Journal of Chitwan Medical College 2024;14(47):55-59
Available online at: www.jcmc.com.np

ORIGINAL RESEARCH ARTICLE

HEMATOCRIT AS AN EARLY PREDICTOR OF SEVERE ACUTE PANCREATITIS: A PROSPECTIVE OBSERVATIONAL STUDY IN TERTIARY CARE CENTRE OF NEPAL

Bishal Acharya[1,*], Harish Chandra Neupane[1], Kishor Kumar Tamrakar[1], Iswar Chandra Ghimire[1], Rajesh Kumar Mandal[1], Rohit Kumar Mishra[1], Birendra Raut[1], Raghavendra Vimal[1], Nimish Raj Bastola[1], Akarsh Kandel[1], Ekanta Timalsina[1], Ajit Shrestha[1]
[1]Department of General Surgery, Surgical Gastroenterology unit, Chitwan Medical College and teaching hospital, Bharatpur, Nepal

Received: 4 Jan, 2024
Accepted: 13 Mar, 2024
Published: 30 Mar, 2024

Key words: Hematocrit; Mild to moderately severe acute pancreatitis; Severe acute pancreatitis.

***Correspondence to:** Bishal Acharya, Department of General Surgery, Surgical Gastroenterology unit, Chitwan Medical College and teaching hospital, Bharatpur, Nepal.*
Email: bishalacharya3@gmail.com

*DOI:*https://doi.org/10.54530/jcmc.1464

Citation

Acharya B, Neupane HC, Tamrakar KK, Ghimire IC, Mandal RK, Mishra RK, Raut B, Vimal R, Bastola NR, Kandel A, Timalsina E, Shrestha A. Hematocrit as an early predictor of severe acute pancreatitis: a prospective observational study in tertiary care centre of Nepal. Journal of Chitwan Medical College.2024;14(47):55-9.

Peer Reviewed

ABSTRACT

Background: The predictor system for acute severe pancreatitis is complex and takes up to 48 hours to complete. This study aimed to investigate the role of admission hematocrit in predicting acute pancreatitis severity.

Methods: This was an observational study, conducted at Chitwan Medical College where 112 patients who met the inclusion criteria and were diagnosed with acute pancreatitis were examined. The patients were followed for 48 hours by evaluating the admission hematocrit and were categorized to have mild to moderately severe pancreatitis or severe pancreatitis according to the modified Atlanta criteria. All analyzes were performed using SPSS statistical software, version 26. Descriptive and inferential statics, sensitivity and specificity were calculated.

Results: The mean age was 44.72 ± 14.5 years and 70.5% were male. The most common etiology was ethanol (52.2%), followed by gallstone disease (26.5%). The prevalence of severe pancreatitis in patients in the high-risk group was 88.89%, and the incidence of severe pancreatitis in patients in the low-risk group was 10.59%. The detection sensitivity of hematocrit level >/= 44% for severe acute pancreatitis was 72.73%, the specificity of 96.2%, the positive predictive value of 88.89%, the negative predictive value of 89.41%, the accuracy of 89.3%, likelihood ratio for positive test result of 19.14 and likelihood ratio for negative test result of 0.283.

Conclusions: Patients with high hematocrit on admission were associated with the occurrence of severe acute pancreatitis, so these patients should be considered as a high-risk group for development of severe acute pancreatitis and monitored meticulously.

INTRODUCTION

Acute pancreatitis is a common gastrointestinal disease, the incidence of which has increased in recent years, with a prevalence of 7.69% in Nepal.[1] After the diagnosis of pancreatitis is made, the severity of disease and organ failure should be evaluated for further treatment.[2,3]

Various scoring systems combining clinical, laboratory and radiographic data have been developed to assess the severity of pancreatitis, however, the biggest limitation of these scores is that they are often difficult and laborious to calculate in daily clinical practice[4,5] Therefore, single independent hematological prognostic markers have been examined to predict the disease severity.[6] CRP, BUN, creatinine and hematocrit are good options because they are readily available in many centers, are inexpensive, and have versatile standards.

The main aim of this study is to determine the role of admission hematocrit in predicting the severity of acute pancreatitis.

