ORIGINAL ARTICLE

Comparison of Harmless Acute Pancreatitis Score with Ranson's Score in Predicting the Severity of Acute Pancreatitis

Hamad Hadi Al-Qahtani[1], Mohammed Khurshid Alam[2] and Muhammad Waheed[1]

Abstract

Objective: To determine the predictability of harmless acute pancreatitis score (HAPS) in determining the severity of acute pancreatitis (AP) and compare it with Ranson's score.

Study Design: Prospective cohort study.

Place and Duration of Study: King Saud Medical City, Riyadh, Kingdom of Saudi Arabia, between January 2012 and December 2015.

Methodology: All patients admitted with AP at King Saud Medical City, Riyadh, during 2012 - 2015 were studied prospectively. Patients were assessed by HAPS and Ranson's score. Predictability values of the two systems were analysed and compared.

Results: Out of 116 patients studied, 104 (89.6%) were HAPS positive and predicted to have mild disease. Pancreatitis was mild in 101 (87%) but severe in 3 (2.6%) patients who scored ≥ 3 Ranson's criteria. Among 12 HAPS negative patients, 10 scored ≥ 3 Ranson's criteria and developed severe pancreatitis while 2 (1.7%) with 2 positive Ranson's criteria developed mild pancreatitis. HAPS correctly predicted the disease severity in 101 (87%) patients, a sensitivity of 98% specificity of 77% and accuracy of 96%. Ranson's system predicted correctly in all but took 48 hours for assessment. Statistical analysis showed moderate agreement (Kappa = 0.776, $p < 0.001$), and positive relation (rs = 0.777, $p < 0.001$) between the two scores.

Conclusion: HAPS is effective in rapid identification of patient who will run non-severe course of AP. Assessment can be completed within one hour from presentation. Ranson's score, although more accurate, takes 48 hours to complete.

Key Words: *Harmless acute pancreatitis score (HAPS). Ranson's score. Acute pancreatitis.*

INTRODUCTION

Acute pancreatitis (AP) is a common abdominal disorder which may represent a major challenge to the treating clinician.[1] It has been classified based on the severity into mild self-limiting acute edematous or rapidly deteriorating acute hemorrhagic necrotizing pancreatitis.[2] Severe acute pancreatitis may lead to multiorgan failure in 10 - 20 % of patients with a serious threat to life.[3] Although the diagnosis of AP is relatively easy where the clinical picture is aided by elevated serum enzymes levels with or without imaging in almost all patients. The main problem is the anticipation of the disease course which has a major impact on the ultimate outcome of the management. Early predictor of the disease severity is important to help triage the patient to an appropriate management setting and to avoid over or under resuscitation of the patients with the adverse outcome.[4]

[1] *Department of Surgery, College of Medicine, King Saud University, Riyadh, Kingdom of Saudi Arabia.*

[2] *Department of Surgery, AL Maarefa Medical College, Riyadh, Kingdom of Saudi Arabia.*

Correspondence: Dr. Hamad Hadi Al-Qahtani, Associate Professor and Consultant Hepatobiliary Surgeon, Department of Surgery, College of Medicine, King Saud University, Riyadh, Kingdom of Saudi Arabia.
E-mail: hamad_qah@hotmail.com

Received: August 05, 2016; Accepted: January 20, 2017.

An ideal predictor needs to be safe, economical, simple, highly sensitive, and specific which can be performed rapidly.[5,6] Several parameters and scoring algorithms have been studied so far including APACHE II, Ranson's[7] and Imrie scores.[4] The aim of these complex scoring algorithms is to predict AP with severe course that requires expensive and sophisticated management in the intensive care unit (ICU). However, these scoring systems have important limitations.[8] The Ranson's score contains data which is not collected routinely at the time of admission and needs 48 hours to be completed. Patients may deteriorate even before completion of assessment of the disease severity.[9] Recently, the harmless acute pancreatitis score (HAPS) has been introduced to identify AP with non-severe course. HAPS contains fewer parameters which can help stratify non-severe disease within a short time after presentation.[9,10] It can be easily remembered and applied in clinical practice to decide whether to admit these patients to a general ward or critical care unit. Its clinical indicator of guarding/rigidity can easily be determined by most experienced clinicians. Its two laboratory indicators can also be determined within an hour of patients' presentation and are generally available in most basic healthcare facilities. HAPS can be completed within one hour from the clinical assessment of the patients.[9]

The aim of this study was to determine the usefulness of HAPS in identifying patients who develop a mild AP, and

evaluate its predictability value in comparison with Ranson's score. No such study has been reported from this region.

