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Original Research Article

CLINICAL UTILITY OF DELTA NEUTROPHIL INDEX AS A SEVERITY AND PREDICTION MARKER IN PATIENTS WITH ACUTE PANCREATITIS

Sirisha Peddi[1], K N Sree Sai Gayathri[2], Vijay Kumar Aitipamula[2], D Keerthana[3]

[1]*Associate Professor, Department of General Medicine: Gandhi Medical College, Secunderabad, Telangana, India.*
[2]*Associate Professor, Department of General Medicine: Government Medical College Vikarabad, Telangana, India.*
[3]*Junior Resident in DM Endocrinology, Department of General Medicine, Sri Venkateshwara institute of Medical sciences, Tirupati, Andhra Pradesh, India.*

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Corresponding Author:
Dr. D Keerthana,
Junior Resident in DM Endocrinology, Department of General Medicine, Sri Venkateshwara institute of Medical sciences, Tirupati, Andhra Pradesh, India.
Email: drkeerthana2134@gmail.com

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ABSTRACT

Background: The aim is to establish the diagnostic usefulness of Delta Neutrophil Index as an early marker of disease severity in patients with acute pancreatitis.

Materials and Methods: Single centre, non-interventional, prospective observational study in department of medicine for a period of 2 years in acute pancreatitis cases admitted diagnosed through clinical, laboratory and radiological parameters.

Results: Mean heart rate was lesser in mild cases (79.20±9.7 bpm) compared to total population (85.05±12.1 bpm). This suggests that heart rate increases with severity of acute pancreatitis. Delta neutrophil index (DNI) had strong positive correlation with CRP, ANC, ATLANTA and BISAP scores , stronger correlation of DNI with severity was seen in mild cases compared to moderate-severe cases.

Conclusion: DNI measured at presentation in emergency has the potential to function as an adjunctive marker for prediction of severity of acute pancreatitis.

Keywords: Delta neutrophil index, Absolute neutrophil count , High sensitivity C reactive protein.

INTRODUCTION

Acute pancreatitis is a severe disease with high mortality and uncertain prognosis. It usually presents in a mild form and recovery is mostly uneventful. However in a few patients, it progresses to a severe form which can be life threatening. Therefore an initial indicator of disease severity is important to predict the clinical course of acute pancreatitis because recognizing severe acute pancreatitis in early clinical phase can lower mortality by applying proper critical care. The incidence of acute pancreatitis (AP) has been increasing, and the primary problem in affected patients is inflammation, which results in a variety of clinical outcomes. Acute pancreatitis affects around 114-200/100,000 population in India and its incidence is higher in south India. AP usually presents in a mild form, and most patients have an uneventful recovery without organ dysfunction. However, about 10–20% of patients with AP progress to a severe form, which can be life-threatening. AP patients may deteriorate and ultimately require critical care in the intensive care unit (ICU) within the first 24–48 hours of hospitalization. Therefore, an initial indicator of disease severity is important to predict the clinical course of AP, because recognizing severe AP (SAP) in the early clinical phase can lower mortality by adequately applying proper critical care.[1,2]

There are several scoring systems to screen patients at risk for severe acute pancreatitis (SAP). The Atlanta classification, a global representative classification of AP, was revised in 2012 based on an international consensus. C-reactive protein (CRP), procalcitonin and interleukin-6 have been studied as possible biomarkers to predict the severity of AP at the initial clinical phase. However, simple and accurate biomarkers for early recognition of SAP are still needed. A convenient and cost-effective serum biomarker that predicts disease severity within the initial 24 hours of symptom onset is desirable.[3]

