



RESEARCH ARTICLE

PROCALCITONIN AS A PREDICTIVE VALUE FOR INTESTINAL ISCHEMIA AND NECROSIS IN ACUTE INTESTINAL OBSTRUCTION PATIENTS UNDERWENT URGENT OPERATION

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ABSTRACT

**OBJECTIVE:** To assess the value of procalcitonin for early detection of intestinal ischemia and necrosis in patients suffering from acute intestinal obstruction on presentation at emergency department before urgent operation.

**METHODS PATIENTS AND:** This was a prospective study of 80 patients who suffering from acute intestinal obstruction and underwent urgent operation in emergency department of Aswan University Hospital. This 80 patients were divided into two groups. Group A (non-ischemia group, n=28). Group B (ischemia group, n=52), this group was divided into two subgroups (B1 and B2). B1 (32 patients) was considered as reversible ischemia group and B2 (20 patients) was considered as irreversible ischemia group. Group A and subgroup B1 (60 patients) was considered as non-necrosis group but subgroup B2 (20 patients) was considered as necrosis group. Group C (20 patients) was considered as control group. The important analyzed data included age, sex, special habits, the time between symptom onset and arrival at the emergency department, vital signs, symptoms, clinical findings, white blood cells count, base deficit, metabolic acidosis, serum procalcitonin levels on presentation at emergency department, the time between arrival and operation, operative findings and what's done, post-operative complications and outcome.

**RESULTS:** Serum Procalcitonin level shows insignificant deference between control and non-ischemia groups 0.33 vs 0.47 ng/ml (P= 0.15). But there was significant increase in the serum procalcitonin levels in ischemia than non-ischemia groups 4.40 vs 0.47 ng/ml (P = <0.0001) and in necrosis than non-necrosis groups 9.46 vs 0.88 ng/ml (P = <0.0001). Multivariate analysis identified serum procalcitonin as an independent predictor of intestinal ischemia in acute intestinal obstruction (P = 0.001, odds ratio 7.17, 95% confidence interval 2.31-22.21) and necrosis (P = 0.002, odds ratio 1.71, confidence interval 1.21- 2.42). Using receiver operating characteristic (ROC) curve analysis, the area under the curve (AUC) of serum procalcitonin for intestinal ischemia and necrosis was 0.85 and 0.81 respectively. A high negative predictive value (NPV) for intestinal ischemia and necrosis of serum procalcitonin levels at <0.36 ng/ml (cut off point) was 80% and 90% respectively. Also a positive predictive value (PPV) for intestinal ischemia and necrosis of serum procalcitonin at >1.77ng/ml were 92% and 70% respectively.

**CONCLUSION:** Serum procalcitonin levels on presentation at emergency department were an independent predictor of intestinal ischemia and necrosis. Thus procalcitonin may be useful for early detection or exclusion of intestinal ischemia and necrosis in acute intestinal obstruction and can be used as an additional diagnostic tool to improve clinical decision-making.

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INTRODUCTION

Acute intestinal obstruction is a common surgical emergency and frequently encountered as a problem in abdominal surgery (Lo *et al.*, 2007). It is a dangerous condition that requires immediate diagnosis and urgent appropriate treatment (Takeachi *et al.*, 2004). Surgeons are mainly concerned with intestinal obstruction because accurate and early recognition of bowel strangulation still remains a difficult problem and is associated with high morbidity and mortality (Fevang *et al.*, 2000).

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The majority of surgeons, therefore, continue to support the idea that the sun should not rise or set on patients with acute intestinal obstruction, in other words, they contend that early operation is always necessary (Firoozmand, 2001). Intestinal ischemia is a major health problem that accounts for 1-2% of intestinal diseases (Kassahun *et al.*, 2008). In United States, intestinal ischemia accounts for 0.1% of all hospital admissions, the incidence of this condition has increased over the last few decades (from 1 in 1000 to 1 in 200 hospitalization for abdominal pain). In most cases, intestinal ischemia requires emergency treatment to avoid tissue necrosis, infectious outcomes, septic shock or lethal multiple organ failure (Higgins *et al.*, 2004).

Intestinal ischemia has been defined as impairment of the intestinal blood supply from celiac axis, superior mesenteric artery and inferior mesenteric artery, this results in tissue injury and a low-flow state with poor intestinal arterial perfusion (Renner *et al.*, 2011). Procalcitonin (PCT) is a 116-amino-acid (AA) precursor of calcitonin (Maruna *et al.*, 2000), that was first described in 1993 by Assicot *et al.*, as a marker of infection. It has three domains:- a 57AA N-terminal domains, the 32 AA calcitonin fragment (involved in the regulation of calcium and phosphorus metabolism) and the 21 AA katacalcin fragment (measured in PCT assays) (Assicot *et al.*, 1993). Procalcitonin is a member of the calcitonin gene-related peptide family and is encoded by the *CALC-I* gene located on the chromosome 11 (11p) (Maruna *et al.*, 2000). In healthy subjects, the "hormokine" PCT is released from the C cells of the thyroid (Becker *et al.*, 2004). In a disease context, PCT production can be stimulated by trauma (Nanda *et al.*, 2009), bacterial endotoxins, pro-inflammatory cytokines (tumour necrosis factor alpha (TNF- $\alpha$ ) and interleukin-6(IL-6)) or cardiogenic shock (Assicot *et al.*, 1993). It is thought that this PCT is released by the liver parenchyma (Lavrentieva *et al.*, 2007). The half-life-time of PCT is between 18 and 24 hours, in patients with kidney failure, between 24 and 30 hours (with a peak at 24 h), the procalcitonin normal level ranged from 0.10-0.50 ng/ml (Barute *et al.*, 2010). The kinetics of serum PCT are not influenced by age, gender or renal function (because only a proportion of PCT is excreted by the kidneys) (Meisner *et al.*, 2000). A number of clinical studies have shown that procalcitonin is a marker for sepsis and inflammation (Barute *et al.*, 2010), for colonic ischemia after aortic surgery (Nagata *et al.*, 2008), and also for intestinal ischemia after acute intestinal obstruction (Markogiannakis *et al.*, 2011). The aim of this study was to assess the value of procalcitonin for early detection of intestinal ischemia and necrosis in patients suffering from acute intestinal obstruction on presentation at emergency department before urgent operations.

