DOI: 10.5152/UCD.2013.1955



Predictors of mortality and morbidity in peritonitis in a developing country

Parwez Sajad Khan¹, Latif Ahmad Dar², Humera Hayat³

Objective: Generalized peritonitis is still a common emergency managed by most general surgeons throughout the world. If the outcome in these patients can be correctly predicted, then better management can be instituted to those patients in need. This study aims to identify factors in patients with peritonitis which have a significant bearing on morbidity and mortality. These factors could be later used to predict the outcome in patients with generalized peritonitis.

Material and Methods: A total of one hundred patients with peritonitis were studied. Factors including age, pulse and respiratory rate, temperature, hemoglobin, total leukocyte count, hematocrit, urea, creatinine, sodium, potassium, pH, PaO₂ levels at the time of surgery along with peritoneal contamination and duration of surgery were noted. Using the Students t test, factors were identified which had a statistically significant influence on the outcome.

Results: Thirty-six patients in the study developed complications and 17 died. Statistics showed that 8 factors influenced morbidity and 11 influenced mortality.

Conclusion: Identifying variables which influence the outcome of patients with peritonitis is an important initial step. Once these factors have been identified, the outcome of patients can be correctly predicted and better management can be instituted to those patients in need.

Key Words: Peritonitis, mortality, morbidity

INTRODUCTION

Generalized peritonitis remains a severe condition despite a dramatic improvement since Kirchner (1) showed that mortality rates could be reduced by strict implementation of surgical principles. At present, mortality is reported to be between 13-43% (2). The prognosis and outcome of peritonitis depend on the complex interaction of many factors, patient related, disease related and intervention related. The chronic health status is also noted to influence the outcome. Whittman demonstrated that age, duration of symptoms, white cell count, mechanisms and origin of infection are related to outcome (2). To establish the effects of various factors affecting the morbidity and mortality, we did an extensive search on PubMed and Google but could not find the overall impact of various variables on the outcome of peritonitis. So, we thought it worthwhile to plan a study on 100 adult patients with secondary/generalized peritonitis in our department. Our study is an attempt to evaluate the influence of multiple factors on morbidity and mortality of patients admitted with generalized peritonitis and evaluate their significance statistically.

MATERIAL AND METHODS

A total of one hundred adult patients with the diagnosis of generalized peritonitis admitted to our department of general surgery over a period of two and a half years, were taken as subjects for the present study. Any patients with primary peritonitis, traumatic peritonitis, localized peritonitis or peritonism were excluded from the study. All patients were resuscitated prior to surgery and examined clinically and evaluated by routine investigation as per the set protocol. The pulse rate, systolic blood pressure (SBP), respiratory rate (RR), weight (kg) and temperature (Celsius) recordings were noted as taken at the time of admission. All the investigations were performed after admission prior to resuscitation so as not to alter the results. Subsequently, all the patients underwent exploratory laparotomy with a surgical procedure tailored to the operative findings. Post operatively all patients were followed during their hospital stay. Statistical analysis of distribution was done and equal variances found. Two statistical comparisons were then performed. The patients were divided into 2 groups,

¹Department of Health and Medical Education, Surgery, Srinagar, India

²Sher-i-kashmir Institute of Medical Sciences, Surgery, Srinagar, India

³Sher-i-kashmir Institute of Medical Sciences, Community Medicine, Srinagar, India

Address for Correspondence Dr. Parwez Sajad Khan

Department of Health and Medical Education, Surgery, Srinagar, India Phone.: 5329906663725 e-mail: parwezsajad@yahoo.co.in

Received: 14.01.2013 Accepted: 11.08.2013

©Copyright 2013 by Turkish Surgical Association Available online at www.ulusalcerrahidergisi.org those who developed complications and those who did not develop any complications. In these 2 groups, preoperative findings and results of investigations were statistically compared in order to note whether there were any factors responsible for complications. Then we again statistically compared the same parameters between survivors and nonsurvivors to note which factors had a bearing on mortality. Finally, a multivariate analysis was done using logistic regression, and the morbidity and mortality was analysed with respect to various laboratory and non laboratory parameters. The statistical package used was SPSS 15.0 and the cut off value for significance was p<0.05.

