

# Haemoconcentration as a predictor of severity in acute pancreatitis

Bohara TP<sup>1</sup>, Karki D<sup>2</sup>, Parajuli A<sup>3</sup>, Rupakheti S<sup>4</sup>, Joshi MR<sup>5</sup>

<sup>1</sup>Tanka Prasad Bohara, Assistant Professor; <sup>2</sup>Dimindra Karki, Resident; <sup>3</sup>Anuj Parajuli, Lecturer; <sup>4</sup>Shail Rupakheti, Lecturer; <sup>5</sup>Mukund Raj Joshi, Associate Professor; Department of Surgery, Kathmandu Medical College Teaching Hospital, Kathmandu, Nepal

## Abstract

**Background:** Acute pancreatitis is usually a mild and self-limiting disease. About 25% of patients have severe episode with mortality up to 30%. Early identification of these patients has potential advantages of aggressive treatment at intensive care unit or transfer to higher centre. Several scoring systems are available to predict severity of acute pancreatitis but are cumbersome, take 24 to 48 hours and are dependent on tests that are not universally available. Haematocrit has been used as a predictor of severity of acute pancreatitis but some have doubted its role.

**Objectives:** To study the significance of haematocrit in prediction of severity of acute pancreatitis.

**Methods:** Patients admitted with first episode of acute pancreatitis from February 2014 to July 2014 were included. Haematocrit at admission and 24 hours of admission were compared with severity of acute pancreatitis. Mean, analysis of variance, chi square, pearson correlation and receiver operator characteristic curve were used for statistical analysis.

**Results:** Thirty one patients were included in the study with 16 (51.61%) male and 15 (48.4%) female. Haematocrit at 24 hours of admission was higher in severe acute pancreatitis (P value 0.003). Both haematocrit at admission and at 24 hours had positive correlation with severity of acute pancreatitis (r: 0.387; P value 0.031 and r: 0.584; P value 0.001) respectively. Area under receiver operator characteristic curve for haematocrit at admission and 24 hours were 0.713 (P value 0.175, 95% CI 0.536 - 0.889) and 0.917 (P value 0.008, 95% CI 0.813 - 1.00) respectively.

**Conclusion:** Haematocrit is a simple, cost effective and widely available test and can predict severity of acute pancreatitis.

**Key words:** Acute necrotizing pancreatitis, Haematocrit

## INTRODUCTION

Acute pancreatitis (AP) is an acute inflammatory disease of pancreas that frequently affects the peripancreatic tissue and less frequently the systemic organs<sup>1</sup>. Severe form of disease is characterized by persistent organ failure, pancreatic necrosis and other local and systemic complications with mortality up to 30%<sup>2-5</sup>. Early identification of these patients has advantages of early triage into severe group and hence, early fluid resuscitation, early admission to intensive care unit or transfer to another centers with intensive care facility.

There are several clinical, biochemical and imaging systems and scores to predict the severity of AP like Ranson's criteria, Acute Physiology and Chronic Health Evaluation (APACHE II), Computerized Tomography (CT) criteria, Bedside Index for Severity in Acute Pancreatitis (BISAP) score etc<sup>4,6-8</sup>. However most of these scoring systems are cumbersome, take around 24 to 48 hours to calculate or rely on diagnostic tests that are not available widely<sup>9</sup>. Therefore, the search of a simple, readily available, economical, non-invasive and reproducible tool is going on<sup>10</sup>. Studies have shown that haematocrit at admission is a useful tool to evaluate the severity of AP however some doubt the role of haematocrit as a prognostic marker<sup>6,11-17</sup>. Hence, the role of haematocrit in determination of severity of acute pancreatitis is still controversial.

We conducted a descriptive study with an aim to study the significance of haematocrit at admission and at 24 hours of admission in predicting the severity of AP.