## PATIENTS AND METHODS

This was a prospective study of acute intestinal obstruction victims (patients with incomplete or chronic intestinal obstruction, paralytic ileus and acute mesenteric ischemia were excluded) submitted to urgent surgical treatment from any age and sex, admitted to the emergency department of Aswan University Hospital-Egypt. In a period from 1st January 2016 to 30th December 2016. The number of cases were eighty (80) patients underwent urgent surgical operations and analysis of their records. Data were collected by us and our residents in emergency departments of Aswan University Hospital. The consent was taken from patients or their guardians and relatives. Diagnosis of acute intestinal obstruction was made based on clinical and radiological criteria. Those patients were divided into two groups (A & B). Group A (28 patients) was considered as non-ischemia group. Group B (52 patients) was considered as ischemia group. This group B was divided into two subgroups (B1 and B2). B1 (32 patients) was considered as reversible ischemia group and B2 (20 patients) was considered as irreversible ischemia group (Necrosis group). Also group A and subgroup B1 were considered as non-necrosis group (60 patients). Group C (control group) include 20 patients not suffering from acute bowel obstruction, other emergency diseases or anybody infections, but complaining from other elective surgical problems e.g:- lipoma, non-infected sebaceous cyst...etc. The collected data include :- a) Socio-demographic data for each patient in the form of (age,

sex, special habits, time interval between onset of symptoms and arrival to emergency department). b) Clinical data:-Vital signs ( pulse rate, blood pressure, temperature and respiratory rate). Symptoms (nausea and abdominal discomfort, vomiting, abdominal pain (colicky pain, constant pain or no pain) absolute constipation (no passage of flatus and/or stool)). bdominal clinical examination ( distension, involuntary muscle guarding, tenderness on palpation and rebound tenderness ). c) Investigations:- 1- Laboratory investigations:- (Serum Procalcitonin, white blood cells and blood gases (metabolic acidosis and base deficit)). 2- Radiological investigations:- (plain -x- ray abdomen (erect and supine), abdominal sonography and abdominal C-T ). d) Time interval between arrival and operation. e) Causes of acute intestinal obstruction (band of adhesions, obstructed hernias with or without strangulated, volvulus or intestinal mass). f) Intraoperative findings (the cause of obstruction, normal intestinal loop, reversible ischemic loop or irreversible ischemic loop). g) What is done during the operation (release of constricting ring of hernias, release of adhesions and bands, resection anastomosis, colostomy and also determination of the affected part of small or large intestine). Intra-operative assessment of intestinal viability was done, based on color, vessel pulsation after restoration of the arterial blood pressure in patient with hypotension at operation and peristalsis. h) Admission in I C U and length of hospital stay. i) Post-operative complications (wound hematoma, wound infection, wound dehiscence, abdominal distension, fecal fistula, chest infection, vomiting and re-exploration). j) Outcome discharge of patients. Procalcitonin methodology:- by ELISA technique.

## STATISTICAL ANALYSIS

Data was analyzed using Medcalc for Windows (version 11.0) and STATA (version 9.2). Quantitative data was represented as mean, standard deviation, and median. Data was analyzed using student t-test to compare means of two groups. When the data was not normally distributed Mann-Whitney test was used to compare two groups. Qualitative data was presented as number and percentage and compared using either Chi square test or fisher exact test. All significant variable in univariate analysis were entered in multivariate logistic regression analysis. Only final model with significant variables were presented in this study. The results of multivariate analysis also were calculated as odds ratio (OR) and 95% confidence interval (95% CI). Data were analyzed by sensitivity, specificity, positive, and negative predictive value derived from the receiver operating characteristic (ROC) curve. The diagnostic accuracy of PCT for predicting ischemia and necrosis were expressed as the area under the ROC curve (AUC). Graphs were produced by using Excel or STATA program. P value was considered significant if it was less than 0.05.

## RESULTS

There was insignificant difference between control and non-ischemia groups regarding to age, gender and procalcitonin, as shown in Table (1). Regarding to age, gender and special habits there was insignificant difference between non-ischemia and ischemia groups, as shown in Table (2). Regarding to time interval between onset of symptoms and arrival to emergency department, pulse rate, temperature and respiratory rate were significantly increased in ischemia group in comparison to non-ischemia groups (P = <0.0001), but there was significant decrease in systolic and diastolic blood

**Table (1): Comparison between control and non-ischemia groups**

Variables	Controls N=20	Non-ischemia N=28	P value
Age *	55.10±11.77 (55.5)	53.00±17.20 (56)	0.90
Gender, n (%)			
Females	8 (40.00%)	10 (35.71%)	0.76
Males	12 (60.00%)	18 (64.29%)	
Procalcitonin *	0.33±0.10 (0.33)	0.47±0.53 (0.29)	0.15

\* Values are expressed as mean ± SD; the median is shown in parentheses

**Table (2): Comparison between non-ischemia and ischemia groups as regard to general characteristics of studied population**

Variables	Non-ischemia N=28	Ischemia N=52	P value
Age *	53.00±17.20 (56)	58.77±12.82 (59.5)	0.20
Gender, n (%)			
Females	10 (35.71%)	18 (34.62%)	0.92
Males	18 (64.29%)	34 (65.38%)	
Special habits, n (%)			
No	10 (35.71%)	12 (23.08%)	0.49
Cigarette	10 (35.71%)	20 (38.46%)	
Goza	8 (28.57%)	18 (34.62%)	
Both	0	2 (3.85%)	

\* Values are expressed as mean ± SD; the median is shown in parentheses

**Table (3): Comparison between non-ischemia and ischemia groups as regard to clinical features of studied population**

Variables	Non-ischemia N=28	Ischemia N=52	P value
Time interval bet. Onset of symp. and arrival to emerg. Depart. *	13.64±4.91 (13.5)	21.19±8.17 (19.5)	<0.0001
Pulse rate*	80.93±6.94 (80)	91.00±10.41 (90)	<0.0001
Systolic blood pressure*	121.07±14.17 (120)	109.04±9.60 (110)	<0.0001
Diastolic blood pressure*	75.00±8.61 (75)	65.96±7.00 (67.5)	<0.0001
Temperature*	37.14±0.27 (37)	37.67±0.67 (37.5)	0.0001
Respiratory rate*	15.64±1.25 (15.5)	17.31±1.42 (17.5)	<0.0001
Nausea and abdominal discomfort, n (%)	18 (64.29%)	34 (65.38%)	0.92
Vomiting, n (%)	18 (64.29%)	42 (80.77%)	0.10
Abdominal pain, n (%)			0.001
No pain	2 (7.14%)	10 (19.23%)	
Colicky pain	20 (71.43%)	12 (23.08%)	
Constant	4 (14.29%)	20 (38.46%)	
Both	2 (7.14%)	10 (19.23%)	
No passage of flatus, n (%)	20 (71.43%)	36 (69.23%)	0.84
No passage of stool, n (%)	22 (78.57%)	40 (76.92%)	0.87
Abd. distention, n (%)	22 (78.57%)	38 (73.08%)	0.59
Involuntary muscle guarding, n (%)	14 (50.00%)	24 (46.15%)	0.74
Tenderness on palpation, n (%)	22 (78.57%)	30 (57.69%)	0.06
Rebound tenderness, n (%)	8 (28.57%)	34 (65.38%)	0.002

\* Values are expressed as mean ± SD; the median is shown in parentheses

**Table (4): Comparison between non-ischemia and ischemia groups as regard to Lab. and radiological findings of studied population**

Variables	Non-ischemia N=28	Ischemia N=52	P value
W.B.C (in thousand)*	7.67±1.99 (7)	12.27±2.59 (12)	<0.0001
Serum Procalcitonin [PCL(ng/ml)]*	0.47±0.53 (0.29)	4.40±6.20 (1.5)	<0.0001
Base deficit [HCO <sub>3</sub> ] (mmol/l)	23.57±1.68 (24.15)	22.28±1.58 (22.4)	0.001
Metabolic acidosis	4 (14.29%)	24 (46.15%)	0.004
Multiple fluid level (X-ray), n (%)	20 (71.43%)	40 (76.92%)	0.59
Dilated bowel loops (X-ray), n (%)	18 (64.29%)	36 (69.23%)	0.65
Dilated bowel loops (Sonar), n (%)	24 (85.71%)	48 (92.31%)	0.35
Abdominal CT, n (%)			
Adhesive band	8 (28.57%)	14 (26.92%)	0.77
Dilated bowel loop	16 (57.14%)	28 (53.85%)	
Mass	4 (14.29%)	8 (15.38%)	
Thick wall	0	2 (3.85%)	

\* Values are expressed as mean ± SD; the median is shown in parentheses

pressure in ischemia group in comparison to non-ischemia groups (P = <0.0001). Constant pain and rebound tenderness were significantly increased in ischemia group in comparison to non-ischemia group (38.46% vs 14.29% and 65.38% vs 28.57%, respectively, P = 0.001 and 0.002 respectively), while

colicky pain was significantly increased in non-ischemia group in comparison to the ischemia group (71.43% vs 23.08%, P = 0.001). Other clinical features shows insignificant difference between non-ischemia and ischemia groups, as shown in Table (3).