METHODS

This was a hospital based observational study conducted from July 2023 till December 2023 in department of General Surgery, Surgical Gastroenterology unit of Chitwan Medical College, Chitwan, Nepal. Using the consecutive sampling technique, all patients of both the genders who were 18 years or older and admitted to the hospital for acute pancreatitis were considered. They were diagnosed according to the revised Atlanta criteria within the study period and inclusion required them to have given informed written consent. All eligible individuals meeting these criteria were included in this study. Patients with pancreatic malignancy, immunocompromised states, referred cases and pregnant women were excluded. Also, patients with traumatic pancreatitis, chronic liver disease, chronic kidney disease, and with systemic diseases that affect haematocrit estimation were excluded from this study.

Regardless of the etiology, all patients diagnosed with acute pancreatitis were admitted and their demographic information was recorded in predesigned proforma. Hematocrit level, measured at the time of admission, by automated hematology analyzer in laboratory of CMCTH was recorded. Patients with

hematocrit value <44% were included in low-risk group (HL) and ≥ 44% were included in high-risk group (HH) for development of severe acute pancreatitis.[5] Enrolled patients were treated according to the institution's standard of practice. Diagnostic tests and tests to confirm the presence or absence of local and systemic complications or organ failure included serum lipase or amylase, ultrasonography of abdomen and pelvis, arterial blood gas analysis, serum creatinine and CECT abdomen and pelvis if complications of acute pancreatitis was suspected.

Patients were followed for 48 hours for the development of any local complications, systemic complications or organ failure. Patients who did not develop complications or had transient organ failure/ transient local or systemic complications as per Revised Atlanta criteria were grouped under mild to moderately severe acute pancreatitis (MM) and those who developed persistent organ failure/ persistent local or systemic complications were grouped under severe acute pancreatitis group (SP). The end of the survey was the end of 48 hours exposure.

All analyzes were performed using SPSS statistical software, version 26. Categorical predictor variables were analyzed using χ^2 test. P value < 0.05 was considered statistically significant. Then sensitivity and specificity of hematocrit measured at the time of admission to predict severe acute pancreatitis were calculated.

RESULTS

One hundred and twelve patients met the inclusion criteria and were included in the study. Mean age of patients with acute pancreatitis was 44.72 years (SD: 14.5) (Figure 1).

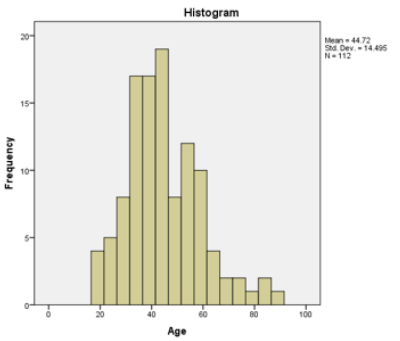

Figure 1: Distribution of the cases according to the age

Majority of the patients with acute pancreatitis were male accounting for 70.5% (Figure 2).

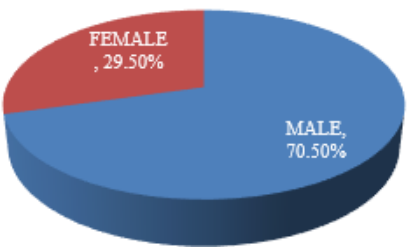

Figure 2: Distribution of the cases according to the gender

Most common etiology for acute pancreatitis was ethanol (52.2%) followed by gall stone disease (26.5%) (Table 1).

Table 1: Etiology of acute pancreatitis

Etiology	Frequency (%)
Ethanol	59 (52.68)
Gall stone	30 (26.78)
Hypertriglyceridemia	6 (5.36)
Unknown	17 (15.18)

Total of twenty-seven patients (24.1%) were under high-risk group and out of which twenty-four patients (88.89%) developed severe acute pancreatitis, according to revised Atlanta criteria. Nine out of eighty-five patients under low-risk group developed severe acute pancreatitis (10.59%) (Table 2).

Patients under high-risk group developed severe pancreatitis significantly higher than patients in low risk group (Pearson chi square value: 60.449, p value <0.0001). Hence hematocrit level at the time of admission was found to be a significant predictor of severe acute pancreatitis.

The sensitivity and specificity of hematocrit, measured at the time of admission and categorized as <44% and >/=44% to predict the development of acute severe pancreatitis, were calculated. The results revealed a sensitivity of 72.3% and a specificity of 96.2% (Table 3).

There was no mortality during the study period (within 48 hours of admission).

