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A Prospective Clinical Study of Disease Pattern and Outcomes of Emergency Laparotomy in Acute Abdomen

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ABSTRACT

Background

Term acute abdomen inherently implies a suddenness of onset; the clinical course of abdominal symptoms can range from minutes to hours to weeks, and is often used synonymously for a condition that requires immediate operative intervention[2]. There are mainly four pathologic processes that occur in the GI tract: hemorrhage, ischemia, obstruction, and infection. Most abdominal pathology involves one or a combination of these processes. It is ironic that given this pathophysiologic simplicity the diagnosis of abdominal complaints is often so imprecise. Laparotomies are one of the most commonly performed surgeries on an emergency basis

Materials and Methods

The study was conducted in the Department of General surgery, GREAT EASTERN MEDICAL SCHOOL AND HOSPITAL BETWEEN between SEPTEMBER 2022 TO SEPTEMBER 2023. 60 patients were included in the study. Data were collected from all patients. Patient's age and sex were noted, history was obtained, Thorough clinical examination was performed. Investigations like xray erect abdomen, ultrasonography and CT abdomen done if necessary. Patients underwent surgery. The

details of all the patients were collected and analyzed.

Result

In this study 50 patients underwent emergency exploratory laparotomy and 6 of them had wound dehiscence. The incidence of wound dehiscence in the present study is 12%. Male preponderance is noted in wound dehiscence patients with ratio of 2.8:1, but female patients are found to be more vulnerable for wound dehiscence with 15.38% of female developing wound dehiscence out of 13 female patients who underwent emergency exploratory laparotomy.In the current study, most of the patients belonged to age group of < 45 years .Wound dehiscence is common in patients less than 46 years of age in this study due to more patients belonging to less than 46 years of age

Perforation peritonitis is the most common cause for which emergency laparotomy took place. In wound dehiscence group, 3 out of 6 patients had perforation peritonitis

Pre-operative anemia and hypoalbuminemia showed to have extremely significant association with wound dehiscence. Diabetes mellitus was seen in 2 out of 6 wound dehiscence patients. Wound infection, 5 out of 27 wound dehiscence. In current study mean value of post-operative day of wound dehiscence is 5.8 days.

Keywords

Term acute abdomen inherently implies hemorrhage, ischemia, obstruction,

INTRODUCTION

The accurate diagnosis and management of a patient with acute abdominal pain remains one of the most challenging tasks for the surgeon. The wide range of causes and the varied spectrum of patient presentation pose a formidable diagnostic and therapeutic challenge[2].

Term acute abdomen inherently implies a suddenness of onset; the clinical course of abdominal symptoms can range from minutes to hours to weeks, and is often used synonymously for a condition that requires immediate operative intervention[2].

There are mainly four pathologic processes that occur in the GI tract: hemorrhage, ischemia, obstruction, and infection. Most abdominal pathology involves one or a combination of these processes. It is ironic that given this pathophysiologic simplicity the diagnosis of abdominal complaints is often so imprecise. Laparotomies are one of the most commonly performed surgeries on an emergency basis (1)

In emergency laparotomies, postoperative problems are common. (2) Emergency laparotomies are performed in patients with acute abdomen or trauma abdomen(3) after completing the bare minimum of examinations to arrive at a preliminary diagnosis, while the patient is being optimized/resuscitated. The research focuses on the aetiology, patient pre-operative conditions, and post-operative outcomes following emergency laparotomies conducted at a tertiary care facility.Perforation of the hollow viscus, intestinal obstruction owing to various reasons, and visceral organ damage are all common findings during emergency laparotomies. These circumstances are critical since any delay in surgical intervention can result in the patient's death. The site of onset of symptoms, etiology, time from onset of symptoms to laparotomy, general condition of the patient, associated comorbidities, recovery from anaesthesia, and postoperative care all play a role in the morbidity and mortality of patients after emergency laparotomy(4). Patients with faecal peritonitis have a

higher mortality rate than patients with other acute abdomen cases.The prevalence of post-operative complications and mortality after emergency laparotomy are also being investigated.

AIMS AND OBJECTIVES OF THE STUDY

1) Determine the risk of morbidity and mortality in patients with acute abdomen.
2) A Comparison of morbidity and death in patients with acute abdomen who presented at different times.
3) Assessment of factors responsible for morbidity in these patients after follow up.

MATERIALS AND METHODS

Study Design

Hospital based prospective observational study.

Study sample

60 Subjects

Source of Sample

Present study will be conducted in patients attending to GEMS hospital with clinical features suggestive of acute abdomen and undergoing emergency laparotomy.

Methodology

The subjects will be randomly divided based on intraop findings as

1) Bowel hollow viscus perforation
   A) gastric perforation / duodenal perforation
   B) upper small bowel perforation
   C) lower small bowel perforation
   D) large bowel perforation
2) Obstruction
   A) with gangreneous changes in bowel
   B) without gangrenous changes (ADHESIONS)

INCLUSION CRITERIA

Patients presented with acute abdomen and underwent emergency laparotomy in gems hospital.

EXCLUSION CRITERIA

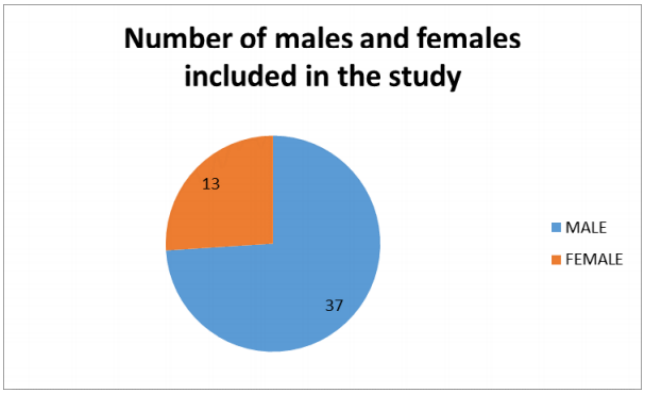
Gynaecological and obstetrics cases

Paediatric cases

RESULTS

1. SEX DISTRIBUTION

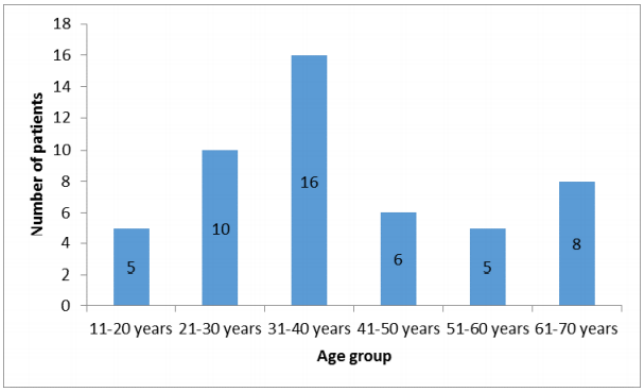
There were 37 men and 13 women among 50 patients who underwent emergency exploratory laparotomy, a ratio of 2.8:1. There were four males and two females among the six wound dehiscence patients. In the instance of dehiscence, the male to female ratio is 2:1. That instance, 37 male patients had laparotomies and four of them developed wound dehiscence, while 13 female patients had emergency laparotomies and two of them acquired wound dehiscence.



Number of Males and Females Included in The Study

2. AGE DISTRIBUTION

Five of the patients who had an emergency laparotomy were between the ages of 11 and 20, ten were between the ages of 21 and 30, and sixteen were between the ages of 31 and 40. Six people were between the ages of 41 and 50, five were between the ages of 51 and 60, and eight were over the age of 60. In the dehiscence group, 66.6 percent of patients were under the age of 45. 33.4 percent were over 45 years old.



Number of Patients Belonging to Different Age Groups

3. DISEASE PATTERN IN EMERGENCY EXPLORATORY LAPAROTOMY

Disease	Male	Female
Perforation peritonitis	22	5
Intestinal obstruction	3	4
Blunt trauma	6	1
Penetrating Trauma	6	1
Malignancy	0	2

Disease Pattern in Emergency Exploratory Laparotomy

Perforation peritonitis forced 54 percent of patients to have an emergency exploratory laparotomy. In 18% of the cases, there was intestinal blockage. For physical damage to the abdomen, 14% had surgery. In 14% of patients, penetrating trauma caused an emergency laparotomy. In 4% of individuals, malignancy manifested itself as acute abdomen. Three of the six patients with wound dehiscence developed perforation peritonitis, and one had peritonitis without perforation. Two of them had cancer and had intestinal obstructions. Perforation peritonitis affected 27 people. Wound dehiscence struck three of them. Perforation peritonitis affects 11.1 percent of the population. Wound dehiscence was more likely in people who had an emergency exploratory laparotomy. Wound dehiscence was discovered in 14.2 percent of intestinal obstruction patients. There was no one there.