METHODOLOGY

This prospective cohort study was conducted at King Saud Medical City, Riyadh, Kingdom of Saudi Arabia, between January 2012 and December 2015. Ethical approval was obtained from the Hospital Research Committee before commencement of the study. All consecutive patients with a primary diagnosis of first attack of AP, of either gender, over the age of 14 years, were included in the study. They were followed prospectively for 6 months after discharge from the hospital or till death, whichever was earlier. Patients were excluded from the study, if they refused participation, or had recurrent AP, or had known comorbid disorders of respiratory, cardiovascular or renal systems. The data was collected in a prepared proforma for the study. Informed consent was obtained from all patients.

Patients were assessed upon presentation to the emergency department (ED), by a board certified general surgeon. The diagnosis of AP was based on presence of upper abdominal pain and increased serum amylase (triple the normal level). Investigations obtained in the ED for all patients included: Complete blood count, hematocrit level, blood sugar, amylase, creatinine, blood urea nitrogen, liver function test (LFT), serum LDH, serum calcium, arterial blood gases, chest X-ray, and abdominal ultrasound. Computed tomography (CT) scan was used selectively for predicted severe disease. The etiology of AP was considered biliary, if imaging studies revealed gallstones disease; and alcoholic, if the patient reported frequent alcohol consumption. AP was labelled 'idiopathic' if it was non-biliary, non-alcoholic with a normal lipid and calcium level, and the patient was not on any medication known to cause AP.

All patients were evaluated according to HAPS (Table I) and Ranson's score. The patient was expected to have a harmless AP course in the absence of all three parameters (HAPS positive). Ranson's score (adjusted to etiology) data was collected at admission and completed at 48 hours. Patients with Ranson's score ≥ 3 were expected to have severe AP. All HAPS negative patients were admitted to ICU, and others were admitted to general surgical ward. At 48 hours, on completion of Ranson's assessment, those patients in general ward scoring ≥ 3 Ranson's criteria were transferred to ICU.

The initial management included nil orally, intravenous fluids, oxygen, narcotic analgesia, nasogastric intubation, if necessary, and continuous monitoring. Patients admitted to ICU were managed according to the critical care standards management protocols. Abdominal CT scan with oral and intravenous contrast was performed on all HAPS negative patients and those predicted to have severe AP by Ranson's score. CT findings were classified according to modified CT severity index (MCTSI).[11] Patients with persistent elevated LFTs for more than 2 - 3 days were submitted to MRCP ± ERCP. Intravenous imipenem were given to all patients with ≥3 Ranson's score at 48 hours or diagnosed/suspected to have infection. Oral feeding was introduced on clinical improvement and decline in serum amylase level. Cholecystectomy was performed during the index admission for all patients with mild acute gallstone pancreatitis. All patients were followed up in the outpatient department (OPD) with a weekly visit in the first month and a monthly visit in the next 5 months with clinical assessment and imaging studies, if indicated. They were discharged from the OPD, if remained completely asymptomatic. The following outcomes were studied: clinical features, diagnosis, imaging results, length of hospital stay, ICU admission, length of stay in ICU, local complications (fluid collections, necrosis), organs failure, need for organ support, nosocomial infection (pneumonia, urinary tract infections, infection of pancreatic necrosis, central line sepsis), the need for image guided interventions, necrosectomy, and mortality. Patients were classified to severe AP according to the revised Atlanta Criteria 2012.[12,13] Any organ failure for more than 48 hours, despite adequate resuscitation and support, was considered to be persistent organ failure.

Statistical analysis was performed using Statistical Package for the Social Sciences (SPSS) version 21.0 software (SPSS Inc., Chicago, IL, USA). Kolmogorov-Smirnov test of normality were used to check if scores are following normal distribution or not. Measure of agreement (Kappa) between the two scores (Ranson's score and HAPS) were calculated. The Spearman correlation coefficient between the two scores was also calculated. We used Fisher's exact test to find if there is association between CT findings and HAPS.

