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COMPARATIVE PREDICTIVE ACCURACY OF HAPS AND BISAP FOR EARLY SEVERITY ASSESSMENT IN ACUTE PANCREATITIS

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ABSTRACT

Background and Objectives: Acute pancreatitis (AP) ranges from mild self-limiting disease to life-threatening severe acute pancreatitis (SAP) with multi-organ failure. Early and accurate risk stratification is essential for guiding triage and management decisions. The Bedside Index of Severity in Acute Pancreatitis (BISAP) and the Harmless Acute Pancreatitis Score (HAPS) represent two widely used early scoring tools with contrasting designs. This retrospective study compared their diagnostic performance for predicting SAP and clinical outcomes.

Methods: A retrospective study was conducted on 41 patients admitted with acute pancreatitis over a 24-month period. BISAP and HAPS scores were calculated using clinical and laboratory data obtained at admission. Disease severity was classified according to the Revised Atlanta Classification (2012). The primary outcomes were severe acute pancreatitis in-hospital mortality and ICU admission. Diagnostic performance was assessed using receiver operating characteristic analysis together with sensitivity, specificity, positive predictive value and negative predictive value. Wilson 95% confidence intervals were calculated for all estimates.

Results: Among the 41 patients included in the study, 26 (63.4%) developed severe acute pancreatitis and 5 (12.2%) died during hospital admission. The mean ICU stay was 5.7 ± 7.8 days. BISAP performed better than HAPS in predicting severe disease with an AUROC of 0.712 compared with 0.494. Using a cutoff value of 2 or higher, BISAP achieved 100% specificity and a positive predictive value of 100% but sensitivity remained low at 42.9% with a negative predictive value of 44.4%. HAPS showed lower overall performance with a sensitivity of 37.5% and specificity of 66.7% when scores of 1 or more were considered indicative of non-harmless disease. Neither score showed a statistically significant association with severe acute pancreatitis at the predefined thresholds on Chi-square testing (BISAP p=0.120 and HAPS p=0.814) which was likely related to the small sample size.

Conclusions: In this cohort BISAP performed better than HAPS for identifying patients at risk of severe acute pancreatitis mainly because of its high specificity. However neither score was sensitive enough to exclude severe disease when used alone. HAPS was originally designed to identify low-risk patients suitable for early discharge and therefore performed poorly in this population where severe disease was common. Neither BISAP nor HAPS predicted in-hospital mortality with acceptable accuracy in our cohort. The inclusion of biomarkers such as CRP or procalcitonin may improve prediction and deserves further evaluation. Because only 41 patients were available for analysis these findings should be interpreted cautiously and require confirmation in larger studies.

KEYWORDS: Acute pancreatitis; BISAP; HAPS; severity scoring; organ failure; mortality; retrospective study; Revised Atlanta Classification.

1. INTRODUCTION

Acute pancreatitis (AP) is a frequent cause of emergency hospital admission worldwide with an incidence of 4.9 to 73.4 cases per 100,000 population each year [1]. Most patients have a mild illness and recover without major complications. Around 15-20% develop severe acute pancreatitis (SAP) which is associated with a mortality rate of 15-35% [2]. The number of severe cases differs between hospitals because referral centres and surgical units often receive patients with more complicated disease. Early recognition of patients who may develop severe pancreatitis is important during the first 24 hours of admission because it influences monitoring, fluid resuscitation and decisions regarding higher levels of care [3]. Acute pancreatitis results from activation of pancreatic enzymes within the gland leading to local inflammation. In some patients the inflammatory response extends beyond the pancreas and leads to systemic inflammatory response syndrome (SIRS) followed by multi-organ dysfunction syndrome (MODS) [4]. The Revised Atlanta Classification (2012) divides acute pancreatitis into three categories. Mild disease is characterised by the absence of organ failure and local complications. Moderately severe disease includes transient organ failure lasting less than 48 hours or local complications.

Severe disease is defined by persistent organ failure lasting more than 48 hours [5]. This classification forms the basis of most current approaches to severity assessment in acute pancreatitis. Several scoring systems have been developed to identify patients at risk of severe disease. Older systems such as Ranson's criteria and the Modified Glasgow Score require data collected over 48 hours which limits their value during early assessment [6,7]. More recently the Bedside Index for Severity in Acute Pancreatitis (BISAP) and the Harmless Acute Pancreatitis Score (HAPS) have become popular because both can be calculated at admission.