In both blunt and penetrating injuries, dehiscence developed. One out of every two patients has Wound dehiscence occurred in both malignancies. In cancer patients, 100 percent of the time wound dehiscence had formed The most likely cause, according to this research, is malignancy. Dehiscence, followed by perforation peritonitis, is a risk factor for the patient. There was a smaller number of cases.

Causative factor for laparotomy	Number of cases underwent laparotomy	Number of cases developed dehiscence	% vulnerable to dehiscence
Perforation peritonitis	27	3	11.1%
Intestinal obstruction	7	1	14.2%
Blunt trauma abdomen	7	0	-
Penetrating trauma	7	0	-
Malignancy	2	2	100%

Disease Pattern and Wound Dehiscence in Emergency Laparotomy

4. POST-OPERATIVE COMPLICATIONS:

	No of patients (out of 50)	Percentage (%)
Fever	34	68%
Nausea and vomiting	28	56%
Chest infection	19	38%
Wound infection	16	32%
Paralytic ileus	13	26%
Wound dehiscence	6	12%
Anastomotic leak	4	8%
Death	4	8%

Post-operative problems were discovered in 34 of the 50 laparotomy cases treated. The most prevalent problems were post-operative fever (68%) and nausea and vomiting (56%) The most prevalent consequences of a prolonged hospital stay were wound infection (33%) and wound dehiscence (16%). COPD was the

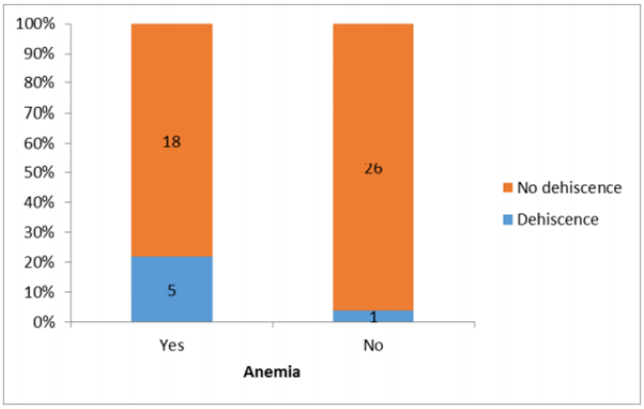
most common co morbid disease among patients who had chest and wound problems (22 percent).

5. ANEMIA AND WOUND DEHISCENCE

According to the established criteria, 23 out of 50 patients had anaemia. Those who had dehiscence Five of the six patients had anaemia, and one patient who did not have anaemiagot it dehiscence. Wound dehiscence did not occur in 26 of the individuals who had anaemia.

Anemia	No wound dehiscence	Wound dehiscence
YES	18	5
NO	26	1
Total		6

Wound Dehiscence and Anemia



Association between anemia and dehiscence (P=0.050)

P 0.050. and was significant using a chi-square test

6. DIABETES MELLITUS IN WOUND DEHISCENCE

7 out of 50 patients had Diabetes mellitus, among which 2 of them had wound dehiscence

Diabetes mellitus	No wound dehiscence	Wound dehiscence
YES	5	2
NO	39	4
Total		6

Dehiscence and Diabetic Mellitus

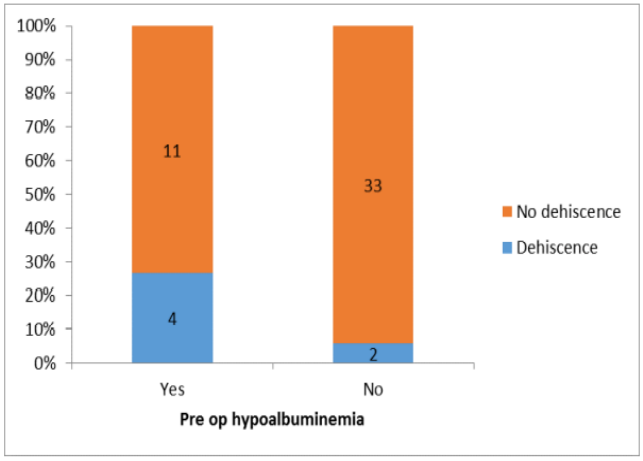
P=0.146.NOT significant using a chi-square test

7. HYPOALBUMINEMIA IN WOUND DEHISCENCE

Among 50 patients 15 had pre-operative hypoalbuminemia. Out of which 4 developed wound dehiscence and 11 did not have dehiscence.

Hypoalbuminemia	No wound dehiscence	Wound dehiscence
YES	11	4
NO	33	2
Total		6

DEHISCENCE AND HYPOALBUMINEMIA



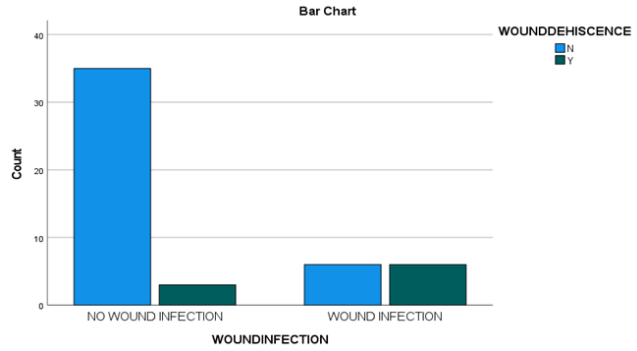
ASSOCIATION BETWEEN PRE-OPERATIVE HYPOALBUMINEMIA AND DEHISCENCE

(P=0.037) P 0.037 and significant using a chi-square

1. WOUND INFECTION/SEPSIS IN WOUND DEHISCENCE:

Wound Infection/Sepsis	No Wound Dehiscence	Wound Dehiscence
PRESENT	11	5
ABSENT	33	1
TOTAL		6

DEHISCENCE AND SEPSIS



2. HYPOALBUMINEMIA* WOUNDINFECTION

Count

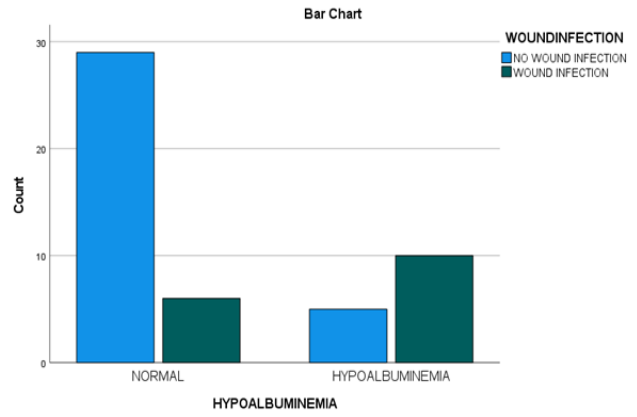
		WOUNDINFECTION		
		NO WOUND INFECTION	WOUND INFECTION	Total
HYPOALBUMINEMIA	NORMAL	29	6	35
	HYPOALBUMINEMIA	5	10	15
Total		34	16	50

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	11.835[a]	1	<.001		
Continuity Correction[b]	9.668	1	. 002		
Likelihood Ratio	11.521	1	<.001		
Fisher's Exact Test				.002	.001
N of Valid Cases	50				

a. 1 Cells (25.0%) have expected count less than 5. The minimum expected count is 4.80.

b. Computed only for a 2x2 table



3. HYPOALBUMINEMIA * CHESTINFECTION

Count

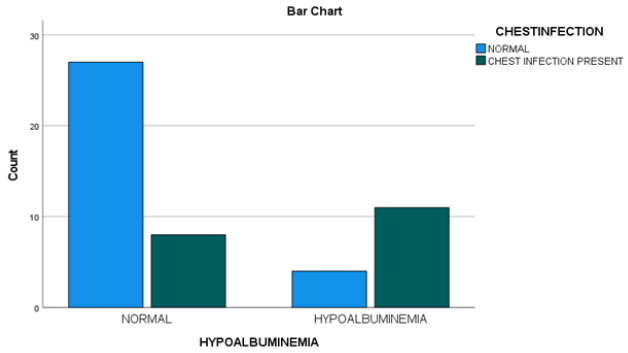
		CHESTINFECTION		
		NORMAL	CHEST INFECTION PRESENT	Total
HYPOALBUMINEMIA	NORMAL	27	8	35
	HYPOALBUMINEMIA	4	11	15
Total		31	19	50

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	11.355[a]	1	<.001		
Continuity Correction[b]	9.314	1	.002		
Likelihood Ratio	11.381	1	<.001		
Fisher's Exact Test				.001	.001
N of Valid Cases	50				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 5.70.

