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Management of Acute Biliary Pancreatitis in Practice Setting of a Multidisciplinary Hospital

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ABSTRACT: Management of acute biliary pancreatitis involves collaboration between medical specialties-surgery, gastroenterology, interventional endoscopy, intensive care in different algorithms. We conducted an analytical retrospective study of 139 subjects diagnosed with acute biliary pancreatitis admitted in the time interval between January 2017 and September 2025. Pancreatitis form was mild or moderate in 128 cases and severe from the onset in 11 cases. All patients benefited from early conservative treatment and most (109) from ERCP and/or cholecystectomy during hospitalization, with favorable outcome in all cases of mild and moderate pancreatitis. Statistical analysis showed that for predictors of severe disease, prolonged evolution and poor outcome at admission, only glycemia was significantly higher in patients with unfavorable evolution (p=0.031). At 48 hours, statistically significant differences were identified for urea (p=0.0047), fibrinogen (p=0.041), lipase (p=0.049), and glycemia (p=0.01). At 72 hours, significant differences persisted for urea (p=0.0024), fibrinogen (p=0.018), and lipase (p=0.016). Exploratory predictive analysis performed showed urea at 72 hours with the strongest positive association with unfavorable clinical outcome (OR 1.06) in the logistic regression model, while Random Forest analysis identified urea at 72 hours, lipase at 72 hours, and admission laboratory parameters as the most relevant contributors to unfavorable clinical evolution. Both tested models have limited predictive performance, and the findings should be interpreted as exploratory. The results emphasize the value of a stepwise, multidisciplinary management strategy combined with dynamic biological assessment for optimizing therapeutic decisions and prognostic evaluation in acute biliary pancreatitis.

KEYWORDS: *Acute biliary pancreatitis, cholecystectomy, ERCP.*

Introduction

Acute biliary pancreatitis is an inflammatory condition in which the inflammation of the pancreatic parenchyma is due to the migration of one or more gallstones from the gallbladder or the bile duct to the ampulla of Vater, with the consequent obstruction of the pancreatic duct, and of the biliary duct in most cases. This event triggers complex physiopathological processes that involve the biliary tree, the gallbladder, the liver and the pancreatic tissue, the extent of which can be of minor or larger development, ranging from mild inflammation to severe pancreatic tissue damage. About 40% of all forms of pancreatitis are of biliary origin. Of these, 80% are mild or medium forms, and the remaining 20% severe forms [1,2,3,4,5].

Management of acute biliary pancreatitis has a treatment algorithm that involves collaboration between medical specialties-surgery, gastroenterology, interventional endoscopy, intensive care and that evolved over time along with the development of different treatment strategies and technologies, as well as intensive care possibilities. This algorithm is still up to debate as to how the treatment steps are involved and the timing of those steps [1,2,3,4,6].

Patients are admitted in the surgery or intensive therapy units and are subjected to initial medical treatment simultaneously with clinical and paraclinical examination. As part of this initial treatment, the prognostic assessment of the severity of acute pancreatitis takes place according to the Ranson criteria and the stratification of patients [5].

The management of severe forms of acute pancreatitis with one or more organ failure (circulatory, pulmonary or renal) must be done in intensive care units so that patients with severe pancreatitis (Ranson score ≥3) are urgently transferred to intensive care units and patients with mild pancreatitis (Ranson score>3), who have oedematous pancreatitis will be treated on the general surgery wards [5].

The treatment options of acute biliary pancreatitis involve the following:

Conservative-treatment objectives

1. Supportive therapy: appropriate hydroelectrolytic rebalancing, maintaining diuresis >0.5ml/kg, supplementing with oxygen (maintaining SaO2 >95%), nasogastric tube only in case of intractable vomiting

10.12865/CHSJ.52.01.10

2. Reduction of pancreatic secretion: through digestive rest-oral feeding of patients is possible after 48 hours from the cessation of pain; in case of moderate and severe forms of acute pancreatitis nutritional support through an enteral tube is indicated, in case of paralytic ileus parenteral nutrition is used.

3. Pain relief-ranging from ordinary analgesics up to continuous pain relief through peridural catheter

4. Prevention of complications. There are no specific therapies to ensure prevention of complications. Antibiotherapy is not currently used for prophylactic purposes.

Endoscopic de-obstruction

It has been proven that a large number of cases of acute biliary pancreatitis evolve with spontaneous transpapillary passage of the obstructive gallstone, more frequently in the first 48 hours and much less likely thereafter.

De-obstruction of the common bile duct in case of acute cholangitis is an element that considerably improves the prognosis of an acute biliary pancreatitis, decreasing the rate of septic complications.

Thus, ERCP-endoscopic retrograde cholangiopancreatography and SE-endoscopic sphincterotomy performed in the first 3 days reduces morbidity and length of hospitalization (but not mortality) in acute biliary pancreatitis.