Table (5): Comparison between non-ischemia and ischemia groups as regard to operative details of studied population

Variables	Non-ischemia N=28	Ischemia N=52	P value
Time interval between arrival and operation (hours)*	21.57±11.42 (17.5)	11.42±4.65 (11)	<0.0001
Diagnosis			
Bands of adhesions, n (%)	8 (28.57%)	14 (26.92%)	0.88
Obstructed hernia without strangulation, n (%)			<0.0001
No			
Yes (femoral hernia)	14 (50.00%)	52 (100%)	
Yes (inguinal hernia)	2 (7.14%)	0	
Yes (paraumbilical hernia)	6 (21.43%)	0	
	6 (21.43%)	0	
Obstructed hernia with strangulation, n (%)			0.001
No			
Yes (inguinal hernia)	28 (100%)	28 (53.85%)	
Yes (femoral hernia)	0	6 (11.54%)	
Yes (incisional hernia)	0	4 (7.69%)	
Yes (paraumbilical hernia)	0	4 (7.69%)	
	0	10 (19.23%)	
Volvulus, n (%)	2 (7.14%)	6 (11.54%)	0.71
Intestinal mass, n (%)	4 (14.29%)	8 (15.38%)	1.00
What is done			
Release of constructing ring of hernia, n (%)	14 (50.00%)	24 (46.15%)	0.74
Release of adhesions and bands, n (%)	8 (28.57%)	14 (26.92%)	0.88
Resection anastomosis, n (%)	2 (7.14%)	16 (30.77%)	0.02
Colostomy, n (%)	4 (14.29%)	14 (26.92%)	0.20
Part affected, n (%)			
Ileum	14 (50.00%)	22 (42.31%)	
Ileal mass	2 (7.14%)	0	0.33
Jejunum	8 (28.57%)	16 (30.77%)	
Rectal mass	2 (7.14%)	6 (11.54%)	
Sigmoid volvulus	2 (7.14%)	6 (11.54%)	
Sigmoid mass	0	2 (3.85%)	

\* Values are expressed as mean ± SD; the median is shown in parentheses

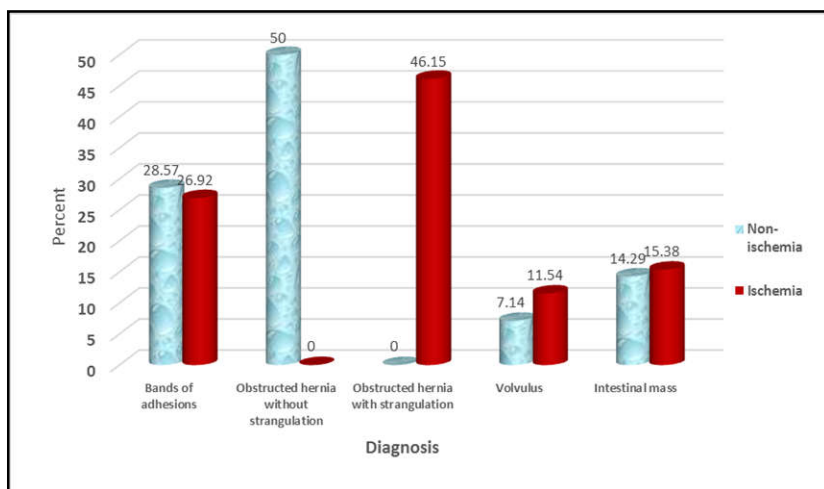


Fig (1): Comparison between non-ischemia and ischemia groups as regard to diagnosis

Table (6): Comparison between non-ischemia and ischemia groups as regard to postoperative finding of studied population

Variables	Non-ischemia N=28	Ischemia N=52	P value
Admission in the ICU*	2 (7.14%)	24 (46.15%)	<0.0001
Length of ICU stay*	5±0 (5)	3.5±1.06 (3)	0.07
Length of hospital stay*	8.07±2.94 (85)	11.77±4.41 (11)	0.0001
Postoperative complications			
Wound Infection, n (%)	4 (14.29%)	12 (23.08%)	0.35
Wound Hematoma, n (%)	2 (7.14%)	8 (15.38%)	0.48
Wound Dehiscence, n (%)	2 (7.14%)	8 (15.38%)	0.48
Abdominal Distention, n (%)	4 (14.29%)	10 (19.23%)	0.76
Fecal Fistula, n (%)	2 (7.14%)	8 (15.38%)	0.48
Chest Infection, n (%)	6 (21.43%)	16 (30.77%)	0.37
Vomiting, n (%)	4 (14.29%)	16 (30.77%)	0.10
Re-Exploration, n (%)	2 (7.14%)	8 (15.38%)	0.48
Death, n (%)	0	4 (7.69%)	0.29

\* Values are expressed as mean ± SD; the median is shown in parentheses

Regarding to laboratory results, serum PTC, WBC and metabolic acidosis were significantly increased in ischemia group in comparison to non-ischemia group ( $P = <0.0001$ ,  $<0.0001$  and  $0.004$ , respectively), but there was significant decrease in base deficit in ischemia group in comparison to non-ischemia group ( $P = 0.001$ ). Regarding to radiological findings there were insignificant difference between ischemia and non-ischemia groups, as shown in Table (4).

Ilium was the most affected part of small intestine in acute intestinal obstruction in both non-ischemia and ischemia groups (57.14% and 42.31% respectively), followed by the jejunum (28.57%) in non-ischemia and (30.77%) in ischemia groups, while the sigmoid volvulus and rectal mass were the commonest cause of large bowel obstruction (7.14%) in non-ischemia and (11.54%) in ischemia groups, but sigmoid mass represent (3.85%) in ischemia group only, as shown in Table (5) and Fig (1).