b. Computed only for a 2x2 table



4. HYPOALBUMINEMIA*ANASTAMOTICLEAK

Count

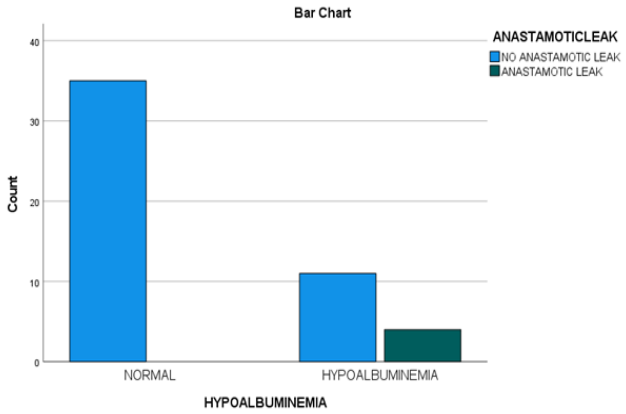
		ANASTAMOTICLEAK		
		NO ANASTAMOTIC LEAK	ANASTAMOTIC LEAK	Total
HYPOALBUMINEMIA	NORMAL	35	0	35
	HYPOALBUMINEMIA	11	4	15
Total		46	4	50

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	10.145[a]	1	.001		
Continuity Correction[b]	6.845	1	.009		
Likelihood Ratio	10.479	1	.001		
Fisher's Exact Test				.006	.006
N of Valid Cases	50				

a. 2 cells (50.0%) have expected count less than 5. The minimum expected count is 1.20.

b. Computed only for a 2x2 table



5. HYPOALBUMINEMIA * DEATH

Count

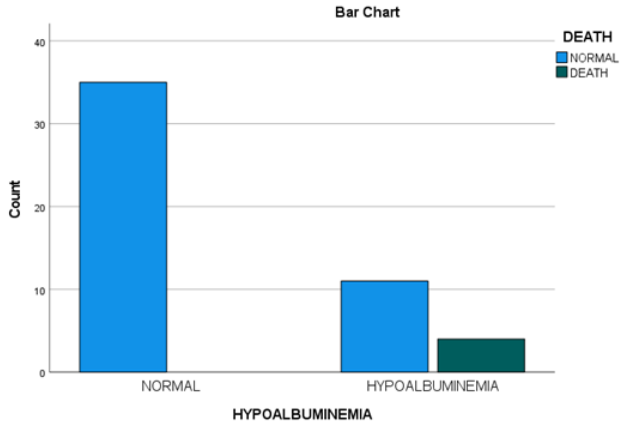
		DEATH		
		NORMAL	DEATH	Total
HYPOALBUMINEMIA	NORMAL	35	0	35
	HYPOALBUMINEMIA	11	4	15
Total		46	4	50

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	10.145[a]	1	.001		
Continuity Correction[b]	6.845	1	.009		
Likelihood Ratio	10.479	1	.001		
Fisher's Exact Test				.006	.006
N of Valid Cases	50				

a. 2 Cells (50.0%) have expected count less than 5. The minimum expected count is 1.20.

b. Computed only for a 2x2 table



6. DIABETES * CHESTINFECTION

Count

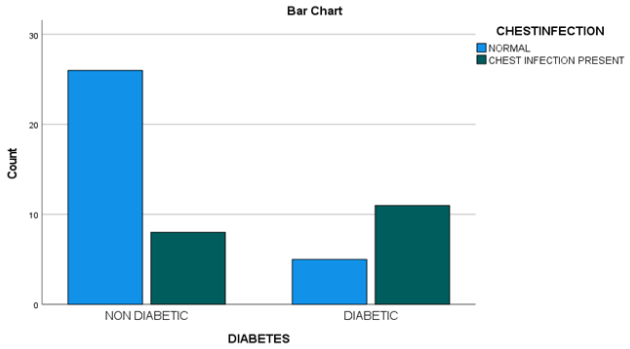
		CHESTINFECTION		
		NORMAL	CHEST INFECTION PRESENT	Total
DIABETES	NON DIABETIC	26	8	34
	DIABETIC	5	11	16
Total		31	19	50

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	9.443[a]	1	.002		
Continuity Correction[b]	7.621	1	.006		
Likelihood Ratio	9.431	1	.002		
Fisher's Exact Test				.004	.003
N of Valid Cases	50				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 6.08.

b. Computed only for a 2x2 table



7. DIABETES * WOUNDINFECTION

Count

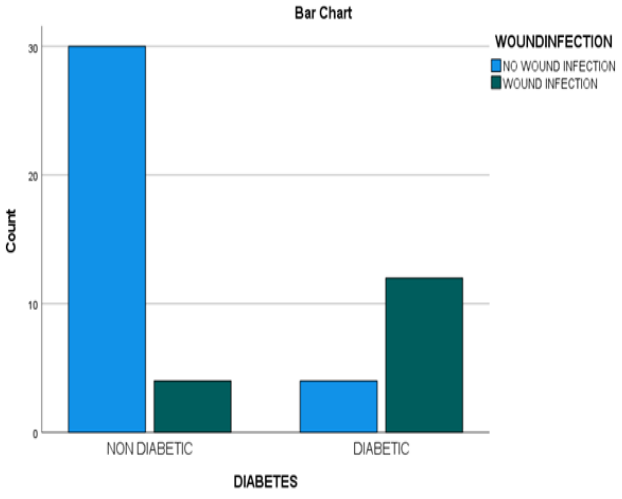
		WOUNDINFECTION		
		NO WOUND INFECTION	WOUND INFECTION	Total
DIABETES	NON DIABETIC	30	4	34
	DIABETIC	4	12	16
Total		34	16	50

Chi-Square Tests

	Value	df	Asymptotic Significance (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	19.994[a]	1	<.001		
Continuity Correction[b]	17.193	1	<.001		
Likelihood Ratio	20.062	1	<.001		
Fisher's Exact Test				<.001	<.001
N of Valid Cases	50				

a. 0 cells (0.0%) have expected count less than 5. The minimum expected count is 5.12.

b. Computed only for a 2x2 table



DISCUSSION

Acute wound failure has many names, including wound dehiscence, disruption, and burst abdomen. The fact that any element is uncommon to play a role in wound healing and dehiscence adds to the subject's complexity, and determining which element is more significant in a given context is difficult. It goes without saying that this case will be difficult to solve. One of the most common and serious postoperative consequences after major abdominal surgery is abdominal wound dehiscence. Mechanical wound separation or dehiscence are symptoms of acute wound failure. Although dermal wound separation degrades the appearance of the wound, it is unlikely to cause any harm, whereas abdominal wall wound failure can be lethal.Regardless of how the dehiscence manifests, the initial treatment comprises discharging the contents of the intestine into the peritoneal cavity and filling it with wet saline packs, stomach decompression with a nasogastric tube, intravenous fluids, and broad-spectrum antibiotics. Despite the fact that the patient is in critical condition, the patient should be stabilised and any antecedent reasons of dehiscence should be addressed if they are recoverable before surgery. Reopening and inspecting the entire surgical wound, Exploratory Laparotomy to look for any intra-abdominal abscesse or anastomotic leak, thorough peritoneal lavage, and a good re-closure should be done , as well as In this study, fifty patients had an emergency midline exploratory laparotomy. Six individuals developed partial or complete wound dehiscence.12.0% of patients experience wound dehiscence as a result of this. In the international literature, the wound dehiscence rate ranges from 1% to 2.6 percent. 116 Dehiscence rate was observed to

be 6.7 percent 119 in emergency laparotomy, according to Penninckx et al. Local studies reveal a greater prevalence, ranging from 6% 117, 118 to 12%. In a research by Pandey S et al, the overall incidence of wound dehiscence was 11.5 percent overall and 17 percent in patients who had Vicryl120 mass closure. The increased incidence of wound dehiscence in our study could be due to the fact that most studies covered both elective and emergency cases, resulting in a lower incidence. Only emergency cases were included in this study, which could explain the increased incidence when compared to international studies .Poor patient preparation, severe inflammatory disease, premorbid conditions, infected wounds, and operating at unusual hours may all play a role in this

Table No. 13 Incidence of Wound Dehiscence

STUDIES	YEAR	INCIDENCE OF WOUND DEHISCENCE	REMARK
Timothy E et al	1983	1.7%	
White et al	1962-71	1.5%	10 years study including both elective and emergency cases with all types of incisions
Jakub et al	2008-11	2.9%	Both emergency and elective cases
Gabriel et al	1985-05	2.8%	Both emergency and elective cases and all abdominal surgeries were included.
Khan MN et al	1998-04	7.8%	Emergency and elective laparotomy patients were included
Hanif et al[117]	2000	14.7%	Both midline and paramedian incisions were included in their study
Waqar SH et al[118]	2005	12% in emergency and 4% in elective cases	All patients underwent midline laparotomy like this study.