DISCUSSION

Early identification of patients at risk for severe acute pancreatitis is an important aspect for both current and future management.[7] Large-cohort studies show that the majority of the deaths (40%) occur early in the course of the disease, with most occurring within the first 72 hours after admission to hospital.[8] Hemoconcentration at admission, defined as initial hematocrit (<44% or ≥ 44%), reflects intravascular volume status, which is closely associated with the tissue perfusion, development of organ failure and pancreatic necrosis.[4] Earlier detection of patients at risk for pancreatic necrosis and organ failure can improve their care through immediate intensive care unit admission and subsequent management.[9]

Acute Pancreatitis is thought to be a result of damage to the pancreatic parenchyma causing a local inflammatory response that spreads and gets generalized. Studies have shown the role of various signaling molecules playing pivotal role in acute pancreatitis, such as cytokines and inflammatory mediators.[10] All these factors lead to increase in vascular permeability causing intravascular fluid to enter the extravascular space.[11,12] This increased extravascular fluid volume reduces pancreatic perfusion pressure leading to microcirculatory changes and contributes to pancreatic ischemia and subsequent necrosis.[13] It has been demonstrated that the severity of acute pancreatitis may be related to hemoconcentration brought on by this fluid loss.

Table 2: High risk and low risk patients with their severity outcome

Characteristics		Acute pancreatitis		P value
		Severe Pancreatitis (SP)	Mild to Moderately severe pancreatitis (MM)	
Risk Groups	High risk group (HH)	24	3	<0.0001
	Low risk group (HL)	9	76	

Table 3: Prediction of development of acute severe pancreatitis by admission hematocrit level

Parameter	Sensitivity (%)	Specificity (%)	Positive predictive value (%)	Negative predictive value (%)	Accuracy (%)	Likelyhood ratio for positive test result	Likelyhood ratio for negative test result
Hematocrit level measured at the time of admission (<44%/≥44%)	72.73	96.2	88.89	89.41	89.3	19.14	0.283

In the present study, the mean age of the patients with acute pancreatitis was 44.72 (SD:14.5) years. Basukala et al, in a hospital based research in Kathmandu, in 2023 reported that the median age of patients with acute pancreatitis was 42 years, which is similar to our study.[1] This study identified the prevalence of acute pancreatitis was higher in men, despite research suggesting that the age and sex distribution of the condition varies depending on its aetiology.[14] Tanweer Karim et al in 2020 also showed male predominance in Indian population (54.84%).[14] Similar finding was given by Dhiraj Yadav et al in New York.[15] However, Basukala et al showed that female were in majority with 52.50% and gall stone being the most common cause of acute pancreatitis.[1] In our study we found ethanol as the most common etiology (52.68%) and gall stone being the second most common cause (26.78%). Tanweer Karim et al documented gallstone as a most common cause (67.74%) followed by ethanol (17.74%).[14] According to Steinberg et al., the most frequent cause of acute pancreatitis in the US, Asia, and the majority of Western Europe is biliary disease.[16]
High hematocrit level on admission is associated with pancreatic necrosis, organ failure, prolonged hospital stay and the need for intensive care.[17-19] The cutoff value of hematocrit in these studies ranged from 39% to 47%, with 44% being the most commonly used.[17-19]

Lankisch PG et al reported different cutoff value for men (hematocrit >43%) and women (>39.6%).[18] In a study by Koutroumpakis E et al, hematocrit level of ≥44% at presentation was similar to APACHE-II in predicting persistent organ failure and was also found to be better at predicting of pancreatic necrosis.[4] Our study showed the similar result. Hematocrit level at the time of admission ≥ 44% was found to be a significant predictor of severe acute pancreatitis (Pearson chi square value: 60.449, p value <0.0001). In our study, a total of twenty-seven patients (24.1%) had hematocrit level ≥ 44% (high risk group) at admission and twenty four of them (88.89%) developed severe acute pancreatitis. Gan SI et al reported the prevalence of high-risk subgroup to be 37%, and as hematocrit exceeded 44%, the risk of severe disease exceeded 50%.[19] Gray and Rosenman concluded in 1965 that hemoconcentration at presentation was a sign of poor prognosis in patients with acute pancreatitis.[20]
In contrast, Talamini et al. found no significant difference in hematocrit level measured within 24 hours of admission between acute pancreatitis survivors and non-survivors.[21] Similarly, hemoconcentration was a poor predictor of severity in acute pancreatitis, according to José M. Remes-Troche et al., who found that neither its presence nor absence at admission was linked to severity, necrosis, or organ failure.[22] The reason might be that, it was a retrospective study and only 69% of the patients had hematocrit measured at 24 hours after admission.

