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A study on clinical profile of patients with peritonitis secondary to hollow viscous perforation

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Abstract
Peritonitis involves the rapid removal of contaminants from the peritoneal cavity into the systemic circulation. It occurs because contaminated peritoneal fluid moves cephalad in response to pressure gradients generated by the diaphragm. The fluid passes through stomata in the diaphragmatic peritoneum and is absorbed into lymphatic lacunae. The lymph flows into the main lymphatic ducts via the substernal nodes. The resultant septicemia predominantly involves gram-negative facultative anaerobes and is associated with high morbidity. A prospective clinical study was conducted on 80 consecutive patients who presented to the surgical department of Hospital and Research Centre with peritonitis secondary to hollow viscus perforation. Study population consisted of 80 consecutive patients with peritonitis secondary to hollow viscus perforation which were confirmed on emergency laparotomy. In the study group of 80 patients, majority of the patients had duodenal perforation (40%). Highest survival rate was seen among duodenal perforation 32 of 32(100%) and the highest mortality was seen among patients with gastric, unknown and colonic perforations. The time of presentation of patients ranged from < 24 hours to 10 days. Most of the patients presented within 1-2 days. Mortality increased correspondingly with delay in presentation to the hospital. It was 25% for 1-2days, 62.5% for 3-5 days and 12.5% for 6 to 10 days. Delayed presentation was usually seen in cases of peritonitis secondary to appendicular perforation which had better prognosis compared to other hollow viscus perforation presenting late.

Keywords: Peritonitis, hollow viscus perforation, septicemia

Introduction
The peritoneal cavity is the largest cavity in the body, the surface area of its lining membrane (2 m^2 in an adult) being nearly equal to that of the skin. It can be divided into parietal and visceral portions. The parietal layer lines the abdominal and pelvic cavities and the abdominal surface of the diaphragm. The visceral layer covers the abdominal and pelvic viscera and includes the mesenteries. The peritoneum consists of a fibrous layer (the tunica subserosa) and a surface layer of mesothelium (the tunica serosa) [1].

The parietal peritoneum is only loosely connected with the body wall, separated from it by an adipose layer, the telasubserosa; whereas the visceral peritoneum is usually tightly attached to the organs it covers [2].

The large surface area of the peritoneal cavity allows infection and malignant disease to spread easily throughout the abdomen. If malignant cells enter the peritoneal cavity by direct invasion (e.g. from colon or ovarian cancer) spread may be rapid.

The peritoneal cavity can also act as a barrier to, and container of disease. Intra-abdominal infection therefore tends to remain below the diaphragm rather than spread into other body cavities [2].

Some compartments collect fluid or pus more often than others. These compartments include the pelvis (the lowest portion), the subphrenic spaces on the right and left sides, and Morrison's pouch, which is a postero-superior extension of the subhepatic spaces and is the lowest part of the paravertebral groove when a patient is recumbent. The falciform ligament separating the right and left subphrenic spaces appears to act as a barrier to the spread of infection; consequently, it is unusual to find bilateral subphrenic collections [3].

Phase I of peritonitis involves the rapid removal of contaminants from the peritoneal cavity into the systemic circulation. It occurs because contaminated peritoneal fluid moves cephalad in response to pressure gradients generated by the diaphragm. The fluid passes through stomata in the diaphragmatic peritoneum and is absorbed into lymphatic lacunae.

The lymph flows into the main lymphatic ducts via the substernal nodes. The resultant septicemia predominantly involves gram-negative facultative anaerobes and is associated with high morbidity [4].

Phase II of peritonitis involves synergistic interactions between aerobes and anaerobes as they encounter host complement and phagocytes. The activation of complement is a first-line event in peritonitis and involves innate and acquired immunity; activation occurs mainly by the classical pathway, with the alternative and lectin pathways in support. Phospholipid surfactants produced by the peritoneal mesothelial cells work synergistically with complement to increase opsonization and phagocytosis. Peritoneal mesothelial cells are also potent secretors of pro-inflammatory mediators, including interleukin-6, IL-8, monocyte chemoattractant protein-1, macrophage inflammatory protein-1α and tumor necrosis factor-α. Therefore, peritoneal mesothelial cells play a central role in the cell signaling pathways leading to the recruitment of phagocytes to the peritoneal cavity and the up regulation of mast cells and fibroblasts in the sub-mesothelium [5].