BISAP was described by Wu et al. in 2008 and includes five variables: blood urea nitrogen greater than 25 mg/dL, impaired mental status with a Glasgow Coma Scale score of 13 or less, SIRS, age of 60 years or more and pleural effusion [8]. Higher BISAP scores are associated with increased mortality and organ failure. HAPS was introduced by Lankisch et al. in 2009 and was designed to identify patients unlikely to develop a severe course [9]. The score uses three variables: absence of peritonism, serum creatinine of 2.0 mg/dL or less and hematocrit of 43% or less. Patients with none of these abnormalities are classified as having harmless acute pancreatitis and generally have a favourable prognosis. Comparisons between BISAP and HAPS have been reported in several studies but information from low and middle income countries remains limited. We compared the performance of BISAP and HAPS in predicting severe acute pancreatitis and other clinical outcomes in 41 patients admitted over a 24-month period using routinely collected clinical data.

2. MATERIALS AND METHODS

2.1 Study Design and Setting

This retrospective study included patients admitted with acute pancreatitis to a general surgery unit over a 24-month period from January 2023 to December 2024. Clinical data had been recorded prospectively during routine patient care and were reviewed retrospectively for the purpose of this study. The study was registered with the institutional research or clinical governance office under registration number and was carried out according to institutional governance requirements. The protocol was submitted to the Institutional Review Board and approval was pending at the time of manuscript preparation (IRB reference: [insert reference number]). No patient identifiers were collected or reported and all data were handled in accordance with local data protection policies.

2.2 Inclusion and Exclusion Criteria

Patients aged ≥18 years admitted with a new diagnosis of AP were included. A diagnosis of AP required that patients meet at least two of three diagnostic criteria drawn from the Revised Atlanta Classification: a clinical presentation with acute-onset abdominal pain typical of pancreatitis, biochemical confirmation via serum amylase and/or lipase elevated to three or more times the upper limit of normal, or radiological confirmation through contrast-enhanced CT (CECT) imaging showing findings characteristic of the disease [5].

Patients were excluded if they were under 18 years of age, had a confirmed traumatic, hereditary, or autoimmune cause for their pancreatitis, had pre-existing chronic pancreatitis or pancreatic malignancy, had incomplete clinical or laboratory records, or left against medical advice within the first 24 hours of admission (precluding calculation of admission scores). No a priori sample size calculation was performed given the retrospective study design; all consecutive eligible patients admitted during the study period were included.

2.3 Scoring Systems

BISAP was calculated within 24 hours of admission using the five standard components described by Wu et al.: a BUN level above 25 mg/dL; an altered mental status corresponding to a GCS of 13 or below; the presence of SIRS; age of 60 years or older; and radiographic pleural effusion. SIRS status was determined using the standard consensus criteria, requiring at least two of the following to be met: body temperature outside the range of 36-38°C, a heart rate above 90 beats per minute, either a respiratory rate above 20 breaths per minute or a $PaCO_2$ under 32 mmHg, and a white cell count outside the 4,000-12,000/μL range or a differential showing more than 10% band forms. BISAP ≥2 was used as the threshold for high-risk classification based on published evidence [8,10]. HAPS was calculated at admission using the three components originally described by Lankisch et al.: the absence or presence of peritoneal signs on physical examination, a serum creatinine value above 2.0 mg/dL, and a hematocrit of 43% or higher. A score of 0 (none of the three criteria present) is classified as “harmless” AP while a score of 1 or more indicates the disease may not be harmless [9]. For the purposes of comparative analysis, HAPS ≥1 was used as the threshold for predicting severe disease.

2.4 Outcome Measures

The primary outcome was SAP as defined by the Revised Atlanta Classification (2012) - persistent organ failure of one or more systems lasting >48 hours. Secondary outcomes included in-hospital mortality, ICU length of stay, CT-confirmed local complications (pancreatic necrosis, pseudocyst, peripancreatic collection), and cardiovascular and respiratory organ failure.

