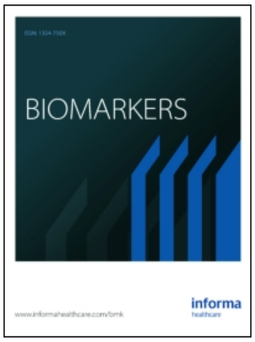

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Can serum resistin predict severity of acute pancreatitis?

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ABSTRACT

Objective: Acute pancreatitis (AP) is a common disorder with high mortality in severe cases. Several markers have been studied to predict development of severe AP (SAP) including serum resistin with conflicting results. This study aimed at assessing the role of baseline serum resistin levels in predicting the SAP.

Methods: This prospective study collected data from 130 AP patients from July 2017 to Nov 2018. Parameters measured included the demographic profile, serum resistin at admission, severity scores, hospital stay, surgery, and mortality. Patients were divided into two groups, severe and non-severe AP. Two groups were compared for baseline characteristics, serum resistin levels, hospital stay, surgery and mortality.

Results: Among 130 patients, 53 patients had SAP. SAP patients had higher BMI, baselines CRP, APACHE II and CTSI scores (p value 0.045, <0.001, <0.001 and 0.001, respectively). Both groups had comparable serum resistin levels. Serum resistin levels were also not different for obese and non-obese patients (p value 0.62). On multivariate analysis, BMI and high APACHE II score and CRP levels were found to independently predict SAP.

Conclusion: We found that serum resistin is not a useful marker for predicting severity of AP and does not correlate with increasing body weight.

Keywords: Acute Pancreatitis; SAP; serum resistin; obese; BMI

INTRODUCTION

Acute pancreatitis (AP), an acute inflammatory disorder of pancreas can present as either acute interstitial pancreatitis (AIP) or severe acute pancreatitis (SAP). Severe disease which occurs in around 10 % patients can have potentially lethal outcome. Thus, it is imperative to predict severe disease right at presentation. Multiple clinical, radiological and biochemical markers have been evaluated to predict severity at disease onset (1). As our understanding of underlying pathophysiological processes causing severe disease increases, various cytokines and inflammatory markers are being investigated for their role as a reliable biomarker.

Obesity is a chronic low-grade inflammatory state characterized by high levels of circulating proinflammatory cytokines (2). Obesity has been shown to be associated with an increased risk of AP development and often accompany severe pancreatitis (3–5). Pancreatic microcirculation is poorer in obese patients which increases the risk of ischemic injury and subsequent local infections (6). There are a number of gaps in our understanding of the pathophysiological changes which amplify the inflammatory response in obese patients with AP. Studies have shown that obese people have increased tumor necrosis factor-α (TNF-α) production, increased number of receptors (TNF-R1 and TNF-R2) as well as increased circulating levels of TNF-α (7,8). Thus, while we know that obese patients with AP have increased inflammatory markers, whether any of these can reliably predict disease severity in AP, is not clearly known.

Human resistin, a 12.5 kDA peptide, belongs to a family of biologically active resistin like molecules (RELMs). Resistin has been identified in pancreatic islets, the gastrointestinal tract, monocytes, adipocytes, spleen, white blood cells and plasma (8). Serum resistin has a regulatory role in a number of metabolic disorders associated with obesity like insulin resistance, diabetes mellitus (DM) and atherosclerotic heart disease (8). Serum resistin has been postulated to have a role as a prognostic biomarker predicting survival in critically ill patients suffering from diseases other than sepsis (9). Release of pancreatic lipases during an episode of AP leads to digestion of peri-pancreatic adipose tissue and causes accumulation of monocytes rich infiltrates and increase the release of resistin. Thus, resistin has been studied in AP for the detection of early inflammation of peripancreatic adipose tissue which is infiltrated by monocytes (10).