When the sex ratio is taken into account, 37 of the 50 patients who had anemergency exploratory laparotomy were male and 13 were female.It is discovered that the ratio is 2.8:1. When wound dehiscence was taken into account, the ratio was 2:1. Looking at the sex ratio, there appears to be a male majority. However, investigation revealed that four male patients out of 37 developed wound dehiscence, whereas two female patients out of 17 developed wound dehiscence. In this study, 10.81 percent of males were sensitive to wound dehiscence, compared to 15.38 percent of females. As a result, females were

shown to be somewhat more susceptible to wound dehiscence in this investigation. There were 75 males and 48 females in the group analysed by White H et al, resulting in a male to female ratio of 1.6:1.This study also discovered the nature of male preponderance. Male: female sex ratio was 3:1 in Munieah NS et al's study, which matches this one. In their study, Kenig J et al looked at 56 individuals who suffered wound dehiscence after various abdominal procedures. There were 37 men and 19 women among the cases; there was a statistically significant difference in gender, with men accounting for more instances (p = 0.034). In a study by Ramneesh G[7] et al, male predominance (37/50) was discovered, with a male-to-female ratio of 2.84:1, which is similar to the current study. The majority of the patients who underwent laparotomy in this study were between the ages of 26 and 45. Four of the fifty patients were between the ages of 26 and 45, with two more between 46 and 60. Sixty-six percent of patients with wound dehiscence were under the age of 45. This is in contrast to most research, which found that wound dehiscence was more common among the elderly.

Table No. 14 Mean Age and Wound Dehiscence

STUDY	YEAR	INFERENCE
Kenig J et al	2008-2011	Mean age in wound dehiscence patient was 66.8±12.6 years.
Pandey S et al	2009-2011	Mean age: 54 years with prolene for closure Mean age: 56 years with vicryl for closure
Madsen G et al	1992	Mean age: 66 years
Khan MN et al	1998-2000	Age > 55 years showed significant value for wound dehiscence.

This study's inconsistency could be due to the fact that there were more patients under 45 years old. Vascularization, collagen deposition, and collagen remodelling are all slowed or delayed as a result of age-related changes in macrophage functions123.The vasoregulation of the microcirculation in old skin is disrupted, which is reflected in alterations in inflammatory responses, fewer progenitor cells, and decreased circulatory mediators. Age-related delays in microvascular responses to stressors result in decreased temperature regulation and a higher risk of tissue hypoperfusion, which prevents wounds from progressing to the angiogenic stage of healing124. According to Gould L[10] et al, optimum healing

procedures following surgery and other stressors must employ multifactorial approaches to address alterations in microcirculation in older persons. Only cases undergoing emergency laparotomy were examined in this investigation, as wound dehiscence has been shown to be more common in emergency cases than elective procedures. The majority of studies included both emergency and elective cases, making it impossible to compare disease patterns. Ramneesh G[7] et alfound that 35 patients (70 percent) developed perforation of the hollow viscus with peritonitis in their study. There were 10 patients (20%) who had intestinal blockage but no signs of peritonitis. Trauma caused harm to solid organs or the mesentery, resulting in hemoperitoneum in five cases (10%). The cancer of the big intestine was found in three patients (six percent).This study, like Ramneesh G[7] et al, had a disease pattern. Perforation peritonitis was the most prevalent illness in patients who experienced dehiscence in this study. This could be because perforation peritonitis affected 54 percent of emergency laparotomy patients. Patients with cancer were shown to be more susceptible to wound dehiscence in this investigation. Wound dehiscence occurred in 100% of patients with malignancy (albeit the number of cases was lower), compared to 11.6 percent for 76 perforation peritonitis and 14.2 percent for blockage. In this investigation, none of the patients with blunt or penetrating abdominal injuries who did not have peritonitis developed wound dehiscence. In cases of blunt trauma without peritonitis, this can be explained by a clean wound. The illness pattern was similar to that seen in the current investigation by Waqar SH[3] et al. With 27 percent of wound dehiscence, perforation peritonitis was the major cause. Peritonitis caused by gastrointestinal tract perforation is the most prevalent surgical emergency worldwide, according to Ramakrishnan K[11] et al. The majority of patients appear late, with purulent peritonitis and septicemia, according to Afridi SP[12] et al in their study. Wound infection and wound dehiscence have been noted as major consequences.These data back up the illness pattern found in this investigation. Pre-operative anaemia was found to be strongly linked to the development of wound dehiscence in this investigation. Anemia affected 23 individuals, five of whom suffered wound dehiscence. Wound dehiscence occurred in 1 patient who did not have anaemia. The chi-square test yielded a p value of 0.050, which is extremely significant. When we look at the literature, we can see that Joergenson and Smith found a greater incidence of burst abdomen in patients with anaemia in their study. Chronic anaemia as part of a generalised nutritional 77 shortage, as proven by Besser and Ehrenhoft13, is a crucial element in wound healing, while acute anaemia from blood loss is not. In a similar study, Makela JT[4] et al discovered that anaemia has a strong relationship with wound dehiscence. Kenig J[6] et al and Riou[15] et al, on the other hand, did not detect a link between anemia and wound dehiscence. According to Carson JL[16] et al, anemia is a risk factor linked to perioperative stress, blood transfusions, and decreased tissue oxygenation, all of which might impact the immune system and wound healing.Out of 50 emergency exploratory laparotomies, 15 patients had hypoalbuminemia prior to surgery. Pre-operative hypoalbuminemia was found in four of the six patients who had wound dehiscence. In statistical analysis, the Chi square test was performed and found to be

significant with a p value of 0.037. The importance of hypoalbuminemia in wound dehiscence is supported by most studies in the literature.

Table No. 15 Hypoprotinemia and Wound Dehiscence

STUDY	YEAR	INFERENCE
Makela JT et al	1989-92	Hypoproteinemia- a strong predictor of wound dehiscence
Hennessey et al[133].	2010	Hypoalbuminemia (<35 g/dL) an independent predictor of surgical site infection
Meena K et al[134]	2013	Hypoproteinemia <60g/l was proved to be a significant variable for wound dehiscence.

This finding is similar to Wolff, Alexander, and Pavdden's and Keill et al's findings that hypoproteinemia was related with 62 percent, 71 percent, and 85 percent of wound dehiscences, respectively. Hypoproteinemia prolongs the inflammatory phase, affects fibroplasia, inhibits proliferation, proteoglycan and collagen formation, neoangiogenesis, and wound remodelling. Protein is essential at all stages of wound healing, as well as for maintaining a positive nitrogen balance. Severe protein depletion causes epidermal and fascial wound breaking strength to deteriorate, as well as an increase in wound infection rates. 135,79 Diabetes mellitus was examined as a risk factor in this study. Seven of the patients had Diabetes Mellitus, and two of them had wound dehiscence. Diabetes was not shown to be associated with a significant P value.In all cases, this could be attributable to careful glycemic control or the younger age group attained. As a result, the negative effects of inadequate glycemic control are reduced, as is their vulnerability to wound infection and dehiscence. Only 4 out of 50 wound dehiscence patients were found to be diabetes in a research by Ramneesh G[7] et al. However, Bybee and Roger[20] et alfound that granulocyte activity in diabetes individuals was reduced. In his study, Spiliotis J discovered that 40 percent of patients with wound dehiscence were diabetes. Diabetes mellitus is mentioned as a risk factor for wound dehiscence138 in the literature. The contentious conclusion in this study could be due to the fact that the majority of the participants were under 45 years old, a group with a lower prevalence of diabetes. Obesity was observed in two of six individuals with wound dehiscence when BMI >30 was included in the variables for assessment.The P value was 0.015, which was considered significant. Overweight (BMI > 30) was found to be a risk factor for wound infection in a study conducted at Sundsvall County Hospital in Sweden, although these effects might be prevented if patients were sutured with a suture length to wound length ratio of 4–4.99.

