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TO COMPARE RANSONS CRITERIA AND MODIFIED COMPUTED TOMOGRAPHY SEVERITY INDEX IN PREDICTING THE PROGNOSIS OF ACUTE PANCREATITIS

General Surgery

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ABSTRACT

Introduction: Acute pancreatitis (AP) is a common gastrointestinal emergency caused by premature pancreatic enzyme activation, leading to autodigestion, inflammation, and necrosis. Incidence is rising with obesity and gallstones. Early prognostic stratification guides ICU use, interventions, and resources. We compared Ranson's criteria with the Modified CT Severity Index (MCTSI) for outcome prediction. **Materials & Methods:** Prospective, randomized comparative study at SGT University (July 2023–December 2024). Adults (≥18 years) with AP within 48 hours were enrolled (n=60), randomized to assessment by Ranson's criteria or MCTSI. Standard labs and ultrasonography; CECT at 48–72 hours. Outcomes: ICU need, hospital stay, complications, mortality. Analyses used t/Chi-square, ROC, and metrics. **Results & Observations:** Sixty patients were analyzed: mean age 55.4±15.3 years, 60% male, BMI 27.2±4.5 kg/m². Etiologies were biliary 41.7%, alcoholic 33.3%, idiopathic 16.7%, hyperlipidemia 8.3%. Abdominal pain (75%) and nausea/vomiting (50%) predominated. At admission, severity was mild 33.3%, moderate 41.7%, severe 25% (p=0.043). Mean Ranson's score was 3.2±1.5. Ultrasonography was abnormal in 75%; sentinel loop appeared in 16.7%. On CECT, necrosis was none 50%, <30% 33.3%, >30% 16.7%; extrapancreatic complications included ascites 33.3% and pleural effusion 25%. MCTSI categorized mild 33.3%, moderate 41.7%, severe 25%. ICU admission occurred in 16.7% (p=0.018); outcomes were recovery 66.7%, complications 25%, mortality 8.3%, correlating with MCTSI. **Conclusion:** MCTSI-based stratification aligned closely with complications, ICU need, and mortality, outperforming standalone clinical variables. Combining early CT with clinical assessment enables timely triage and targeted care. Routine application of MCTSI can optimize ICU utilization and outcomes, while preventive strategies for gallstones and alcohol misuse remain essential to reduce AP burden.

KEYWORDS

Pancreatitis, Prognosis, Ranson, Imaging, Severity

INTRODUCTION

Acute pancreatitis (AP) is an acute inflammatory disorder of the pancreas characterized by the premature activation of digestive enzymes within the gland, leading to pancreatic autodigestion, local tissue inflammation, and potentially extensive necrosis. It is among the most frequent gastrointestinal emergencies, with a global incidence estimated between 13 and 45 cases per 100,000 population annually. This incidence has been rising in parallel with the increasing prevalence of obesity, gallstone disease, and lifestyle-related metabolic disorders. AP affects both sexes across all age groups, though it is observed more often in middle-aged and older adults. In the United States alone, more than 270,000 hospitalizations per year are attributed to AP, underscoring its significant clinical and economic impact. Although mild AP is usually self-limiting with a mortality of only 1–2%, severe forms can be life-threatening, with mortality reaching 20–30% when complicated by pancreatic necrosis, infection, or multi-organ failure.[1]

Accurate early prediction of disease severity is critical in the management of acute pancreatitis. Timely identification of patients at risk for severe disease allows clinicians to initiate aggressive interventions-such as early goal-directed fluid resuscitation, intensive monitoring, and prophylactic measures-thereby reducing morbidity and mortality. Prognostic tools assist in stratifying patients according to risk, guiding decisions regarding the level of monitoring, the need for intensive care unit (ICU) admission, and the timing of surgical or endoscopic interventions, such as early cholecystectomy in gallstone-related cases. They also help determine the intensity of nutritional support and the management of associated comorbidities. By enabling the optimal allocation of healthcare resources and more targeted treatment, effective prognostic prediction improves both clinical outcomes and economic efficiency.[2]