RESULTS

The etiology of peritonitis in the 100 patients is demonstrated in Table 1. Small bowel perforation was due to typhoid in 6 and tuberculosis in 4, while the etiology of perforation in the large bowel was malignancy in 4, volvulus in one and intussusception in another. The miscellaneous group consisted of three patients with ruptured liver abscess, one with pancreatic necrosis, one with twisted enterogenous cyst, and the other with a perforated Meckel's diverticulum. The 12 postoperative peritonitis patients consisted of two patients who had undergone hysterectomy, dilatation and curettage in three, laparoscopic cholecystectomy in three, oesophageal dilatation for esophagus cancer in two, endoscopic polypectomy in one, and oesophago-gastrectomy with feeding jejunostomy in one with an anastomotic leak.

Overall, 54 complications developed in 36 patients (36%) (Table 2). Of the 11 patients with shock 6 patients died. Septicemia was documented in 10 patients. *E. coli* was the most commonly isolated organism from blood. Patients were managed by culture sensitivity guided antibiotics but 5 of these patients died. Six patients developed postoperative renal failure, three of these were complicated by multiorgan failure, and 4 died. 5 patients developed anastomotic leaks, 2 died despite re-exploration. Bile leak was noted in one patient where CBD was repaired at first laparotomy. Respiratory tract infection developed in 5, urinary tract infection in 2, wound infection in 10 and burst abdomen in 4. None of these patients succumbed.

In the study 17 patients died, a mortality of 17%. None of the patients died before surgery. The following factors were compared between the survivors and non-survivors and are represented in Table 3. Out of the 100 patients four patients were from outside the state. One was a labourer from Bihar, one a businessman from Maharashtra and two were from the armed forces stationed in Srinagar. Of the remaining 96, 44 (45.8%) patients were from within the city limits and 52 (54.2) from the rural areas of our state. There were 5 deaths among the urban group of patients -11.36% mortality rate (MR).

The factors found to significantly affect the mortality and morbidity are demonstrated in Tables 3-6. These observations regarding these factors are described below.

Table 1. Etiology of peritonitis and respective mortality rates (n=100)

Etiological group	No of patients	Survivors (S)	Non Survivors (NS)	Mortality Rate. (MR)
Peptic ulcer perforation	31	28	3	9.6%
Appendicular perforation	20	20	0	0
Small gut perforation	10	8	2	20%
Colonic perforation	6	2	4	66.7%
Genital organs	6	6	0	0
Gangrene of gut	4	3	1	25%
Stomach perforation (other than peptic)	3	3	0	0
GB Perforation	2	2	0	0
Postoperative peritonitis	12	8	4	33.3%
Miscellaneous	6	3	3	50%
Total	100	83	17	17%

Table 2. Complications and related mortality (n=36)

	No of patients	No of deaths
Septicemia	10	5 (50%)
Shock	11	6 (54.5%)
Renal failure	6	4 (66.6%)
Anastomotic leak	5	2 (40%)
Respiratory tract infection	5	0
Urinary tract infection	2	0
Wound infection	10	0
Burst Abdomen	4	0
Open Abdomen	1	0

Age

The age in the study ranged from 15-90 years with an overall average of 40.06 ± 17.60 . We divided the patients into three groups as shown in Table 6. Complications were most common in the elderly, as was the mortality. However, although the age was a significant factor in depicting the mortality (Table 3) it did not significantly affect the morbidity (Table 4).

Pulse and Respiratory Rate

The average pulse in the study was 102.329±17.497 (58-162) while the average respiratory rate was 24.060±8.723 (12-48). Both pulse and respiratory rate were found to be significant factors for the development of complications and death (Table 3, 4).