In this study, the authors experienced no mortality during the study period (within 48 hours of admission). The mortality rate among patients with acute pancreatitis in study done by Basukala et al was 0.83% during the course of hospital stay.[1] This mortality rate is relatively lower than another study done by Banks P et al which reported the overall mortality in acute pancreatitis to be approximately 5%.[23]

In this study, sensitivity of hematocrit level measured at the time of admission to predict severe acute pancreatitis was 72.73%, specificity of 96.2%, positive predictive value of 88.89% and negative predictive value of 89.41%. Gan SI et al documented sensitivity and specificity of hematocrit level <45% and ≥ 45% as 71% and 63%, respectively.[19] They also documented negative predictive value of 96%, which is higher than our study and positive predictive value of 26%.[19] Lankisch PG et al included a hematocrit cutoff value of 43.0% in male patients and 39.6% in female patients, resulting in a sensitivity and specificity for the diagnosis of necrotizing pancreatitis of 74% and 45% respectively with the positive predictive value of 24% and the negative predictive value of 88%.[18] Brown A et al reported hematocrit (<44% or ≥ 44%) as a marker for necrotizing pancreatitis, with a sensitivity of 72%, specificity of 83%, positive predictive value of 68% and the negative predictive value of 85%.[24]

Accuracy of hematocrit level at admission to predict severe acute pancreatitis in our study was 89.3%, likelihood ratio for positive test result was 19.14 and likelihood ratio for negative test result was 0.283. Lankisch et al, for hematocrit level >43.0% for male patients and >39.6% for female patients, calculated accuracy of 50%.[18]

According to our findings, the value of hemoconcentration lies in its high negative predictive value (89.41%) and high specificity (96.2%), which means the absence of hemoconcentration usually excludes a suspicion of development of severe acute pancreatitis. Hence, a CECT of abdomen may not be required to confirm pancreatic necrosis unless the patient fails to improve. This conclusion may help to reduce the hospital expenditure. In addition, sensitivity of the test is 72.73% in our study, indicating that hematocrit level at the time of admission can be used as a simple screening tool to predict the severity of acute pancreatitis. Probability of developing severe acute pancreatitis in a patient with increased hematocrit level is high as the positive predictive value is 88.89%. Similarly, likelihood of developing severe acute pancreatitis in a patient with raised hematocrit level $\geq$ 44%, according to this study, is 19.14 which means, a patient with raised hematocrit level on admission is likely to develop severe acute pancreatitis compared to those with hematocrit level <44%.

Many of the patients were referred to our hospital with the diagnosis of acute pancreatitis after 48 hours of diagnosis. These patients were more often of severe acute pancreatitis. Therefore in order to avoid the selection bias, these group of patients who presented to our hospital with prior diagnosis of acute pancreatitis were excluded from the study. This was single institute based study conducted in short duration of six months with a sample size of 112 patients, so a large multicentric study can be conducted to approve the conclusion. The cost effectiveness is yet to be compared with other laboratory parameters and scoring systems to predict severity of acute pancreatitis.

CONCLUSION

Admission hematocrit level is the preferred clinical marker for predicting severe acute pancreatitis based on its accuracy and availability. Therefore, it is recommended that patients with high hematocrit level at admission should be monitored closely for fluid resuscitation, supportive care along with other treatments to reduce the complications, morbidity and mortality. We also recommend conducting a large multicentre study to validate our results and compare the cost effectiveness of hematocrit at admission with other scoring systems to predict the severity of acute pancreatitis.

CONFLICT OF INTEREST: None

FINANCIAL DISCLOSURE: None

REFERENCES:

1. Basukala S, Pathak BD, Dawadi P, Bohara S, Tamang A, Pahari S, et al. Acute Pancreatitis among Patients Visiting the Department of Surgery in a Tertiary Care Centre: A Descriptive Cross-sectional Study. J Nepal Med Assoc. 2023;61(258):106–10. [DOI]
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;62(1):102–11. [DOI]
3. Garg PK, Singh VP. Organ Failure due to Systemic Injury in Acute Pancreatitis. Physiol Behav. 2018;176(5):139–48.[DOI]
4. Koutroumpakis E, Wu BU, Bakker OJ, Dudekula A, Singh VK, Besselink MG, et al. Admission hematocrit and rise in blood urea nitrogen at 24 h outperform other laboratory markers in predicting persistent organ failure and pancreatic necrosis in acute pancreatitis: A post hoc analysis of three large prospective databases. Am J Gastroenterol. 2015;110(12):1707–16. [DOI]
5. Papachristou GI, Muddana V, Yadav D, Oconnell M, Sanders MK, Slivka A, et al. Comparison of BISAP, Ranson's, APACHE-II, and CTSI scores in predicting organ failure, complications, and mortality in acute pancreatitis. Am J Gastroenterol. 2010;105(2):435–41.[DOI]
6. Papachristou GI, Clermont G, Sharma A, Yadav D, Whitcomb DC. Risk and Markers of Severe Acute Pancreatitis. Gastroenterol Clin North Am. 2007;36(2):277–96. [DOI]
7. Toouli J, Bassi C, Telford J, Freeny P. Guidelines Pancreatitis Aguda 2002. J Gastroenterol Hepatol. 2002;17:1–25. [DOI]
8. McKay CJ, Evans S, Sinclair M, Carter CR, Imrie CW. High early mortality rate from acute pancreatitis in Scotland, 1984-1995. Br J Surg. 1999;86(10):1302–5. [DOI]
9. Ernst Klar, MD, Thomas Foitzik, MD, Heinz Buhr, MD, Konrad Messmer, MD, Christian Herfarth M. Isovolemic hemodilution with dextran 60 as treament of pancreatic ischaemia in Acute Pancreatitis. Ann Surg. 1993;217(4):369–74. [DOI]
10. Gómez-Cambronero LG, Sabater L, Pereda J, Cassinello N, Camps B, Viña J, et al. Role of cytokines and oxidative stress in the pathophysiology of acute pancreatitis: therapeutical implications. Curr Drug Targets Inflamm Allergy. 2002;1(4):393–403. [DOI]
11. Eibl G, Hotz HG, Faulhaber J, Kirchengast M, Buhr HJ, Foitzik T. Effect of endothelin and endothelin receptor blockade on capillary permeability in experimental pancreatitis. Gut. 2000;46(3):390–4. [DOI]
12. Foitzik T, Eibl G, Hotz HG, Faulhaber J, Kirchengast M, Buhr HJ. Endothelin receptor blockade in severe acute pancreatitis leads to systemic enhancement of microcirculation, stabilization of capillary permeability, and improved survival rates. Surgery. 2000;128(3):399–407. [DOI]
13. Zhou ZG, Chen YD. Influencing factors of pancreatic microcirculatory impairment in acute panceatitis. World J Gastroenterol. 2002;8(3):406–12. [DOI]
14. Karim T, Jain A, Kumar V, Kumar RB, Kumar L, Patel M. Clinical and severity profile of acute pancreatitis in a hospital for low socioeconomic strata. Indian J Endocrinol Metab. 2020;24(5):416–21. [DOI]
15. Yadav D, Lowenfels AB. The epidemiology of pancreatitis and pancreatic cancer. Gastroenterology. 2013;144(6):1252–61. [DOI]
16. William Steinberg, M.D., Scott Tenner, M.D. MP. Acute Pancreatitis. N Engl J Med. 1994;330(17):1198–210. [DOI]
17. Baillargeon JD, Orav J, Ramagopal V, Tenner SM, Banks PA. Hemoconcentration as an early risk factor for necrotizing pancreatitis. Am J Gastroenterol. 1998;93(11):2130–4. [DOI]
18. 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;96(7):2081–5. [DOI]
19. Gan SI, Romagnuolo J. Admission hematocrit: A simple, useful and early predictor of severe pancreatitis. Dig Dis Sci. 2004;49(11–12):1946–52. [DOI]
20. Samuel H. Gray, MD, Leonard D. Rosenman M. Acute pancreatitis, The Significance of Hemoconcentration at Admission to the Hospital. Arch

Surg. 1965;91(2):485–8. [DOI]

21. Talamini G, Bassi C, Falconi M, Sartori N, Frulloni L, Di Francesco V, et al. Risk of death from acute pancreatitis - Role of early, simple "routine" data. Int J Pancreatol. 1996;19(1):15–24. [DOI]

22. 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;11(44):7018–23. [DOI]

23. Banks PA, Freeman ML, Fass R, Baroni DS, Mutlu EA, Bernstein DE, et al. Practice guidelines in acute pancreatitis. Am J Gastroenterol. 2006;101(10):2379–400. [DOI]

24. Brown A, Orav J, Banks PA. Hemoconcentration is an early marker for organ failure and necrotizing pancreatitis. Pancreas. 2000;20(4):367–72. [DOI]