2.5 Statistical Analysis

Categorical variables are reported as frequencies and percentages. Continuous variables are reported as means ± standard deviations (SD) or medians with interquartile range (IQR) as appropriate. The Mann-Whitney U test was used for non-parametric comparison of continuous outcomes between severity groups. Chi-square or Fisher’s exact test was used for categorical comparisons. Receiver operating characteristic (ROC) analysis was performed and area under the ROC curve (AUROC) calculated for both scoring systems for SAP prediction and mortality. Sensitivity, specificity, PPV, and NPV were calculated with 95% Wilson score confidence intervals. A formal statistical comparison of the BISAP and HAPS

ROC curves (e.g., DeLong test) was not performed given the limited sample size and correspondingly low power for such a comparison; AUROC values are therefore presented descriptively rather than as a formally tested difference. A p-value <0.05 was considered statistically significant. All analyses were performed using Python 3.12 (pandas, scipy, scikit-learn).

3. RESULTS

3.1 Patient Characteristics

Forty-one patients were included in the study. The mean ICU stay was 5.71 ± 7.82 days. In-hospital mortality occurred in 5 patients (12.2%). Six patients (14.6%) left against medical advice after the first day of admission. Based on the clinical picture at admission, 24 patients (58.5%) presented with AP without severe features, 6 (14.6%) had severe acute pancreatitis (SAP) already evident at admission, and 11 (26.8%) had infected necrosis at admission. Following completion of the hospital course and application of the Revised Atlanta Classification (accounting for persistent organ failure or local complications that developed or became apparent after admission), 26 patients (63.4%) were classified as severe acute pancreatitis (***Table 1***).

Table 1. Clinical Outcomes and Hospital Course (N=41)

Parameter	n (%)
Total patients	41
In-hospital mortality	5 (12.2%)
Severe AP (final)	26 (63.4%)
AMA discharge after Day 1	6 (14.6%)
Mean ICU stay (days)	5.71 ± 7.82
Infected necrosis	11 (26.8%)
CT: Necrosis	9 (22.0%)
CT: Pseudocyst	2 (4.9%)
CT: Peripancreatic collection	7 (17.1%)
CT: Edematous AP	25 (61.0%)
CVS organ failure (vasopressors)	9 (22.0%)
Respiratory failure (ventilator/NIV)	8 (19.5%)
Systemic complication	16 (39.0%)

3.2 BISAP Score Distribution and Components

BISAP scores ranged from 0 to 4. The majority of patients had low scores: 23 patients (56.1%) scored 0, 12 (29.3%) scored 1, 5 (12.2%) scored 2, and 1 patient (2.4%) scored 4. Accordingly, 6 patients (14.6%) had BISAP ≥2, constituting the high-risk group. Among individual BISAP components, BUN >25 mg/dL was the most prevalent (13 patients; 31.7%), followed by age >60 years (6 patients; 14.6%), SIRS (4 patients; 9.8%), impaired mental status (3 patients; 7.3%), and pleural effusion (2 patients; 4.9%) (***Table 2***).

Table 2. BISAP and HAPS Component Frequencies (N=41)

Component	n	Percentage (%)
BISAP Components		
BUN >25 mg/dL	13	31.7%
Impaired Mental Status (GCS ≤13)	3	7.3%
SIRS	4	9.8%
Age ≥60 years	6	14.6%
Pleural Effusion	2	4.9%
HAPS Components		
Peritonism	2	4.9%

Component	n	Percentage (%)
Hematocrit ≥43%	19	46.3%
Creatinine elevated (≥2.0 mg/dL)	7	17.1%

3.3 HAPS Score Distribution

HAPS scores were distributed as follows: 16 patients (39.0%) scored 0 (harmless AP), 22 (53.7%) scored 1, and 3 (7.3%) scored 2. Thus, the majority of patients (25/41; 61.0%) were classified as potentially non-harmless by HAPS. Hematocrit ≥43% was the most commonly positive HAPS component (19 patients; 46.3%), followed by elevated creatinine (7 patients; 17.1%) and peritonism (2 patients; 4.9%). Score distributions are shown in ***Figure 2***.