Phase III of peritonitis is an attempt by host defenses to localize infection, mainly via production of fibrinous exudates that traps microbes within its matrix and promotes local phagocytic effectors mechanisms. It also serves to promote the development of abscesses. Regulation of the formation and degradation of fibrinous exudates is vital to this process. The plasminogen-activating activity generated by peritoneal mesothelial cells determines whether the fibrin that forms after peritoneal injury is lysed or organized into fibrous adhesions. In particular, tumor necrosis factor-α stimulates the production of plasminogen activator-inhibitor-1 by peritoneal mesothelial cells, which inhibits degradation of fibrin.

The commonest organisms are Escherichia coli, aerobic and anaerobic streptococci, and bacteroides. Less frequently Clostridium welchii is found; still less frequently staphylococci or Klebsiella pneumoniae (Friedländer's bacillus) [6].

Methodology

A prospective clinical study was conducted on 80 consecutive patients who presented to the surgical department of Hospital and Research Centre with peritonitis secondary to hollow viscus perforation.

Study population consisted of 80 consecutive patients with peritonitis secondary to hollow viscus perforation which were confirmed on emergency laparotomy.

Inclusion criteria

All patients diagnosed to have peritonitis secondary to hollow viscus perforation

Exclusion criteria

1. Patients less than 16 years of age.
2. Post-operative peritonitis
3. Gynaecological causes of peritonitis.
4. Spontaneous bacterial peritonitis.
5. Peritonitis secondary to ventriculo-peritoneal shunts.
6. Blunt and penetrating abdominal injuries.

All the patients were subjected to emergency exploratory laparotomy. The surgical procedure performed depended upon the operative findings and the surgeon's choice, as no guidelines could be laid down due to the varied etiology with peritonitis due to hollow viscus perforation.

Results

Age of the patients in this study ranged from 16years to 75years. The mean age of the patients at the time of admission was 45.55 years (SD 16.43).

Maximum number of patients 17(21.3%) were in the age group of 41-50years, followed by 20% (n= 16) in age group of 21-30years, 18.8% (n=15) in 61-70years, 16.3% (n=13) in both 31-40years and 51-60years. 5% (n=4) of cases were in the age group of <20years, 2.5% (n=2) cases in >70years, 5.33% (n=8) in age group of more than 70years as depicted in the table.

Table 1: Age Group

Age in years	No. of patients	%
<20	4	5.0
20-30	16	20.0
31-40	13	16.3
41-50	17	21.3
51-60	13	16.3
61-70	15	18.8
>70	2	2.5
Total	80	100.0

Highest mortality is in the age group of 41-50years and 61-70years (37.5%). There were 3 patients in each age group. The next highest mortality (25%) is seen in age group of 51-60years. Other age groups did not have any mortality. Mortality rate of 20% (3 of 15 patients) seen in age group of 61-70years. Similarly 17.64% (3 of 17patients) of mortality rate between 41-50years, 15.38% (2 of 13 patients) between 51-60years and is depicted in table no 11. Thus in our study mortality rate is more in the middle and older age group and with increase in age as depicted in the table.

Table 2: Status of Mortality by Age Groups

Age in years	Outcome		Total
	Survived	Expired	
<20	4(5.6%)	0(0%)	4(5%)
20-30	16(22.2%)	0(0%)	16(20%)
31-40	13(18.1%)	0(0%)	13(16.3%)
41-50	14(19.4%)	3(37.5%)	17(21.3%)
51-60	11(15.3%)	2(25%)	13(16.3%)
61-70	12(16.7%)	3(37.5%)	15(18.8%)
>70	2(2.8%)	0(0%)	2(2.5%)
Total	72(100%)	8(100%)	80(100%)

P=0.314, Not significant, Fisher Exact test

Out of 72 patients who had survived 63(87.5%) were males and 9(12.5%) were females. Out of 8 patients who had expired 5(62.5%) were males and 3(37.5%) were females. This is depicted in the table. Thus in our study mortality was observed more in males.