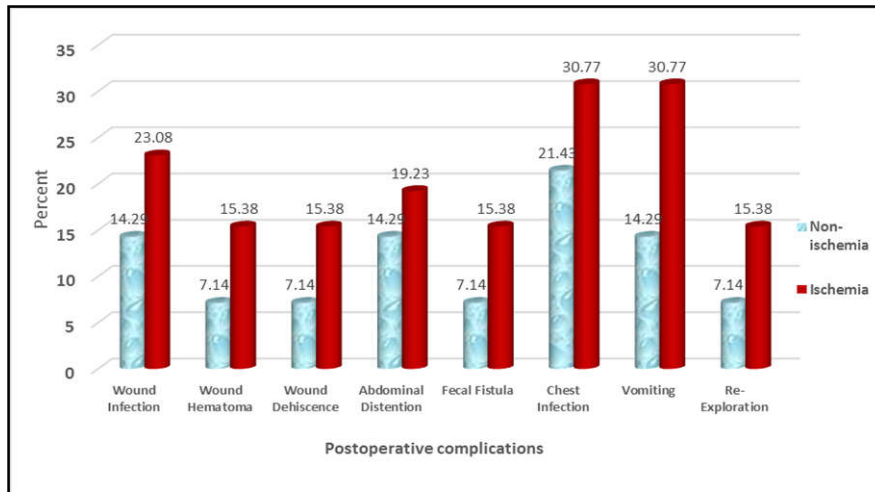


Fig (2): Comparison between non-ischemia and ischemia groups as regard to Postoperative complications

Table (7): Final Multivariate analysis for predictive factors of bowel ischemia

Variable	Odds ratio (95% CI)	P value
Pulse rate/min	1.33 (1.03-1.25)	0.009
S. procalcitonin	7.17 (2.31-22.21)	0.001
Baseline deficit	3.91 (1.31-11.62)	0.01

Table (8): Comparison between non-necrosis and necrosis groups as regard to general characteristics of studied population

Variables	Non-Necrosis N=60	Necrosis N=20	P value
Age *	55.77±15.18 (56)	59.7±12.90 (60)	0.22
Gender, n (%)			
Females	22 (36.67%)	6 (30.00%)	0.59
Males	38 (63.33%)	14 (70.00%)	
Special habits, n (%)			
No	18 (30.00%)	4 (20.00%)	0.08
Cigarette	22 (36.67%)	8 (40.00%)	
Goza	20 (33.33%)	6 (30.00%)	
Both	0	2 (10.00%)	

\* Values are expressed as mean ± SD; the median is shown in parentheses

Time interval between arrival and operation was significantly decreased in ischemia in comparison to non-ischemia groups,  $P = <0.0001$ . Regarding to diagnosis, bands of adhesions were more in non-ischemia group in comparison to ischemia groups (28.5% vs 26.92%), obstructed hernias without strangulation represent (50%) of non-ischemia group, while obstructed hernias with strangulation represent (46.15%) of ischemia group, volvulus were more in ischemia than non-ischemia groups (11.54% vs 7.14%), also intestinal masses were more in ischemia than non-ischemia groups (15.38% vs 14.29%). Regarding to operative intervention, resection anastomosis was significantly increased in ischemia group in comparison to non-ischemia group (30.77% vs 7.14%,  $P = 0.02$ ), while there were insignificant differences between the non-ischemia and ischemia groups regarding to release of constricting ring of hernias, release of adhesions and bands and colostomy.

There was significant increase in I C U admission and length of hospital stay in ischemia group in comparison to non-ischemia group ( $P = <0.0001$  and  $0.0001$ , respectively), but there was insignificant difference regarding to post-operative complications and outcome in both studied groups, as shown in Table (6) and Fig (2). The final results of multivariate analysis for predictive factors of bowel ischemia were serum PCT ( $P = 0.001$ , OR. 7.17, 95% CI. 2.31-22.21), pulse rate ( $P = 0.009$ , OR. 1.33, 95% CI. 1.03-1.25) and base deficit ( $P = 0.01$ , OR. 3.91, 95% CI. 1.31- 11.62), those were identified as independent predictors of intestinal ischemia, as shown in Table (7). Regarding to age, gender and special habits there were insignificant difference between non-necrosis and necrosis groups, as shown in table (8). Regarding to time interval between onset of symptoms and arrival to emergency department ( $P = 0.001$ ), pulse rate, temperature and respiratory

**Table (9): Comparison between non-necrosis and necrosis groups as regard to clinical features of studied population**

Variables	Non-Necrosis N=60	Necrosis N=20	P value
Time interval bet. Onset of symp. and arrival to emerg. Depart.*	16.23±5.30 (17.5)	25.5±10.63 (23.5)	0.001
Pulse rate*	83.8±7.17 (85)	98.5±11.25 (97.5)	<0.0001
Systolic blood pressure*	116.17±12.87 (110)	104.5±7.05 (102.5)	0.0002
Diastolic blood pressure*	71.33±8.02 (70)	62.5±7.34 (60)	<0.0001
Temperature*	37.31±0.33 (37.3)	37.99±0.94 (37.85)	<0.0001
Respiratory rate*	16.3±1.43 (16)	18±1.30 (18)	<0.0001
Nausea and abdominal discomfort, n (%)	38 (63.33%)	14 (70.00%)	0.59
Vomiting, n (%)	44 (73.33%)	16 (80.00%)	0.55
Abdominal pain, n (%)			
No pain	8 (13.33%)	4 (20.00%)	
Colicky pain	30 (50.00%)	2 (10.00%)	0.02
Constant	14 (23.33%)	10 (50.00%)	
Both	8 (13.33%)	4 (20.00%)	
No passage of flatus, n (%)	42 (70.00%)	14 (70.00%)	1.00
No passage of stool, n (%)	46 (76.67%)	16 (80.00%)	0.76
Distention, n (%)	44 (73.33%)	16 (80.00%)	0.55
Involuntary muscle guarding, n (%)	28 (46.67%)	10 (50.00%)	0.80
Tenderness on palpation, n (%)	40 (66.67%)	12 (60.00%)	0.59
Rebound tenderness, n (%)	26 (43.33%)	16 (80.00%)	0.004

\* Values are expressed as mean ± SD; the median is shown in parentheses

**Table (10): Comparison between non-necrosis and necrosis groups as regard to Lab and radiological finding of studied population**

Variables	Non-Necrosis N=60	Necrosis N=20	P value
W.B.C (in thousand)*	9.58±2.79 (9.25)	13.91±2.21 (14.35)	<0.0001
Serum Procalcitonin [PCL(ng/ml)]*	0.88±0.65 (0.78)	9.46±7.69 (10.44)	<0.0001
Metabolic acidosis, n (%)	14 (23.33%)	14 (70.00%)	<0.0001
Base deficit [HCO <sub>3</sub> ] (mmol/l)	23.14±1.52 (23.3)	21.45±1.72 (21.1)	0.0001
Multiple fluid level (X-ray) , n (%)	44 (73.33%)	16 (80.00%)	0.55
Dilated bowel loops (X-ray) , n (%)	42 (70.00%)	12 (60.00%)	0.41
Dilated bowel loops (Sonar) , n (%)	8 (13.33%)	0	0.19
Abdominal CT, n (%)			
Adhesive band	16 (26.67%)	6 (30.00%)	
Dilated bowel loop	34 (56.67%)	10 (50.00%)	0.09
Mass	10 (16.67%)	2 (10.00%)	
Thick wall	0	2 (10.00%)	