The subsequent treatment will be directly related to the severity of the pancreatic inflammation and the presence of obstructive jaundice or acute cholangitis [5,7,8,9,10].

Surgical treatment

Studies have shown that surgery in acute biliary pancreatitis performed within the first 48 hours increases the mortality rate regardless of the severity of the pancreatitis.

In case of severe acute pancreatitis (Ranson score >3), complicated with obstructive jaundice or acute cholangitis, surgical intervention can aggravate the evolution of pancreatitis. In such a situation, surgical intervention should be avoided, and the biliary complication should be resolved by endoscopic retrograde cholangiography (ERCP) and endoscopic sphincterotomy (ES). Cholecystectomy should be postponed and can be performed after at least 6 weeks after the patient's recovery from pancreatitis and discharge [5].

Patients with oedematous pancreatitis have a positive evolution, in 3-5 days the inflammation symptoms resolve and the patients can be either discharged with indication to undergo cholecystectomy, or in cases of mild pancreatitis, cholecystectomy can be performed during the same hospitalization.

In such situations, recurrence of acute pancreatitis was frequently observed in 29-63% of cases, if cholecystectomy was not performed [11,12,13].

For these reasons, the term of cholecystectomy depends on the clinical evolution of acute biliary pancreatitis. In case of mild pancreatitis, if gallstones have been confirmed by ultrasound, cholecystectomy is indicated during the same period trade-winds [5,6,14,15].

Cholecystectomy in patients with acute pancreatitis is rational to be solved laparoscopically. The success rate is between 80 and 100% with a conversion rate between 0 and 16% [15,16,17].

Thus, cholecystectomy should be performed to avoid recurrence of acute biliary pancreatitis.

In case of acute biliary pancreatitis with mild evolution, the surgical intervention must be performed after the normalization of the analyses and ideally during the same hospitalization. In severe biliary pancreatitis cholecystectomy will be postponed until there is a resolution of the inflammatory response and sufficient clinical recovery [5].

Materials and methods

We conducted an analytical retrospective study regarding patients diagnosed with acute biliary pancreatitis admitted to the Ist Surgery Clinic at the University Emergency Hospital Bucharest in the time interval between January 2017 and September 2025, in order to review the current state of treatment options and the usage of treatment algorithms in practice.

The study enrolled a number of 139 subjects included in the study of which 90 were females, 49 males.

An analytical survey of the database for predictors of severe disease, prolonged evolution and poor outcome was conducted using univariate analysis. We used the threshold of 7 days hospital stay as unfavorable evolution, as most mild biliary pancreatitis are discharged in this time interval; also, cases with severe pancreatitis that had a fulminant evolution to death were included as unfavorable evolution.

An exploratory predictive analysis was conducted to assess the ability of selected clinical and biological variables to predict unfavorable clinical evolution, defined as death and/or prolonged hospitalization (>7 days).

Two multivariable prediction models were developed, one using logistic regression and one with the aid of supervised machine learning, using a Random Forest algorithm. Continuous variables were standardized where appropriate, and missing data were handled using median imputation.

Machine learning analysis was performed using Python with scikit-learn libraries. The outcome was defined as a composite of in-hospital death or prolonged hospitalization (>7 days).

The dataset was cleaned and missing values were handled using median imputation.

Continuous variables were used as predictors, including age, urea, fibrinogen, leukocyte count, glucose, lipase, and amylase.

Data were split into training and testing sets using stratified sampling (70/30 split) to preserve class distribution. Logistic regression was applied with standardized variables. A Random Forest classifier was used to explore nonlinear relationships. No extensive hyperparameter tuning was performed due to the exploratory nature of the analysis.

The dataset was evaluated using stratified cross-validation to improve robustness. Given the class imbalance (115 unfavorable vs 24 favorable cases) and the number of variables relative to the sample size, the multivariable model may be subject to overfitting and should be interpreted cautiously.

Model performance was assessed using the area under the receiver operating characteristic curve (AUC-ROC), accuracy, and F1-score, with AUC-ROC considered the primary metric.

Feature importance was analyzed using the Random Forest model.

No external validation cohort was available.

Given the limited sample size, all machine learning analyses were considered exploratory.

Results

Descriptive statistics:

- From the study group, all 139 patients benefited from early conservative treatment and follow-up in the same hospital setting

- Pancreatitis form defined by the prognostic scores used were mostly mild or moderate forms (128 cases), 11 cases with severe forms from the onset

- 35 cases required ERCP (performed between 0 and 13 days after onset, with 8 cases requiring early endoscopic etiologic treatment within 48 hours of admission), 107 benefited from cholecystectomy during hospitalization (between day 2 and 20 after hospitalization, after normalization of pancreatic blood enzymes)

- The outcome was favorable (patients were discharged in good condition) in all 128 cases of mild and moderate pancreatitis, from which 24 patients were within the range of 7 days or less hospital stay and 104 patients required longer than 7 days of hospitalization. The outcome was poor in the 11 cases of severe pancreatitis resulting in the patients' demise. We considered that death and the threshold of 7 days hospitalization as unfavorable evolution.