Temperature

The average temperature was 37.762±0.634 (37-39.5). Although the temperature was found to be a significant factor for the development of complications, it did not produce a statistically significant effect on the mortality (Table 3, 4).

Table 3. Factors influencing outcome in patients (n=100)							
Surviving (n=83)				Non Surviving (n=17)			
Parameter	Min	Max	Mean±SD	Min	Max	Mean±SD	
Age	15	90	36.57±15.30	28	75	56.24±13.21	.000
Pulse	58	162	100.11±16.65	90	140	110.94±13.95	.014
SBP	60	150	108.99±17.25	55	180	105.82±30.93	.556
RR	12	42	22.59±7.67	18	48	33.53±10.71	.000
Weight	20	75	61.26±9.54	45	68	58.35±7.98	.243
Temperature	37	39	37.66±0.57	37	39	37.85±0.49	.206
Hemoglobin	7.0	16.9	11.53±2.20	6	12.6	9.51±1.75	.001
TLC	4.20	29.50	10.87±3,95	3.9	32	14.60±7.46	.004
DLC- N%	51	93	78.86±8.69	57	93	80.76±9.71	.420
Hct %	26	48	36.79±4.88	26	48	39.88±7.34	.033
Urea	18	107	40.29±19.67	43	138	76.53±26.97	.000
Creatinine	0.5	2.5	1.25±0.41	1.7	4.3	2.56±0.68	.000
Na+	112	152	133.14±4.96	121	145	129.9±6.06	.024
K+	2.1	5.6	3.47±0.68	2.4	6.3	3.67±1.31	.360
рН	7.03	7.52	7.37±0.08	7.0	7.4	7.25±0.10	.000
PaO ₂	45	98	81.54±11.21	46.9	78	63.42±6.98	.000
SBP: systolic blood pre	SBP: systolic blood pressure, RR: respiratory rate, TLC: total leukocyte count, DLC: differential leukocyte count, Hct: hematocrit						

Table 4. Factors influencing occurrence of complications (n=100)									
	Complications Absent (n=64)		Compl	Complications Present (n=36)			P (2 tail)	Sig	
Parameter	Min	Max	Mean±SD	Min	Max	Mean±SD	t test		
Age	18	90	38.34±15.86	15	75	42.83±17.90	1.290	.200	NS
Pulse	58	120	98.66±13.66	76	162	108.06±19.97	2.778	.007	S
SBP	60	150	110.31±17.04	55	180	105.00±24.63	-1.266	.209	NS
RR	12	44	22.34±8.51	16	48	28.37±9.22	3.284	.001	S
Weight	45	75	63.26±7.51	20	70	56.13±10.63	1.183	.239	NS
Temp (C)	37	39	37.13±0.55	37	39	37.79±0.56	-3.901	.000	S
Hb (gm/dL)	7	16.9	11.7±2.32	6	14	10.23±1.79	-3.254	.002	S
TLC (x10 ⁹)	5.3	29.50	11.28±4.39	3.90	32	11.91±5.75	0.611	.542	NS
DLC- N%	56	92	79.06±7.95	51	93	79.40±10.44	0.182	.856	NS
Hct %	26	48	37.34±4.82	26	48	37.27±6.56	-0.060	.953	NS
Urea	18	107	40.08±20.20	22	138	58.29±29.02	3.688	.000	S
Creatinine	0.5	2.9	1.35±0.50	0.5	4.3	1.70±0.87	2.577	.011	S
Na	112	152	133.33±5.65	121	139	131.25±4.23	-1.910	.059	NS
К	2.5	5.6	3.49±0.72	2.1	6.3	3.52±0.98	0.171	.865	NS
рН	7.00	7.52	7.37±0.07	7.03	7.48	7.32±0.10	-2.803	.006	S
PaO ₂	45	98	81.87±12.08	51.20	90	72.15±11.16	-3.938	.000	S

Hemoglobin

The average Hb in the study was 11.8±2.25 (6-16.9). It significantly affected both the outcome and morbidity (Table 3-5). Subsequently, we divided the patients into three groups based on the Hb levels to note the influence of individual levels on morbidity and mortality.