In patients with AP, use of serum resistin as a biomarker has shown conflicting results. Serum resistin was initially thought to be a promising biomarker, one which can predict severe acute pancreatitis (SAP) in the early phase (10). However other studies have reported contradictory findings. Thus the aim of this study was to assess the role of baseline serum resistin levels in predicting severity of AP patients

METHODS

Patients

This prospective observational study was carried out from July 2017 to Nov 2018 at a tertiary care center in North India. All patients admitted with a diagnosis of AP with age >12 years were enrolled after taking written informed consent and study approval from the institutes' ethical approval committee (INT/IEC/2017/1379). Exclusion criteria included patients of acute on chronic pancreatitis, those who had history of preexisting co morbidities like cardiopulmonary disease, hepatic disease, renal disease or severe immune system disorders, patients who had underlying pancreatic malignancy or tumor of any other organ and patients who had undergone prior endoscopic/ radiological or surgical intervention for AP.

Diagnosis of AP was made using revised Atlanta criteria, by the presence of at least 2 of the following 3 features: pain abdomen consistent with AP, raised serum lipase (or amylase) at least

3 times the upper limit of normal (ULN), and characteristic findings of AP on trans-abdominal USG, contrast enhanced CT abdomen or MRI abdomen. Severity classification of AP was done using revised Atlanta Classification (11).

Obesity was defined as per body mass index (BMI). Patients were categorized into normal, overweight and obese categories based on "Consensus statements for Asian Indians" (12). Studies have shown higher body fat, excess metabolic perturbation, and cardiovascular risk factors at lower BMI in Asian versus white population and a lower cut off for overweight and obesity has been suggested for Asian Indians (13). Patients with BMI < 18.5 kg/ m^2 were categorized as underweight. Those with BMI from 18.5 – 22.9 kg/m^2 were labeled normal. BMI ranging from 23.0 – 24.9 kg/m^2 were overweight and those with BMI > 25 kg/m^2 were classified as obese.

Other parameters included were: complete blood counts, liver function tests, renal function tests, fasting serum triglycerides, baseline serum C-reactive protein (CRP) levels and procalcitonin levels. Severity scores assessed were APACHE II, presence of SIRS at admission, organ failure criteria (according to modified Marshall scoring system for organ failure) and computed tomography severity index (CTSI). Serum resistin level was measured at the time of admission. Outcome was assessed in the form of hospital stay, ICU stay, mechanical ventilation requirement, requirement of surgery and mortality. Mortality was assessed for the period of hospitalization at out center.

Estimation of resistin levels

The blood sample for serum preparation was obtained according to standardized venipuncture procedure. Blood sample was mixed with EDTA citrate and then centrifuged for 30 minutes at a speed of 3000 rpm. Supernatant was collected and stored at – 80 ºC. The samples were analyzed using double antibody sandwich enzyme linked immunosorbent one step process assay (ELISA) to assay the level of resistin in the samples (QAYEE-BIO).

Treatment protocol

All the patients of AP underwent contrast enhanced computed tomography (CECT) abdomen on day 5-7 of pain onset or at admission (14). Antibiotics were used for extrapancreatic infections and suspected or confirmed infected pancreatic necrosis (suspected on clinical worsening, neutrophilia or elevation of procalcitonin). 'Step-up' approach was used for the management of peri/extra-pancreatic fluid collections (15). Image guided percutaneous drain (PCD) was inserted and upgraded or additional PCDs were placed when there was worsening of OF or sepsis, or an inadequate drainage despite significant residual collection. Endoscopic or surgical intervention was done when percutaneous intervention failed.

Statistical analysis:

All data were entered in Excel spread sheet and analysed using IBM SPSS version 23.0 (Chicago, IL, USA). Quantitative or numerical variable was represented with measures of central location like mean, median, measures of dispersion i.e. standard deviation, standard error. Normal distribution of data was checked with Shapiro-Wilk test. During analysis of data, continuous variables were compared using the Student t-test for normally distributed data while Mann-Whitney U test was used non-normal data. For comparison of more than two groups with continuous variable, ANOVA or Kruskal wallis test was used. Dichotomous variables (e.g. sex, mortality) were compared using Chi square test or fisher's exact test when appropriate. Multivariable logistic regression analysis was done for all the significant variables on univariate variables to find the association and predictors of severe disease. For continuous variables, cut offs was identified using the receiver operator curve (ROC) to optimize the multivariate logistic regression model. Results are presented as adjusted odds ratio (OR) with 95% confidence interval (CI). The p value of less than 0.05 was taken as statistically significant.