Therapeutic management involved

1. Early medical conservative treatment-supportive therapy, reduction of pancreatic secretion, pain relief, prevention of complications-in our group all patients underwent this step, including intensive therapy, pain therapy and advanced support of vital functions adapted to the severity of the disease and intensity of pain symptoms.

This included early intensive parenteral hydration (more than 2 l PEV/24 hours), food rest, analgesics, antispasmodic, antiemetics, gastric antisecretory drugs, including antibiotics in most cases. Despite the fact that prophylactic antibiotic therapy no longer has a firm indication at present, the majority of patients in our cohort required antibiotic therapy for the treatment of associated acute cholecystitis. The objectives of the treatment were to maintain an adequate water balance with diuresis greater than 0.5ml/kg, minimizing pain, maintaining vital functions within physiological limits, the possibility of early oral feeding to avoid infectious complications, normalization of the inflammatory syndrome and of the value of blood pancreatic enzymes. More than half of the cases in the study group had a rapid favorable evolution within 48-72 hours of the initiation of treatment, allowing continuation of the etiological treatment.

2. The etiological treatment of pancreatitis-decompression of the main bile duct and removal of the stone reservoir (gallbladder)-among the cases studied, 8 had an indication for early endoscopic retrograde cholangiopancreatography (ERCP) for CBP decompression (in the first 48 hours)-papillosphincterotomy with the extraction of bile stones obstruction and subsequently underwent laparoscopic cholecystectomy at a later time. The surgical therapy is differentiated according to the severity of the pancreatitis and the response to conservative medical treatment, with or without minimally invasive exploration of the CBP:

10.12865/CHSJ.52.01.10

- laparoscopic cholecystectomy after remission of the clinical and biological syndrome in mild forms-early after 48h (average first 5 days)-70 cases were self-limited mild forms that benefited from early cholecystectomy, 37 had medium forms that allowed cholecystectomy t more of 7 days from the onset

- delayed surgery (10-14 days) for severe forms: open/laparoscopic cholecystectomy+ treatment of choledochal lithiasis+glandular interventions in cases with infected necrosis.

In our group, among the four deaths with severe forms from the onset, one case was of high severity, with death less than 24 hours after presentation, for the other three cases open intervention was preferred due to compartment syndrome at variable time intervals, without therapeutic success.

In addition, 32 cases in the group did not benefit from surgical intervention, either due to previous cholecystectomy, or serious pathology associated with major operative risk, or the presence of an evolving pregnancy in the first trimester; among these, two cases presented residual CBP lithiasis that required ERCP.

Analytical statistics

An analytical survey of the database for predictors of severe disease, prolonged evolution and poor outcome was conducted, the results shown in tables 1,2,3. When comparing the biological parameters of patients with unfavorable evolution versus patients with favorable evolution, results with an alpha level of 0.05 were considered statistically significant ($p \leq 0.05$).

Table 1. Univariate analysis of admission data set.

Admission	Unfavorable evolution			Favorable evolution			p Mann-
Variables	**Min**	**Max**	**Mean±Std.dev.**	**Min**	**Max**	**Mean±Std.dev.**	**Whitney**
Serum Amylase	29	6767	1459.80±1196.32	111	5272	1356.96±1546.43	0.3078
Lipase	17	25054	4447.69±4606.24	336	18971	3603.50±4713.02	0.1289
Total Bilirubin	0.27	10.39	2.80±2.17	0.44	8.83	2.45±2.04	0.4654
Direct Bilirubin	0.08	6.1	1.56±1.49	0.09	5.55	1.34±1.35	0.4569
AST	7	1107	262.57±210.28	13	669	232.92±182.42	0.5754
ALT	15	912	292.59±219.15	22	1111	309.88±278.79	0.9622
Urea	13	154.2	47.99±29.47	17	96	36.82±18.75	**0.0525**
Creatinine	0.43	4.63	1.12±0.68	0.48	1.84	0.91±0.31	0.3391
Fibrinogen	243	834	428.83±121.42	312	495	398.67±55.37	0.6219
Leukocyte	4.5	38.3	13.42±5.92	6.2	21.8	11.33±3.82	0.1783
Glucose	49	710	161.29±96.50	71	309	130.29±59.21	**0.0312**
Serum Na+	124	147	138.76±3.64	128	147	139.63±4.18	0.2275
Serum K+	2.6	5.7	4.03±0.57	2.72	6.19	4.12±0.62	0.5234

Table 2. Univariate analysis of 48 h data set.