Total Leukocyte Counts

The mean TLC in the study was 11.5 ± 4.89 (3.9-32). Surprisingly, the TLC counts were not significantly different in the patients with complications and those without them (Table 4). However they were a significant predictor for mortality (Table 3, 5). Leukocyte counts of less than 4×10^9 were not associated with survival.

Haematocrit

The mean haematocrit was noted to be 37.12±5.96, range 26-49. It was noted to be a significant factor in predicting the mortality, but again not a significant factor for morbidity (Table 3-5). It was also noted that when the haematocrit was less than 40, both the morbidity and mortality were significantly lower than seen in those with a haematocrit of over 40.

Creatinine

The creatinine levels were found to range from 0.5-4.3 with an average of 1.5 \pm 0.73. Creatinine levels were found to be significant predictors of both morbidity and mortality (Table 3-5). Maximum morbidity and mortality was noted in the group of patients who had creatinine levels of more than 1.5.

Urea

The average urea levels in the series were 48.70±27.86 (15-138). It was noted to be a significant factor for both morbidity and mortality (Table 3-5). The incidence of both complications and deaths were found to rise with levels of more than 40 (Table 5).

In multivariate analysis of various laboratory parameters, most of the variables present in the table above were significantly associated with morbidity and mortality in patients with peritonitis (Table 5). PaO_2 tops the list with a score of 318.617 followed by urea, creatinine, pH, Hb and TLC in that order.

Sodium

The average sodium levels in the series were 132.43±5.53 (112-152). Sodium levels, although noted to be a significant factor for mortality, did not influence the occurrence of complications significantly (Table 3, 4). Maximum morbidity and mortality were noted in the patients who had sodium levels of lower than 135 (Table 5).

Potassium

Potassium levels ranged from 2.1-6.3 with an average of 3.53±0.87. Potassium was not found to be a significant factor for morbidity and mortality (Table 3, 4).

рΗ

In this series the pH ranged from 7-7.5 with an average of 7.35 ± 0.09 . The pH was a significant factor for both morbidity and mortality (Table 3, 4). The morbidity and mortality were maximum in the group of patients with a pH of less than 7.35 (Table 5).

PaO,

The average PaO_2 in the series was 79.49 ± 13.4 with a range of 45-98. Both morbidity and mortality were significantly affected by PaO_2 levels (Table 3, 4). A level below 60 was associated with the highest morbidity and mortality (Table 5).

Delay in Treatment

The minimum duration of symptoms before treatment was started was 3 hours, while the maximum was 5 days. Maximum mortality was noted in the group who presented after 48 hours (Table 6).

Table 5. Multi-logistic regression analysis -of lab parameters (n=100). Rank order of regression scores with respect to mortality and morbidity