Delta neutrophil index is a convenient and cost effective serum biomarker which predicts the severity of the disease. Delta neutrophil index correlates with the immature granulocytes circulating in the blood. Delta neutrophil index is the leucocyte differentials measured in the Myeloperoxidase channel and those measured in the nuclear lobularity channel. Changes in immature granulocyte cells are seen earlier than absolute leucocyte counts, hence Delta neutrophil index can contribute to predicting the development of severe acute pancreatitis. The delta neutrophil index (DNI) identifies the difference in leukocyte subfractions through a cytochemical myeloperoxidase reaction and a nuclear lobularity assay through blood cell analysis. DNI strongly correlates with manual immature granulocyte count. The DNI can represent changes in absolute white blood cell (WBC) or neutrophil count because the course of granular leukocyte differentiation in inflammatory and infectious conditions starts with formation of immature granulocytes. Therefore, DNI has been studied by several research groups as a serum biomarker in infectious and inflammatory conditions. In addition, DNI may serve as an efficient test to represent inflammation and infection in patients in the emergency department (ED) because DNI can be included with routine complete blood counts.[4]

No past studies have assessed the clinical efficacy of DNI as an initial biomarker for severity of acute pancreatitis in the Emergency setting. Therefore, this thesis studied the clinical usefulness of DNI as an early indicator for estimating severity of AP.

MATERIALS AND METHODS

Type of study: single centre, non-interventional, prospective observational study.

Site of study: Department of medicine, Gandhi hospital, secunderabad.

Duration of study: November 2020 to June 2022

Ethical clearance: prior approval was obtained from institutional ethics committee of Gandhi medical college, secunderabad.

Informed consent: all participants were explained about purpose and procedure of study in their understandable language. Patients had willfully agreed and signed on inform consent document.

Study subjects: acute pancreatitis cases admitted in Gandhi hospital were selected based on following criteria:

Inclusion Criteria

- Age > 18 years
- Acute pancreatitis diagnosed through clinical, laboratory and radiological parameters

Exclusion Criteria

- Acute pancreatitis caused by malignancies.
- Patients with haematological abnormalities, malignancies, or concomitant infections.
- Patients who received Granulocyte colony stimulating factor, glucocorticoids and other immunosuppressive agents.
- Patients with neutropenic fever following chemotherapy.
- Post endoscopic retrograde cholangio pancreatography pancreatitis.
- Acute exacerbation of chronic pancreatitis.

Study Procedure: After recruitment into study, every patient was asked for detailed history, examined clinically and investigated for hemoglobin, total and differential WBC count, C reactive protein, liver function tests with serum albumin, renal function tests, serum electrolytes, serum amylase, serum lipase, blood urea nitrogen, serum creatinine, arterial blood gas analysis. Radiographic tests were done when indicated. Delta neutrophil index will be determined in these patients using a specific blood cell analyzer. Revised 2012 Atlanta score, Bed side index of severity in acute pancreatitis (BISAP) score were used to classify the severity of acute pancreatitis. History, clinical examination details and investigational results from each patient were recorded in pre designed patient information sheet.

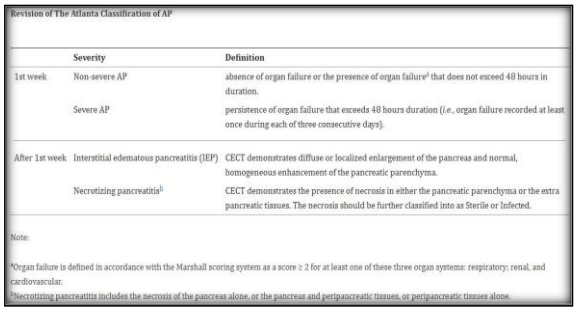
Statistical analysis: Data was tabulated in excel spreadsheet. For every parameter mean and SD were calculated for total population and mild pancreatitis group. Correlations were checked using Pearson's correlation. Statistical significance was set at $p < 0.05$. SPSS was used for statistical analysis.