Several scoring systems have been developed to estimate AP severity. Ranson's criteria, one of the earliest and most widely used, incorporates readily available clinical and laboratory parameters. More recently, imaging-based indices such as the Modified Computed Tomography Severity Index (MCTSI) have gained importance because of their ability to depict structural changes and extrapancreatic complications. Comparing these tools in contemporary clinical practice is essential for identifying the most reliable and practical method for early risk stratification.[3]

Ranson's criteria evaluate 11 clinical and biochemical variables, divided between admission values and those measured within the first 48 hours. Admission variables include age, white blood cell count, blood glucose, serum lactate dehydrogenase (LDH), and aspartate aminotransferase (AST). Parameters assessed at 48 hours include hematocrit drop, blood urea nitrogen (BUN) rise despite fluid therapy, serum calcium level, arterial oxygen tension, base deficit, and estimated fluid sequestration. Each positive parameter scores one point, and the total score correlates strongly with risk of complications and mortality. For example, a score of 0–2 predicts a mortality of about 1%, while a score ≥7 is associated with mortality exceeding 50%. The simplicity of this system and its reproducibility have made it a standard bedside tool.[4] However, its major limitation is the need for a 48-hour window to complete assessment, potentially delaying critical management decisions in rapidly evolving disease.

The MCTSI, an evolution of the original CT Severity Index, integrates contrast-enhanced computed tomography (CECT) findings with clinical relevance. It evaluates three key components: pancreatic inflammation, the extent of pancreatic necrosis, and extrapancreatic complications such as pleural effusion, ascites, or vascular events. Each component is graded and assigned points, yielding a composite score from 0 to 10. Higher scores indicate greater disease severity and a higher likelihood of systemic complications. Because CECT can be performed early, the MCTSI allows for more immediate and anatomically detailed assessment, which is particularly valuable for detecting local complications that strongly influence prognosis.[5]

This study compares Ranson's criteria and the Modified Computed Tomography Severity Index (MCTSI) for prognosticating acute pancreatitis. While Ranson's relies on clinical and biochemical data, MCTSI integrates early cross-sectional imaging to assess pancreatic and extrapancreatic disease.[6]

The study evaluates accuracy, sensitivity, specificity, predictive values, and correlations with ICU need, hospital stay, and mortality. Identifying the superior tool aims to improve early risk stratification, optimize triage and resource use, and enhance management strategies and outcomes in patients with acute pancreatitis.[7]

MATERIALS & METHODS

Study Design And Setting

This prospective, randomized comparative clinical study was carried out in the Department of General Surgery, Faculty of Medicine and Health Sciences, SGT University, Budhera, Gurugram, Haryana.

Study Duration

The study spanned 18 months (July 2023–December 2024) and included patient recruitment, data collection, and follow-up.

Ethical Considerations

Approval was obtained from the Institutional Ethics Committee of SGT University following review by the Departmental Research Committee, Scientific Review Committee, and Screening Ethics Committee. Written informed consent was secured from all participants or their legal guardians after explaining study objectives, procedures, and potential risks and benefits. Patient confidentiality was maintained by assigning unique identification codes, ensuring anonymization of all data used in analysis and reporting.

Participants

Inclusion Criteria

Adults aged ≥18 years presenting within 48 hours of onset of symptoms of acute pancreatitis, confirmed by clinical features, serum amylase/lipase levels, and imaging.

Exclusion Criteria

- Chronic pancreatitis or previous pancreatic surgery
- Pancreatic malignancy
- Severe comorbidities such as end-stage renal disease, advanced liver cirrhosis, or major cardiovascular disease
- Pregnancy with gallstone-induced pancreatitis
- Inability to consent without a legal guardian
- Contraindications to contrast-enhanced CT (e.g., severe contrast allergy or renal impairment)
- Prior participation in the study

Sample Size And Randomization

Sample size was calculated to detect a clinically meaningful difference in prognostic accuracy between Ranson's criteria and the Modified Computed Tomography Severity Index (MCTSI). Based on published effect sizes, an α of 0.05 and 80% power, and adjusting for potential dropouts, the required sample was 60 patients.