3.4 Diagnostic Performance for Severe AP Prediction

ROC analysis revealed BISAP had better discriminatory ability compared to HAPS for SAP prediction (AUROC 0.712 vs 0.494) (***Figure 1***). For mortality prediction, both scores performed at or below chance level (BISAP AUROC 0.492 vs HAPS 0.469); neither score demonstrated clinically useful discrimination for in-hospital mortality in this cohort. At a threshold of BISAP ≥2, sensitivity for SAP was 42.9% (95% CI: 23.7-64.2%), specificity was 100.0% (95% CI: 72.2-100%), PPV was 100.0% (95% CI: 54.1-100%), and NPV was 44.4% (95% CI: 28.0-62.0%). HAPS ≥1 as a threshold for predicting severe disease demonstrated sensitivity 37.5% (95% CI: 20.7-57.3%), specificity 66.7% (95% CI: 38.4-86.9%), PPV 37.5% (95% CI: 20.7-57.3%), and NPV 66.7% (95% CI: 38.4-86.9%). These results are summarised in ***Table 3*** and visualised in the forest plot (Figure 3). The wide confidence intervals throughout reflect the limited sample size and should be considered when interpreting these point estimates.

Table 3. **Diagnostic Performance of BISAP and HAPS for Predicting Severe Acute Pancreatitis**

Metric	BISAP ≥2	95% CI	HAPS ≥1	95% CI
AUROC	0.712	-	0.494	-
Sensitivity	42.9%	23.7-64.2%	37.5%	20.7-57.3%
Specificity	100.0%	72.2-100%	66.7%	38.4-86.9%
PPV	100.0%	54.1-100%	37.5%	20.7-57.3%
NPV	44.4%	28.0-62.0%	66.7%	38.4-86.9%
Chi² p-value	p = 0.120		p = 0.814	

AUROC = Area under ROC curve; PPV = Positive Predictive Value; NPV = Negative Predictive Value; CI = Wilson 95% Confidence Interval

3.5 Outcomes by Severity Group

Patients classified as SAP experienced longer ICU stays (mean 6.81 ± 9.61 days vs 3.80 ± 1.93 days in non-severe AP; Mann-Whitney U p=0.566). The non-significant p-value likely reflects the small sample size and the marked overlap in ICU stay distributions between groups. All in-hospital deaths occurred among patients classified as severe acute pancreatitis (5/26; 19.2% vs 0/15; 0% in non-severe AP). CVS organ failure and respiratory failure were also predominantly seen in the SAP group. These data are illustrated in ***Figure 4*** and summarised in ***Table 4.***

Table 4. **Clinical Outcomes Stratified by Severity (Severe vs Non-Severe AP)**

Outcome	Severe AP (n=26)	Non-Severe AP (n=15)	p-value
In-hospital Mortality	5 (19.2%)	0 (0%)	0.076*
ICU Days (mean ± SD)	6.81 ± 9.61	3.80 ± 1.93	0.566†
CVS Organ Failure	8 (30.8%)	1 (6.7%)	0.076*
Respiratory Failure	7 (26.9%)	1 (6.7%)	0.130*
Systemic Complication	14 (53.8%)	2 (13.3%)	0.011*
CT Necrosis	8 (30.8%)	1 (6.7%)	0.076*
AMA Discharge	3 (11.5%)	3 (20.0%)	0.436*