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RESULTS

Of the 215 patients of AP seen by us during the study period, 130 patients with first attack were included in the study (Figure 1). Excluded patients had prior intervention (n=43), recurrent pancreatitis (n=16), chronic pancreatitis (n=15) and severe comorbidities (n=14). The mean age of the patients was 39.16±13.25 years. Among 130 patients, 100 (76.9%) were males. Mean BMI at presentation was 22.78±3.91 kg/m2. Based on 'Consensus statement for Asian-Indians', 61 (46.9%) patients had normal weight, 14 (10.8%) were overweight and 44 (33.8%) were categorized as obese. Table 1 gives demographic details of study population.

Patients were divided into two groups based on severity of AP i.e. non-severe AP vs. severe AP and compared. Table 2 shows that BMI, baseline CTSI scores and APACHE II scores were significantly higher for the severe AP as compared to mild and moderately severe AP. The parameters were also compared among the mild, moderately severe and severe pancreatitis (Supplementary Table 1). Mean serum resistin value at admission in patients with severe AP was 1.24 ng/ml (±1.72 ng/ml) and among patients with mild/ moderately severe AP was 1.39 ng/ml (±2.45 ng/ml), p = 0.685 (Table 2). Serum resistin values was also not different for mild, moderately severe and severe AP (1.08±0.67 ng/ml, 1.52±2.89 ng/ml and 1.24±1.72 ng/ml respectively, p = 0.802) (Supplementary Table 1) (Figure 2). Serum resistin levels were also compared among obese versus non-obese, for all patients with AP and for those with severe AP, and were not statistically different (p value 0.62 and 0.4 respectively). Among the two common etiologies, patients who had AP related to intake of alcohol had higher baseline resistin levels than those who had gall stone related AP, though statistically non-significant (p = 0.19) (Table 4). Serum CRP was also measured at the time of presentation. Mean CRP levels were significantly higher in patients with SAP vs. those with mild/ moderately severe AP (217.31 mg/dL vs. 93.98 mg/dL respectively, p <0.001).

On univariate analysis, BMI, APACHE II score, CTSI score and CRP levels were found to be significantly higher in severe AP. Using the ROC, optimal cutoffs were identified for BMI, APACHE II score, CTSI score and CRP levels to predict the severe pancreatitis which were applied in logistic regression model. On multivariate logistic regression analysis, APACHE II score ≥8, BMI ≥21.5Kg/m^2 and CRP ≥64 mg/dL were found to predict the severe AP with increased chances of developing severe pancreatitis by 19.40, 4.95 and 4.35 times (Table 3).

Duration of hospital stay, ICU stay and mechanical ventilation were significantly higher in severe AP compared to mild/moderately severe AP (*p* value <0.001, <0.001 and 0.001, respectively). Overall mortality was 8.5% in the present study and it was significantly more in severe AP compared to mild/moderately severe AP, 16.9 % versus 2.59% respectively (p = 0.009) (Table 2).

DISCUSSION

We evaluated 130 consecutive patients of acute pancreatitis for their disease course with 44 (33.8%) patients obese and 14 (10.7%) patients were overweight based on BMI, as per the 'Asian-Indian consensus for obesity'. Our results showed that high BMI, APACHE II and serum CRP predicted the development of severe AP, but baseline serum resistin levels failed to serve as a predictive marker of severity of AP.

The role of resistin in obesity has been found to be variable across studies. Initial studies had shown higher serum resistin levels and gene expression in abdominal depots in obese patients compared to lean patients. The studies showed a positive correlation between serum resistin levels and change in BMI and visceral fat area (16–19). A significant reduction in circulating serum levels was also shown with weight loss and post gastric bypass (19,20). Contrary to these initial studies suggesting a role of resistin in obesity, recent studies have shown no correlation of serum or plasma levels of resistin with any marker of adiposity (21). Heilbronn et al. found no correlation between serum resistin levels and percentage body fat, visceral adiposity and BMI (22). Mabrouk et al. in an Egyptian study found a positive correlation of serum resistin levels with insulin, HOMA-IR and hsCRP only in obese diabetics and not in obese non-diabetic patients (23). Parreno-Caparros et al. found no correlation between obesity and resistin levels with levels being no different among obese and lean patients (24). Thus also found no change in serum resistin levels with weight reduction after gastric bypass in obese patients (24). Recent studies suggest a correlation between resistin with insulin resistance in obese diabetes patients, but no correlation with obese non-diabetes (23,25). In our study, we also found no difference in the mean resistin levels in obese patients compared to non-obese patients (1.20±1.55 ng/ml vs. 1.39±2.4 ng/ml respectively, $p = 0.62$).