### Address for correspondence

Dr. Tanka Prasad Bohara  
Assistant Professor  
Department of Surgery  
Kathmandu Medical College Teaching Hospital  
Sinamangal, Kathmandu, Nepal  
E-mail: tankaprasad.bohara@gmail.com

## METHODS

Thirty one patients admitted in the Department of Surgery, Kathmandu Medical College Teaching Hospital, a tertiary level hospital, with diagnosis of first episode of acute pancreatitis from February 2014 to June 2014 were included in the study. Patients with chronic liver disease, chronic kidney disease, and patient with systemic disease which has effect on haematocrit estimation such as anaemia or haematological malignancies and those not giving consent were excluded. Patients referred from other centre whose haematocrit level at admission was not available were also excluded from the study because fluid resuscitation at other centre can alter the haematocrit levels. Informed consent was taken from each patient. Ethical clearance was taken from Institutional Review Committee at Kathmandu Medical College.

Diagnosis of AP was made if two of the following three conditions were present (1) abdominal pain consistent with AP; (2) serum amylase (or lipase) more than three times the normal value; (3) characteristic findings of AP on abdominal ultrasound, Contrast Enhanced Computed Tomography (CECT) abdomen or Magnetic Resonance Imaging (MRI)<sup>2</sup>. Severity of pancreatitis was classified as per Revised Atlanta Classification as mild acute pancreatitis, moderately severe pancreatitis and severe acute pancreatitis<sup>2</sup>.

Demographic data, history, relevant physical findings, serum amylase, liver function test including haematocrit at admission and at 24 hours of admission, abdominal ultrasound, CECT abdomen and MRI abdomen (if done) findings were noted. Haematocrit at admission and 24 hours of admission were compared with severity of AP based on revised Atlanta classification.

## Statistical Analysis

Categorical variables were expressed as absolute or relative frequencies and continuous variables were expressed as mean±SD. Analysis of Variance (ANOVA) test was used to analyze continuous variables between

different groups while chi square test was used on categorical variables. Pearson correlation was used to test correlation of haematocrit with the severity of pancreatitis. Receiver Operator Characteristic (ROC) curve was plotted for range of haematocrit levels at admission and at 24 hours of admission as prognostic factor for severity of acute pancreatitis. Classification of severity based on revised Atlanta classification was used as gold standard of severity. p value of < 0.05 was considered statistically significant. Statistical Package for the Social Science (SPSS) version 20.0 was used for analysis of data.

## RESULTS

Thirty one patients with first episodes of acute pancreatitis were included in the study. Seven patients were excluded from the study out of them five patients had associated liver disease and two patients had chronic kidney disease. Mean age of the patients was 50.16 years (range 21 – 82 years) with 16 (51.6%) male and 15 (48.4%) female. Biliary pathology was the cause of AP in 16 (51.61%) patients, alcohol was the cause in nine (29.03%) patients, one (3.23%) had post Endoscopic Retrograde Cholangiopancreatography (ERCP) and five (16.13%) had idiopathic pancreatitis.

There were 15 (48.4%) patients with mild AP, 12 (38.7%) patients with moderately severe AP and four (12.9%) patients with severe AP.

Both haematocrit at admission and haematocrit at 24 hours had positive correlation with severity of AP with correlation coefficient of 0.387 (P value 0.031) and 0.584 (P value 0.001) respectively.

Area under ROC curve for haematocrit at admission (figure 1) was 0.713 (P value 0.175, 95 % CI 0.536 - 0.889) and that for haematocrit at 24 hours of admission (figure 2) was 0.917 (P value 0.008, 95 % CI 0.813 – 1.00). Coordinates of ROC curve shows that cut-off value of 44 % is 100 % sensitive and 88.9 % specific in prediction of severity of pancreatitis for haematocrit at 24 hours of admission.