48 hours	Unfavorable evolution			Favorable evolution			p Mann-
Variables	**Min**	**Max**	**Mean±Std.dev.**	**Min**	**Max**	**Mean±Std.dev.**	**Whitney**
Serum Amylase	18	3514	473.50±540.58	34	1710	305.79±401.81	0.0753
Lipase	14	9989	946.93±1565.32	26	1563	387.21±460.91	**0.049**
Total Bilirubin	0.24	10.76	2.25±2.08	0.45	7.83	1.90±1.90	0.2236
Direct Bilirubin	0.1	8.7	1.28±1.59	0.09	4.62	1.01±1.26	0.1593
AST	13	879	113.01±135.18	12	405	104.75±91.23	0.8286
ALT	11	631	197.06±149.44	15	688	244.88±204.75	0.4973
Urea	11	212	47.88±34.06	8.7	65.3	29.40±13.27	**0.0047**
Creatinine	0.32	5.59	1.19±0.86	0.47	1.52	0.86±0.26	0.1148
Fibrinogen	206	811	455.72±134.61	188	769	394.57±106.80	**0.0414**
Leukocyte	4.2	37.2	12.58±6.26	5.2	18.5	9.61±3.31	0.06
Glucose	47	376	111.34±55.11	40	145	84.08±23.19	**0.0139**
Serum Na+	126	150	139.91±3.99	129	144	140.46±3.09	0.3539
Serum K+	2.8	6.1	3.90±0.57	3.18	4.92	3.99±0.39	0.15

Table 3. Univariate analysis of 72 h data set.

72 hours	Unfavorable evolution			Favorable evolution			p Mann-
Variables	**Min**	**Max**	**Mean±Std.dev.**	**Min**	**Max**	**Mean±Std.dev.**	**Whitney**
Serum Amylase	17	11325	306.37±1124.94	28	3197	262.21±712.31	0.3537
Lipase	13	91457	1282.20±9001.62	13	227	97.00±70.43	**0.0162**
Total Bilirubin	0.22	8.6	1.56±1.55	0.33	5.34	1.42±1.23	0.6809
Direct Bilirubin	0.07	5.8	0.83±1.08	0.08	2.68	0.74±0.79	0.7417
AST	6	1627	68.16±161.89	6	340	70.85±78.19	0.3519
ALT	11	950	132.24±134.45	25	570	180.55±146.69	0.085
Urea	8	225.7	41.22±33.35	8.8	43.6	22.76±9.83	**0.0024**
Creatinine	0.44	4.74	1.06±0.73	0.5	1.44	0.81±0.21	0.3449
Fibrinogen	215	1037	488.04±159.30	304	658	411.95±101.25	**0.0187**
Leukocyte	3.4	34.1	11.47±5.60	3.8	20.1	9.86±4.06	0.2892
Glucose	31	285	103.33±46.12	50	197	86.80±32.21	0.082
Serum Na+	128	151	140.51±3.56	136	146	140.70±2.99	0.9537
Serum K+	2.71	5.43	3.81±0.53	3.12	4.85	3.92±0.38	0.1978

At admission (Table 1), no statistically significant differences were observed between patients with favorable and unfavorable evolution for amylase (p=0.307), lipase (p=0.128), total bilirubin (p=0.465), AST (p=0.575), and ALT (p=0.962). Admission glycemia was significantly higher in patients with unfavorable evolution (p=0.031).

At 48 hours (Table 2), statistically significant differences were identified for urea (p=0.0047), fibrinogen (p=0.041), lipase (p=0.049), and glycemia (p=0.01), with higher values in patients with unfavorable evolution.

At 72 hours (Table 3), significant differences persisted for urea (p=0.0024), fibrinogen (p=0.018), and lipase (p=0.016).

Other non-numerical data analyzed were imagistic diagnosis studies. Abdominal ultrasound was performed in all 139 patients (100%). Computed tomography (CT) was performed in 77 patients (55.4%), mainly in cases with more severe clinical evolution or in diagnostic uncertainties. CT use was not significantly associated with ICU admission (p=0.10) or with the need for ERCP (p=0.41).

Mortality occurred in 9 patients who underwent CT compared with 2 patients without CT examination; however, the difference was not statistically significant (p=0.13). Magnetic resonance cholangiopancreatography (MRCP) was performed in 4 patients (2.9%) and was selectively indicated in cases with suspected common bile duct lithiasis. No significant association was found between MRCP and ICU admission (p=0.77) or mortality (p=1.00). MRCP was more frequently associated with ERCP, although the difference did not reach statistical significance (p=0.08). Endoscopic ultrasound (EUS) was performed in 18 patients (12.9%), mainly for the evaluation of suspected choledocholithiasis. No statistically significant association was observed between EUS and ICU admission (p=1.00) or mortality (p=0.39).

Patients evaluated by EUS underwent ERCP more frequently, although this difference was not statistically significant (p=0.25).