Atlanta Classification: The Atlanta Classification is a clinically based classification system resulting from an international meeting, the 1992 International Symposium on Acute Pancreatitis. Briefly, the Atlanta Classification categorizes acute pancreatitis (AP) as "mild" to "severe." The latter is distinguished by organ failure and/or local complications. The Atlanta symposium attempted to offer a global "consensus" and a universally applicable classification system for AP. The definitions of AP, its severity, and organ failure and local complications in the Atlanta Classification are widely accepted and used by physicians and radiologists, representing an important step forward in the classification of AP. Although the Atlanta Classification has proved useful in the years since 1992, many of its definitions proved confusing and have not been accepted or utilized by the pancreatic community. Increased knowledge of the pathophysiology of necrotizing pancreatitis, improved imaging of the pancreatic parenchyma and peri-pancreatic collections, and the development of new interventions to manage complications, such as minimally invasive radiologic, endoscopic, and laparoscopic procedures have resulted in several studies identifying shortcomings in the Atlanta Classification. The limitations of Atlanta Classification can be summarized as follows:

- Patients identified as having "severe AP" consist of subgroups with very different outcomes,
- Forms of AP with higher risks of mortality, such as necrotizing pancreatitis (sterile or infected?

pancreatic or peripancreatic?), were inadequately described or categorized,

- And organ failure was not adequately categorized (transient or persistent?).
- In order to establish a more accurate classification system, the Acute Pancreatitis Classification Working Group revised the Atlanta Classification in 2008.



Revision of The Atlanta Classification of AP

	Severity	Definition
1st week	Non-severe AP	absence of organ failure or the presence of organ failure[a] that does not exceed 48 hours in duration.
	Severe AP	persistence of organ failure that exceeds 48 hours duration (*i.e.*, organ failure recorded at least once during each of three consecutive days).
After 1st week	Interstitial edematous pancreatitis (IEP)	CECT demonstrates diffuse or localized enlargement of the pancreas and normal, homogeneous enhancement of the pancreatic parenchyma.
	Necrotizing pancreatitis[b]	CECT demonstrates the presence of necrosis in either the pancreatic parenchyma or the extra pancreatic tissues. The necrosis should be further classified into as Sterile or Infected.

Note:

[a]Organ failure is defined in accordance with the Marshall scoring system as a score ≥ 2 for at least one of these three organ systems: respiratory; renal, and cardiovascular.

[b]Necrotizing pancreatitis includes the necrosis of the pancreas alone, or the pancreas and peripancreatic tissues, or peripancreatic tissues alone.

An obvious feature of the revised classification is that AP is classified into two phases: an early phase (usually within the first week of onset) and a subsequent phase occurring after the first week of onset of the disease.

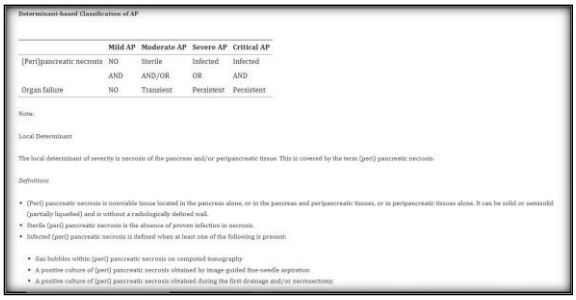
These two phases have a distinct pathophysiology. Because the first phase is characterized more by the presence or absence of organ failure and less by morphologic findings in and around the pancreas, AP should be classified as being in the first phase based on "functional" or "clinical" parameters.

In the second phase, the need for treatment is determined by the presence of symptoms and/or complications. Therefore, "morphologic" criteria should be used to classify AP in the second stage because morphologic criteria can be used to guide treatment. Briefly, the clinical classification is used during the early phase of disease (within the first week of onset) while the morphologic classification is used during the subsequent phase (usually after the first week after onset).

Several comprehensive reviews of the available evidence have noted several flaws with this revised classification:

a. "mild" and "severe" are not sufficient to categorize the severity of AP and cannot differentiate between subgroups with different outcomes
b. the classification of severity should be based on key factors that are causally associated with severity, rather than on descriptions of events that may correlate with severity but are not causally associated with it
c. There are insufficient grounds for ending the first phase 1 week after onset of symptoms. Further, clinical events can occur in individual patients in any order on any day, so severity should be categorized based on key events when they occur and without regard to the sequence they occur in.