Only a limited numbers of studies have evaluated the role of serum resistin in AP and have shown conflicting results (10,26–29). A proof of concept study in murine model of AP showed serum resistin having a positive correlation with occurrence and development of AP (30). Early human studies also showed elevated levels of serum resistin in AP patients compared to control group (10,28), and serum resistin was thought to be a promising biomarker for predicting disease severity early in patients of AP. Schaffler et al. showed that serum resistin, at admission, predicted development of peripancreatic necrosis and severe AP better than serum CRP (10). A subsequent study by Kibar et al. also showed serum resistin levels, at 72 hours, could predict development of necrotizing pancreatitis better than CRP (29). Yu et al. compared serum resistin and leptin levels at admission in AP and found only resistin levels to be significantly elevated in patients with severe AP and comparable to APACHE II in predicting severe AP, and not leptins (26). These initial studies provided evidence for serum resistin as a clinical predictor of severity. However, all these studies had included small number of patients with the maximum of 90 patients.

However other studies showed conflicting results with no correlation of serum resistin with severity of pancreatitis (27,28). Al-Maramhy et al. studied 102 patients of gall stone induced AP and found no difference in serum resistin levels between mild and severe AP. No correlation of serum resistin levels was found with BMI and weight (27). Similarly, our study also showed no difference in serum resistin levels between severe and non-severe AP, irrespective of the presence of obesity or etiology of AP. We also noted BMI and APACHE II to be significantly higher in severe AP and predictors for the development of severe AP.

These conflicting results in literature could be attributed to various reasons including differences in age groups studied, number of patients with severe AP, ethnicity, day of assessment of serum resistin levels or different methods used for serum resistin assessment. The study population was relatively younger (mean age <50 years) in the study (27) showing no correlation between serum resistin and severity compared to studies with positive correlation (mean age >50 years) (10,29). All studies had included patients irrespective of etiology of AP except the study by Al-Maramhy et al. where only gallstone induced AP was studied. It showed no correlation between obesity and serum resistin (27). Severe AP contributed 1/4th to 1/3rd of the study patients in all the studies but comparison arms differed in different studies, with comparison between mild and severe (27) and between severe and non-severe AP in other studies (26,29). Timing of measurement of resistin levels has also varied in different studies, with some researchers having measured it daily till day 7 (10), some on day 1, 3 and 7 of admission (29) while others estimated it on the day of hospitalization only (26,27). Regarding the definition of obesity, among the 3 studies with a positive correlation of serum resistin levels (10,26,29), 2 studies had included only overweight or obese patients (10,26) and one study included patients with normal BMI (20-25 Kg/m^2) (29), while in studies showing no correlation patients were included irrespective of BMI class (27,28), as in our study. However, we categorise the obese patient based on lower BMI as suggested by Asian Indian Consensus because in for Indian population studies have shown higher body fat, excess metabolic perturbation, and cardiovascular risk factors at lower BMI compared to white population (13).

Our study is the first from the South East Asia based on Asian-Indian consensus for obesity in AP and showed no correlation between serum resistin levels and severity of disease. Our study had a significant number of patients with adequate contribution from different etiologies, and enough patients of different severity grades. There are a few limitations, though. First, we did not include controls since patients with different severity served as controls for each other. All the published studies except one (27) had also studies only different severity grades. Second, we did not have data to quantify pancreatic and extrapancreatic necrosis to be correlated with serum resistin that could have added more value to the study. Thirdly, serum resistin levels were measured only once at the time of admission in our study. Serial measurement of resistin levels with change in the levels could have provided some useful results and should be evaluated further in the future studies.