No statistically significant associations were observed between clinical evolution and age (p=0.059), common bile duct diameter (p=0.77), presence of choledocholithiasis (p=0.13).

ERCP was performed in 35 patients; no statistical significance was found between the favorable and unfavorable evolution groups (p=0.11), nor between mortality and survival groups (p=0,358).

Exploratory predictive analysis

An exploratory predictive analysis was conducted to evaluate the ability of selected clinical and biological parameters to predict unfavorable clinical evolution, defined as prolonged hospitalization (>7 days) and/or death.

Two models were developed: a multivariable logistic regression model and one using a Random Forest algorithm.

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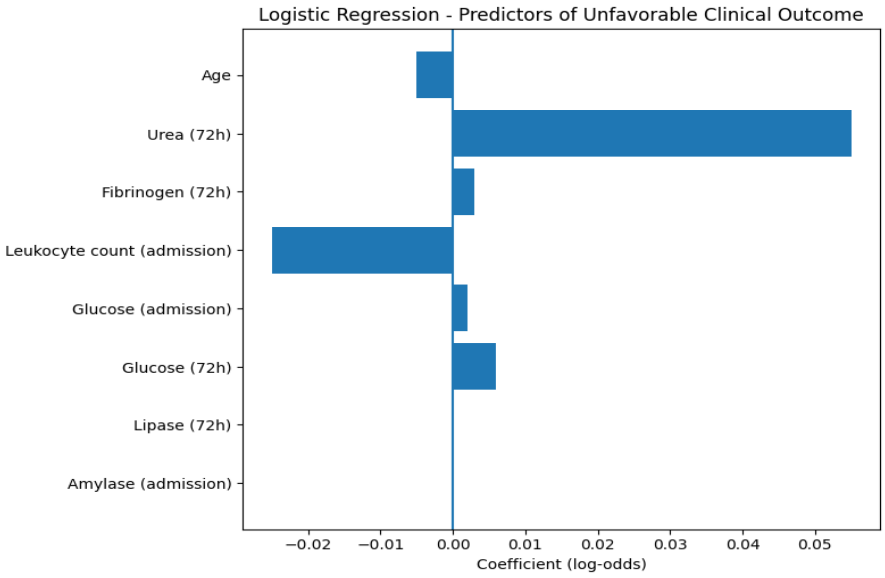

Figure 1. Logistic regression coefficients for predictors of unfavorable clinical outcome. Positive coefficients indicate increased odds, while negative coefficients suggest a potential protective effect.

Logistic regression analysis showed that urea at 72 hours had the strongest positive association with unfavorable clinical outcome (OR 1.06, 95% CI 0.99-1.12). Fibrinogen and glucose levels demonstrated minimal positive effects (OR≈1.00-1.01 for fibrinogen, OR≈1.01 for glucose), while leukocyte count at admission showed a negative association. Other variables, including age and pancreatic enzymes, had negligible contributions (Figure 1).

Overall, the model showed limited predictive capacity, with no independent predictors identified.

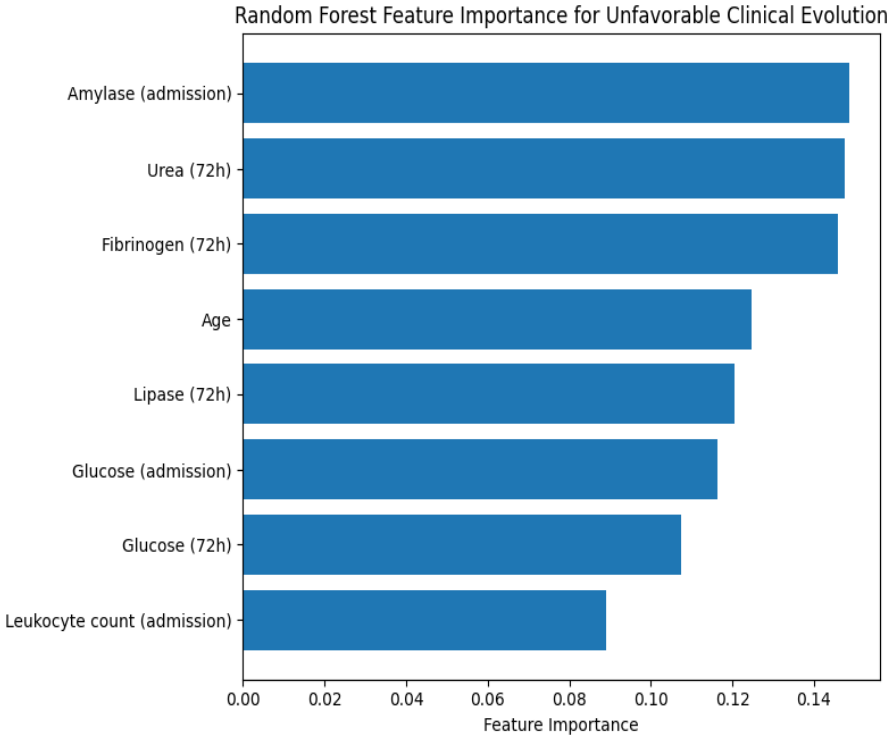

Figure 2. Feature importance derived from the Random Forest model for predicting unfavorable clinical evolution. Higher values indicate greater contribution of each variable to the model.