Hb (gm/dL) >12		No of patients	Morbidity	Mortality	Regression score
9-12	Hb (gm/dL)				
6-9 11 7 4 <6 0 0 0 0 TLC (x10°) <4 4 4 2 2 4-11 60 11 0 198.675 >11 38 23 15 Hct % <25 0 0 0 0 26-40 73 19 9 169.956 >40 27 10 6 Creatinine <1.5 71 15 0 1.5-3.0 27 11 16 268.885 >3.0 2 1 1 Urea <40 56 7 1 41-80 28 17 11 298.706 >80 16 9 5 Sodium <135 63 27 9 136-150 35 9 8 94.142 >150 2 0 0 pH <7.35 37 23 14 7.36-7.5 61 11 3 249.593 >7.5 2 1 0 PaO ₂ <60 9 0 9 61-90 68 26 8 318.617	>12	35	4	1	
6-9	9-12	54	21	12	213 273
TLC (x10°) <4	6-9	11	7	4	213.273
<4	<6	0	0	0	
4-11 60 11 0 198.675 >11 38 23 15 Hct % <25 0 0 0 0 26-40 73 19 9 169.956 >40 27 10 6 Creatinine <1.5 71 15 0 1.5-3.0 27 11 16 268.885 >3.0 2 1 1 Urea <40 56 7 1 41-80 28 17 11 298.706 >80 16 9 5 Sodium <135 63 27 9 136-150 35 9 8 94.142 >150 2 0 0 pH <7.35 37 23 14 7.36-7.5 61 11 3 249.593 >7.5 2 1 0 PaO₂ <60 9 0 9 61-90 68 26 8 318.617	TLC (x10°)				
>11 38 23 15 Hct % <25 0 0 0 0 26-40 73 19 9 169.956 >40 27 10 6 Creatinine <1.5 71 15 0 1.5-3.0 27 11 16 268.885 >3.0 2 1 1 Urea <40 56 7 1 41-80 28 17 11 298.706 >80 16 9 5 Sodium <135 63 27 9 136-150 35 9 8 94.142 >150 2 0 0 pH <7.35 37 23 14 7.36-7.5 61 11 3 249.593 >7.5 2 1 0 PaO ₂ <60 9 0 9 61-90 68 26 8 318.617	<4	4	2	2	
Hct % <25	4-11	60	11	0	198.675
<pre><25 0 0 0 26-40 73 19 9 169.956 >40 27 10 6 Creatinine <1.5 71 15 0 1.5-3.0 27 11 16 268.885 >3.0 2 1 1 Urea <40 56 7 1 41-80 28 17 11 298.706 >80 16 9 5 Sodium <135 63 27 9 136-150 35 9 8 94.142 >150 2 0 0 pH <7.35 37 23 14 7.36-7.5 61 11 3 249.593 >7.5 2 1 0 PaO₂ <60 9 0 9 61-90 68 26 8 318.617</pre>	>11	38	23	15	
26-40 73 19 9 169.956 >40 27 10 6 Creatinine <1.5 71 15 0 1.5-3.0 27 11 16 268.885 >3.0 2 1 1 Urea <40 56 7 1 41-80 28 17 11 298.706 >80 16 9 5 Sodium <135 63 27 9 136-150 35 9 8 94.142 >150 2 0 0 pH <7.35 37 23 14 7.36-7.5 61 11 3 249.593 >7.5 2 1 0 PaO ₂ <60 9 0 9 61-90 68 26 8 318.617	Hct %				
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Creatinine <1.5 71 15 0 1.5-3.0 27 11 16 268.885 >3.0 2 1 1 Urea <40 56 7 1 41-80 28 17 11 298.706 >80 16 9 5 Sodium <135 63 27 9 136-150 35 9 8 94.142 >150 2 0 0 pH <7.35 37 23 14 7.36-7.5 61 11 3 249.593 >7.5 2 1 0 PaO ₂ <60 9 0 9 61-90 68 26 8 318.617	26-40	73	19	9	169.956
<pre><1.5</pre>	>40	27	10	6	
1.5-3.0 27 11 16 268.885 >3.0 2 1 1 Urea <40 56 7 1 41-80 28 17 11 298.706 >80 16 9 5 Sodium <135 63 27 9 136-150 35 9 8 94.142 >150 2 0 0 pH <7.35 37 23 14 7.36-7.5 61 11 3 249.593 >7.5 2 1 0 PaO ₂ <60 9 0 9 61-90 68 26 8 318.617	Creatinine				
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Urea <40 56 7 1 41-80 28 17 11 298.706 >80 16 9 5 Sodium <135 63 27 9 136-150 35 9 8 94.142 >150 2 0 0 pH <7.35 37 23 14 7.36-7.5 61 11 3 249.593 >7.5 2 1 0 PaO ₂ <60 9 0 9 61-90 68 26 8 318.617	1.5-3.0	27	11	16	268.885
<40 56 7 1 41-80 28 17 11 298.706 >80 16 9 5 Sodium <135 63 27 9 8 94.142 >150 2 0 0 pH <7.35 37 23 14 7.36-7.5 61 11 3 249.593 >7.5 2 1 0 9 61-90 68 26 8 318.617	>3.0	2	1	1	
41-80 28 17 11 298.706 >80 16 9 5 Sodium <135 63 27 9 136-150 35 9 8 94.142 >150 2 0 0 pH <7.35 37 23 14 7.36-7.5 61 11 3 249.593 >7.5 2 1 0 PaO ₂ <60 9 0 9 61-90 68 26 8 318.617	Urea				
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Sodium <135 63 27 9 136-150 35 9 8 94.142 >150 2 0 0 pH <7.35 37 23 14 7.36-7.5 61 11 3 249.593 >7.5 2 1 0 PaO ₂ <60 9 0 9 61-90 68 26 8 318.617	41-80	28	17	11	298.706
<pre><135 63 27 9 136-150 35 9 8 94.142 >150 2 0 0 pH </pre> <pre><7.35 37 23 14 7.36-7.5 61 11 3 249.593 >7.5 2 1 0</pre> PaO ₂ <pre><60 9 0 9 61-90 68 26 8 318.617</pre>	>80	16	9	5	
136-150 35 9 8 94.142 >150 2 0 0 pH <7.35 37 23 14 7.36-7.5 61 11 3 249.593 >7.5 2 1 0 PaO ₂ <60 9 0 9 61-90 68 26 8 318.617	Sodium				
>150 2 0 0 pH <7.35 37 23 14 7.36-7.5 61 11 3 249.593 >7.5 2 1 0 PaO ₂ <60 9 0 9 61-90 68 26 8 318.617	<135	63	27	9	
pH <7.35 37 23 14 7.36-7.5 61 11 3 249.593 >7.5 2 1 0 PaO ₂ <60 9 0 9 61-90 68 26 8 318.617	136-150	35	9	8	94.142
<7.35 37 23 14 7.36-7.5 61 11 3 249.593 >7.5 2 1 0 PaO ₂ <60 9 0 9 61-90 68 26 8 318.617	>150	2	0	0	
7.36-7.5 61 11 3 249.593 >7.5 2 1 0 PaO ₂ <60 9 0 9 61-90 68 26 8 318.617	рН				
>7.5 2 1 0 PaO ₂ <60 9 0 9 61-90 68 26 8 318.617	<7.35	37	23	14	
PaO ₂ <60 9 0 9 61-90 68 26 8 318.617	7.36-7.5	61	11	3	249.593
<60	>7.5	2	1	0	
61-90 68 26 8 318.617	PaO ₂				
	<60	9	0	9	
>90 25 4	61-90	68	26	8	318.617
	>90	25	4		