Given the aforementioned flaws of the Atlanta Classification, a determinant-based classification of AP severity was developed in 2012.[5]



Determinant-based Classification of AP

	Mild AP	Moderate AP	Severe AP	Critical AP
(Peri)pancreatic necrosis	NO	Sterile	Infected	Infected
	AND	AND/OR	OR	AND
Organ failure	NO	Transient	Persistent	Persistent

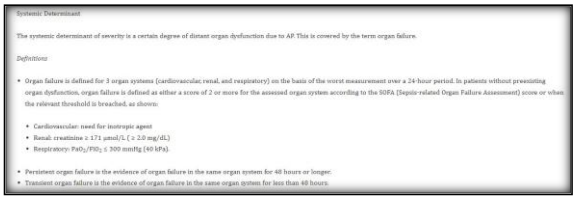
Note:

Local Determinant

The local determinant of severity is necrosis of the pancreas and/or peripancreatic tissue. This is covered by the term (peri) pancreatic necrosis.

Definitions

- (Peri) pancreatic necrosis is nonviable tissue located in the pancreas alone, or in the pancreas and peripancreatic tissues, or in peripancreatic tissues alone. It can be solid or semisolid (partially liquefied) and is without a radiologically defined wall.
- Sterile (peri) pancreatic necrosis is the absence of proven infection in necrosis.
- Infected (peri) pancreatic necrosis is defined when at least one of the following is present:
  - Gas bubbles within (peri) pancreatic necrosis on computed tomography
  - A positive culture of (peri) pancreatic necrosis obtained by image-guided fine-needle aspiration
  - A positive culture of (peri) pancreatic necrosis obtained during the first drainage and/or necrosectomy.



Systemic Determinant

The systemic determinant of severity is a certain degree of distant organ dysfunction due to AP. This is covered by the term organ failure.

Definitions

- Organ failure is defined for 3 organ systems (cardiovascular, renal, and respiratory) on the basis of the worst measurement over a 24-hour period. In patients without preexisting organ dysfunction, organ failure is defined as either a score of 2 or more for the assessed organ system according to the SOFA [Sepsis-related Organ Failure Assessment] score or when the relevant threshold is breached, as shown:
  - Cardiovascular: need for inotropic agent
  - Renal: creatinine ≥ 171 μmol/L (≥ 2.0 mg/dL)
  - Respiratory: $PaO_2/FiO_2 \leq 300$ mmHg (40 kPa).
- Persistent organ failure is the evidence of organ failure in the same organ system for 48 hours or longer.
- Transient organ failure is the evidence of organ failure in the same organ system for less than 48 hours.

Systematic reviews of the evidence and expert opinions have favored this classification over the revised Atlanta Classification. New data and international consultation may lead to a different answer in the future and necessitate further revisions, but the transition from a classification based on "clinical experience" to one based on "evidence-based determinants" is a step in the right direction.

Bedside Index of Severity in Acute Pancreatitis (BISAP)

A scoring system that can be used at the time of admission to predict severity is the Bedside Index of Severity in Acute Pancreatitis (BISAP). This scoring system specific to AP includes five variables: blood urea nitrogen (BUN) > 25, impaired mental status, age > 60 years, pleural effusion, and >= 2 SIRS criteria. The observed mortality rate of patients has been shown to increase with an increasing number of positive variables; the mortality in patients with 0 variables was about 0.20%, and the mortality of patients with all five variables was shown to be around 22–27%. The BISAP scoring system has been further validated in several prospective cohort studies and has proven useful in clinical settings, particularly for prediction of necrosis and mortality. In a comparative study of several scoring methods, including BISAP, Ranson, APACHE II, and CTSI, it was found that the BISAP scoring system was similar to the others in terms of risk stratification, as well as early identification of patients at risk for in-hospital mortality. To calculate BISAP, a history and physical examination, basic labs, and a chest x-ray study are needed, all of which are reasonably obtained in an ED work-up. Although BISAP does include a parameter for pleural effusion, it does not require evidence of inflammation or necrosis of the pancreas, so CT imaging is unnecessary.