**Table 1: Mean age and sex distribution according to the severity of AP**

	Severity of Acute Pancreatitis based on revised Atlanta classification			p value
	Mild	Moderately Severe	Severe	
Mean Age in years (± SD)	50.8 (± 13.83)	49.17 (± 19.25)	46 (± 18.81)	0.874 <sup>a</sup>
<b>Sex</b>				
Male	6 (37.5 %)	8 (50.0%)	2 (25.0%)	0.386 <sup>b</sup>
Female	9 (60.0%)	4 (26.7%)	2 (13.3%)	

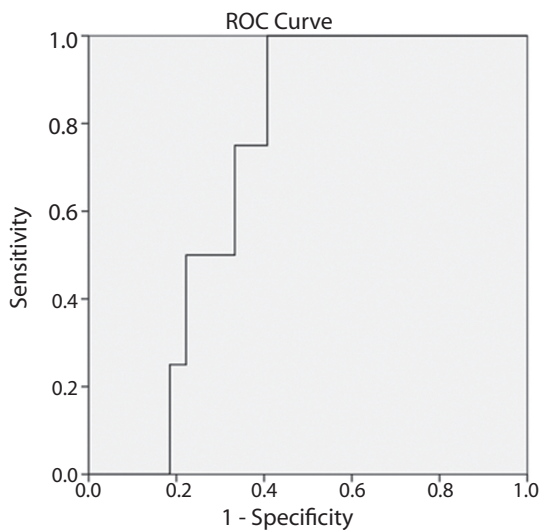
<sup>a</sup> Calculated by ANOVA

<sup>b</sup> Calculated by Chi square test

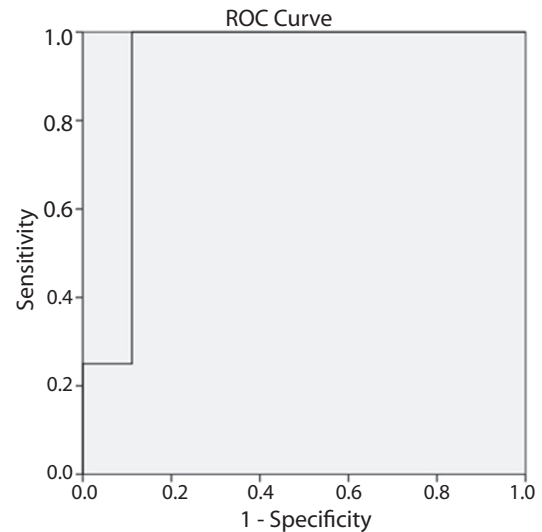
**Table 2: Haematocrit in different grades of severity**

	Severity of Acute Pancreatitis based on revised Atlanta classification			p value
	Mild	Moderately Severe	Severe	
Mean of haematocrit in % at admission	40.57 ( $\pm$ 6.29)	45.35 ( $\pm$ 7.74)	47.42 ( $\pm$ 2.28)	0.090
Mean of haematocrit in % at 24 hours of admission	36.72 ( $\pm$ 4.47)	41.12 ( $\pm$ 5.55)	46.45 ( $\pm$ 3.16)	0.003

<sup>a</sup> P value calculated by ANOVA



**Figure 1:** Receiver Operator Characteristic (ROC) curve for haematocrit at admission as prognostic factor for severity of pancreatitis



**Figure 2:** Receiver Operator Characteristic (ROC) curve for haematocrit at 24 hours of admission as prognostic factor for severity of pancreatitis

## DISCUSSION

Severe acute pancreatitis can occur in about 25% of patients with AP with mortality up to 30%<sup>5</sup>. Severe form of disease is characterized by persistent organ failure, pancreatic necrosis and other local and systemic complications<sup>2</sup>. Identification of patients at risk of severe disease early in the course of disease allows early triage of the patients, early fluid resuscitation, admission to a high care or intensive care units and transfer of patients to specialized tertiary centers if patient present to a primary care centers. Similarly proper identification of patients not at risk of severe disease may be managed out of intensive care units and at general hospitals. This can help in judicious use of resources and avoid unnecessary referral and overload at tertiary care hospital.