Nature of Peritoneal Contaminant

The majority of the patients had purulent peritoneal fluid at laparotomy; however the patients with feculent peritoneal contamination had maximum morbidity and mortality (Table 6).

Duration of Surgery

In our series, the patients who underwent longer procedures had a worse outcome (Table 6).

Table 6. Multi-logistic regression analysis of other factors (non-lab parameters) (n=100). Rank order of regression scores with respect to mortality and morbidity

	No of patients	Morbidity	Mortality	Regression score
Age in years				
<30	36	7	-	
31-60	54	22	13	216.889
>60	10	3	4	
Duration of syn	nptoms (dela	ay in treatmen	t)	
<12 hours	24	5	1	
12-24 hours	22	9	2	289.946
24-48 hours	19	8	1	203.5 10
>48 hours	35	18	13	
Peritoneal fluid	nature			
Exudative	14	2	-	
Purulent	62	24	12	
Feculent	15	8	5	230.123
Bilious	5	1	-	
Haemorrhagic	4	1	-	
Duration of Sur	gery			
<2 hours	71	18	5	93.347
>2 hours	29	18	12	75.5 17
Comorbidity				
Present	41	24	16	271.397
Absent	59	11	1	2, 1,02,

Comorbidity

Comorbid conditions were recorded for which the patient was taking any treatment. Patients with comorbidity had the maximum mortality and morbidity (Table 6).