In his article, Wolff stated that malignancy did not cause wound dehiscence. Neoplastic disorders were not shown to be a risk factor for wound dehiscence in a study conducted by Kenig J et al. In a research by Waqar SH et al., 15% of patients with cancer had wound dehiscence. In their study, Sinhai A et al found that 26.31 percent of wound dehiscence patients had cancer.Malignancy was found to be a significant risk factor for wound dehiscence in the current investigation. On the role of malignancy in wound dehiscence, there is conflicting evidence in the literature. In the current investigation, six individuals experienced pulmonary problems after surgery. Many studies also included pulmonary complications as a risk factor. This is because coughing and laboured breathing as a result of pulmonary problems causes an increase in intra-abdominal pressure, which has been linked to dehiscence.The second reason is because pulmonary problems cause tissue hypoxia, which impedes wound healing. In this case, one out of every six patients suffered pulmonary problems following surgery. The P value was not significant at all (83). The contentious finding in this study could be due to the fact that the majority of the study population was under 45 years old, a group with a lower risk of pulmonary complications.

Hypoalbuminemia is a common and vexing complication seen not only in patients undergoing emergency surgeries but also in majority of hospitalized patients. Laparotomy is one of the commonest surgical procedure performed in tertiary care hospitals. The indications for the same can be varying. It may be a planned elective surgery or an emergency surgical procedure. Although laparoscopy has made extensive advances in diagnosing and treating intra-abdominal pathologies, laparotomy still is important when laparoscopy fails and in most centres in an emergency setting. The complications encountered are much more in emergency laparotomy compared to elective surgery. My study took into account emergency laparotomy surgeries through midline skin incision for uniformity.

Various factors have been studied to correlate and predict complications and adverse outcomes following laparotomies namely age, obesity, comorbidities like Diabetes mellitus, renal parenchymal disease, duration of sepsis, cause of sepsis, inherent lung diseases, smoking, fever, hypothermia, liver diseases and coronary artery disease. Various prognostic scores also have been put into use like APACHE II, APACHE III, GLASGOW and SOFA. But none have been fool proof nor has there been a single and simple marker or risk factor which could effectively predict the complications or adverse post-operative outcomes without taking into account the confounding factors. Serum albumin is one such marker. The significance of albumin in homeostasis and wound healing is well known. But it has not been studied as an independent prognostic risk factor especially in surgical setting

Indications for Laparotomy

Peritonitis due to perforation of hollow viscus was the commonest cause for laparotomy in my study. This is similar to most studies done around the world. Site of perforation commonly found in my study was duodenum followed by appendix, ileum, colon and stomach. In the study done by Greenallet al found appendix as the commonest site of perforation while Stulz et al had ileum and duodenum as the common site of perforation. Site of perforation differed in studies done in Asian countries compared to the West.

Ileum and appendicular perforations are common in the Asians (tropical climate) possibly due to the increased incidence of TB and enteric fever as compared to the West where duodenal and even colonic perforations are common.

Post-operative recovery is considerably different in different perforations. Duodenal and appendicular perforations carry significantly low morbidity and mortality as compared to ileal or colonic perforation. Patients with appendicular perforation usually tolerate early enteral feeds and have shorter hospital stay with fewer complications. This is true with duodenal perforation also. Patients who had ileal, jejunal or colonic perforations have higher morbidity possibly due to delayed presentation and gross contamination of peritoneal cavity. These patients have prolonged hospital stay too. This was shown in the study done by Reinhardt[41] et al

COMPLICATIONS

Surgical site infection was one of the common complication noted in my study. This was similar to the studies done by Lohsiriwaat90 et al, Gibbs[63] et al, Schultze[59]et al and Haukipuro et al. Incidence of surgical site infection is much higher in emergency laparotomy as compared to elective surgeries. It is also seen more in long standing sepsis and large intestinal perforation as compared to duodenal or gastric perforations probably because severity of sepsis is usually mild and degree of contamination is lesser. Surgical site infection may be minimal with some discharge and redness at the incision site which is managed conservatively with antibiotics and wound dressings or more significant in the form of collections needing debridement and secondary suturing of the wound. This prolonged the hospital stay in most patients. The predisposing N factors to developing surgical site infections are many. Hypoalbuminemia is a major risk factor for the same. But in my study this was not statistically significant (p value>0.05). There are other factors like duration of sepsis, cause of peritonitis, comorbidities like Diabetes, immunocompromised state, excess abdominal fat, surgical technique to some extent, fomite borne infections in hospital wards and presence of concomitant infections like respiratory tract, urinary tract infection. These confounding factors were not taken into consideration in my study.

The microbial growth in culture of discharge from the surgical wound was mostly same as the pathogen grown in the peritoneal fluid namely Escherichia coli, Klebsiella sp Pseudomonas species, Proteus species and unidentified Gram negative bacilli. Second commonest was gram positive cocci including Methicillin Resistant Staphylococcus Aureus especially from hospital wards and skin commensals otherwise normally seen on the body surface like Staphylococcus epidermidis. This was not part of my study and was not studied in detail.

Patients who developed surgical site infection in my study was mostly managed by wound dressing and culture specific antibiotics. There were a few patients who did not grow any microbe even after repeated wound swab cultures, but responded to empirical antibiotics and wound dressings.

Common antibiotics used were Ciprofloxacin, Metronidazole, Amikacin, Piperacillin-tazobactam,Cefazolin, Cefuroxime and Amoxycillin-clavulanate. Other rarely used ones were Meropenem, Cefoperazone-sulbactam and Tigecycline

Those who needed secondary suturing when the wound was left open underwent the same in wards as bedside procedure under local anesthesia and rarely in operating room. Very few patients were put on vacuum assisted dressing which accelerated wound closure.

WOUND DEHISCENCE

There was statistically significant positive correlation between hypoalbuminemia and wound dehiscence. This was shown in other studies by those done by Fleck[61] et al, Hoye[62] et al, Cochrane meta-analyses review83, Rosa et al and Hennessey et al. Hypoalbuminemia was the most significant risk factor that delayed wound healing and hence predisposing to wound dehiscence. Wound dehiscence included partial giving away of wound to burst abdomen exposing the intestines needing immediate, mostly tension suturing. Wound dehiscence was noted mostly between 5th to 9th postoperative days.

Other risk factors like chronic cough, development of lower respiratory tract infection in the post-operative period causing increased intra-abdominal pressure were not seen in my study. Inherent factors that cause delayed wound healing like Vitamin C deficiency, connective tissue disorders, and immunocompromised states might be coexisting in some patients in my study too. But these were not found to be statistically significant in any of the studies done worldwide.

ANASTOMOTIC LEAK

18% of patients underwent resection and anastomosis of bowel in my study. 8% of these patients developed anastomotic leak. 82% had hypoalbuminemia. This was statistically significant. Incorrect surgical technique of anastomosis was once thought to be an important risk factor in developing anastomotic leak. But with the advent of surgical stapler devices and better suture materials and changed schools of thought in techniques of bowel anastomosis, incorrect technique is rarely seen as a risk factor although it still exists. Resection anastomosis done in a setting of TB, inflammatory bowel diseases like ulcerative colitis and Crohn's disease, malignancy like adenocarcinoma of colon and immunocompromised states like HIV or in gross peritoneal contamination are significant and known risk factors that predispose to anastamotic leak needing relook laparotomy. Most of the times in these cases the patients end up having a diversion stoma. This was true in my study also. Those patients who needed relook laparotomy for anastomotic leak underwent diversion stoma and reresection anastomosis.

PULMONARY COMPLICATIONS

Prevalence of hypoalbuminemia in patients who developed pulmonary complications was 76% , statistically significant. Those patients who already had a known pulmonary disease like bronchial asthma, COPD, pulmonary TB, lung malignancy and restrictive lung diseases were excluded. Patients who developed complications namely, lower respiratory tract infection, pneumonia, pleural effusion and ARDS in the post-operative period were counted in my study. Study done by Ryan[25] et al and Knaus[27]et al had similar pulmonary complication rates although the former studies post-esophagectomy complications and Latter Postoperative Complications In Icu.

MORTALITY

There was 100% prevalence of hypoalbuminemia in all deceased patients (pvalue 0.001) Total number of patients who died were 4 (8%). Cause of death in majority were ARDS, acute kidney injury and cardiac

arrest alone and combined as they went into MODS. Mortality encountered on table was seen in 2 patients who had fatal hypovolemia secondary to blood loss due to blunt trauma of abdomen (RTA). The results were similar to studies done by Ryan[25] et al (93%), Gibbs[63] et al (91%) and Pasha[64] et al (94%).

LIMITATIONS OF THE STUDY

1. Although major confounding factors were eliminated, there were other factors like duration and severity of sepsis and inherent wound delaying factors which would contribute to post-operative adverse outcomes but were not studied independently.
2. There were other unexpected adverse events like acute coronary event or cerebrovascular accident that would adversely affect the post-operative recovery and rehabilitation and prolong the hospital stay of patients which were not taken into account.

SUMMARY

In this study 50 patients underwent emergency exploratory laparotomy and 6 of them had wound dehiscence. The incidence of wound dehiscence in the present study is 12%.