\* Values are expressed as mean ± SD; the median is shown in parentheses

**Table (11): Comparison between non-necrosis and necrosis groups as regard to operative details of studied population**

Variables	Non-Necrosis N=60	Necrosis N=20	P value
Time interval between arrival and operation (hours)*	16.97±9.59 (14)	9±2.5 (9)	0.0005
Diagnosis			
Bands of adhesions, n (%)	16 (26.67%)	6 (30.00%)	0.77
Obstructed hernia without strangulation, n (%)			
No			0.13
Yes (femoral hernia)	46 (76.67%)	20 (100%)	
Yes (inguinal hernia)	2 (3.33%)	0	
Yes (paraumbilical hernia)	6 (10.00%)	0	
6 (10.00%)		0	
Obstructed hernia with strangulation, n (%)			0.23
No			
Yes (femoral hernia)	46 (76.67%)	10 (50.00%)	
Yes (incisional hernia)	2 (3.33%)	2 (10.00%)	
Yes (inguinal hernia)	2 (3.33%)	2 (10.00%)	
Yes (Para umbilical hernia)	4 (6.67%)	2 (10.00%)	
6 (10.00%)		4 (20.00%)	
Volvulus, n (%)	6 (10.00%)	2 (10.00%)	1.00
Intestinal mass, n (%)	10 (16.67%)	2 (10.00%)	0.47
What is done			
Release of constructing ring of hernia, n (%)	28 (46.67%)	10 (50.00%)	0.80
Release of adhesions and bands, n (%)	16 (26.67%)	6 (30.00%)	0.77
Resection anastomosis, n (%)	2 (3.33%)	16 (80.00%)	<0.0001
Colostomy, n (%)	14 (23.33%)	4 (20.00%)	1.00
Part affected, n (%)			
Ileum	26 (43.33%)	10 (50.00%)	
Ileal mass	2 (3.33%)	0	
Jejunum	18 (30.00%)	6 (30.00%)	0.92
Rectal mass	6 (10.00%)	2 (10.00%)	
Sigmoid colon	6 (10.00%)	2 (10.00%)	
Sigmoid mass	2 (3.33%)	0	

\* Values are expressed as mean ± SD; the median is shown in parentheses

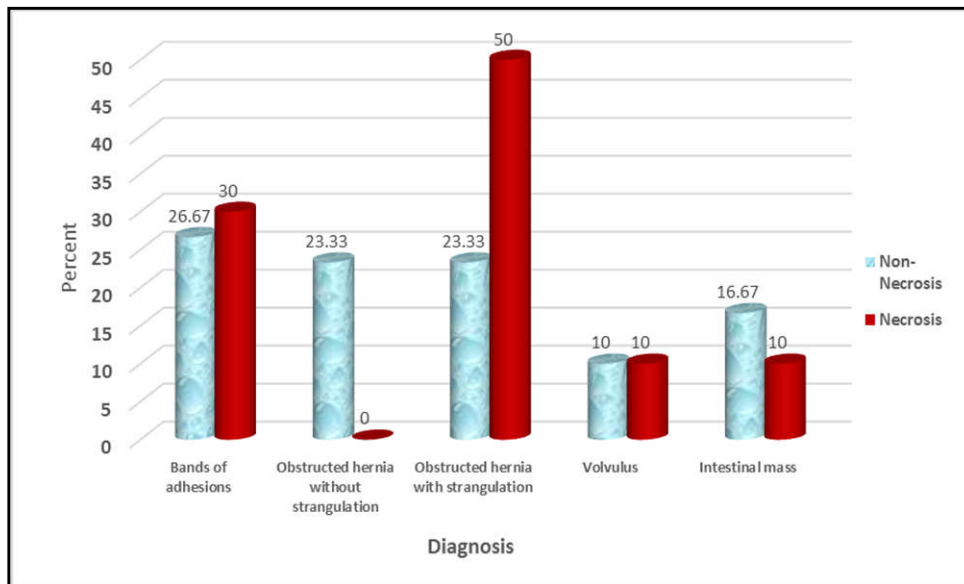


Fig (3): Comparison between non-necrosis and necrosis groups as regard to diagnosis

Table (12): Comparison between non-necrosis and necrosis groups as regard to postoperative finding of studied population

Variables	Non-Necrosis N=60	Necrosis N=20	P value
Admission in the ICU*	12 (20.00%)	14 (70.00%)	<0.0001
Length of ICU stay*	3.5±1.17 (3)	3.71±1.06 (4)	0.59
Length of hospital stay*	9.4±2.94 (9)	13.7±6.02 (14.5)	0.0004
Postoperative complications			
Wound Infection, n (%)	10 (16.67%)	6 (30.00%)	0.20
Wound Hematoma, n (%)	6 (10.00%)	4 (20.00%)	0.24
Wound Dehiscence, n (%)	6 (10.00%)	4 (20.00%)	0.26
Abdominal Distention, n (%)	8 (13.33%)	6 (30.00%)	0.10
Fecal Fistula, n (%)	4 (6.67%)	6 (30.00%)	0.01
Chest Infection, n (%)	14 (23.33%)	8 (40.00%)	0.16
Vomiting, n (%)	12 (20.00%)	8 (40.00%)	0.13
Re-Exploration, n (%)	4 (6.67%)	6 (30.00%)	0.01
Death, n (%)	0	4 (20.00%)	0.003

\* Values are expressed as mean ± SD; the median is shown in parentheses

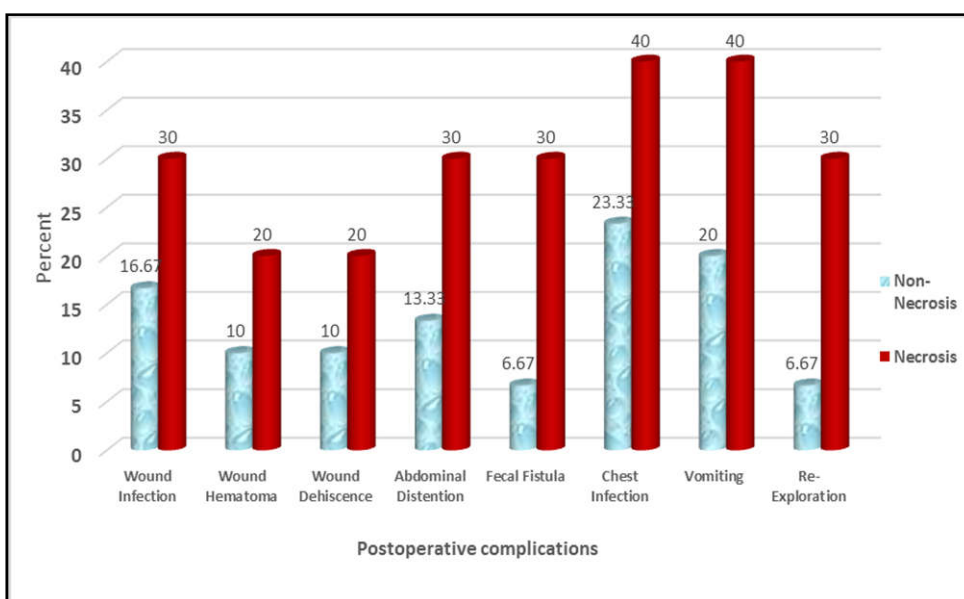


Fig (4): Comparison between non-necrosis and necrosis groups as regard to postoperative complications

Table (13): Final Multivariate analysis for predictive factors of necrosis

Variable	Odds ratio (95% CI)	P value
Pulse rate/min	1.35 (1.09-1.68)	0.007
S. procalcitonin	1.71 (1.21-2.42)	0.002