Advantages of the BISAP score include its simplicity and its ability to be calculated during the patient's stay in the ED. A few clinical and laboratory values have been found to be very closely linked to in-hospital mortality in patients with AP. Of these predictive single variables, many are included in the BISAP. One study found that an elevation of BUN at

admission (>20 mg/dL) that did not experience a decline by at least 5 mg/d: within 24 h correlated with the highest mortality risk of 15–21%. This study suggests that increased BUN at admission, as well as an increase in BUN over 24 h, are risk factors for mortality secondary to AP and may be used as a sole predictor of severity. Pleural effusion and pulmonary infiltration within the first 24 h are also useful single variable predictors, and are appropriately included in the BISAP scoring system. Additional single variable predictors not included in the BISAP scoring system may aid the stratification of patients with AP presenting to the ED. C-reactive protein (CRP) has peak induction at 72 h and is therefore not helpful in the early presentation of AP, but may be useful in stratifying pancreatitis into mild and severe cases.

RESULTS

100 patients of acute pancreatitis were recruited in this study. Their mean age was 35.60±6.3 years. All patients were male. Among these 50 patients were having mild acute pancreatitis based on ATLANTA and BISAP scores; their mean age was similar to entire study population. BISAP SCORE of entire population ranged from 0 to 5 with mean score of 2.40±1.4; while that in mild cases was 1.20±0.8.

Table 1: Baseline demographics.

	Total study population (Mean±SD)	Only mild cases (Mean±SD)
N	100	50
Age	35.60±6.3 years	35.60±7.3 years
Males	100 (100%)	50 (100%)
Females	0	0
BISAP	2.40±1.4	1.20±0.8

On clinical examination, impaired mental status was seen in 30 patients, and all these had moderate to severe grade of acute pancreatitis. Mean heart rate was 85.05±12.1 bpm, mean body temperature was 37.51±0.9 0C, and mean respiratory rate was 19.35±2.7 bpm. Similar means were seen in mild cases excepting the heart rate which was lesser in mild pancreatitis (79.20±9.7 bpm).

Table 2: Clinical evaluation.

	Total study population (Mean±SD)	Only mild cases (Mean±SD)
Impaired Mental Status (N)	30	0
Heart Rate (Bpm)	85.05±12.1	79.20±9.7
Body Temperature (0C)	37.51±0.9	37.19±0.7
Respiratory Rate (Bpm)	19.35±2.7	19.30±3.0

Lab investigations revealed mean blood urea of 28.35±9.0 mg/dl, mean PaCO2 of 34.40±2.9 mm Hg, mean CRP of 3.93±2.6 mg/dl, mean total WBC count of 11614.50±3874.3 cells/mcl, mean absolute neutrophil count of 7812.80±3131.4 cells/mcl and mean DNI value of 3.63±2.8. Means of all these parameters were lesser in mild cases compared to total population.

Pleural effusion was identified clinically and confirmed radiologically in 60 (60%) cases among total study population; while among mild cases, 20 (40%) had pleural effusion. Other laboratory parameters like BUN, CRP and ANC were lesser in mild cases compared to total population

Table 3: Laboratory and radiological evaluation

	Total study population (Mean±SD)	Only mild cases (Mean±SD)
BUN (mg/dl)	28.35±9.0	22.30±9.3
PaCO2 (mm Hg)	34.40±2.9	34.50±2.8
CRP (mg/dl)	3.93±2.6	2.13±1.0
ANC (cells/mcl)	11614.50±3874.3	9908.00±2656.3
DNI	3.63±2.8	1.50±0.9
Pleura effusion n (%)	60 (60%)	20 (40%)

Correlations of DNI: Delta neutrophil index (DNI) had strong positive correlation with CRP and ANC with R2 values of 0.94 and these associations were statistically significant with $p < 0.05$. DNI levels proportionately increased with increasing ATLANTA and BISAP scores. Spearman's correlation revealed strong positive correlation with rho value of 0.93 and 0.84 and these associations were statistically significant with $p < 0.05$.