** Fisher's exact test; † Mann-Whitney U test; SD = Standard Deviatio*

4. FIGURES

Figure 1: ROC Curves - BISAP and HAPS for SAP and Mortality Prediction

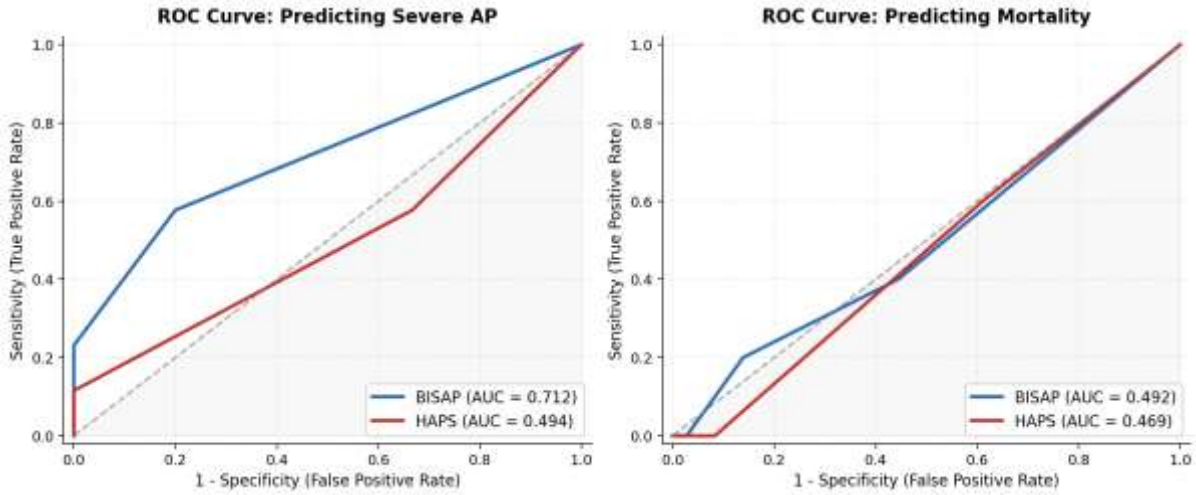

Figure 1. *Receiver Operating Characteristic (ROC) curves comparing BISAP and HAPS scoring systems for predicting (A) Severe Acute Pancreatitis and (B) In-hospital Mortality. BISAP demonstrates better discriminatory ability for SAP prediction (AUROC 0.712 vs 0.494); neither score performs above chance level for mortality prediction.*

Figure 2: Score Distributions

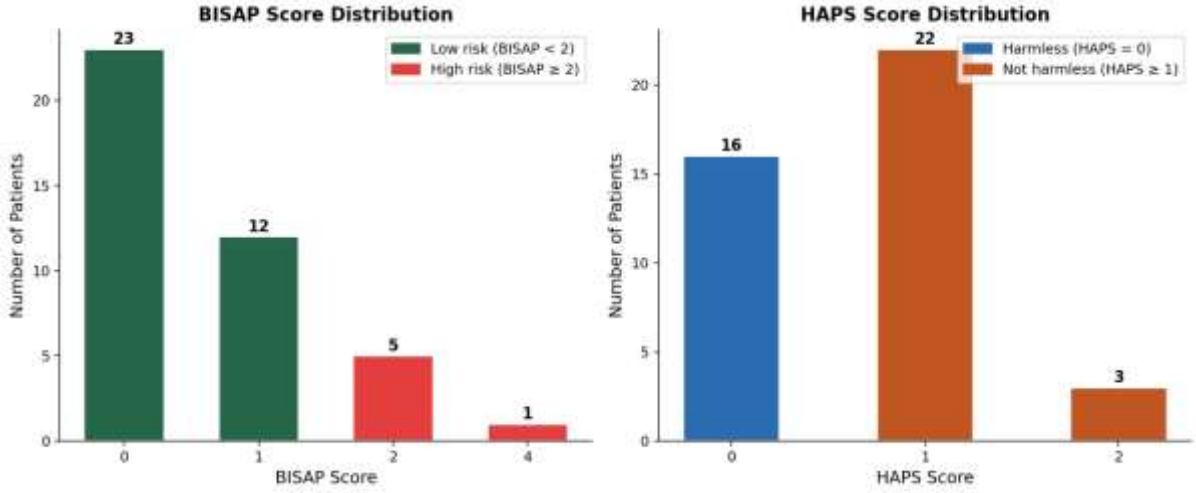

Figure 2*. Distribution of BISAP (left) and HAPS (right) scores among the 41 study patients. Green bars represent low-risk categories; red/orange bars represent elevated risk. The majority of patients had BISAP 0-1 and HAPS 0-1.*