RESULTS

The study included 116 patients with a diagnosis of AP (Figure 1). Their mean age was 42 ±25.4 years (range 18 - 67 yeas) with a female to male ratio of 5:1 [97 (83.6%) females and 19 (16.4%) males. The median duration from the onset of symptoms to the ED presentation was 11 hours (range 5 - 27). Epigastric pain was the commonest presenting symptom, followed by nausea and vomiting. Yellowish discoloration of the sclera and dark urine was complained by 9 (7.8%) patients. Causes of AP among the study group were gallstones disease (n=102, 88%), alcohol intake (n=4, 3.5%), hyperlipidemia (n=2, 1.7%), and idiopathic (n=8, 7%). All patients were classified into HAPS positive or negative by a senior board certified general surgeon within one hour of arrival to ED. The assessment also included the Ranson's score at admission and at 48 hour.

All patients with positive HAPS (n=104, 89.6%) were admitted to the general surgical ward. Three of them scored > 3 positive Ranson's criteria at 48 hours after admission. They were transferred to the ICU for appropriate management. All three developed severe AP and ran a complicated course in ICU. HAPS were negative in 12 patients: All with positive epigastric rebound tenderness, 5 patients with abnormal hematocrit, and 2 patients with abnormal serum creatinine level. All HAPS negative patients (n=12, 10.4%) were admitted to the ICU. The initial (at admission) Ranson's scores did not identify any patients as severe AP. However, 10 out of 12 (83.3%) HAPS negative patients scored > 3 positive Ranson's criteria at 48 hours. All 10 patients ran a severe course in ICU. The remaining 2 (16.7%) HAPS negative patients scored 2 Ranson's criteria at 48 hours. Both had a mild AP. CT scans, performed on patients expected to have severe AP by both scores (n=15, 13%), have been classified by MCTSI. MRCP was performed on 9 patients with persistent elevated LFTs. Only one was found with an impacted stone at the lower end of common bile duct, which was removed by ERCP. This patient subsequently died of complications. Details of the patients who had severe AP are summarised in Table II. Hospital stay and mortality among patients with severe AP were 54 ±28 days and 5 out of 13 patients (38.5%) respectively.

Patients classified as mild AP (n=103, 89%) were treated in the surgical ward until complete resolution of the attack. Those with acute gallstone pancreatitis underwent cholecystectomy during the index admission. Non-

Table I: Harmless acute pancreatitis score.

	Score = 0 (Positive)	Score = 1 (Negative)
Epigastric rebound tenderness/guarding	No	Yes
Creatinine	<2 mg / dl	≥2 mg/dl
Hematocrit	<43% for male or 39.6% for female	≥43% for male or 39.6% for female

Positive HAPS: Absence of all criteria.
Negative HAPS: Presence of any of the criteria

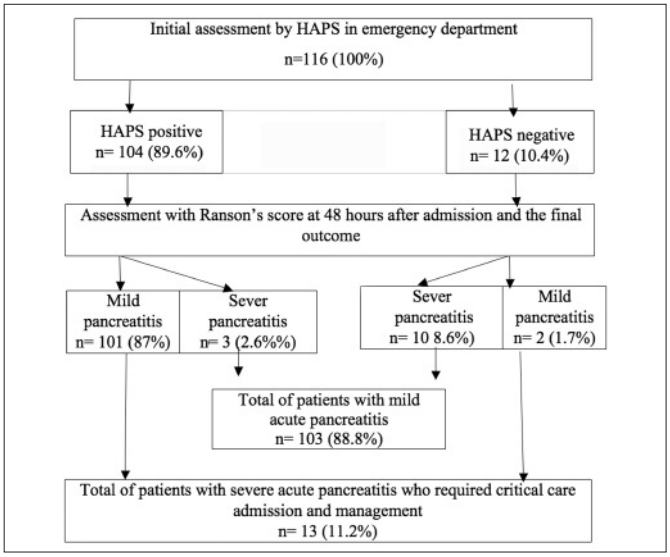

Figure 1: Algorithm for severity assessment of acute pancreatitis by HAPS and Ranson's score.