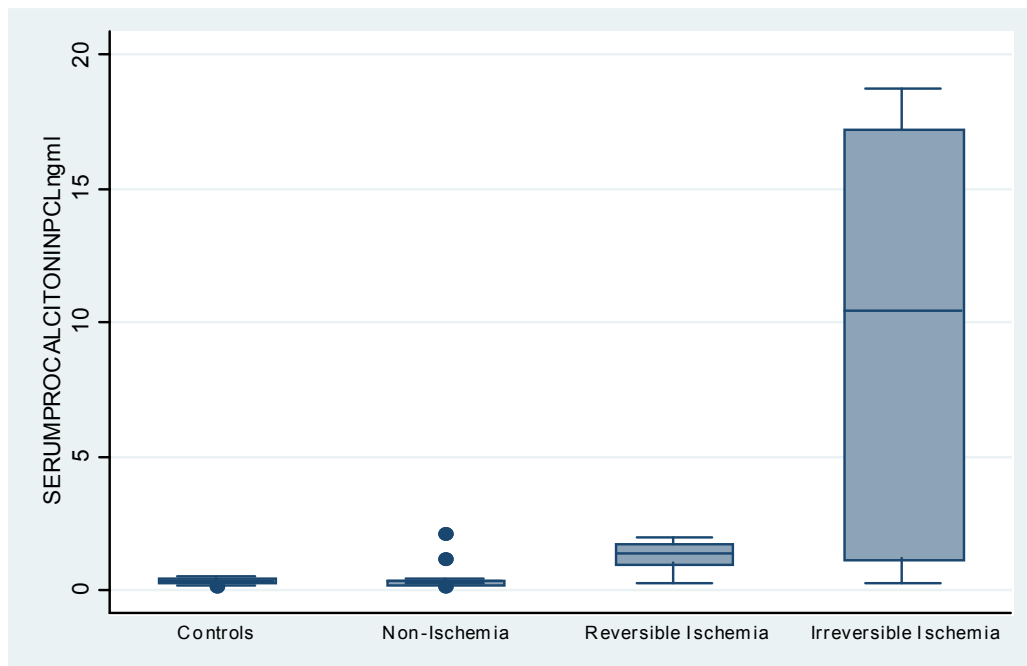


Fig (5): Procalcitonin levels among different study groups

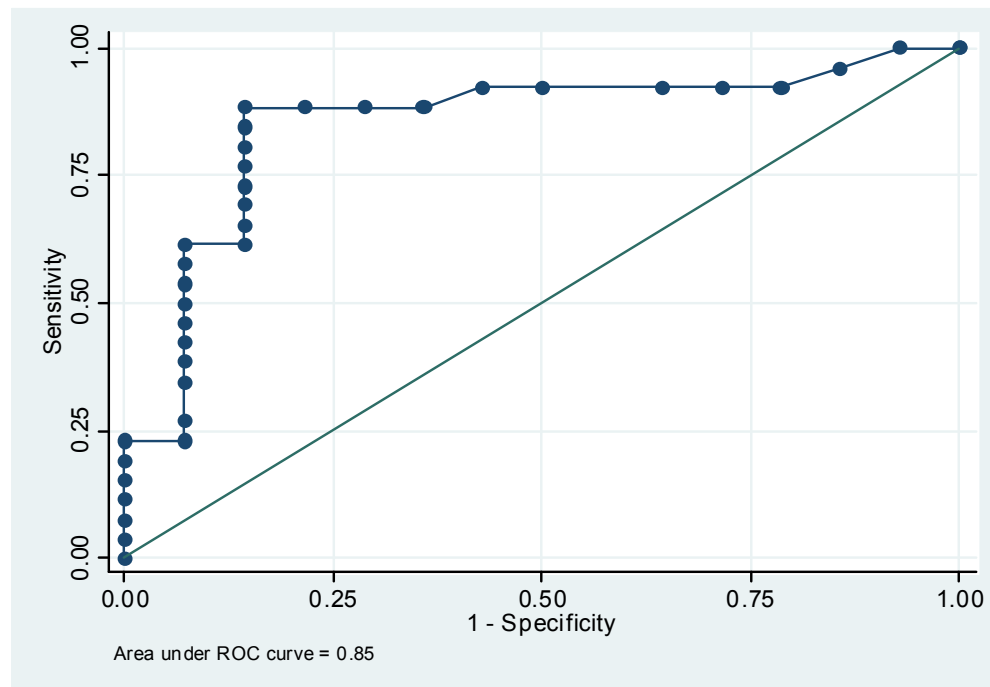


Fig (6): Roc curve of procalcitonin in predicting presence of ischemia

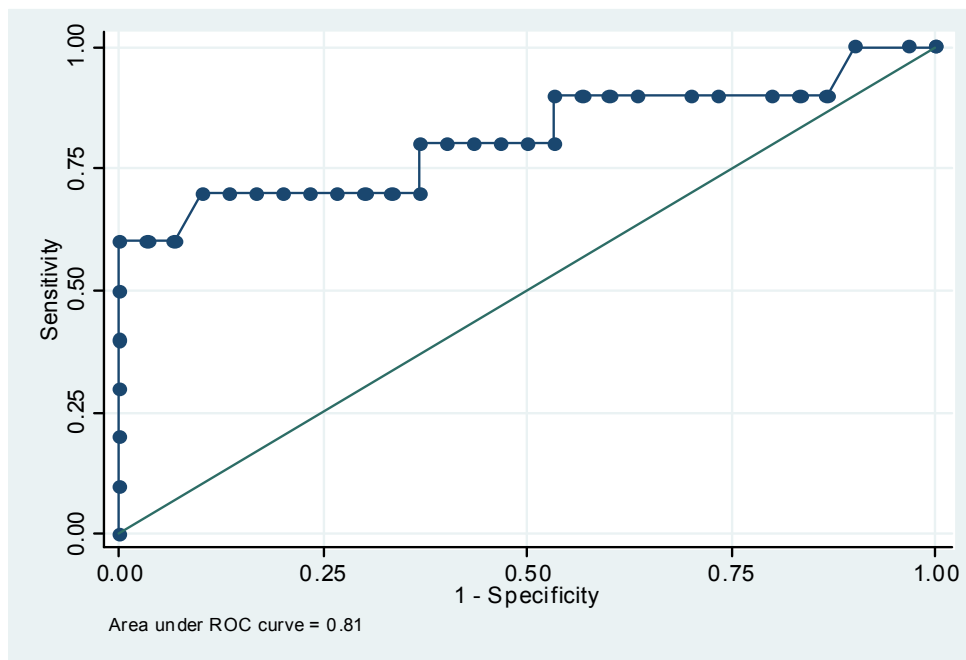
rate were significantly increased in necrosis group in comparison to non-necrosis group ( $P = <0.0001$ ), but there was significant decrease in systolic and diastolic blood pressure in necrosis group in comparison to non-necrosis group ( $P = 0.0002$  and  $<0.0001$  respectively). Constant pain and rebound tenderness were significantly increased in necrosis group in comparison to non-necrosis group (50% vs 23.33% and 80% vs