The study concludes that serum resistin is not a useful marker of predicting severity of AP. It does not correlate with increasing body weight and while obesity is associated with increased severity of AP.

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Table 1: Baselines characteristics of the patients, n=130

Parameter		**Results**
Age (mean±SD) years		39.16±13.25
Gender	Male	100 (76.9%)
Etiology of pancreatitis	Alcohol	75 (57.7%)
	Gallstone	39 (30%)
	Others	16 (12.3%)
BMI (Kg/m^2, mean±SD)		22.78±3.91
Underweight*		11 (8.5%)
Normal*		61 (46.9%)
Overweight*		14 (10.8%)
Obese*		44 (33.8%)
Waist circumference (cm, mean±SD)		89.24±7.88
Severity of AP	Mild	23 (17.7%)
	Moderately severe	54 (41.5%)
	Severe	53 (40.8%)
SIRS		82 (63.1%)
BISAP	<2	75 (57.7%)
	≥2	55 (42.3%)
APACHE II score (mean±SD)		7.6±3.86
CTSI (mean±SD)		7.35±2.61
CRP (mg/dL, mean±SD)		150.37±187.5
Organ Failure	No OF	75 (57.7%)
	Single OF	42 (32.3%)
	Multiple OF	13 (10%)
	ALI	49 (37.7%)
	AKI	13 (10%)
	CVS	9 (8%)

*: as per the Asian guidelines(12)

ALI: Acute lung injury, AKI: Acute kidney injury, AP: Acute Pancreatitis, APACHE II: Acute physiology and chronic health evaluation, CRP: C-reactive protein, BMI: Body mass index, CRP: C-reactive protein, CVSF: cardiovascular system failure, OF: organ failure, SIRS: systemic inflammatory response syndrome

Table 2: Comparison between patients in the Mild/Moderately Severe AP and Severe AP

Parameters	**Mild+Moderately severe AP (77)**	**Severe AP (53)**	**P value**
Serum resistin (ng/ml)	1.39±2.45	1.24±1.72	0.685
Body mass index	22.19±3.73	23.83±4.03	0.045*
APACHE II	5.52±2.16	10.62±3.79	<0.001*
CTSI[#]	6.61±2.82	8.40±1.88	0.001*
CRP (mg/dl, median)[$]	93.98±96.27	217.31± 246.37	<0.001*
Total duration of hospitalization (days, median)	7.44±10.52	25.70±19.76	<0.001*
Duration of ICU stay (days, median)	0.62±2.23	7.49±11.62	<0.001*
Duration of mechanical ventilation (days, median)	0.13±0.73	2.98±9.50	0.001*
Surgery	3 (3.89%)	4 (7.54%)	0.648
Mortality	2 (2.59%)	9 (16.9%)	0.009*

*: significant value, APACHE II: Acute physiology and chronic health evaluation, CRP: C-reactive protein, CTSI: CT severity index, ICU: Intensive care unit

[#]<10%missing data

[$]<15% missing data

Table 3: Multivariate analysis[@] for severe AP

Parameters	**p value**	**Adjusted OR**	**95% CI for Odds ratio**
BMI ≥21.5Kg/m^2	0.005*	4.956	1.605-15.303
Serum C-reactive protein ≥64mg/dL	0.009*	4.352	1.445-13.107
APACHE II ≥8	<0.001*	19.409	6.307-59.729
Constant	<0.001	0.025	

*: significant value, APACHE II: Acute physiology and chronic health evaluation, BMI: Body mass index
[@]Multivariate model of logistic regression was built on 112 cases

Table 4: Comparison between patients of alcohol versus gallstone induced acute pancreatitis