Table 4: Correlation of DNI with other parameters of pancreatitis in total population

Variable	DNI	ATLANTA	BISAP	CRP	ANC
DNI (Correlation)	1	0.93**	0.84**	0.94*	0.94*
Sig. (2-tailed)		<0.05	<0.05	<0.05	<0.05
N	100	100	100	100	100

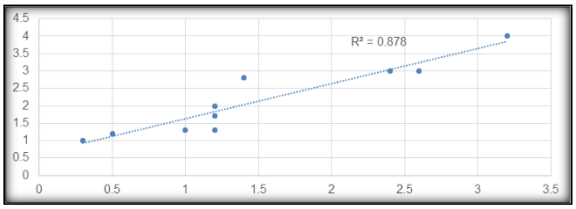


Figure 1: Pearson's correlation of DNI and CRP in all cases

Correlation of DNI with CRP and ANC was higher in mild acute pancreatitis cases with R2 value of 0.96 and these associations were statistically significant with $p < 0.05$. DNI levels proportionately increased with increasing BISAP score; Spearman's correlation revealed strong positive correlation with rho value of 0.88 and this associations were statistically significant with $p < 0.05$.

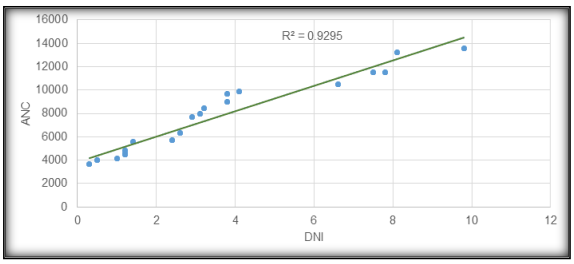


Figure 2: Pearson's correlation of DNI and ANC

Table 5: Correlation of DNI with other parameters of pancreatitis in mild cases

		BISAP	CRP	ANC
DNI	Correlation	0.88**	0.96**	0.96**
	Sig. (2-tailed)	<0.05	<0.05	<0.05
	N	50	50	50