On multivariate analysis (multilogistic regression) of various non-lab parameters (Table 6), most of the parameters were found to be significantly associated with morbidity and mortality in the patients. Delay in management has the highest score of 289.946 followed by comorbidity in the patients. Other significant factors included the nature of the peritoneal fluid and age of the patient.

DISCUSSION

Secondary peritonitis is the condition with which most of the general surgeons frequently deal in emergency situations. It still carries a high morbidity and mortality despite a dramatic decrease. In our study of 100 adult patients admitted as generalized peritonitis, the most common etiology was peptic ulcer perforation (31%), followed by appendicular perforation (20%) and small gut perforation (10%). This is in contrast to Western literature, where lower gastrointestinal tract perforation predominates (3-5). This is in agreement with other studies from India, except for the difference that in the remainder of our country small bowel perforation secondary to typhoid may constitute a higher percentage (4-6).

The overall morbidity in our series was 36% (Table 2), with some patients developing more than one complication. A higher morbidity of 50% was recently reported in 2006 by Jhobta in his review of 504 cases in a similar demographic region (4). The nature of complications in our series is similar to that reported by others. Desa and Mehta (7) reported wound infection in 17, burst abdomen in 10, renal failure in 13 and anastamotic leaks in 11 of his series of 161 patients. Stephen (8) reported wound infection in 30, anastomotic breakdown in 5, gut fistulae in 8, renal complications in 30 and septicemia in 20 patients. In our series, we noted a mortality of 17%. The mortality reported for secondary peritonitis in the literature varies (5, 9-11). Desa and Mehta (7) reported a mortality of 24.8%, while Angelo Nespoli (12) reported it to be 20.5%. A higher mortality was seen by Stephen-50% (8). In a study by Koperna and Schulz (13), a mortality of 18.5% was noted. The average age in the survivors was 36.57 and that of the non-survivors was 56.24, signifying higher mortality in elderly patients. Similar results have been documented by other studies (7, 14). However, Boey (15) did not find age to be a significant factor of mortality in his study of peritonitis secondary to duodenal perforations.

However, age was not found to affect the morbidity significantly. The effect of age on the outcome is probably due to the presence of co-morbidity with advanced age and decreased physiological reserves. The presence of co-morbidity was shown to have a significant effect on both the morbidity and mortality in our series. 58.5% of the patients with co morbidity developed complications and 39% died. In comparison, only 18.6% developed complications and only 1.6% died of the patients who had no co-morbid condition. A similar influence of underlying disease on the outcome is well substantiated in literature (13-15). However, the nature of the underlying condition and its influence on the outcome has not been studied.

In our study the patients who survived had a mean duration of symptoms of 34.1 hours, while those who died had an average of 56.2 hours. Patients with delayed presentation for treatment fared the worst in our study. A similar conclusion has been documented by most other studies (16, 17). The average pulse in the survivors was 100.11±16.65 and in the non survivors was 110.94±13.95. This difference was found to be statistically significant. Similarly, the average pulse in those without complications was 98.66±13.66, while in those who developed complications it was 108.06±19.97 and again the difference was significant.

Therefore, in our series pulse was found to be an important factor influencing both the morbidity and mortality. The average respiratory rate in survivors, when compared to that of the non survivors, was again found to be statistically significant, as was the difference in those with complications and those without (Table 3, 4). Therefore respiratory rate was also found to be an influencing factor for the development of complications and mortality. Surprisingly, our study showed that the systolic blood pressure did not influence the morbidity nor the mortality. This is in striking contrast to the study by Boey (15) who demonstrated that outcome was significantly related to preoperative shock.