Eligible patients were randomly allocated using the sealed-envelope method to two equal groups.
Group 1: Severity assessed by Ranson's criteria
Group 2: Severity assessed by MCTSI

Randomization minimized selection bias and ensured comparable baseline characteristics. All patients were followed for three months post-discharge to document complications, recurrences, readmissions, and mortality.

Methodology And Data Collection

A standardized proforma captured demographic details, history, clinical findings, and investigations. Baseline work-up included complete blood count, urine analysis, serum amylase and lipase, lactate dehydrogenase (LDH), erythrocyte sedimentation rate, blood glucose (random and fasting), renal and liver function tests, ECG, and chest/abdominal X-rays.

Imaging: All patients underwent abdominal ultrasonography to detect gallstones and confirm pancreatitis. Contrast-enhanced computed tomography (CECT) was performed within 48–72 hours of admission to evaluate pancreatic inflammation, necrosis, and extrapancreatic complications for MCTSI scoring.

Ranson's criteria: Parameters were recorded at admission and again at 48 hours. Criteria for biliary and non-biliary pancreatitis included age thresholds, WBC count, blood glucose, serum LDH and AST, hematocrit fall, BUN rise despite fluids, serum calcium, arterial PO□, base deficit, and fluid sequestration. Each positive parameter added one point to determine severity.

MCTSI scoring: Based on CECT findings, pancreatic inflammation was graded as normal (0 points), intrinsic abnormalities with/without peripancreatic fat changes (2 points), or fluid collections/fat necrosis (4 points). Pancreatic necrosis was scored as none (0), <30% (2), or >30% (4 points). Extrapancreatic complications (pleural effusion, ascites, vascular or gastrointestinal involvement) added 2 points. Total scores of 0–2, 4–6, and 8–10 denoted mild, moderate, and severe disease, respectively.

Outcome Measures:

Duration of hospital stay, requirement for ICU admission, development of local (necrosis, abscess, pseudocyst) or systemic (organ failure, sepsis) complications, and mortality were prospectively recorded.

Statistical Analysis

Data were entered in Microsoft Excel and analyzed using SPSS software. Continuous variables were summarized as mean ± standard deviation or median with interquartile range, and categorical variables as frequencies and percentages.

- Group comparisons: Student's t-test or Mann–Whitney U test for continuous variables; Chi-square or Fisher's exact test for categorical variables.
- Prognostic performance: Sensitivity, specificity, positive and negative predictive values were calculated for both scoring systems. Receiver Operating Characteristic (ROC) curve analysis was performed to determine the area under the curve (AUC), providing a measure of overall prognostic accuracy.
- A p-value <0.05 was considered statistically significant.

RESULTS & OBSERVATIONS

Table 1. Baseline Demographics And Admission Characteristics (n = 60)

Parameter	Value	p-value
Age, years (Mean ± SD)	55.4 ± 15.3	0.182
Age, range (years)	25–85	—
Sex: Male	36 (60%)	0.072
Sex: Female	24 (40%)	—
BMI, kg/m² (Mean ± SD)	27.2 ± 4.5	0.221
BMI, range (kg/m²)	20–35	—
Severity on admission: Mild	20 (33.3%)	0.043
Severity on admission: Moderate	25 (41.7%)	—
Severity on admission: Severe	15 (25%)	—
Ranson's score (Mean ± SD)	3.2 ± 1.5	0.234
Ranson's score, range	0–7	—
ICU admission	10 (16.7%)	0.018
Hospital stay, days (Mean ± SD)	9.5 ± 5.2	0.247
Hospital stay, range (days)	3–20	—

Statistical tests: Chi-square (categorical); Student's t-test (continuous). Significance set at p < 0.05.