Figure 3: Forest Plot - Diagnostic Performance

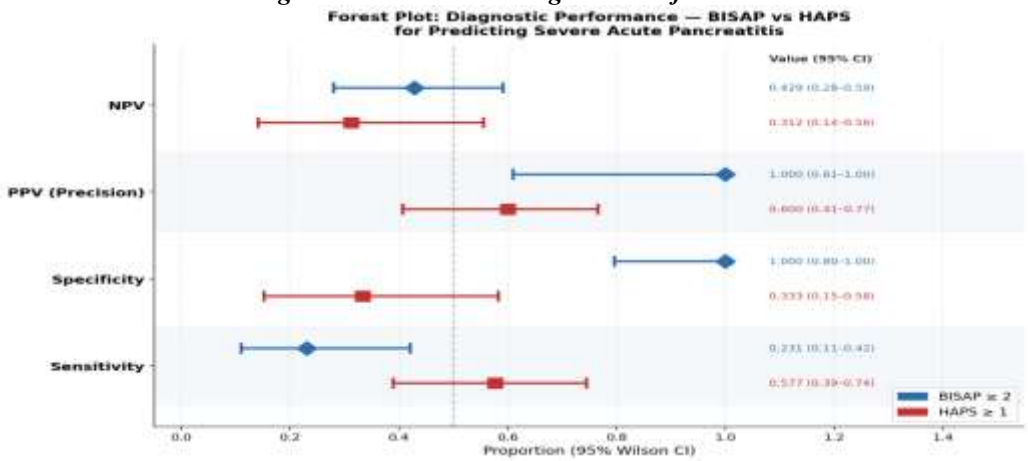

Figure 3. *Forest plot displaying sensitivity, specificity, PPV, and NPV with 95% Wilson confidence intervals for BISAP ≥2 (blue diamonds) and HAPS ≥1 (red squares) in predicting Severe Acute Pancreatitis. BISAP ≥2 achieves perfect specificity at the cost of low sensitivity.*

Figure 4: Clinical Outcomes by Severity Group

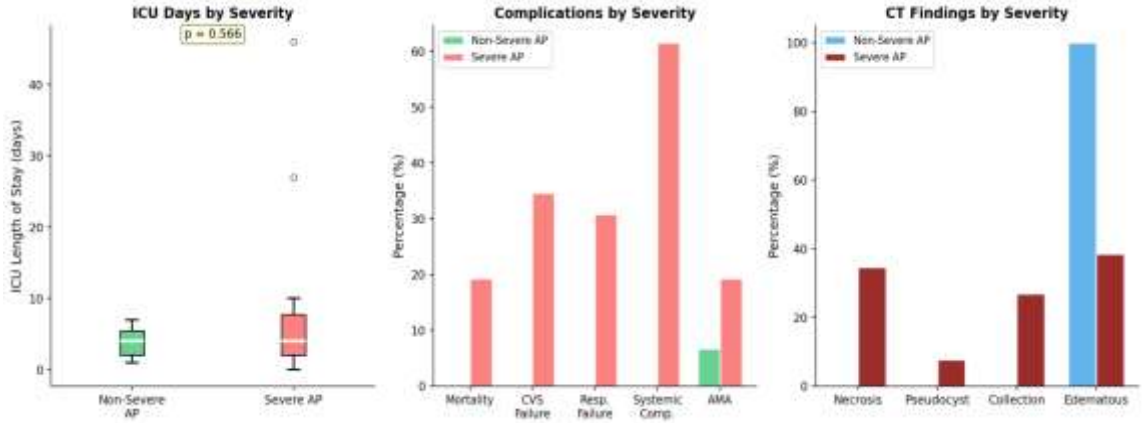

Figure 4. *Clinical outcomes stratified by severity. (Left) ICU length of stay by severity group (Mann-Whitney U test). (Centre) Complication rates by severity. (Right) CT imaging findings by severity group.*