Male preponderance is noted in wound dehiscence patients with ratio of 2.8:1, but female patients are found to be more vulnerable for wound dehiscence with 15.38% of female developing wound dehiscence out of 13 female patients who underwent emergency exploratory laparotomy.

In the current study, most of the patients belonged to age group of < 45 years

Wound dehiscence is common in patients less than 46 years of age in this study due to more patients belonging to less than 46 years of age

Perforation peritonitis is the most common cause for which emergency laparotomy took place. In wound dehiscence group, 3 out of 6 patients had perforation peritonitis. Next leading cause was malignancy with 2 out of 6 patients.

Pre-operative anemia and hypoalbuminemia showed p- value 0.050 & 0.037 respectively found to have extremely significant association with wound dehiscence.

Diabetes mellitus was seen in 2 out of 6 wound dehiscence patients and p value obtained was 0.146 which means an insignificant association with wound dehiscence.

Increased BMI had p value more than 0.015 and found to have significant association with wound dehiscence.

Wound infection, 5 out of 27 wound dehiscence patients had p value less than 0.001 and showed significant association with wound dehiscence.

Even when 100% of malignant patients had wound dehiscence ,malignancyshow significant association with wound dehiscence on statistical analysis, the number of cases were less.

In current study mean value of post-operative day of wound dehiscence is 5.8 days.

If there is absence of combination of all the three factors- pre-operative anemia, hypoalbuminemia and obesity, there is higher chance that the patient will not develop wound dehiscence.

CONCLUSION

Wound dehiscence is a significant complication of sluggish wound healing. Despite advances in medicine, wound dehiscence in emergency laparotomies is still a common occurrence. This severe complication is caused by a combination of

causes. Knowledge of the more common mechanisms and highly contributory factors can aid in keeping high-risk patients under close observation in order to limit the incidence of this infamous complication.Preoperative hypoalbuminemia, preoperative anaemia, intra-abdominal sepsis/wound infection, and obesity were all identified as significant risk factors for abdominal wound dehiscence in this investigation. Wound dehiscence was more common in the elderly, females, and cancer patients.Our goal was to see if pre-operative anaemia, hypoalbuminemia, and obesity might be utilised to predict patients who are less likely to develop wound dehiscence rather than individuals who are more likely to develop wound dehiscence. We may therefore deduce from the findings of this study that a variety of risk factors for abdominal wound dehiscence can be reduced during the perioperative phase.As a result, we suggest that if the aforesaid predisposing factors are fully understood before to performing any emergency abdominal surgery, the current incidence and burden on the health care system can be further reduced.

Serum albumin is a major inflammatory marker whose levels drop dramatically in acute inflammation such as sepsis, generating negative results in the patient's recovery.

Hypoalbuminemia is an independent prognostic risk factor which has strong positive correlation to adverse post-operative outcomes and effectively shows the degree of inflammation rather than the nutritional status of the patient contrary to common belief. The scope of this study could be extended in such a way as to know whether early intervention to correct hypoalbuminemia would enhance the post-operative recovery by decreasing the complications. So, serum albumin stands as a simple, easy and effective marker for predicting postoperative complications in patients undergoing emergency laparotomy.

From this study it can be concluded that serum albumin is a significant inflammatory marker whose levels decrease drastically in acute inflammation like sepsis causing adverse outcomes in the recovery of the patient. Hypoalbuminemia is an independent prognostic risk factor which has strong positive correlation to adverse post-operative outcomes and effectively shows the degree of inflammation rather than the nutritional status of the patient contrary to common belief.

The scope of this study could be extended in such a way as to know whether early intervention to correct hypoalbuminemia would enhance the post-operative recovery by decreasing the complications.

So, serum albumin stands as a simple, easy and effective marker for predicting post-operative complications in patients undergoing emergency laparotomy.

REFERENCES

1. Adrian S, Peter L. Wound dehiscence, incisional hernia and para-stomal hernia. In: Moris PJ, William W (eds): Oxford Textbook of Surgery. 22nd ed. Oxford University Press, 2000; 1983-1990.
2. Hanif N, Ijaz A, Niazi UF, Akhtar I, Zaidi AA, Khan MM. Acute wound failure in emergency and elective laparotomies. J Coll Physicians Surg Pak 2000; 11: 23-26.
3. Waqar SH, Malik ZI, Razzaq A, Abdullah MT, Shaima A, Zahid MA. Frequency and risk factors for wound dehiscence/burst abdomen in midline

laparotomies. J Ayub Med Coll Abbottabad. 2005 Oct-Dec; 17(4): 70-73

4. Penninckx FM, Poelmans SV, Kerremens RP, Beckers JP. Abdominal wound dehiscence in gastroenterological surgery. Ann Surg. 1979; 189(3): 345–352.
5. Pandey S, Singh M, Singh K, Sandhu S. A Prospective Randomized Study Comparing Non-absorbable Polypropylene (Prolene®) and Delayed Absorbable Polyglactin 910 (Vicryl®) Suture Material in Mass Closure of Vertical Laparotomy Wounds. The Indian Journal of Surgery. 2013; 75(4): 306-310. doi:10.1007/s12262-012-0492-x. xvi
6. Kenig J, Richter P, Żurawska S, Lasek A, Zbierska K. Risk factors for wound dehiscence after laparotomy - clinical control trial. Pol PrzeglChir 2012; 84: 565-573 [PMID: 23399620 DOI: 10.2478/v10035-012-0094-0]
7. Garg Ramneesh, Shah Sheerin, Singh Surinder, Singh Bir. A Prospective Study of Predictors for Post Laparotomy Abdominal Wound Dehiscence. Journal of Clinical and Diagnostic Research. 2014 | January | Volume 8 | Issue 1 | Page 80-83
8. Swift ME, Burns AL, Gray KL, et al. Age-related alterations in the inflammatory response to dermal injury. J Invest Dermatol. 2001; 117:1027–1035
9. Bentov I, Reed MJ. Anesthesia, microcirculation, and wound repair in aging. Anesthesiology. 2014; 120:760–772.
10. Gould L, Abadir P, Brem H, et al. Chronic Wound Repair and Healing in Older Adults: Current Status and Future Research. J Am Geriatr Soc. 2015 March; 63(3): 427–438.
11. Ramakrishnan K, Salinas RC. Peptic Ulcer disease. Am Fam Physician. 2007; 1: 1005–12.
12. Afridi, S. P., Malik, F., Ur-Rahman, S., Shamim, S., & Samo, K. A. (2008). Spectrum of perforation peritonitis in Pakistan: 300 cases Eastern experience. World Journal of Emergency Surgery : WJES, 3, 31. http://doi.org/10.1186/1749-7922-3-31 xvii 128. Joergenson EJ, Smith ET. Postoperative abdominal wound separation and evisceration. Am J Surg. 1950 Feb;79(2):282-7.
13. Besser, E. L. and Ehkekhoft, J. L. Relationship of acute anemia to wound healing: experimental suffciently to allow gentle probing of the study. Surgery, 14: 239, 1943.
14. Makela JT, Kiviniemi H, Juvonen T, et al. Factors Influencing Wound Dehiscence After Midline Laparotomy. Am J Surg. 1995; 170: 387-390.
15. Riou J-P, Cohen JR, Johnson H Jr. Factors influencing wound dehiscence. Am J Surg. 1992; 163: 324-330.
16. Carson JL, Duff A, Poses RM et al. Effect of anaemia and cardiovascular disease on surgical mortality and morbidity. Lancet 1996; 348:1055–1060.
17. Hennessey DB, Burke JP, Ni-Dhonochu T, Shields C, Winter DC, Mealy K.: Preoperative hypoalbuminemia is an independent risk factor for the development of surgical site infection following gastrointestinal surgery: a multi-institutional study. Ann Surg 2010, 252: 325–9.
18. Meena K, Ali S, Chawla AS et al . A prospective study of factors influencing wound dehiscence after midline laparotomy.SS. 2013; 04: 354–358. doi:10.4236/ss.2013.48070