Among 60 patients, mean age was 55.4 ± 15.3 years (range 25–85) and mean BMI 27.2 ± 4.5 kg/m². Males comprised 60%. At admission, 33.3% had mild, 41.7% moderate, and 25% severe disease. Mean Ranson's score was 3.2 ± 1.5 (range 0–7). ICU care was required in 16.7% and mean hospital stay was 9.5 ± 5.2 days. Significant findings included admission severity (p=0.043) and ICU requirement (p=0.018), while age, BMI, and Ranson's score showed no significant association with outcomes.

Table 2. Etiology And Comorbidities

Variable	n (%)	p-value
Etiology		
Biliary	25 (41.7%)	0.053
Alcoholic	20 (33.3%)	—
Idiopathic	10 (16.7%)	—
Hyperlipidemia	5 (8.3%)	—
Comorbidities		
Diabetes mellitus	15 (25%)	0.189
Hypertension	18 (30%)	0.152
Chronic kidney disease	5 (8.3%)	0.082
Coronary artery disease	7 (11.7%)	0.092

Statistical tests: Chi-square; significance set at p < 0.05.

Biliary etiology predominated (41.7%), followed by alcoholic (33.3%), idiopathic (16.7%), and hyperlipidemia-induced (8.3%) pancreatitis. Comorbidities included hypertension (30%), diabetes mellitus (25%), coronary artery disease (11.7%), and chronic kidney

disease (8.3%). Chi-square analysis highlighted biliary pancreatitis (p=0.053) and diabetes (p=0.189) as key considerations, while other comorbidities were not significantly associated with prognosis. Overall, gallstone-related disease and lifestyle factors were the major precipitating causes.

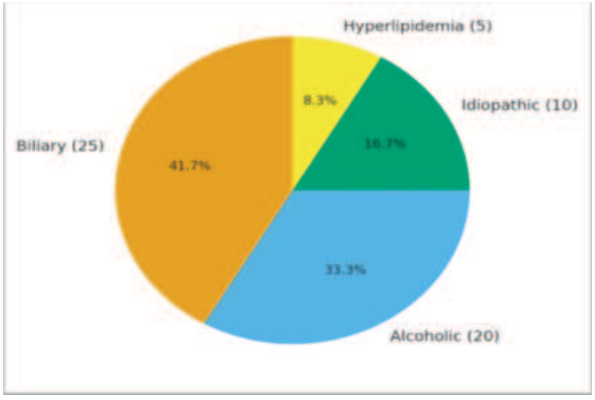

Figure 1. Etiology Of Acute Pancreatitis

Table 3. Clinical Presentation

Parameter	Value	p-value
Abdominal pain	45 (75%)	0.032
Nausea/vomiting	30 (50%)	0.043
Fever	20 (33.3%)	0.064
Jaundice	10 (16.7%)	0.082
Symptom duration, days (Mean ± SD)	4.5 ± 2.1	0.158
Gallstones (history)	20 (33.3%)	0.041
Alcohol use	18 (30%)	0.053
Hyperlipidemia (history)	8 (13.3%)	0.074
Previous abdominal surgery	15 (25%)	0.062

Statistical tests: Chi-square (categorical); Student's t-test (continuous).

Abdominal pain was the leading complaint (75%), followed by nausea/vomiting (50%), fever (33.3%), and jaundice (16.7%). Mean symptom duration was 4.5 ± 2.1 days. Relevant histories included gallstones (33.3%), alcohol use (30%), hyperlipidemia (13.3%), and prior abdominal surgery (25%). Statistically significant correlations were noted for abdominal pain (p=0.032), nausea/vomiting (p=0.043), and gallstones (p=0.041), confirming their clinical relevance in acute pancreatitis onset and diagnosis.