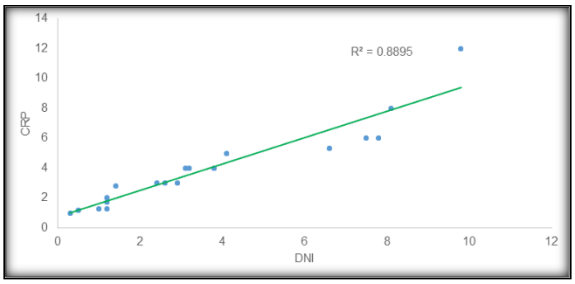


Figure 3: Pearson's correlation of DNI and CRP in mild cases

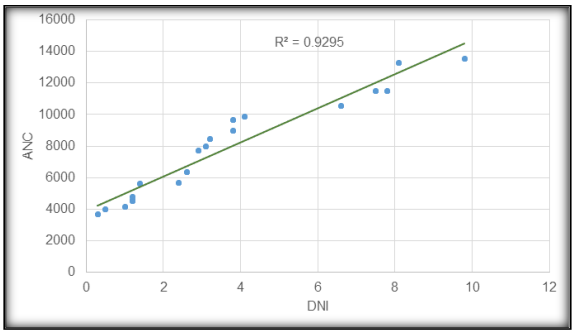


Figure 4: Pearson's correlation of DNI and ANC in mild cases

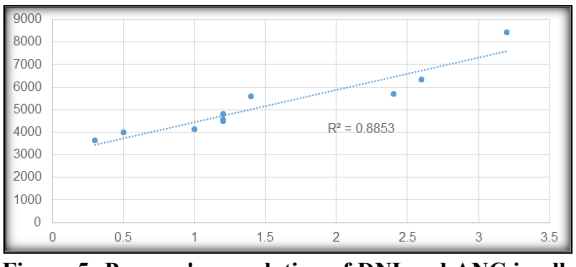


Figure 5: Pearson's correlation of DNI and ANC in all cases

DISCUSSION

This study was done to evaluate DNI as predictive marker for severity of acute pancreatitis. In this study, 100 acute pancreatitis cases were analyzed. Their mean age was 35.60±6.3 years. These results are similar to Weiss F.U. et al,[6] who reported peak incidence age of acute pancreatitis as between 35 and 44 years. All patients were male. Drake et al,[7] also reported that pancreatitis is more common in males.

ATLANTA and BISAP scores were used to grade the severity of pancreatitis. It was found that 50 of 100 patents had mild acute pancreatitis. Among the mild cases, their mean age was similar to that of total population, telling us that severity is not related to age. Kara et al,[8] also proved that any age group can be effected with mild, moderate or severe pancreatitis; age is not correlated to severity.

Mental impairment was not seen in mild acute pancreatitis but only in 30 of 50 moderate to severe acute pancreatitis cases. These results are similar to Kantly and Medikeri who reported zero cases with mental impairment in acute pancreatitis cases with low BISAP score.

Mean heart rate was lesser in mild cases (79.20±9.7 bpm) compared to total population (85.05±12.1 bpm). This suggests that heart rate increases with severity of acute pancreatitis. Takeshi Okamoto et al,[9] had demonstrated that mild acute pancreatitis patients had lower heart rate and shorter hospital stay, while those with higher heart rates had higher severity of acute pancreatitis and longer hospital stay.

Body temperature and respiratory rate were within normal limits for all cases and there was no difference between mild cases and total population. These results are similar to Bohidar et al,[1] and Zhang et al.[10]

Pleural effusion was identified clinically and confirmed radiologically in 60 (60%) cases among total study population; while among mild cases, 20 (40%) had pleural effusion. These results are similar to George W Browne and CS Pitchumoni.[11]

Other laboratory parameters like BUN, CRP and total ANC were lesser in mild cases compared to total population. Suhan Lin et al,[12] and Wu et al,[13] also

reported that BUN, CRP and ANC increases with severity and mild acute pancreatitis cases have lower BUN, CRP and ANC.

Delta neutrophil index (DNI) was lower (1.50±0.9) in mild cases compared to total population (3.63±2.8). Delta neutrophil index (DNI) had strong positive correlation with CRP, ANC, ATLANTA and BISAP scores. Kim et al,[14] also analyzed 209 acute pancreatitis cases; they reported higher DNI in severe cases and established positive correlation of DNI with Atlanta classification. In this study stronger correlation of DNI with severity was seen in mild cases compared to moderate-severe cases. Werner et al,[15] also established stronger correlation of DNI with severity in mild cases compared to moderate-severe cases.

There are multiple etiologies of the inflammation observed in Acute Pancreatitis. Activation of intra-acinar trypsinogen causes an acinar injury in the initial phase of Acute Pancreatitis. However, local and systemic inflammation occur independently in Acute Pancreatitis. In addition, the inflammatory response and inflammatory mediators have important pathophysiologic roles in the clinical course of Acute Pancreatitis. Autodigestion causes excessive transfer of leukocytes to the pancreas. The leukocyte's significant role is to release pro-inflammatory cytokines and oxygen-derived free radicals, which cause necrotic changes of the acinar. If these reactions continue, activated proteolytic enzymes and various inflammatory cytokines induce an acute respiratory failure, systemic inflammatory response syndrome, septic shock and multi-organ dysfunction. If the disease process resolves after the local inflammatory process, the disease will regress to mild AP. Thus, leukocytes are an important contributor to disease pathogenesis, progression and severity, and changes in DNI precede changes in absolute leukocyte count. Therefore, DNI is a useful biomarker to predict severity of acute pancreatitis in mild cases.