19. Joyce K. Understanding the Role of Nutrition and Wound Healing. Nutr Clin Pract. 2010; 25: 61-68. Xviii
20. Bybee JD and Rogers DE. The phagocytic activity of polymorphonuclear leukocytes obtained from patients with diabetes mellitus. J Lab Clin Med. 1964; 64: 1-13. 137. Spiliotis J, Tsiveriotis K, Datsis AD, et al. Wound dehiscence: is still a problem in the 21th century: a retrospective study. World Journal of Emergency Surgery 2009; 4:12.
21. Col C, Soran A, Col M: Can postoperative abdominal wound dehiscence be predicted? Tokai J Exp Clin Med 1998; 23(3): 123-127.
22. Cruse PJE, Foord R. The epidemiology of wound infection: A 10 year prospective study of 62939 wounds. Am., 1980; 60: 27.
23. Webster C, Neumayer L, Smout R, Horn S, Daley J, Henderson W, Khuri S, Qua NVAS. Prognostic models of abdominal wound dehiscence afterlaparotomy. J Surg Res 2003; 109: 130–7
24. Chang Y, Tsai Y, Lin P, Chen M, Liu C. Prevalence and risk factors for postoperative delirium in a cardiovascular intensive care unit. Am J Crit Care 2008; 17(6):567–575.
25. Ryan AM, Hearty A, Prichard RS, Cunningham A, Rowley SP, Reynolds JV. Association of hypoalbuminemia on the first postoperative day and complications following esophagectomy. J Gastrointest Surg 2007; 11(10):1355–1360.
26. Ueda M, Endo I, Nakashima M, Minami Y, Takeda K, Matsuo K, et al. Prognostic factors after resection of pancreatic cancer. World J Surg 2009; 33(1):104–110.
27. Knaus WA, Wagner DP, Draper EA, Zimmerman JE, Bergner M, Bastos PG, et al. The APACHE III prognostic system. Risk prediction of hospital mortality for critically ill hospitalized adults. Chest 1991; 100(6):1619–1636.
28. Doweiko JP, Nompleggi DJ. The role of albumin in human physiology and pathophysiology, Part III: Albumin and disease states. Journal of Parenteral and Enteral Nutrition JPEN 1991; 15(4):476–483.
29. Hoerr SO, Allen R, Allen K. The closure of the abdominal incision; a comparison of mass closure with wire and layer closure with silk. Surgery 1951; 30:166-173.
30. Hermann RE. Abdominal wound closure using a new polypropylene monofilament suture. Surg GynecolObstet 1974; 138:84-86.
31. Kato TS, Cheema FH, Yang J, Kawano Y, Takayama H, Naka Y, Farr M, Lederer DJ, Baldwin MR et al. Preoperative serum albumin levels predict 1-year postoperative survival of patients undergoing heart transplantation. Circ Heart Fail. 2013 Jul; 6(4):785-91.
32. Dudley HAF. Layered and mass closure of the abdominal wall: a theoretical and experimental analysis. Br J Surg 1970; 57:664-667. 69
33. Sanders RJ, DiClementi D, Ireland K. Principles of abdominal wound closure I: Animal studies. Arch Surg 1977; 112:1184-1187.
34. Wantz GE, Chevrel JP, Flament JB, Guillou Kingsnorth A, Schumpelick V, Verhaeghe P. Incisional hernia: the problem and the cure. J Am Coll Surg 188(4):429-447, 1999.
35. Takeda Y, Reeve EB. Studies of the metabolism and distribution of albumin with autologous I131-albumin in healthy men. J Lab Clin Med 1963; 61:183–202.

36. Alderson P, Bunn F, Lefebvre C, Li Wan Po A, Li L, Roberts I, et al. Human albumin solution for resuscitation and volume expansion in critically ill patients. Cochrane Database Syst Rev 2004; Oct 18(4):CD001208.
37. Ruot B, Papet I, Bechereau F, Denis P, Buffiere C, Gimonet J, et al. Increased albumin plasma efflux contributes to hypoalbuminemia only during early phase of sepsis in rats. Am J PhysiolRegulIntegr Comp Physiol 2003; 284(3):R707–R713.
38. Lee G, Dennis A, editors. Cecil Medicine. Philadelphia: Saunders Elsevier; 2008. p. 1097.
39. Durward A, Mayer A, Skellett S, Taylor D, Hanna S, Tibby SM, et al. Hypoalbuminaemia in critically ill children: incidence, prognosis, and influence on the anion gap. Arch Dis Child 2003; 88(5):419–422.
40. Schirmer-Mikalsen K, Vik A, Gisvold SE, Skandsen T, Hynne H, Klepstad P. Severe head injury: control of physiological variables, organ failure and complications in the intensive care unit. Acta Anaesthesiol Scand 2007; 51(9):1194–1201.
41. Reinhardt GF, Myscofski JW, Wilkens DB, Dobrin PB, Mangan JE, Stannard RT. Incidence and mortality of hypoalbuminemic patients in hospitalized veterans. JPEN J Parenter Enteral Nutr 1980; 4(4):357–359. 70
42. Mankertz J, Schulzke J. Altered permeability in inflammatory bowel disease: pathophysiology and clinical implications. Curr Opin Gastroenterol 2007; 23(4): 379–383.
43. Russell JA. Management of sepsis. N Engl J Med 2006; 355(16):1699–1713.
44. Fishel RS, Are C, Barbul A. Vessel injury and capillary leak. Crit Care Med 2003; 31(8 Suppl):502–511.
45. Mehta D, Malik AB. Signaling mechanisms regulating endothelial permeability. Physiol Rev 2006; 86(1):279–367.
46. Aghajanian A, Wittchen ES, Allingham MJ, Garrett TA, Burridge K. Endothelial cell junctions and the regulation of vascular permeability and leukocyte transmigration. J ThrombHaemost 2008; 6(9):1453–1460.
47. Clayburgh DR, Shen Le, Turner JR. A porous defense: the leaky epithelial barrier in intestinal disease. Lab Invest 2004; 84(3):282–291.
48. Griffiths AM, Drobnies A, Soldin SJ, Hamilton JR. Enteric protein loss measured by fecal alpha 1-antitrypsin clearance in the assessment of Crohn's disease activity: a study of children and adolescents. J Pediatr Gastroenterol Nutr 1986; 5(6):907– 911.
49. Schulzke J, Tröger H, Amasheh M. Disorders of intestinal secretion and absorption. Best Pract Res Clin Gastroenterol 2009; 23(3):395–406.
50. Laine L, Garcia F, McGilligan K, Malinko A, Sinatra FR, Thomas DW. Proteinlosing enteropathy and hypoalbuminemia in AIDS. AIDS 1993; 7(6):837–840.
51. Bode L, Salvestrini C, Park PW, Li J, Esko JD, Yamaguchi Yu, et al. Heparan sulfate and syndecan-1 are essential in maintaining murine and human intestinal epithelial barrier function. J Clin Invest 2008; 118(1):229–238. 71
52. Ma TY, Anderson JM. Tight junctions and the intestinal barrier. In: Johnson LR, editor. Physiology of the gastrointestinal tract. Vol. 2.

Burlington (MA): Elsevier Academic Press; 2006. p. 1559–1594.

53. Lencer WI. Patching a leaky intestine. N Engl J Med 2008; 359(5):526–528.
54. Perel P, Roberts I. Colloids versus crystalloids for fluid resuscitation in critically ill patients. Cochrane Database Syst Rev 2007; Oct 17(4):CD000567.
55. Liberati A, Moja L, Moschetti I, Gensini GF, Gusinu R. Human albumin solution for resuscitation and volume expansion in critically ill patients. Intern Emerg Med 2006; 1(3):243–245.
56. Minson CT, Wladkowski SL, Pawelczyk JA, Kenney WL. Age, splanchnic vasoconstriction, and heat stress during tilting. Am J Physiol 1999; 276(1 Pt 2):R203–R212.
57. Cohen DC, Winstanley A, Engledow A, Windsor AC, Skipworth JR. Marathoninduced ischemic colitis: why running is not always good for you. Am J Emerg Med 2009; 27(2):e5–e7.
58. Wilkes MM, Navickis RJ. Patient survival after human albumin administration. A meta-analysis of randomized, controlled trials. Ann Intern Med 2001; 135(3):149– 164. 59. Schultze HE, Heremans JF. Molecular biology of human proteins, with special reference to plasma proteins. Amsterdam: Elsevier; 1966.
59. Boutros A. Intra- and postoperative use of albumin. Clinical indications and results. Curr Stud Hematol Blood Transfus 1986; 53(53):180–185.
60. Fleck A, Raines G, Hawker F, Trotter J, Wallace PI, Ledingham IM, et al. Increased vascular permeability: a major cause of hypoalbuminaemia in disease and injury. Lancet 1985; 1(8432):781–784. 72
61. Hoye RC, Bennett SH, Geelhoed GW, Gorschboth C. Fluid volume and albumin kinetics occurring with major surgery. JAMA 1972; 222(10):1255–1261.
62. Gibbs J, Cull W, Henderson W, Daley J, Hur K, Khuri SF. Preoperative serum albumin level as a predictor of operative mortality and morbidity: results from the National VA Surgical Risk Study. Arch Surg. 1999; 134:36–42.
63. Pasha J. Nisar, Kweku A. Appau, Feza H. Remzi, Ravi P. Kiran. Preoperative Hypoalbuminemia Is Associated with Adverse Outcomes After Ileoanal Pouch Surgery Inflamm Bowel Dis 2012; 18:1034–1041.
64. Remuzzi G, Cravedi P, Perna A, Dimitrov BD, Turturro M, Locatelli G, et al. Longterm outcome of renal transplantation from older donors. N Engl J Med 2006; 354(4):343-52