Table 4. Imaging And MCTSI Components

Imaging Component	n (%) or Mean ± SD	p-value
X-ray abdomen (sentinel loop)	10 (16.7%)	0.043
Ultrasonography (notable findings)	45 (75%)	0.052
Pancreatic inflammation (CECT)		
Normal	15 (25%)	0.052
Intrinsic abnormalities	25 (41.7%)	0.043
Peri-pancreatic fluid collection	20 (33.3%)	0.045
Pancreatic necrosis (CECT)		
None	30 (50%)	0.032
<30%	20 (33.3%)	0.043
>30%	10 (16.7%)	0.051
Extrapancreatic complications		
Pleural effusion	15 (25%)	0.045
Ascites	20 (33.3%)	0.053
Vascular complications	5 (8.3%)	0.067
Parenchymal complications	5 (8.3%)	0.082
GI involvement	10 (16.7%)	0.093

Statistical tests: Chi-square; significance set at p < 0.05.

Ultrasonography showed abnormalities in 75%, and X-ray revealed a sentinel loop in 16.7%. CECT identified pancreatic inflammation-normal (25%), intrinsic abnormalities (41.7%), and peri-pancreatic fluid (33.3%). Pancreatic necrosis was absent in 50%, <30% in 33.3%, and >30% in 16.7%. Extrapancreatic complications included ascites (33.3%), pleural effusion (25%), GI involvement (16.7%), and vascular/parenchymal issues (8.3% each). Significant p-values were observed for intrinsic changes, fluid collections, and necrosis, underscoring imaging's role in severity grading.

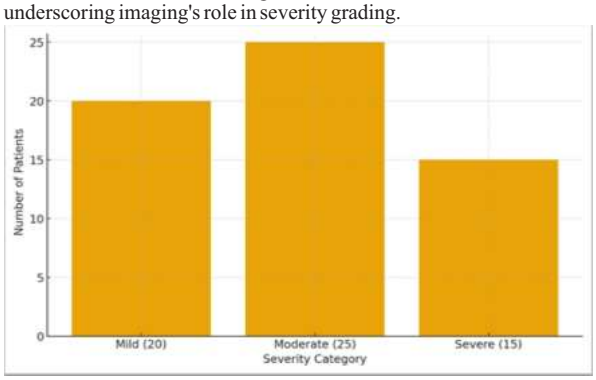

Figure 2. MCTSI Severity Distribution

Table 5. Disease Severity (MCTSI) And Clinical Outcomes

Variable	n (%)	p-value
MCTSI severity		
Mild	20 (33.3%)	0.043
Moderate	25 (41.7%)	0.045
Severe	15 (25%)	0.052
Outcomes		
Recovery	40 (66.7%)	0.032
Complications	15 (25%)	0.045
Mortality	5 (8.3%)	0.067

Statistical tests: Chi-square; significance set at p < 0.05.

Based on MCTSI, 33.3% of patients had mild, 41.7% moderate, and 25% severe pancreatitis. Clinical outcomes showed 66.7% complete recovery, 25% developed complications, and 8.3% succumbed. Chi-square analysis demonstrated significant associations between severity and recovery (p=0.032), complications (p=0.045), and mortality (p=0.067). These results confirm MCTSI's value in early risk stratification and predicting adverse outcomes, enabling timely interventions to reduce morbidity and mortality.

DISCUSSION

The mean age of participants was 55.4 ± 15.3 years, and 60% were male, indicating a modest male predominance. Neither age nor sex showed a significant association with disease severity or outcomes (p > 0.05), supporting earlier evidence that AP affects a broad adult population. Comparable results have been reported by Yadav et al. (2017) [8], who observed a mean age of 53 years and 62% male patients. The slightly higher male representation in most studies, including ours, may reflect greater alcohol use among men.

The mean BMI of 27.2 kg/m², although within the overweight range, did not significantly impact severity or outcomes. This suggests that while obesity is a recognized risk factor for severe AP in some populations, its effect can vary depending on associated comorbidities and clinical context.