C-reactive protein (CRP) levels are well known to in response to injury, infection, and inflammation anywhere in the body. CRP (C-reactive protein) is mainly classified as an acute marker of inflammation (acute phase reactant), but main research is now starting to indicate its important roles that CRP plays in inflammation. CRP is the principal downstream mediator of the acute-phase response (APR) following an inflammatory event and is primarily manufactured by IL-6-dependent hepatic biosynthesis. CRP research has been looked mostly on the role of CRP and its subtypes on cardiovascular disease and stroke. CRP is utilized as a bio clinical marker of inflammation anywhere in the body, with the rise of serum levels being a strong independent predictor/marker of cardiovascular disease in asymptomatic individuals. CRP levels have been linked and is related to prognosis in patients with atherosclerotic disease, congestive heart failure (CHF), atrial fibrillation (AF), myocarditis, aortic valve disease (AVD), and heart transplantation, reflecting that it has an active role in the pathophysiology of cardiovascular disease (CVD). Higher levels of CRP have been seen in patients with acute appendicitis, acute cholecystitis, acute pancreatitis, and meningitis. In patients suffering possible symptoms of acute appendicitis, acute appendicitis can be ruled out in those with CRP (c reactive protein) levels lower than 25 mg/L, blood taken 12 hours after the onset of symptoms. In acute pancreatitis, CRP levels of above than 210 mg/L were able to differentiate between mild and severe cases, with 83% sensitivity and 85% specificity. Critical levels of CRP level is given as various in numerous studies be that as it may, CRP level >100 mg/dl as given in concentrate by research associates with values got in our concentrate as is taken as critical incentive for correlation. Distribution of etiology and gender in pancreatitis couldn't be contemplated as a large portion of the review bunch is comprised by young and middle ages.

The BISAP score is easy to use for judging severity of acute pancreatitis but it has the disadvantage of not distinguishing between patients with temporary organ dysfunction and those with long-term organ dysfunction, and it can overestimate severity. The Atlanta classification is laborious and requires clinicians to reassess many factors 48 hours after the initial assessment. Therefore, early prediction of patients with higher risk of developing severe acute pancreatitis is difficult with the conventional scoring systems.

However, DNI is easy to calculate in emergency since it can be simultaneously obtained with routine complete blood counts to evaluate inflammatory and infectious diseases. DNI is a single, cost-effective, accurate and fast serum biomarker that can be used in emergency to identify patients in the early phase of pancreatitis with a higher risk of developing severe acute pancreatitis.

This study has certain limitations. Firstly, because of the retrospective design and single-center study setup, some data, such as subjective symptoms and physical examinations, were missing and/or inaccurate. Additionally, a selection bias could have resulted from patient exclusion. To reduce possible bias, all acute pancreatitis patients were included who were admitted to hospital because the DNI was available for all these patients. Secondly, DNI values were not serially assessed after admission, so we were unable to assess the meaning of changes in DNI for predicting treatment response. Thirdly, Ranson's criteria was not used because it includes several factors that has to be measured, and severity cannot be determined until 48 hours after admission, limiting their use in emergency. Fourthly, We also did not calculate computed tomography severity index, which can be evaluated by Balthazar grade and presence of necrosis. However, this index focuses on local complications and does not represent a systemic inflammatory response.

Despite these limitations, this study has strengths. This is the first study done in India to evaluate the

predictive value of DNI in acute pancreatitis. Because DNI can be quickly and conveniently calculated, it can easily be used in an emergency setting. However, future prospective studies are needed to confirm the outcomes.

CONCLUSION

DNI measured at presentation in emergency has the potential to function as an adjunctive marker for prediction of severity of acute pancreatitis. Intensive care and careful management should be considered for acute pancreatitis patients with a DNI value greater than 1.8% upon presentation.

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