A case control study comparing haemoconcentration in patients with necrotizing pancreatitis with that in mild pancreatitis showed that the haematocrit at admission of  $\geq 47\%$  was seen in more patients with necrotizing pancreatitis than mild pancreatitis (11/32 vs 3/32; P value 0.03). At 24 hours of admission failure of

admission haematocrit to decrease was also significant in necrotizing pancreatitis group (26/32 vs 4/32; P value  $< 0.001$ )<sup>11</sup>. Another prospective study showed that haematocrit at admission  $\geq 44\%$  and/or failure of haematocrit to decrease at 24 hours of admission was associated with the development of necrotizing pancreatitis and organ failure with negative predictive value for necrotizing pancreatitis and organ failure of 96% and 97% respectively<sup>12</sup>. However, Troche et al<sup>16</sup> studied association of hematocrit at admission or at 24 hours after admission with severe AP, organ failure, and pancreatic necrosis and found that hematocrit was not a good predictor of severity in AP. Their results showed that the sensitivity, specificity and positive predictive values for necrosis and organ failure were low but negative predictive values was between 61% to 86%, being highest for organ failure<sup>16</sup>. Current study showed that the level of haemoconcentration as evidenced by haematocrit level was higher as the grades of severity increases both at admission and 24 hours of admission but statistically significant difference was seen in the level of haematocrit at 24 hours of admission (P value

0.090 vs 0.003). Both haematocrit at admission and at 24 hours had positive correlation with severity of AP with correlation coefficient of 0.387 (P value = .031) and 0.584 (P value .001) respectively which is statistically significant. Current study also showed that the cut-off value for haematocrit at 24 hours of admission at 44% is 100% sensitive and 88.9% specific in prediction of severity of pancreatitis similar to Brown et al<sup>12</sup>.

A few studies have shown that haemoconcentration predicts pancreatic necrosis and mortality only among patient transferred to their hospital from other hospitals and not in patients directly admitted to their hospital<sup>13,14</sup>. However another study showed that among the patients with haemoconcentration in AP who were directly admitted to their hospital within 12 hours of pain had severe pancreatitis<sup>18</sup>. A study observed high negative predictive value of haematocrit and suggested that in the absence of hemoconcentration, contrast-enhanced CT may be unnecessary on admission unless the patient does not improve<sup>15</sup>. The negative predictable value of haematocrit has been used in Harmless Acute Pancreatitis Score (HAPS) in which absence of haemoconcentration (Haematocrit < 43% for male and < 39.6% for female) along with absence of rebound tenderness and serum creatinine < 2 mg/dl has been found to be sensitive and specific to predict non severe acute pancreatitis<sup>19,20</sup>. Similarly haematocrit has been used in panc 3 score to predict severe pancreatitis on presentation and authors

stated that "The prognostic value of the scoring system seems to depend mainly on the serum haematocrit" signifying the role of haematocrit in prediction of severe AP<sup>9</sup>.

Haematocrit is a simple haematological test. It is cost effective, less time consuming, reproducible and is even available in primary health care facilities. So it can be used even in primary health care centre to predict severe course in AP and initiate early treatment and early transfer to higher centre. It is also important in higher centers for proper risk stratification even by non-specialists for early referral to concerned department and intensive care units for optimal level of care.

Our study has certain limitations. It is a single centre study with small sample size and hence, the results may not be generalized. A multicentre study with larger sample size would be required.

## CONCLUSION

Haematocrit is a simple, cost effective, reproducible and widely available test that can be used for early prediction of severity in acute pancreatitis presenting with the first episode and in acute pancreatitis without fluid resuscitation. It can be used even in primary health centre and emergency department of higher centre for early risk stratification and early transfer to intensive care units.