Table II: Details of patients with severe acute pancreatitis by both scores .

Age/sex	HAPS**	R.S*	Contrasted CT scan findings	MCTSI*** and the outcome	Long-term complications
52 / F	Negative	5	Pancreatic necrosis (> 30%), peripancreatic fluid collection with pleural effusion	MCTSI= 8 Transient renal failure which recovered with hemodialysis (survived)	None
47 / F	Negative	5	Oedematous pancreas with peripancreatic fluid collection with pleural effusion	MCTSI= 8 Transient respiratory and renal failure which recovered with artificial ventilation and hemodialysis respectively (survived)	Pseudocyst resolved with conservative treatment
41 / M	Negative	2	Oedematous pancreas with no fluid collection	MCTSI=2 Clinical and biochemical improvement within few days followed by LC**** during the index admission	None
61 / M	Negative	6	Pancreatic necrosis (> 30%), peripancreatic fluid collection, gas bubbles pleural effusion	MCTSI=10 Persistent multiorgan failure, and irreversible septic shock , underwent pancreatic necrosectomy	Died
38 / F	Positive	6	Patches of pancreatic necrosis (<30%) with peripancreatic fluid collections and gas bubbles and pleural effusion	MCTSI=8 Persistent multiorgan failure, and irreversible septic shock , underwent pancreatic necrosectomy	Died
33 / F	Negative	5	Oedematous pancreas with peripancreatic necrosis and fluid collection pleural effusion and ascites	MCTSI=8 Transient renal and respiratory failure, and hemodynamic instability, which recovered with hemodialysis, artificial ventilation and inotropic support (survived)	Pseudocyst , underwent open cystogastrostomy
49 / F	Negative	5	Oedematous pancreas with peripancreatic fluid collection pleural effusion	MCTSI=8 Transient renal failure which recovered with hemodialysis (survived)	None
55 / M	Negative	5	Emphysematous necrotic pancreas (>30%), ill-defined peripancreatic fluids and pleural effusion	MCTSI=8 Transient renal and respiratory failure, and hemodynamic instability which recovered with hemodialysis, artificial ventilation and inotropic support (survived)	Walled of necrosis, underwent open evacuation
34 / F	Negative	2	Oedematous pancreas with no fluid collection	MCTSI=2 Clinical and biochemical improvement within few days followed by LC during the index admission	None
46 / M	Negative	7	Pancreatic necrosis (>30%), peripancreatic fluids, gas bubbles	MCTSI=10 Persistent multiorgan failure, and irreversible septic shock underwent pancreatic necrosectomy	Died
48 / F	Negative	7	Pancreatic necrosis (<30%), peripancreatic fluids, gas bubbles	MCTSI=8 Persistent multiorgan failure and irreversible septic shock, pancreatic necrosectomy	Died
38 / F	Positive	6	Oedematous pancreas with acute peripancreatic fluid collection and pleural effusion	MCTSI=8 Transient renal failure which recovered with hemodialysis (survived)	None
35 / F	Positive	5	Oedematous pancreas with peripancreatic fluids and pleural effusion	MCTSI=8 Transient renal failure, which recovered with hemodialysis (survived)	WPN required delayed open evacuation
41 / F	Negative	7	Pancreatic necrosis (>30%), peripancreatic fluids, ascites	MCTSI=8 Massive pulmonary embolism	Died
59 / F	Negative	4	Pancreatic necrosis (<30%), peripancreatic fluids and pleural effusion	MCTSI=8 (survived)	None

** R.S: Ranson's score; ** HAPS: Harmless acute pancreatitis score; *** MCTSI: Modified computed tomography severity index; ****LC: Laparoscopic cholecystectomy*

biliary AP patients were discharged from the hospital after full recovery with an appropriate advice. The length of hospital stay among these patients was 5.4 ±1.2 days. There was no local or systemic complication, organ failure or mortality in these patients. HAPS has demonstrated a sensitivity of 98.06%, specificity of 77%, positive predictive value (PPV) of 97.1%, negative predictive value (NPV) of 83%, and accuracy of 96%.