Table 3: Status of Mortality by Gender

Gender	Outcome		Total
	Survived	Expired	
Female	9(12.5%)	3(37.5%)	12(15%)
Male	63(87.5%)	5(62.5%)	68(85%)
Total	72(100%)	8(100%)	80(100%)

P=0.060 +, significant, Chi-Square test

Table 4: Status of Mortality Depending on Site of Perforation

Site of Perforation	Outcome		Total
	Survived	Expired	
Duodenal	32(44.4%)	0(0%)	32(40%)
Pyloric	17(23.6%)	1(12.5%)	18(22.5%)
Gastric	9(12.5%)	3(37.5%)	12(15%)
Ileal	6(8.3%)	0(0%)	6(7.5%)
Appendix	6(8.3%)	0(0%)	6(7.5%)
Unknown	0(0%)	2(25%)	2(2.5%)
Jejunum	1(1.4%)	1(12.5%)	2(2.5%)
Colon	0(0%)	1(12.5%)	1(1.3%)
Rectum	1(1.4%)	0(0%)	1(1.3%)
Total	72(100%)	8(100%)	80(100%)

P< 0.001**, significant, Fisher Exact test

In the study group of 80 patients, majority of the patients had duodenal perforation (40%). Highest survival rate was seen among duodenal perforation 32 of 32(100%) and the highest mortality was seen among patients with gastric, unknown and colonic perforations.

The time of presentation of patients ranged from < 24 hours to 10 days. Most of the patients presented within 1-2 days. Mortality increased correspondingly with delay in presentation to the hospital. It was 25% for 1-2days, 62.5% for 3-5 days and 12.5% for 6 to 10 days. Delayed presentation was usually seen in cases of peritonitis secondary to appendicular perforation which had better prognosis compared to other hollow viscus perforation presenting late.

Table 5: Status of Mortality in Relation to Time of Presentation

Duration (days)	Outcome		Total
	Survived	Expired	
1-2	46(63.9%)	2(25%)	48(60%)
3-5	23(31.9%)	5(62.5%)	28(35%)
6-10	3(4.2%)	1(12.5%)	4(5%)
Total	72(100%)	8(100%)	80(100%)

P=0.106, Not significant, Fisher Exact test

Patients with higher APACHE II score and Mannheims Peritonitis Index (MPI) had more associated complications. 34(42.5%) patients had SSI with a p value of 1.000, a total of 18(22.5%) patients had respiratory complications with a P value of 0.071, a total of 12(15%) patients had renal complications with a P value of <0.001, 21(26.3%) patients had paralytic ileus and none of the patients had burst abdomen.

Table 6: Complications

Complications	Outcome		Total (n=80)	P value
	Survived (n=72)	Expired (n=8)		
Respiratory	14(19.4%)	4(50%)	18(22.5%)	0.071+
Renal	5(6.9%)	7(87.5%)	12(15%)	<0.001**
SSI	31(43.1%)	3(37.5%)	34(42.5%)	1.000
Sepsis	8(11.1%)	6(75%)	14(17.5%)	<0.001**
Burst abdomen	0(0%)	0(0%)	0(0%)	1.000
Paralytic ileus	16(22.2%)	5(62.5%)	21(26.3%)	0.026*

Chi-square test/ Fisher Exact test

Discussion

In hospital, mortality rate due to peritonitis remains high. In the current study, the in hospital mortality rate was 28%, most of them were due to septicemia.