43.33% respectively,  $P = 0.02$  and  $0.004$  respectively), while colicky pain was significantly increased in non-necrosis group in comparison to necrosis group (50% vs 10%,  $P = 0.02$ ). Other clinical features shows insignificant difference between non-necrosis and necrosis groups, as shown in Table (9). Regarding to laboratory results, serum PCT, WBC and metabolic acidosis were significantly increased in necrosis group in comparison to



**Cut off point, Area under the curve, sensitivity, specificity, PPV, NPV and accuracy of serum procalcitonin in predicting presence of ischemia**

Cut off point	Area under the curve	sensitivity	specificity	PPV	NPV	accuracy
>0.36	0.85 (0.76-0.92)	88.5 (76.6-95.6)	85.7 (67.3-96.0)	92 (80.8-97.8)	80.0 (61.4-92.3)	87.10%



**Fig (7): Roc curve of procalcitonin in predicting presence of necrosis**

**Cut off point, Area under the curve, sensitivity, specificity, PPV, NPV and accuracy of serum procalcitonin in predicting presence of necrosis**

Cut off point	Area under the curve	sensitivity	specificity	PPV	NPV	accuracy
>1.77	0.81 (0.71-0.89)	70.0 (45.7-88.1)	90.0 (79.5-96.2)	70 (45.0-88.5)	90 (79.5-96.2)	80%

non-necrosis group ( $P = <0.0001$ ), but there was significant decrease in base deficit in necrosis group in comparison to non-necrosis group ( $P = 0.0001$ ). Regarding to radiological findings there was insignificant difference between necrosis and non-necrosis groups, as shown in Table (10). Time interval between arrival and operation was significantly decreased in necrosis group in comparison to non-necrosis group ( $P = 0.0005$ ). Regarding to diagnosis, bands of adhesions were more in necrosis in comparison to non-necrosis groups (30% vs 26.67%), obstructed hernias without strangulation were more prevalent in non-necrosis group in comparison to necrosis group (23.33 vs 0%), while obstructed hernias with strangulation were more common in necrosis group in comparison to non-necrosis group (50% vs 23.33%), volvulus was equal in incidence in both non-necrosis and necrosis groups (10%), also intestinal masses were more prevalent in non-necrosis than necrosis groups (16.67% vs 10%). Regarding to operative intervention, resection anastomosis was significantly increased in necrosis group in comparison to non-necrosis group (80% vs 3.33%)  $P = <0.0001$ , while there were insignificant differences between non-necrosis and necrosis groups regarding to release of constricting ring of hernias, release of adhesions and bands and colostomy. Ilium was the most affected part of small intestine in both non-necrosis and necrosis groups (46.66% and 50%, respectively), followed by the jejunum (30%) in both non-necrosis and necrosis groups, while the sigmoid volvulus and rectal masses were the

commonest causes of large bowel obstruction in non-necrosis and necrosis groups (10%) for each group, but sigmoid mass represents (3.33%) in non-necrosis group only, as shown in Table (11) and Fig (3). There was significant increase in I C U admission and length of hospital stay in necrosis group in comparison to non-necrosis group ( $P = <0.0001$  and  $0.0004$  respectively). Regarding to post-operative complications and outcome there were significant increase in fecal fistula, re-exploration and death in necrosis group in comparison to non-necrosis group ( $P = 0.01, 0.01$  and  $0.003$  respectively), but other post-operative complication shows insignificant difference between the studied groups, as shown in Table (12) and Fig (4). The final results of the multivariate analysis were serum PCT ( $P = 0.002$ , OR. 1.71, 95% CI. 1.21- 2.42) and pulse rate ( $P = 0.007$ , OR. 1.35, 95% CI. 1.09- 1.68) they were identified as independent predictors of intestinal necrosis, as shown in Table (13). Using ROC curve analysis, the AUC of PCT for ischemia was 0.85(95% CI. 0.76- 0.92), serum PCT cut of point at  $> 0.36$  ng/ml for predicting ischemia yielded 88.5% (95% CI. 76.6-95.6) sensitivity, 85.7% (95% CI. 67.3-96.0) specificity, 92% (95% CI. 80.8- 97.8) PPV, 80% (95% CI. 61.4- 92.3) NPV and 87.1% accuracy, as shown in Fig (6). Using ROC curve analysis, the AUC of PCT for necrosis was 0.81(95% CI. 0.71- 0.89), serum PCT cut of point at  $> 1.77$  ng/ml for predicting necrosis yielded 70.0% (95% CI. 45.7- 88.1) sensitivity, 90% (95% CI. 79.5- 96.2) specificity, 70% (95% CI. 45.0-88.5) PPV, 90% (95% CI. 79.5-96.2) NPV



**Fig.(8) Small intestinal loop shows irreversible ischemia (gangrenous loop)**



**Fig. (9). Small intestinal loop shows reversible ischemia..**

and 80% accuracy, as shown in Fig . (7). A high *NPV* for intestinal ischemia and necrosis of serum procalcitonin levels at  $<0.36$  ng/ml (cut off point) was 80% and 90% respectively. Also a *PPV* at  $>1.77$ ng/ml (cut off point) were 92% and 70% respectively.

## DISCUSSION

In the studies using univariate analysis, strangulation was associated with age (Bizer *et al.*, 1981). Others reported that, age and female sex had a marked effect on the rate of strangulation (Lo *et al.*, 2007). In our study, there was insignificant deference between non-ischemia and ischemia groups regarding to age, sex and special habits ( $P = 0.26, 0.92$  and  $0.49$  respectively), and also between non-necrosis and necrosis groups ( $P = 0.22, 0.59$  and  $0.08$  respectively). Other investigators, found that, heart rate, WBC, rebound tenderness and constant pain correlated with strangulation and that leukocytosis and preoperative hospital stay  $>25$  hours was associated with intestinal necrosis (Otamiri *et al.*, 1987). But another study, concluded that, no preoperative clinical or laboratory parameters or combination of parameters was associated with strangulation (Sarr *et al.*, 1983). In our study, we reported that, there was significant increase regarding to time interval between onset of symptoms and arrival to emergency department, pulse rate, temperature and respiratory rate in ischemia group in comparison to non-ischemia group,  $P = <0.0001$  and also in necrosis group in comparison to non-necrosis group,  $P = <0.0001$ , but there was significant decrease in systolic and diastolic blood pressure in ischemia group in comparison to non-ischemia group,  $P = <0.0001$  and also in

necrosis group in comparison to non-necrosis group,  $P = 0.0002$  and  $<0.0001$  respectively. Constant pain and rebound tenderness were significantly increased in ischemia group in comparison to non-ischemia group,  $P = 0.001$  and  $0.002$  respectively and also in necrosis group in comparison to non-necrosis group,  $P = 0.02$  and  $0,004$  respectively, while colicky pain was significantly increased in non-ischemia group in comparison to ischemia group,  $P = 0.001$  and also in non-necrosis group in comparison to necrosis group,  $P = 0.02$ . Also WBC was significantly increased in ischemia and necrosis groups in comparison to non-ischemia and non-necrosis groups,  $P = <0.0001$ . Other studies, reported that, adhesions were the major cause of obstruction (65.7%), followed by cancer of large intestine (16.5%) and hernias (12.4%) (Markogiannakis *et al.*, 2011). While in our study, hernias were the commonest cause of acute intestinal obstruction (50%), followed by adhesions (28.57%), then intestinal masses (14.29%) and lastly sigmoid volvulus (7.14%). Markogiannakis *et al.*, found that, hernias were the most frequent cause of ischemia (51.4%). Then dhensions (20%) and large bowel cancer (17.2%) and the least common was the sigmoid volvulus (5.7%). Hernias were also the most common cause of necrosis (45.5%), other causes were adhesions (22.7%), large bowel cancer (13.6%) and sigmoid volvulus (9.1%) (Markogiannakis *et al.*, 2011). This concurs with our study, hernias were the commonest cause of intestinal ischemia (46.15%) and necrosis (50%), followed by adhesions (26.92% and 30%, respectively), intestinal masses (15.38% and 10% respectively) and sigmoid volvulus (11.54% and 10% respectively). The time interval between arrival and operation was lesser in ischemia than the non-ischemia groups ( $P = 0.0001$ ) (Bizer *et al.*, 1981).