5. DISCUSSION

This study compared BISAP and HAPS in patients admitted with acute pancreatitis. A large proportion of patients in our series developed severe disease. BISAP performed better than HAPS in identifying severe acute pancreatitis. The AUROC for BISAP was 0.712 compared with 0.494 for HAPS. A BISAP score of 2 or more showed very high specificity for severe disease. The high specificity observed with BISAP was similar to findings from previous studies. Wu et al. reported AUROC values of 0.82 to 0.83 for mortality prediction in three large patient cohorts [8]. Singh et al. reported AUROC values of 0.87 for organ failure and 0.91 for mortality in a prospective Indian study [10]. The lower AUROC seen in our patients was probably related to the small sample size and the wider confidence intervals associated with it. Sensitivity was considerably lower than specificity in our cohort. Only 42.9% of patients with severe acute pancreatitis had a BISAP score of 2 or more. More than half of the severe cases therefore had scores below this threshold. A BISAP score below 2 should not be used to exclude severe disease. Lowering the cutoff to 1 would increase sensitivity but would reduce specificity. HAPS showed poorer performance in our patients and the AUROC was close to 0.5. The score was originally developed to identify patients who were unlikely to develop severe pancreatitis rather than to predict severe disease itself. Lankisch et al. reported negative predictive values of 98% to 99% for HAPS 0 in prospective studies [9]. Severe acute pancreatitis was present in 63.4% of our cohort which was much higher than the proportion reported in most general populations with acute pancreatitis. Because predictive values change with disease prevalence the negative predictive value of HAPS was lower in our study. Eleven of the sixteen patients classified as harmless by HAPS later fulfilled criteria for severe acute pancreatitis. HAPS should therefore be used cautiously in populations with a high proportion of severe disease. Meshram et al. reported a mortality rate of 82.3% in a tertiary referral centre in New Delhi and reported perfect sensitivity and specificity of BISAP for mortality prediction in 102 patients [11]. Referral centres often receive transferred patients with more advanced disease and this may explain the higher mortality reported in that study. The mortality rate in our cohort was 12.2% which is closer to figures reported in unselected populations with acute pancreatitis.Previous studies have also reported good results with BISAP. Papachristou et al. reported AUROC values of 0.78 for severe acute pancreatitis and 0.83 for mortality in 185 patients [12]. A meta-analysis by Gao et al. pooling ten cohorts reported a sensitivity of 56% and a specificity of 91% for a BISAP score of 3 or more in predicting mortality, supporting its value as a bedside tool that can be calculated at admission [13]. A systematic review and meta-analysis of scoring systems for severe acute pancreatitis reported a pooled BISAP sensitivity of 74.4% and specificity of 81.5% across 43 studies [14]. A more recent meta-analysis involving 5476 patients reported pooled AUROC values of 0.94 for severity prediction and 0.92 for mortality prediction [15]. Similar to our findings BISAP showed high specificity but lower sensitivity than Ranson scoring. These findings carry practical implications for bedside triage. A BISAP score ≥2 at admission, given its high specificity in this cohort, can reasonably prompt closer monitoring, early ICU liaison, and more aggressive fluid resuscitation, even though a low score should not be used to defer escalation if clinical concern persists. HAPS, by contrast is best applied to patients with a low pre-test probability of severe disease, such as those presenting to lower-acuity or primary care settings, where a score of 0 may support a more conservative initial management plan [16]; in cohorts with a high baseline prevalence of SAP, as in this study population, HAPS is not a reliable tool for identifying patients suitable for early discharge. In practice, neither score should replace serial clinical reassessment, nor both are best used as adjuncts that flag patients warranting closer attention rather than as stand-alone gatekeepers for triage decisions. Our study has several limitations. The retrospective design and single-centre cohort limit generalisability. The sample size of 41 patients is relatively small; the study was underpowered to detect moderate differences between scores, and several comparisons that did not reach statistical significance (e.g., BISAP p=0.120, HAPS p=0.814) should be interpreted with this limitation in mind rather than as evidence of no effect. The resulting wide confidence intervals around all performance estimates further limit the precision of these findings. The high SAP prevalence (63.4%), well above the 15-20% typically reported in unselected AP populations, raises the possibility of referral or selection bias at our unit and fundamentally limits the applicability of HAPS, which was designed to identify a low-risk subgroup[17,18]. BISAP component scores were derived from routine clinical data, which may introduce measurement variability compared to prospective research-grade data collection. A formal comparison of baseline demographic and laboratory characteristics (age, sex, aetiology,

comorbidities, and admission laboratory values) between severe and non-severe groups would further strengthen this analysis and is recommended if such data can be made available. Given the limited sample size, all estimates of diagnostic performance reported here should be interpreted as exploratory and hypothesis-generating rather than definitive. Future work should include larger prospective datasets, multivariate predictive modelling incorporating both scores and biomarkers (e.g., CRP, procalcitonin, interleukin-6), and validation in diverse healthcare settings.

6. CONCLUSIONS

This retrospective study suggests that BISAP demonstrated better discrimination than HAPS for identifying severe acute pancreatitis in this cohort, achieving 100% specificity at a threshold of ≥2. However both scores demonstrate limitations as standalone risk stratification tools: BISAP's low sensitivity means it cannot independently rule out SAP, while HAPS is poorly suited to populations with high baseline SAP prevalence. Clinically, BISAP ≥2 is a useful red flag for escalating care intensity and early ICU referral. HAPS may retain utility in lower-acuity settings for identifying patients suitable for early discharge, but should not be used to exclude SAP when clinical concern is high. These scores should complement, rather than replace, serial clinical assessment and established clinical judgment. Larger prospective studies with standardised data collection are needed to define the optimal combined scoring strategy and to assess whether integration of additional inflammatory biomarkers can overcome the inherent limitations of these admission-based tools.

Conflict of Interest

The authors declare no conflict of interest.

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