Table 4. Feature importance derived from the Random Forest model-numerical values.

Variable	Importance
Amylase (admission)	0,1487314527
Urea (72h)	0,147563486
Fibrinogen (72h)	0,1457460894
Age	0,1245752445
Lipase (72h)	0,1206039561
Glucose (admission)	0,1162727555
Glucose (72h)	0,1074570499
Leukocyte count (admission)	0,08904996598

Random Forest analysis identified urea at 72 hours, lipase at 72 hours, and admission laboratory parameters as the most relevant contributors to unfavorable clinical evolution.

Other variables, including fibrinogen, glucose levels, leukocyte count, and age, showed lower importance values. These findings suggest that dynamic biological markers may contribute to outcome prediction. However, given the limited sample size, these results should be interpreted cautiously (Figure 2, Table 4).

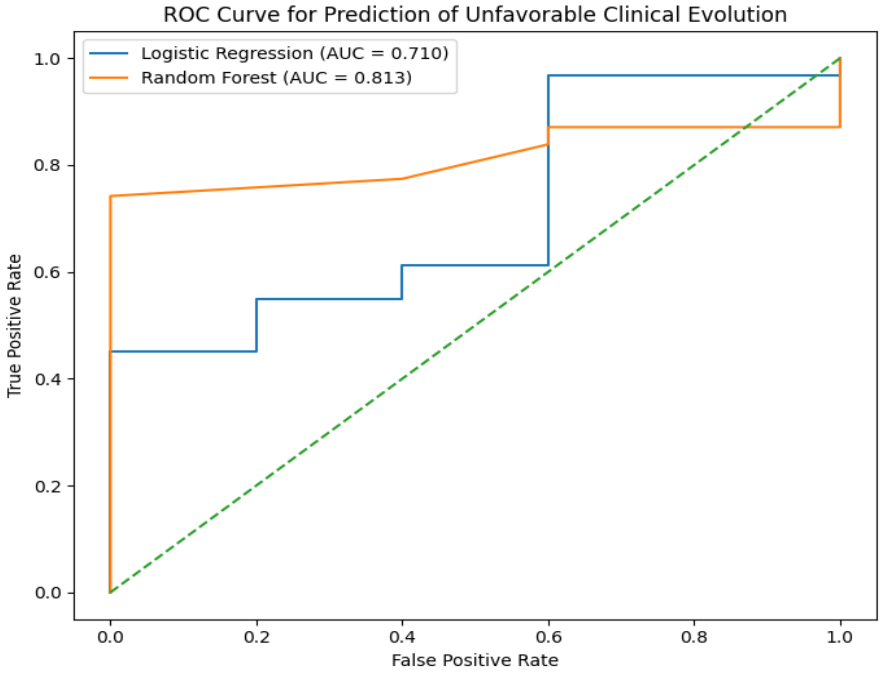

Figure 3. Receiver operating characteristic (ROC) curves for logistic regression and Random Forest models predicting unfavorable clinical evolution.

The ROC analysis (Figure 3) demonstrated modest discriminative ability, with the Random Forest model outperforming logistic regression (AUC=0.813 vs 0.710).

However, both models showed limited overall predictive accuracy, consistent with the exploratory nature of the analysis.

Table 5. Comparison of logistic regression and Random Forest models.

Model	AUC	Accuracy	F1 Score	Accuracy 95% CI	F1 95% CI
Logistic Regression	0.710	0.86	0.93	0.81-0.91	0.88-0.98
Random Forest	0.813	0.83	0.91	0.78-0.88	0.86-0.96

The Random Forest model demonstrated superior discriminative ability, achieving a higher AUC compared to logistic regression (0.813 vs 0.710). However, logistic regression showed slightly better overall classification performance, with higher accuracy (0.86 vs 0.83) and F1 score (0.93 vs 0.91).

These findings suggest that while Random Forest may better capture complex nonlinear relationships, logistic regression remains a robust and reliable model in this dataset (Table 5).

Overall, these results suggest that the tested models have limited predictive performance, and the findings should be interpreted as exploratory.

Future studies on larger cohorts, with better class balance and external validation, are needed to confirm the predictive value of these variables.

10.12865/CHSJ.52.01.10

Discussion

Acute biliary pancreatitis remains a condition with heterogeneous clinical evolution, requiring a multidisciplinary therapeutic approach adapted to disease severity [1-5].