In our study, the systolic pressure readings were recorded at initial reception of the patient in the emergency department before any resuscitation was done. A major portion of these patients become stable after the resuscitation, therefore in our study the initial systolic pressure may not have made a significant difference to the outcome in these patients.

Maybe if only those patients who remained hypotensive despite resuscitation are considered then we would have noted a significant result. Non operative management has been considered in patients with unresponsive shock but in this series we operated on all patients with shock (18-21).

Higher temperature, usually considered an indicator of underlying sepsis, is also a part of the body's inflammatory response. Both of these are components of peritonitis, therefore a fever may not signify overwhelming sepsis and a poor outcome. In our study the temperature did not influence the mortality; however, it had a significant bearing on the morbidity (Table 3, 4).

Haemoglobin also affected both outcome and morbidity (Table 3, 4). The lower the haemoglobin levels, the more the complications and the deaths (Table 5). Haemoglobin is responsible for transfer of oxygen to the tissues and therefore its deficiency would lead to tissue hypoxia and exacerbate organ failure. This would understandably lead to more complications and increase the mortality. Haemoglobin has not been studied as a variable influencing the outcome in patients with peritonitis either directly or as part of any commonly used scoring system such as APACHE II, Mannheims Peritonitis Index or Peritonitis Index Altona etc. Since the influence of haemoglobin seems to be significant both for morbidity and mortality, it may be worthwhile considering including this parameter in scoring systems.

In this study, the total leukocyte counts were not significantly different in patients with complications and those without them, but higher TLC was a significant predictor of mortality (Table 3-5). TLC has been used as a part of scoring systems for predicting the outcome in peritonitis, most notably APACHE II (22). In this scoring system both patients with extremely high TLC levels and those with low levels are allotted maximum points.

This was also noted in the current study where maximum morbidity and mortality was noted in the two extremes of the range (Table 5). Similarly, kidney function tests (urea and creatinine), arterial pH and arterial oxygen concentration (PaO₂) were found to have a significant effect on both morbidity and mortality (Table 3-5). These parameters are used in the APACHE II scoring system to predict outcome (22). Also, haematocrit and sodium levels were significantly related to mortality but not to the morbidity (Table 3-5). Potassium levels had no relation at all either to morbidity or to the mortality (Table 3, 4). However, it is still one of the parameters in the APACHE scoring system (22). There are many studies which have attempted to establish the importance of peritoneal soakage and duration of perforation as a factor contributing to morbidity and mortality (23-26). Our study confirmed that maximum morbidity

and mortality is noted in the patients with feculent peritonitis (Table 6).

When we compared the time of surgery, we found that the morbidity and mortality were significantly higher in the group of patients where surgery lasted more than 2 hours. However, we feel that this variable may be confounded by the etiology of peritonitis. Most (71.8%) of the patients whose surgery lasted less than 2 hours had perforated appendix or duodenal ulcer, while 75.8% of patients having longer surgeries had colonic perforation, postoperative peritonitis or gangrene of the gut, all known to be associated with greater morbidity and mortality.

CONCLUSION

Generalized peritonitis is still a common emergency managed by most general surgeons throughout the world. This study identifies certain predictive factors for morbidity and mortality in such patients, which may be of help in predicting the outcome. Once outcome can be correctly predicted, better management can be instituted to those patients in need. However, further prospective studies would be needed to validate the individual factors identified in this study.

Conflict of Interest: No conflict of interest was declared by the authors.

Peer-review: Externally peer-reviewed.

Author Contributions

Concept - P.S.K., L.A.D., H.H.; Design - P.S.K., L.A.D., H.H.; Supervision - P.S.K., L.A.D., H.H.; Funding - P.S.K., L.A.D., H.H.; Data Collection and/or Processing - P.S.K., H.H.; Analysis and/or Interpretation - P.S.K., H.H.; Literature Review - P.S.K., H.H.; Writer - P.S.K., H.H.; Critical Review - P.S.K., L.A.D., H.H.

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