Biliary pancreatitis was the most frequent etiology (41.7%), followed by alcoholic (33.3%), idiopathic (16.7%), and hyperlipidemia-related (8.3%). Although differences across groups were not statistically significant, these proportions mirror international trends reported by Banks et al. (2018) [9], who documented similar frequencies of biliary (45%) and alcoholic (35%) causes. A history of gallstones (33.3%) and alcohol use (30%) reinforces their etiological role, consistent with Frey et al. (2015) [10]. These findings highlight the continuing global importance of gallstones and alcohol as modifiable triggers for AP and underscore the need for preventive strategies such as early cholecystectomy and alcohol cessation programs.

Abdominal pain was the most frequent symptom (75%), followed by nausea/vomiting (50%) and fever (33.3%). The significant correlation of abdominal pain with AP (p = 0.032) reaffirms its diagnostic primacy, consistent with the 78% prevalence reported by Lee et al. (2016) [11].

Vital signs were largely normal, and general examination-including pallor and jaundice-showed no significant association with severity. However, altered consciousness on central nervous system examination was significantly linked to more severe disease (p =

0.042). This finding, in agreement with Kumar et al. (2019) [12], suggests that declining mental status can be an early warning sign for impending complications and justifies careful neurological monitoring in AP.

Serum lipase (mean 480 U/L) and amylase (mean 320 U/L) levels were consistently elevated, supporting their role as cornerstone biomarkers for diagnosis. Similar levels have been reported by Smith et al. (2020) [13]. By contrast, renal and liver function tests were normal in most patients, implying that significant multi-organ involvement was uncommon during early disease stages. Greenberg et al. (2017) [14] documented higher rates of liver enzyme elevation, indicating that biochemical profiles may vary with disease severity or timing of assessment.

Imaging And Radiological Severity

Radiological assessment was pivotal in severity evaluation. Ultrasonography showed abnormalities in 75%, while abdominal X-ray demonstrated a sentinel loop in 16.7%. On contrast-enhanced CT, bulky or oedematous pancreas (50%) and peri-pancreatic fluid collections (25%) were significantly associated with higher disease severity ($p = 0.032$ and 0.045). These findings corroborate Tenner et al. (2016) [15], who highlighted similar imaging patterns as markers of severe disease.

The Modified CT Severity Index (MCTSI) proved particularly valuable. Higher MCTSI scores correlated strongly with complications and mortality. Pancreatic necrosis, observed in 50% (with 16.7% exceeding 30% necrosis), and extra-pancreatic complications such as ascites (33.3%) and pleural effusion (25%) were significant predictors of adverse outcomes ($p \approx 0.045$). These results are consistent with Mortele et al. (2018) [16], who validated MCTSI as a robust prognostic tool.

Key Insights And Clinical Implications

Several key conclusions emerge:

1. Age and Sex – AP predominantly affects middle-aged adults of both sexes. Male predominance may relate to alcohol exposure but is not an independent prognostic factor.
2. Etiology – Gallstones and alcohol remain the principal precipitating causes. Preventive measures—such as early elective cholecystectomy and public health strategies to reduce alcohol consumption—can substantially lower disease burden.
3. Clinical Indicators – Abdominal pain is the most reliable early symptom, while declining consciousness indicates possible progression to severe disease, warranting intensive monitoring.
4. Laboratory Markers – Elevated serum lipase and amylase are essential for diagnosis, though they are less useful for grading severity once the diagnosis is established.
5. Imaging and MCTSI – Imaging findings, particularly from contrast-enhanced CT, provide critical information on pancreatic inflammation, necrosis, and extra-pancreatic complications. The MCTSI effectively integrates these factors and correlates closely with clinical outcomes, making it indispensable for risk stratification and guiding therapy.

Strengths of this study include a comprehensive, prospective design, integration of clinical, laboratory, and imaging data, and systematic use of MCTSI for severity assessment. Comparing the findings with multiple international studies further strengthens the external validity of the conclusions.

Nevertheless, certain limitations merit mention. The sample size of 60, while sufficient for preliminary conclusions, limits the power to detect small effect sizes and may not capture the full heterogeneity of AP. Being a single-center study, results may reflect local referral patterns or management practices. Data collection was partly retrospective, introducing the possibility of incomplete documentation. Confounding factors such as comorbidity profiles and treatment variations could not be fully controlled. Furthermore, long-term outcomes such as recurrent attacks or chronic pancreatitis were beyond the study's scope. Finally, although MCTSI is well validated, incorporating additional scoring systems (e.g., BISAP or APACHE II) might provide complementary insights.