This concurs with our results, that the time interval between arrival and operation was lesser in ischemia group than the non-ischemia group ( $P = <0.0001$ ) and also in necrosis than the non-necrosis groups ( $P = 0.0005$ ). Moore, reported that, metabolic acidosis is a common development in patients suffering from acute intestinal obstruction, this acidosis is marked after 24 hours of obstruction and becomes very marked if the obstruction is strangulated (Moore, 1959). This concurs with our study, there is significant increase in metabolic acidosis in ischemia and necrosis groups in comparison to non-ischemia and non-necrosis groups ( $P = 0.004$  and  $<0.0001$  respectively). Some studies, found that, the ischemia group had greater mortality, although not statistically significant, greater complication rate and longer ICU and hospital durations of stay than the non-ischemia group. Patients with necrosis also experienced increased complications, ICU admissions and mortality rate (Lo *et al.*, 2007). Also, other studies, reported that, strangulation is related with higher morbidity and mortality (Fevang *et al.*, 2000). In our research, we reported that, there was significant increase in the incidence of ICU admission and length of hospital stay in ischemia and necrosis groups in comparison to non-ischemia and non-necrosis groups ( $P = <0.0001, 0.0001$  and  $<0.0001, 0.0004$  respectively), also the incidence of complications were increased in ischemia than non-ischemia groups with insignificant deference, while there was significant increase in the incidence of fecal fistula, re-exploration and death in necrosis group in comparison to non-necrosis group ( $P = 0.01, 0.01$  and  $0.003$  respectively). Ayten *et al.*, observed that, PCT was normal in both a control and a simple small bowel obstruction groups, PCT elevation was detected after 30 minutes in the strangulated group and becomes significant at

120 minutes compared with the control and simple obstruction groups, suggesting that PCT may be a useful marker for the early detection of strangulation (Ayten *et al.*, 2005). In contrast, Papazogias *et al.* did not identify any no Table PCT elevation in strangulation groups in comparison to control and simple obstruction groups. In particular, PCT levels were normal in all groups, namely, control, simple small bowel obstruction, strangulated small bowel obstruction, simple large bowel obstruction and strangulated large bowel obstruction groups, with no elevation after 30, 90, 120 or 240 minutes (Papazogias *et al.*, 2008). In our study, we observed that there was insignificant difference between control group and non-ischemia group (simple bowel obstruction) regarding to PCT levels (0.33 and 0.47 respectively,  $P = 0.15$ ).

Other studies, reported that, serum procalcitonin levels on presentation at the emergency department were markedly greater in ischemia than non-ischemia groups (9.62 vs 0.30 ng/ml;  $P = 0.0001$ ) and in necrosis than non-necrosis groups (14.53 vs 0.32 ng/ml;  $P = 0.0001$ ). Multivariate analysis identified PCT as an independent predictor of ischemia ( $P = 0.009$ , OR 2.252, 95% CI 1.225-4.140) and necrosis ( $P = 0.005$ , OR 2.762, 95% CI 1.356-5.627). Using ROC curve analysis, the AUC of PCT for ischemia and necrosis were 0.77 and 0.87 respectively. A high NPV for ischemia and necrosis of PCT levels <0.25 ng/ml (83% and 95% respectively) and a PPV of PCT >1 ng/ml were identified (95% and 90% respectively) (Markogiannakis *et al.*, 2011). This concurs with our results, we found that, serum PCT levels on presentation at the emergency department were markedly greater in the ischemia than the non-ischemia groups (4.40 vs 0.47 ng/ml;  $P = <0.0001$ ) and in the necrosis than the non-necrosis groups (9.46 vs 0.88 ng/ml;  $P = <0.0001$ ). Multivariate analysis identified PCT as an independent predictor of ischemia ( $P = 0.001$ , OR 7.17, 95% CI 2.31-22.21) and necrosis ( $P = 0.002$ , OR 1.71, 95% CI 1.21-2.42). Using ROC curve analysis, the AUC of PCT for ischemia and necrosis was 0.85 and 0.81 respectively. A high NPV for ischemia and necrosis of PCT levels <0.36 ng/ml (80% and 90% respectively) and a PPV of PCT >1.77 ng/ml were identified (92% and 70% respectively).

Acute intestinal obstruction cause injury of intestinal mucosa leading to intestinal barrier dysfunction and increase of gut permeability (Van Leeuwen *et al.*, 1994). So, translocation of intestinal bacteria and their endotoxins occurs to extra-intestinal sites that may induce inflammation mediators and acute reaction and which triggering host immune inflammatory defense mechanisms and SIRS (Akçay *et al.*, 1996). Direct correlation between intestinal ischemia and permeability with translocation and acute inflammatory response also has been demonstrated (Meddah *et al.*, 2001). PCT early and closely correlates with inflammatory host response, intestinal barrier dysfunction increased intestinal permeability and endotoxemia (Ammori *et al.*, 2003). Dandona *et al.* observed that, when healthy volunteer were injected with *Escherichia coli* endotoxin, PCT was detected at 4 hours, peaked at 6 hours and remained stable between 8 and 24 hours. Thus our results may be explained by the fact that translocation of endotoxins and bacteria along with the inflammatory host response, which are more prominent in strangulation obstruction, can cause an increase in PCT. This findings imply PCT is an early strangulation marker in the clinical setting because the median period between symptoms onset and presentation is approximately 24 hours (Dandona *et al.*, 1994). In our study, the median period between Onset of symptoms and

presentation at emergency department were 13.5, 19.5, 17.5 and 23.5 hours in non-ischemia, ischemia, non-necrosis and necrosis groups, respectively.

## Conclusion

From the previous results, we concluded that, serum PCT values on presentation at emergency department for detection of ischemia and necrosis in patients suffering from acute intestinal obstruction were an independent predictor of both ischemia and necrosis. Thus, PCT may be very useful for early diagnosis or exclusion of intestinal ischemia and necrosis. So, serum PCT could serve as an additional diagnostic tool to improve clinical decision-making.

## Recommendation

Further researches and studies are needed to evaluate PCT levels on presentation at emergency departments as an accurate and early predictor of ischemia and necrosis in patients suffering from acute intestinal obstruction. Also PCT analysis should be done routinely for patients suffering from acute intestinal obstruction.

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Nil.

## Conflicts of Interest

There are no conflicts of interest.

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