Parameters		**Alcohol induced AP (75)**	**GSD induced AP (39)**	**P value**
Male		74 (98.7%)	17 (43.6%)	<0.001*
Serum Resistin	Mean	1.64±2.77	0.90±0.16	0.19
	Median	0.88 (0.6-20)	0.84 (0.5-12.6)	
Severity of AP	Severe	33 (44%)	15 (38.5%)	0.57
	Mild/Moderately Severe	42 (56%)	24 (61.5%)	
Body mass index (BMI)		22.53±3.4	23.13±4.03	0.408
APACHE II		7.83±3.9	6.82±2.9	0.123
CTSI		7.79±2.42	6.61±2.79	0.025*
CRP (mg/dl, median)		177±227.28	100.9± 86.86	0.015*
Organ Failure	OF	33 (44%)	15 (38.5%)	0.57
	ALI	30 (40%)	14 (35.9%)	0.725
	AKI	12 (16%)	1 (2.6%)	0.032*
	CVSF	5 (6.7%)	2 (5.1%)	0.745
Total duration of hospitalization (days, median)		15.81±18.73	12.23±13.31	0.241
Duration of ICU stay (days, median)		3.44±6.82	2.72±6.38	0.585
Duration of mechanical ventilation (days, median)		1.15±3.68	0.36±1.70	0.122
Surgery		2 (2.7%)	4 (10.3%)	0.092
Mortality		7 (9.5%)	3 (7.7%)	0.73

*: significant value, ALI: Acute lung injury, AKI: Acute kidney injury, AP: Acute pancreatitis, APACHE II: Acute physiology and chronic health evaluation, BMI: Body mass index, CRP: C-reactive protein, CTSI: CT severity index, CVSF: cardiovascular system failure, OF: Organ failure, ICU: Intensive care unit

LEGENDS TO FIGURE:

Figure 1: Flow diagram showing study design and inclusion of patients

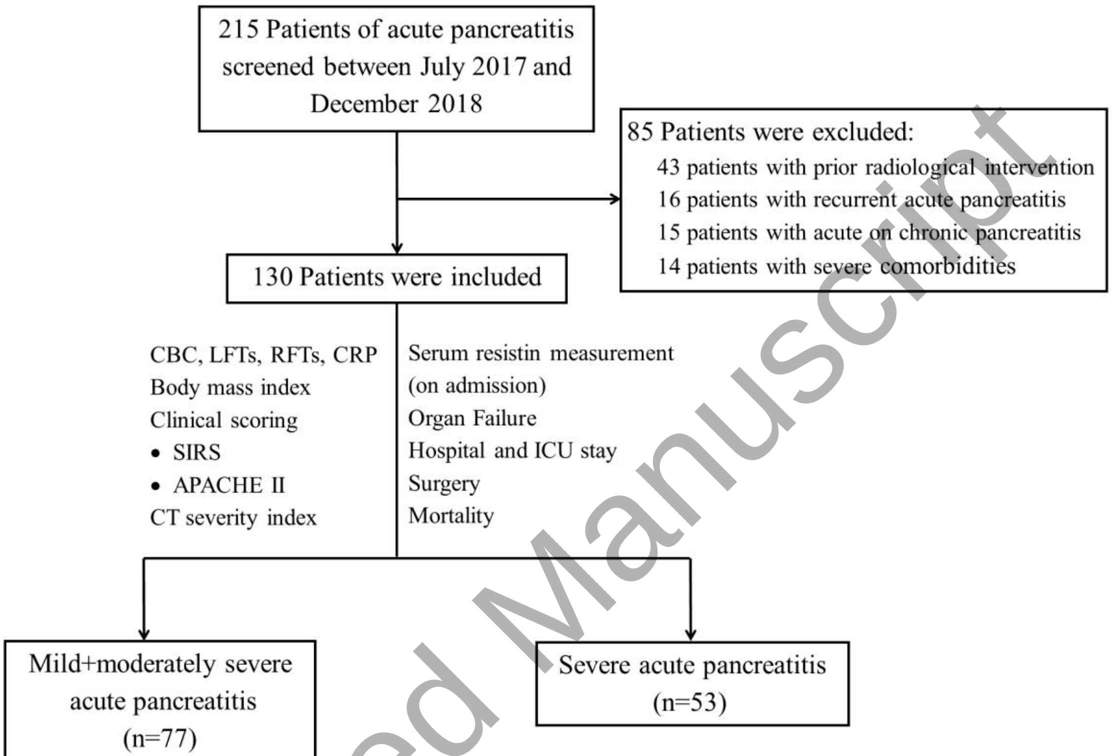
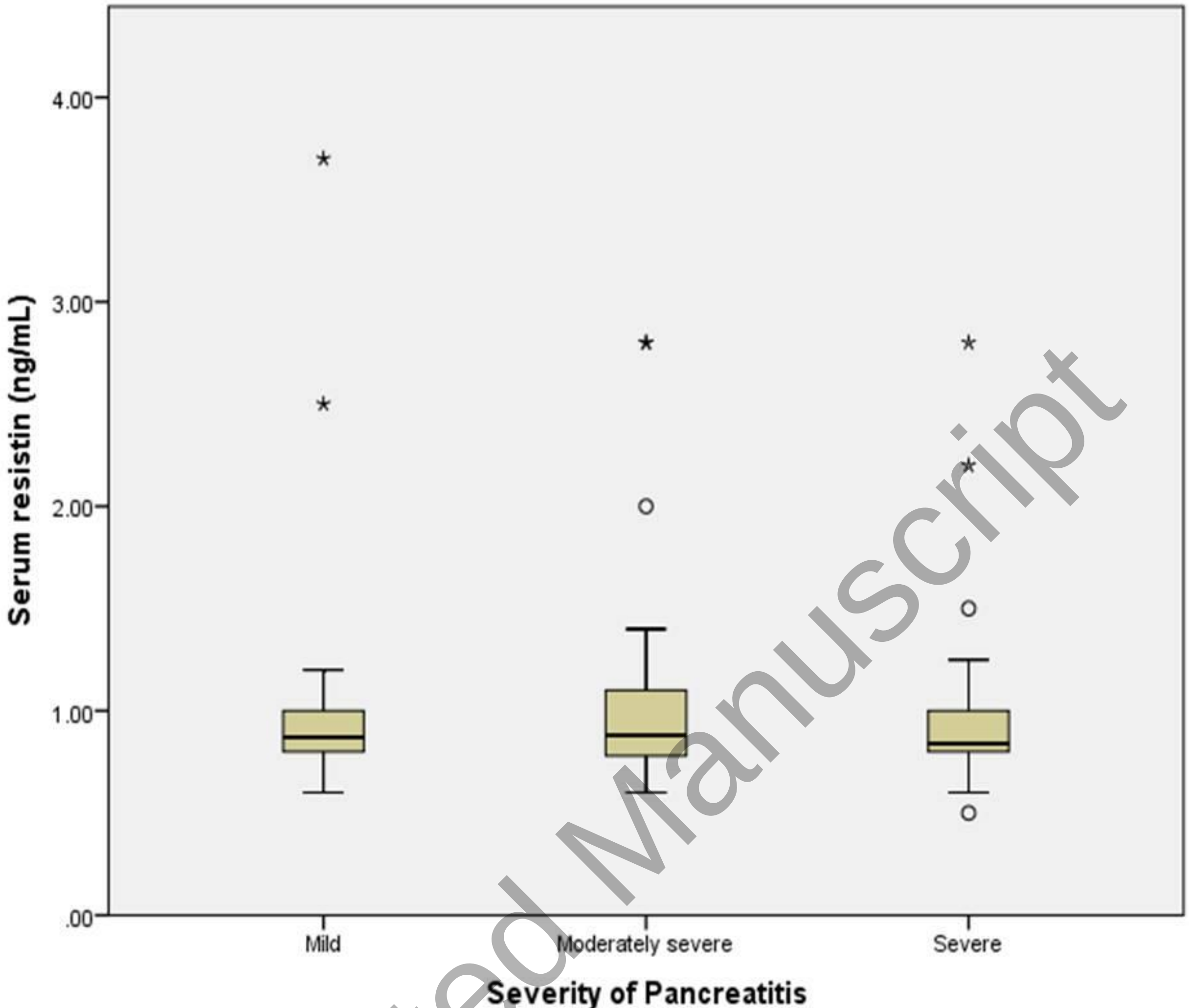


Figure 2: Box plot showing the baseline mean resistin levels in patients with mild, moderately severe and severe pancreatitis.


Serum resistin (ng/mL)
4.00
3.00
2.00
1.00
.00
Mild
Moderately severe
Severe
Severity of Pancreatitis