In the present unicentric retrospective study, the majority of patients presented mild or moderate forms with favorable outcomes under early conservative management, while severe forms were associated with increased mortality, consistent with previously published data [3,4].

Dynamic evaluation of biological markers during the first 72 hours proved to be more informative than admission values alone. Urea, fibrinogen, lipase, and glycemia showed statistically significant associations with unfavorable clinical evolution at 48 and 72 hours, highlighting the importance of serial laboratory monitoring in early risk stratification [4,5].

In contrast, imaging parameters and endoscopic intervention were not independently associated with outcome, findings also reported by other authors [8-10].

Early etiological management, including ERCP when indicated and cholecystectomy during the same hospitalization in selected patients, was feasible and safe in the majority of cases, contributing to favorable outcomes in mild and moderate pancreatitis [7-10,15-17].

Delayed or conservative surgical strategies were reserved for severe forms or patients with increased operative risk, in line with current recommendations and previous clinical studies [5,14-16].

Exploratory predictive modelling demonstrated a modest ability to discriminate unfavorable evolution, with non-linear models outperforming logistic regression. The predictive performance of both models was modest, reflecting the limitations imposed by the relatively small dataset and class imbalance.

Although the Random Forest model achieved a higher AUC, indicating improved discrimination, logistic regression demonstrated better classification stability as reflected by higher accuracy and F1 score. Given the relatively small sample size, simpler models may generalize better, and the use of more complex machine learning approaches should be interpreted with caution. Although some biological markers appeared relevant in exploratory analyses, no independent predictors were identified. These findings should be interpreted cautiously and require validation in larger cohorts. Although not suitable for individual risk prediction, these findings support the role of inflammatory and pancreatic injury markers as relevant contributors to disease severity especially in dynamic measurements, as suggested in recent literature [3-5,18-22].

In conclusion, the results of this study emphasize the value of a stepwise, multidisciplinary management strategy combined with dynamic biological assessment for optimizing therapeutic decisions and prognostic evaluation in acute biliary pancreatitis [1-5].

Serial monitoring of inflammatory and pancreatic injury markers during the first 72 hours may provide clinically relevant prognostic information, while timely etiological treatment remains essential for preventing recurrence and improving outcomes [7-10,15-17].

Early etiological management, including ERCP when indicated and cholecystectomy during the same hospitalization in selected patients, was feasible and safe in the majority of cases, contributing to favorable outcomes in mild and moderate pancreatitis. Delayed or conservative surgical strategies were reserved for severe forms or patients with increased operative risk [21, 22].

Conclusions

Exploratory predictive modelling demonstrated a moderate ability to discriminate unfavorable evolution, with non-linear models outperforming logistic regression.

Although not suitable for individual risk prediction, these findings support the role of inflammatory and pancreatic injury markers as relevant contributors to disease severity.

Overall, the results emphasize the value of a stepwise, multidisciplinary management strategy combined with dynamic biological assessment for optimizing therapeutic decisions and prognostic evaluation in acute biliary pancreatitis. [18-23]

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None to declare.

Author Contributions

Conceptualization, A.L.D. and A.L.M.; Methodology, A.L.D., D.I.V. and D.V. D; Investigation, A.L.D. and A.L.M; Data analysis, A.L.D., A.L.M. and D.I.V.; Manuscript writing and initial draft preparation, A.L.D.; Manuscript review and editing, A.L.D., A.L.M. and D.I.V; Supervision, D.V. D. All authors read and approved the final manuscript.

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Conflicts of interest

The authors declare no competing interests.

Institutional Review Board

The study was conducted according to the guidelines of the Declaration of Helsinki; the study and the protocols utilized therein were approved by the Ethics Committee of Emergency University Hospital of Bucharest (6917/23.11.2022 and extension 40222/03.06.2025).

Consent Statement

Not applicable for a retrospective descriptive study.

Data availability

All data presented in the manuscript are available from the authors upon request.