The hospital mortality rate according to other studies ranged from 10% in Mishra *et al.* and Jhobta *et al.* and reaching up to 63 per cent in case of Nithin Agarwal *et al.*. In all these studies septicemia is the main cause of death.

Table 7: Mortality Rate in Various Studies

	Study	mortality rate
1.	Our study	10%
2	Mishra *et al.* [7]	10%
3	RS Jhobta *et al.* [8]	10%
4	Ajaz *et al.* [9]	16.8%
5	Notash *et al.* [10]	17.5%
6	C Ohmann *et al.* [11]	21%
7	Nithin Agarwal *et al.* [12]	63%

The prospective study involved 80 patients of both sexes with secondary peritonitis. Age of the patients in this study ranged from 16years to 75years. The mean age of the patients at the time of admission was 45.55 years(SD 16.43).Maximum number of patients 17(21.3%) were in the age group of 41-50years, Samir Delibegovic *et al.* and Ashis Ahuja *et al.* stated predominant population from age group 21–40 years. C Ohmann *et al.* study showed predominant population in 50-69years age group. These findings are different from our study.

Table 8: Comparison of Predominant Age Group in Peritonitis.

Study	Predominant age group
Samir Delibegovic *et al.* [13]	21-40 years
Ashis Ahuja *et al.* [14]	21-40 years
C Ohmann *et al.* [11]	50-69years
Our study	41-50 years

Highest mortality in our study was in the age group of 61-70years. Notash *et al.* also stated mortality (58.8%) being more in >60 years of age C Ohmann *et al.* cited highest mortality in age >70yrs with 37%. In our study it was observed that mortality rate increases with increase in age.

Table 9: Age Group with Highest Mortality

Studies	Age group with highest mortality
Notash *et al.* [10]	>60 years
C Ohmann *et al.* [11]	>70years
Our study	>60years

Current study showed the male preponderance in peritonitis with ratio of male: female as 5.6:1. Male preponderance was also found in Samir Delibegovic *et al.* with male to female ratio of 3:1, Ajazahamed Malik *et al.* with 69:32 and also in Sharma R, Huttunen *et al.*. In our study mortality rate was observed more among males (62.5%) than females (37.5%).

The perforations of proximal gastrointestinal tract were six times as common as perforations of distal gastrointestinal tract as has been noted in earlier studies from India, which is in sharp contrast to studies from developed countries like United States, Greece and Japan which revealed that distal gastrointestinal tract perforations were more common.

Gastroduodenal perforations were most common site of etiology for perforation. But many studies had small intestine as most common site.

Table 10: Site of Perforation in Different Study Group

	Study	Site of Perforation		
		Gastroduodenal	**Small intestine**	**Large intestine**
1	Ajaz Ahamed Malik *et al.* [9]	30.6%	9.9%	5.9%
2	Notash *et al.* [10]	60%	42.5	
3	RS Jhobta [8]	65.67%	18.27%	3.7%
4	Nithin Agarwal *et al.* [12]	23%	43%	6%
5	Our study	77.5%	10%	2.6%

Overall mortality rate in peritonitis due to hollow viscus perforation in our study was 10%. The individual mortality according to etiology showed highest with gastroduodenal perforation (50%) as seen in Notash *et al.* study, but Ajaz found highest mortality in large intestine perforation. Most of the study showed maximum mortality with colonic perforation.

Table 11: Comparing Site Specific Mortality Rate

	Study	Site specific mortality rate		
		Gastro duodenal	**Small intestine**	**Large intestine**
1	AjazAhamed Malik *et al.* [9]	9.6%	2%	66.7%
2	Notash *et al.* [10]	23.1%	14.3%	
4	Nithin Agarwal *et al.* [12]	8.2%	43%	19.2%
5	Our study	50%	12.5%	12.5%

Conclusion

Peritonitis secondary to hollow viscus perforation is most common in young males in their prime age. In hospitals, mortality rate for perforative peritonitis remains high in spite of advances in investigation, improved treatment modality, better inpatient care and advanced hospital resources.

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