All survived patients were followed-up in the OPD for 6 months. No patients with mild AP developed any late complication. However, 4 patients with severe AP presented with delayed local complications [n=2 (1.7%) walled of necrosis, n=2 (1.7%) pseudocyst] within 6 weeks from hospital discharge. Both patients with walled off necrosis required open surgical evacuation. One patient with pancreatic pseudocyst was treated with open cystogastrostomy while the other resolved spontaneously. All of them remained well during subsequent follow-up until discharge from the clinic.

The data of the two scores did not follow normal distribution since p-value more than 0.05. Measure of agreement (Kappa) between the two scores was 0.776 ($p<0.0001$). There was positive relation between them ($rs = 0.777$, $p<0.0001$). There was no significant difference between CT findings and HAPS ($p = 0.629$).

DISCUSSION

Majority of patients with AP run a mild course.[14] HAPS was developed to identify mild cases of AP within a short time from presentation. This provides clinician with a basis to confidently admit these patients to general ward for management. There is persistent shortage of ICU beds in most hospitals. HAPS can help minimise unnecessary admission to ICU. In the present study, HAPS correctly identified mild AP in 101 patients (87%), a very similar finding to other reported studies.[15] HAPS helped in the admission triage of these patients to an appropriate management setting within an hour of presentation. Only 3 patients, who were initially admitted to general ward, were subsequently transferred to ICU on the basis of Ranson's score. These patients subsequently developed severe illness and were managed in an appropriate setting. Swedish cohort study where HAPS could predict a non-severe course of acute pancreatitis with a specificity of 96.3% and PPV of 98.7%.[13] In this study, HAPS has demonstrated a sensitivity of 98.06%, specificity of 77%, PPV of 97.1%, NPV of 83%, and accuracy of 96%. The other advantage of HAPS system, observed by clinicians in this study, was the simplicity of its parameters which can easily be remembered and assessed.

Severe AP is likely to develop life-threatening local and systemic complications and higher mortality.[1,13] These patients need to be identified early and cared for in a critical care setup. HAPS was not developed to identify severe AP. However, in this study 10 out of 12 (83%) HAPS negative patients eventually developed severe disease. All HAPS negative patients (n =12) were initially admitted in ICU, only 2 were later transferred to general ward because of non-severe AP.

When we compared HAPS with Ranson's system, 10 HAPS negative and 3 HAPS positive patients (n=13) scored more than 3 Ranson's points. All developed severe AP. Ranson's score, although correctly predicted in all patients, took 48 hours to complete the classification of these patients. HAPS, on the other hand, correctly predicted the course of the disease in 111 patients (96%) within one hour of the arrival to the ED.

CT was used selectively in this study for patients expected by both scores to develop severe AP. The policy of selective use was planned to minimise unnecessary radiation, possible contrast toxicity, and the cost to the majority of patients with mild AP. Moreover, the severity of macroscopic pancreatic changes in CT scan do not always relate to the severity of the illness.[9] When HAPS was compared with MCTSI, 10 out of 12 HAPS negative patients and 3 from HAPS positive (n=104) patients developed severe disease based on MCTSI criteria. In this study, all those found to have severe AP by MCSTI criteria ran a severe course of the disease.

Gallstone is a leading cause of AP in Saudi Arabia.[16] This was also observed in the present study, where 88% of AP was due to gallstones. Majority of patients in this series were females and in their 4th decade of life, like other published series.[17,18]

All patients with mild AP (n=103) recovered within one week with no local or systemic complications. None developed any delayed complication. The incidence of severe AP in this study was 11%, which is lower than the other recently reported studies.[10,18] Local complications such as peripancreatic fluid collections, pseudocysts, abscesses, pancreatic necrosis, and multiorgan failure are the most important determinant of the ultimate outcome of severe AP.[18] The reported incidence of these complications in recent studies varies between 4 - 20%.[18,19] In this study, however, the incidence of pancreatic necrosis was 8%. All patients with > 3 points of Ranson's score developed local complications and/or organ failure. High mortality in these patients has been reported.[18,19] Among patients who have died (n=5) in this study, all had developed pancreatic necrosis and 4 of them also developed multiorgan failure and sepsis. Mortality rate among patients who developed pancreatic necrosis was 55%. All of those who died of complications had > 3 Ranson's score and were HAPS negative.