## REFERENCES

1. Trikudanathan G, Navaneethan U, Vege SS. Current controversies in fluid resuscitation in acute pancreatitis: a systematic review. *Pancreas*. 2012 Aug;41(6):827–34.
2. Banks PA, Bollen TL, Dervenis C, Gooszen HG, Johnson CD, Sarr MG, et al. Classification of acute pancreatitis--2012: revision of the Atlanta classification and definitions by international consensus. *Gut*. 2013 Jan;62(1):102–11.
3. Steinberg W, Tenner S. Acute pancreatitis. *N Engl J Med*. 1994 Apr;330(17):1198–210.
4. Domínguez-Muñoz JE, Carballo F, García MJ, de Diego JM, Campos R, Yangüela J, et al. Evaluation of the clinical usefulness of APACHE II and SAPS systems in the initial prognostic classification of acute pancreatitis: a multicenter study. *Pancreas*. 1993 Nov;8(6):682–6.
5. Tenner S, Sica G, Hughes M, Noordhoek E, Feng S, Zinner M, et al. Relationship of necrosis to organ failure in severe acute pancreatitis. *Gastroenterology*. 1997 Sep;113(3):899–903.
6. Talukdar R, Nageshwar Reddy D. Predictors of adverse outcomes in acute pancreatitis: new horizons. *Indian J Gastroenterol*. 2013 May;32(3):143–51.
7. Lankisch PG, Blum T, Maisonneuve P, Lowenfels AB. Severe acute pancreatitis: when to be concerned? *Pancreatol*. 2003 Jan;3(2):102–10.
8. Woo SM, Noh MH, Kim BG, Hsing C Ter, Han JS, Ryu SH, et al. Comparison of Serum Procalcitonin with Ranson, APACHE-II, Glasgow and Balthazar CT Severity Index Scores in Predicting Severity of Acute Pancreatitis. *Korean J Gastroenterol*. 2011 Jul;58(1):31–7.
9. Brown A, James-Stevenson T, Dyson T, Grunckenmeier D. The panc 3 score: a rapid and accurate test for predicting severity on presentation in acute pancreatitis. *J Clin Gastroenterol*. 2007 Oct;41(9):855–8.
10. Frossard JL, Hadengue a., Pastor CM. New serum markers for the detection of severe acute pancreatitis in humans. *Am J Respir Crit Care Med*. 2001 Jul;164(1):162–70.

11. Baillargeon JD, Orav J, Ramagopal V, Tenner SM, Banks PA. Hemoconcentration as an early risk factor for necrotizing pancreatitis. *Am J Gastroenterol*. 1998 Nov;93(11):2130–4.
12. Brown A, Orav J, Banks PA. Hemoconcentration is an early marker for organ failure and necrotizing pancreatitis. *Pancreas*. 2000 May;20(4):367–72.
13. Wu BU, Johannes RS, Conwell DL, Banks PA. Early hemoconcentration predicts increased mortality only among transferred patients with acute pancreatitis. *Pancreatology*. 2009 Jan;9(5):639–43.
14. Wu BU, Conwell DL, Singh VK, Repas K, Maurer R, Bollen TL, et al. Early hemoconcentration is associated with pancreatic necrosis only among transferred patients. *Pancreas*. 2010 Jul;39(5):572–6.
15. Lankisch PG, Mahlke R, Blum T, Bruns A, Bruns D, Maisonneuve P, et al. Hemoconcentration : An Early Marker of Severe and / or Necrotizing Pancreatitis ? A Critical Appraisal. *Am J Gastroenterol*. 2001 Jul;96(7):2081–5.
16. Remes-Troche JM, Duarte-Rojo A, Morales G, Robles-Díaz G. Hemoconcentration is a poor predictor of severity in acute pancreatitis. *World J Gastroenterol*. 2005 Nov;11(44):7018–23.
17. Gardner TB, Olenec C A, Chertoff JD, Mackenzie TA, Robertson DJ. Hemoconcentration and pancreatic necrosis: further defining the relationship. *Pancreas*. 2006 Aug;33(2):169–73.
18. Kapoor K, Repas K, Singh VK, Conwell DL, Morteale KJ, Wu BU, et al. Does the duration of abdominal pain prior to admission influence the severity of acute pancreatitis? *J Pancreas*. 2013 Mar;14(2):171–5.
19. Lankisch PG, Weber-Dany B, Hebel K, Maisonneuve P, Lowenfels AB. The Harmless Acute Pancreatitis Score: A Clinical Algorithm for Rapid Initial Stratification of Nonsevere Disease. *Clin Gastroenterol Hepatol*. 2009 Jun;7(6):702–5.
20. Talukdar R, Sharma M, Deka A, Teslima S, Dev Goswami A, Goswami A, et al. Utility of the “harmless acute pancreatitis score” in predicting a non-severe course of acute pancreatitis: a pilot study in an Indian cohort. *Indian J Gastroenterol*. 2014 Jul;33(4):316–21.