	Pt name	age	sex	TOP	PATHOLOGY					SP	SYMP	COMORBIDITIES				RX					COMPLICATIONS			
					PP	IO	MI	BT	FT			HTN	DM	ANAE	HYPOAL		FEVER	N&V	CI	WI	WD	PI	AL	DEATH
1	hemanth kumar	31	M	3 D	N	Y	N	N	N		AP , V	N	N	Y	N	R& A	Y	Y	N	N	N	Y	N	N
2	harishankar	24	M	3D	N	Y	N	N	N		AD , AP	N	N	N	Y	R& A	Y	N	N	Y	Y	N	Y	Y
3	jonnada lachayya	32	M	4D	N	Y	N	N	N		AD , AP, C	N	Y	N	N	R& A	N	Y	Y	Y	N	N	N	N
4	gedala tatrao	43	M	1D	Y	N	N	N	N	DP	AP , V	N	N	N	N	GOP	N	N	N	N	N	N	N	N
5	p krishna rao	40	M	1D	Y	N	N	N	N	ileal	AP , V	N	N	N	Y	PC	Y	N	Y	Y	N	N	N	N
6	g krishna rao	34	M	2D	Y	N	N	N	N	ileal	AP , V	Y	N	N	N	IL	Y	Y	N	N	N	N	N	N
7	g tavittamma	68	F	5D	N	Y	N	N	N		AD ,C	N	Y	Y	Y	R& A	Y	N	Y	Y	Y	Y	N	N
8	sambana tavitiraid	22	M	3D	Y	N	N	N	N	DP	AP , V	N	N	N	Y	PC	N	N	N	N	N	N	N	N
9	sapatti malleswara	72	M	2D	Y	N	N	N	N	DP	AP , V	Y	N	N	N	PC	Y	Y	N	N	Y	Y	N	N
10	bora appayya	62	M	3D	Y	N	N	N	N	DP	AP , V	N	N	N	N	GOP	N	Y	N	N	N	N	N	N
11	p narayanamma	57	F	2D	N	Y	N	N	N	DP	AP	N	Y	Y	N	R& A	Y	N	N	Y	N	N	N	N
12	T eswararao	17	M	2D	N	N	N	N	Y		AP	N	N	N	N	PC	Y	Y	Y	N	N	N	N	N
13	p jadamma	54	F	2D	N	N	Y	N	N		AD, C	N	N	Y	N	EC	Y	N	N	N	N	N	N	N
14	veeram setti trinad	20	M	4D	N	N	N	N	N	DP	AP , V	Y	Y	N	N	GOP	N	Y	Y	Y	N	N	N	N
15	sheeksaidulu	62	M	5D	Y	N	N	N	N	IP	AP	N	N	Y	N	IL	N	N	Y	N	N	N	N	N
16	paatro somanath r	35	M	1D	N	Y	N	N	N		AP , V	N	N	N	Y	R& A	Y	Y	Y	N	N	N	Y	Y
17	pillak dandasi	48	M	1D	Y	N	N	N	N	DP	AP,V	N	N	Y	N	GOP	Y	Y	N	N	N	N	N	N
18	s venkata naidu	41	M	1D	Y	N	N	N	N	DP	AP,V	N	N	N	N	PC	Y	Y	Y	N	N	N	N	N
19	paila chandrayya	79	M	1D	Y	N	N	N	N	IP	AP	N	N	Y	Y	IL	Y	N	N	N	N	N	N	N
20	k sundra rao	39	M	2D	Y	N	N	N	N	DP	AP,V	N	Y	N	N	GOP	N	N	N	Y	N	N	N	N
21	yenni aparna	67	M	1D	Y	N	N	N	N	DP	AP,V	N	Y	Y	N	PC	Y	Y	N	N	N	N	N	N
22	t simhadri	36	M	1D	Y	N	N	N	N	IP	AP	N	N	Y	N	PC	Y	Y	N	N	N	N	N	N
23	d sridhar	75	M	1D	Y	N	N	N	N	DP	AP,V	Y	Y	N	N	GOP	N	Y	Y	Y	N	N	N	N
24	K swamy naidu	53	M	1D	Y	N	N	N	N	DP	AP,V	Y	N	Y	N	PC	Y	N	N	N	N	N	N	N
25	Lokonda appalamm	43	F	5D	N	N	Y	N	N		AD, C	Y	Y	N	N	EC	Y	Y	N	N	N	N	N	N
26	K chandra sekhar ra	68	M	1D	N	N	N	Y	N		AP	N	N	Y	Y	S	Y	N	N	Y	N	Y	N	N
27	A guranna	59	M	1D	Y	N	N	N	N	GP	AP,V	N	Y	Y	Y	OC	N	N	Y	Y	Y	N	N	N
28	Ch parvathamma	33	F	2D	Y	N	N	N	N	AP	AP,C	Y	N	N	N	GOP	Y	Y	N	N	N	N	N	N
29	M benarji	37	M	2D	N	Y	N	N	N		AP	N	N	N	Y	R& A	N	N	Y	N	N	N	Y	Y
30	M punyavathi	39	F	5D	Y	N	N	N	N	IP	AD	N	N	Y	N	GOP	Y	Y	N	N	N	N	N	N
31	s poornima	16	F	1D	Y	N	N	N	N	DP	AP,V	N	Y	Y	N	PC	Y	Y	N	N	N	Y	N	N
32	R apparao	33	M	2D	N	N	N	Y	N		AP	N	Y	N	Y	S	N	N	Y	Y	N	Y	N	N
33	b sunitha	25	F	3D	N	N	N	N	Y		AD, C	Y	N	Y	N	PC	Y	Y	N	N	N	N	N	N
34	D lingaraju	29	M	1D	Y	N	N	N	N		AP	N	Y	N	Y	PC	Y	N	Y	Y	N	Y	N	N
35	K narayana rao	53	M	1D	N	N	N	N	Y		AP	N	N	N	N	PC	Y	Y	N	N	N	N	N	N
36	b lakshmi	15	F	1D	Y	N	N	N	N	DP	AP,V	N	N	Y	N	PC	N	N	N	N	Y	N	N	N
37	K venkataramana	23	M	2D	N	N	N	Y	N		AP	N	Y	N	Y	R& A	Y	N	Y	Y	N	N	N	N
38	Nageswara rao	50	M	1D	N	N	N	N	Y		AP	Y	N	Y	N	PC	N	Y	N	N	N	N	N	N
39	anuradha	28	F	1D	Y	N	N	N	N	DP	AP,V	Y	N	N	N	GOP	Y	Y	N	N	N	N	N	N
40	v adhilakshmi	37	F	2D	N	N	N	Y	N		AP	N	Y	Y	N	R& A	N	Y	Y	Y	Y	Y	N	N
41	ganesh	39	M	2D	N	N	N	Y	N		AP	N	Y	N	N	R& A	Y	N	Y	N	N	N	N	N
42	rama rao	41	M	1D	N	N	N	N	Y		AP	N	N	Y	N	R& A	N	N	N	N	N	N	N	N
43	someswara rao	22	M	1D	Y	N	N	N	N	IP	AP	Y	N	Y	N	GOP	Y	Y	N	N	N	N	N	N
44	somayya	30	M	1D	N	N	N	Y	N		AP	Y	Y	N	Y	R& A	N	N	Y	Y	N	N	Y	Y
45	b jagadheswari	40	F	1D	N	N	N	N	Y		AP	Y	N	Y	N	PC	Y	Y	N	N	N	N	N	N
46	muralidhar	31	M	1D	N	N	N	N	Y		AP	N	N	N	N	IL	Y	Y	N	N	N	N	N	N
47	raju	35	M	2D	N	N	N	Y	N		AP	N	N	Y	Y	S	N	Y	Y	N	N	N	N	N
48	a kalyani	31	F	3D	Y	N	N	N	N	DP	AP , V	N	N	Y	Y	PC	Y	Y	Y	Y	Y	N	N	N
49	k raju	52	M	2D	Y	N	N	N	N	DP	AP	N	N	N	N	GOP	N	N	N	N	N	N	N	N
50	D rajesh	19	M	3D	Y	N	N	N	N	DP	AP	N	N	N	N	GOP	N	N	N	N	N	N	N	N