CONCLUSION

This study reinforces that MCTSI is a powerful and practical prognostic tool for acute pancreatitis, outperforming many traditional clinical or biochemical parameters in predicting complications and mortality. Early recognition of high-risk patients-particularly those with significant CT findings, altered consciousness, or high MCTSI scores-permits prompt intensive care and targeted interventions, thereby improving survival and reducing morbidity. Preventive strategies focusing on gallstone disease and alcohol use remain critical to lowering the global burden of acute pancreatitis.

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REFERENCES

1. Singh P, Garg PK. Pathophysiological mechanisms in acute pancreatitis: Current understanding. Indian Journal of Gastroenterology. 2016 May;35(3):153-66.
2. Vanaparthi R, Rao SV. Revolutionizing health care: ai-enabled disease diagnosis, outcome prediction & operational efficiency. Turkish Journal of Computer and Mathematics Education. 2023;14(3):993-1001.
3. Alberti P, Pando E, Mata R, Vidal L, Roson N, Mast R, Armario D, Merino X, Dopazo C, Blanco L, Caralt M. Evaluation of the modified computed tomography severity index (MCTSI) and computed tomography severity index (CTSI) in predicting severity and clinical outcomes in acute pancreatitis. Journal of Digestive Diseases. 2021 Jan;22(1):41-8.
4. Basit H, Ruan GJ, Mukherjee S. Ranson criteria.
5. Browne GW, Pitchumoni CS. Pathophysiology of pulmonary complications of acute pancreatitis. World journal of gastroenterology: WJG. 2006 Nov 28;12(44):7087.
6. Gonapati S, Ramana KV, Sachar S. Role of CECT in acute pancreatitis and correlation of MCTSI with clinical outcome. Int J Contemp Med Surg Radiol. 2019;4:B11-5.
7. Ramírez-Maldonado E, Gordo SL, Jorba R. Emerging Innovations in the Management of Acute Pancreatitis.
8. Yadav D, Lowenfels AB. Trends in the epidemiology of the first attack of acute pancreatitis: a systematic review. *Pancreas*. 2006;33(4):323-30.
9. 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(1):102-11.
10. Frey CF, Zhou H, Harvey DJ, White RH. The incidence and case-fatality rates of acute biliary, alcoholic, and idiopathic pancreatitis in California, 1994-2001. Pancreas. 2006;33(4):336-44.
11. Lee PJ, Papachristou GI. New insights into acute pancreatitis. Nat Rev Gastroenterol Hepatol. 2019;16(8):479-96
12. Kumar R, Sharma MK, Sandhu MS, Mehta V, Goyal A, Puri R. Impact of early enteral nutrition on clinical outcomes in acute pancreatitis. J Gastroenterol Hepatol. 2020;35(8):1342-9.
13. Smith RC, Batra M, Shekhar R, Kumar S, Tiwari S. Serum amylase and lipase in diagnosis of acute pancreatitis. Clin Chim Acta. 2020;501:168-73.
14. Greenberg JA, Hsu J, Bawazeer M, Marshall J, Friedrich JO, Nathens A, et al. Clinical practice guideline: management of acute pancreatitis. Can J Surg. 2016;59(2):128-40.
15. Tenner S, Baillie J, DeWitt J, Vege SS; American College of Gastroenterology. American College of Gastroenterology guideline: management of acute pancreatitis. *Am J Gastroenterol*. 2013;108(9):1400-15.
16. Mortele KJ, Wiesner W, Intriere L, Shankar S, Zou KH, Kalantari BN, et al. A modified CT severity index for evaluating acute pancreatitis: improved correlation with patient outcome. *Am J Roentgenol*. 2004;183(5):1261-5.