References

1. Howard JM. Gallstonepancreatitis. In: Howard JM, Jordan GL, Reber HA (Eds.): Surgical diseases of the pancreas, Lea & Febiger, 1987, Philadelphia, 256-283.
2. Guice KS. Acute Pancreatitis. In: Greenfield LJ (Eds.): Scientific Principle and Practice, Lippincott-RavenPublishers, 1997, Philadelphia, 242-246.
3. Bell D, Keane MG, Pereira SP. Acute pancreatitis. Medicine, 2015, 43(3):174-181.
4. Lankisch PG, Apte M, Banks PA. Acute pancreatitis. Lancet, 2015, 386(9988):85-96.
5. Gologan E, Pantazescu AN, Bălan Gh. Diagnosticul pancreatitei acute biliare. Jurnalul de Chirurgie, Iași, 2009, 5(2):123-135.
6. British Society of Gastroenterology. United Kingdom guidelines for the management of acute pancreatitis. Gut, 1998, 42 (Suppl 2):S1-S13.
7. Neoptolemos JP, Carr-Locke DL, London NJ, Bailey IA, James D, Fossard DP. Controlled trial of urgent endoscopic retrograde cholangiopancreatography and endoscopic sphincterotomy versus conservative treatment for acute pancreatitis due to gallstones. Lancet, 1988, 2(8618):979-983.
8. Fan ST, Lai EC, Mok FP, Lo CM, Zheng SS, Wong J. Early treatment of acute biliary pancreatitis by endoscopic papillotomy. N Engl J Med, 1993, 328(4):228-232.
9. Fölsch UR, Nitsche R, Ludtke R, Hilgers RA, Creutzfeldt W. Early ERCP and papillotomy compared with conservative treatment for acute biliary pancreatitis. The German Study Group on Acute Biliary Pancreatitis. N Engl J Med, 1997, 336(4):237-242.
10. Liu CL, Lo CM, Fan ST. Acute biliary pancreatitis: Diagnosis and management. World J Surg, 1997, 21(2):149-154.
11. Dixon JA, Hillam JD. Surgical treatment of biliary tract disease associated with acute pancreatitis. Am J Surg, 1970, 120(3):371-375.
12. Frei GJ, Frei VT, Thirlby RC, McClelland RN. Biliary pancreatitis: Clinical presentation and surgical management. Am J Surg, 1986, 151(1):170-175.
13. Ranson JH. The timing of biliary surgery in acute pancreatitis. Ann Surg, 1979, 189(5):654-663.
14. Pellegrini CA. Surgery for gallstone pancreatitis. Am J Surg, 1993, 165(4):515-518.
15. Uhl W, Mьller CA, Krahenbuhl L, Schmid SW, Scholzel S, Bьchler MW. Acute gallstone pancreatitis: Timing of laparoscopic cholecystectomy in mild and severe disease. Surg Endosc, 1999, 13(11):1070-1076.
16. Tang E, Stain SC, Tang G, Froes E, Berne TV. Timing of laparoscopicsurgery in gallstonepancreatitis. Arch Surg, 1995, 130(5):496-499.
17. Soper NJ, Brunt LM, Callery MP, Edmundowicz SA, Aliperti G. Role of laparoscopic cholecystectomy in the management of acute gallstonepancreatitis. Am J Surg, 1994, 167(1):42-50.
18. Banks PA, Bollen TL, Dervenis C, Gooszen HG, Johnson CD, Sarr MG, Tsiotos GG, Vege SS. Classification of acute pancreatitis-2012: revision of the Atlanta classification and definitions by international consensus. Gut, 2013, 62(1):102-111.
19. Working Group IAP/APA Acute Pancreatitis Guidelines. IAP/APA evidence-based guidelines for the management of acute pancreatitis. Pancreatology, 2013, 13(4 Suppl 2):e1-e15.
20. Tenner S, Baillie J, DeWitt J, Vege SS. American College of Gastroenterology guideline: management of acute pancreatitis. Am J Gastroenterol, 2013, 108(9):1400-1415.
21. van Dijk SM, Hallensleben NDL, van Santvoort HC, Fockens P, van Goor H, Bruno MJ, Besselink MG. Dutch Pancreatitis Study Group. Acute pancreatitis: recent advances through randomised trials. Gut, 2017, 66(11):2024-2032.
22. Schepers NJ, Hallensleben NDL, Besselink,MG, Anten,MPGF, Bollen TL, da Costa DW, van Delft F, van Dijk SM, van Dullemen HM, Dijkgraaf MGW, van Eijck CHJ, Erkelens GW, Erler NS, Fockens P, van Geenen EJM, van Grinsven J, Hollemans RA, van Hooft JE, van der Hulst RWM, Jansen JM, Kubben FJGM, Kuiken SD, Laheij RJF, Quispel R, de Ridder RJJ, Rijk MCM, Romkens TEH, Ruigrok CHM, Schoon EJ, Schwartz MP, Smeets XJNM, Spanier BWM, Tan ACITL, Thijs WJ, Timmer R, Venneman NG, Verdonk RC, Vleggaar FP, van de Vrie W, Witteman B, van Santvoort HC, Bakker OJ, Bruno MJ, Dutch Pancreatitis Study Group. Urgent endoscopic retrograde cholangiopancreatography with sphincterotomy versus conservative treatment in predicted severe acute gallstone pancreatitis (APEC): a multicentre randomised controlled trial. Lancet, 2020, 396(10245):167-176.
23. Crockett SD, Wani S, Gardner TB, Falck-Ytter Y, Barkun AN. American Gastroenterological Association guideline on initial management of acute pancreatitis. Gastroenterology, 2018, 154(4):1096-1101.

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