of sepsis, diffuse generalized peritonitis and, character of exudate, whether clear, cloudy or fecal.

Conclusion

We conclude that the prognosis of peritonitis has improved due to application of modern surgical techniques, however a severity index is needed to be more objective. Mannheim Peritonitis Index is a useful method to predict outcome in these patients. All MPI adverse factors except for colonic origin behaved as expected. MPI is easy to calculate, does not need any laborious work, economically the cheapest for us and there is a marked difference between survivors and non-survivors. The threshold value of 27 can easily be applied in our environment, above which the prognosis has to be guarded. MPI, together with surgeon's clinical judgment may be used to aid the surgeon in making the always difficult decision of reintervening a patient.

Thus Mannheim Peritonitis Index can be easily used for the prognostic assessment of peritonitis in our environment despite the different etiologies, and can also help in comparison of future studies.

Ethical approval: None required.

Funding:None.

Conflicts of interest: None.

References:

1. Alastair M. Thompson. General surgical anatomy and Exam. Schwartz Principles of Surgery, 7th ed, McGraw Hill: 1999.
2. Wittman. Intra-abdominal infection. Marcel Dekker, inc 1991.
3. Alteimer WA. The treatment of acute peritonitis. The Journal of Am Med Assoc 1949; 139(6): 347-351.
4. Bailey C.A., Jumeaux C.V. Aureomycin in the treatment of peritonitis. BMJ 1950; 1: 271.
5. Long William B, William Gill, John da Costa. Peritonitis. Roy Coll Surg Edinb 1970; 15: 158-163.
6. Bhansali S.K. Gastrointestinal perforations. J Postgrad Med 1967; XIII: 1-12.
7. Sharma L. Generalized peritonitis in India. The tropical spectrum. Jap J Surg 1991; 21: 272-77.
8. Hardy JD, George R. Walker, J. Harold. Perforated peptic ulcer. An analysis of 206 consecutive cases with emphasis on pathophysiologic changes and deaths. Ann Surgery 1961: 911.
9. Stephen L. Wangenstein, Robert C. Wray, Gerald T. Golden. Am J Surg 1972; 123: 539.
10. John Boey, John Wong, Guan B. Ong. Bacteria and septic complications in patients with perforated duodenal ulcers. Am J Surg 1982; 143: 635.
11. Feliciano V. David. Emergency management of perforated peptic ulcers in the elderly patients. Am J Surg 1984: 765.

12. Wittman DH, Mosche Schein and Robert EC. Management of secondary peritonitis. *Ann Surg* 1996; 10-18.
13. Pacelli F, Battista DG, Alferi S. Prognosis of IAI's. *Arch Surg* 1996; 131(6): 641-645.
14. Fugger R, Rogy M, Herbst F, Shemper M, Schultz F. Validation of Mannheim Peritonitis Index. *Chirurg* 1988; 59: 598-601.
15. Billing A, Frohlich D, Schildbery F.W. Peritonitis study group. Prediction of outcome using the Mannheim Peritonitis Index in 2003 patients. *Br J Surg* 1994; 81: 209-13.
16. S. Biondo, E. Ramos, D. Fracalvieri, E. Kreisler, J.M. Ragu_e, E. Jaurieta, Comparative study of left colonic peritonitis severity score and Mannheimperitonitis index, *Br. J. Surg.* 93 (2006) 616e622.
17. K.K. Tan, S.L. Bang, R. Sim, Surgery for small bowel perforation in an Asian population: predictors of morbidity and mortality, *J. Gastrointest. Surg.* 14 (2010) 493e499.
18. Rodolfo L, Bracho-Riquelme MC, Armando Melero-Vela MC, M en C, Aidee Torres-Ramirez MC. Mannheim Peritonitis Index validation study at the hospital General de Durango (Mexico). *Cir Ciruj* 2002; 70: 217-225.
19. Crawford E and Ellis H. Generalized peritonitis- the changing spectrum, a report of 100 cases. *Br. J. Clinical practice* 1985 (May); 177-178.
20. Dawson JL. A study of some factors affecting the mortality rate in diffuse peritonitis. *Gut* 1963; 4: 368-371.
21. Nicholas V Christou, Philip S Barrie, Delinger, Paul Waymock. Surgical infection society intra-abdominal study, prospective evaluation of management techniques and outcome. *Arch Surg* 1993; 128: 193-199.
22. Nwigwe CG, Atoyebi OA. Validation of Mannheim Peritonitis Index (A Nigerian Study). *Ebonyi Medical Journal* 2007; 6(1): 3-8.
23. Dandapat MC and Mukherjee LM. G.I. perforations. *Ind J Surg* 1991; 53(5): 193-198.
24. Desa L A, Mehta S J, Nadkarni K M, Bhalerao R A. Peritonitis: A study of factors contributing to mortality. *Indian J Surg.* 1983; 45: 593-604.
25. Angelo Nespoli. The choice of surgical procedure for peritonitis due to colonic perforation. *Arch Surg* 1993; 128: 814-18.
26. Ajaz A Malik, Latief A Dar, Khurshid A Wani, Mehmood A Wani, Rauf A wani, Fazl Q Parray. Mannheim Peritonitis Index and APACHE-II – Prediction of outcome in patients with peritonitis. *Turkish Journal of Trauma and Emergency Surgery* 2010; 16(1): 27-32.