CONCLUSION

HAPS is an effective tool in early prediction of non-severe AP. It helps in the disposal of these patients to an

appropriate management setting. Moreover, its parameters are easy to remember; can be determined quickly and its laboratory components are available in most health facilities. Ranson's assessment system was more accurate in predicting the course of AP in this study. However, it needs 48 hours to complete the assessment process.

REFERENCES

1. Munsell MA, Buscaglla JM. Acute pancreatitis. *J Hosp Med* 2010; **5**:241-50.
2. Mofidi R, Patil PV, Suttie SA , Parks RW. Risk assessment in acute pancreatitis. *Br J Surg* 2009; **96**:137-50.
3. Van Santvoort HC, Besselink MG, Bakker OJ, Hofker HS, Boermeester MA, Dejong CH, *et al*. A step up approach or open necrosectomy for necrotizing pancreatitis. *N Engl J Med* 2010; **362**:1491-502.
4. Talukdar R, Nageshwar Reddy D. Predictors of adverse outcomes in acute pancreatitis: New horizons. *Indian J Gastroenterol* 2013; **32**:143-51.
5. Layer P. A simple index on the day of admission which predicts outcome in acute pancreatitis. *Gut* 2008 **57**:1645-6.
6. Wang S, Feng X, Li S, Liu C, Xu B, Bai B, *et al.* The ability of current scoring systems in differentiating transient and persistent organ failure in patients with acute pancreatitis. *J Crit Care* 2014; **29**:693.e7-11.
7. Nealon WH, Bawduniak J, Walser EM. Appropriate timing of cholecystectomy in patients who present with moderate to severe gallstone-associated acute pancreatitis with peri-pancreatic fluid collections. *Ann Surg* 2004; **239**:741-9.
8. Wahab S, Khan RA, Ahmad I, Wahab A. Imaging and clinical prognostic indicators of acute pancreatitis: A comparative insight. *Acta Gastroenterol Latinoam* 2010; **40**:283-7.
9. Lankisch PG, WeberDany 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; **7**:702-5.
10. Surco Y, Huerta Mercado J, Pinto J, Piscoya A, De Los Ríos R, Prochazka R, *et al.* Early prediction of severity in acute pancreatitis. *Rev Gastroenterol Peru* 2012 **32**:241-50.
11. Bollen TL, Singh VK, Maurer R, Repas K, van Es HW, Banks PA, *et al.* Comparative evaluation of the modified CT severity index and CT severity index in assessing severity of acute pancreatitis. *AJR Am J Roentgenol* 2011; **197**:386-92.
12. Thoeni RF. The revised Atlanta classification of acute pancreatitis: Its importance for the radiologist and its effect on treatment. *Radiology* 2012; **262**:751-64.
13. Banks PA, Bollen TL, Dervenis C, Gooszen HG, Johnson CD, Sarr MG, *et al.* Acute pancreatitis classification working group. Classification of acute pancreatitis 2012: Revision of the Atlanta classification and definitions by international consensus. *Gut* 2013; **62**:102-11.
14. Oskarsson V, Mehrabi M, Orsini N, Hammarqvist F, Segersvärd R, Andrén Sandberg A, *et al.* Validation of the harmless acute pancreatitis score in predicting non-severe course of acute pancreatitis. *Pancreatology* 2011; **11**:464-8.
15. 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 nonsevere course of acute pancreatitis: A pilot study in an Indian cohort. *Indian J Gastroenterol* 2014; **33**:316-21.
16. Abu Eshy SA. Pattern of acute pancreatitis. *Saudi Med J* 2001; **22**:215-8.
17. Afzal N, Iqbal A, Zafar I, Hassan M. Acute pancreatitis. *Professional Med J* 2010; **17**:199-204.
18. Shabbir S, Jamal S, Khaliq T, Khan ZM. Comparison of BISAP score with Ranson's score in determining the severity of acute pancreatitis. *J Coll Physicians Surg Pak* 2015; **25**:328-31.
19. Kaya E, Dervisoglu A, Polat C. Evaluation of diagnostic findings and scoring systems in outcome prediction in acute pancreatitis. *World J Gastroenterol* 2007; **13**:3090-4.