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Original Research Article

Comparison of pediatric liver injury unit score and King's college hospital criteria as a predictor of outcome in children with acute liver failure

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ABSTRACT

Background: Pediatric acute liver failure (ALF) is a life-threatening condition with high morbidity and mortality, necessitating accurate prognostic tools for early risk stratification. The pediatric liver injury unit (PLIU) Score is a relatively new scoring model developed to improve mortality prediction in pediatric ALF. This study compares PLIU Score and King's College Hospital Criteria (KCHC) in predicting clinical outcomes in pediatric ALF patients.
Methods: This prospective observational study included 28 pediatric ALF patients admitted to a tertiary care hospital in Bangladesh. Clinical and laboratory parameters were assessed at admission, and both KCHC and PLIU Scores were calculated. The primary outcome measure was survival without liver transplantation vs. mortality. Sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) were analyzed for both scoring systems. ROC curve analysis was performed to determine the predictive performance of PLIU Score.
Results: Among the 28 patients, 64.29% survived without liver transplantation, while 35.71% succumbed to ALF. Wilson's disease (50% mortality) and indeterminate ALF (40% mortality) were associated with the highest fatality rates. PLIU Score >233 was significantly correlated with mortality (p=0.04), whereas KCHC showed no significant association with mortality (p=0.09). PLIU Score demonstrated higher sensitivity (80%), specificity (66.7%), PPV (57.1%), and NPV (85.7%) compared to KCHC (40%, 22.2%, 22.2%, and 40%, respectively). ROC curve analysis confirmed the superior predictive ability of PLIU (AUC=0.75) over KCHC.
Conclusion: PLIU Score outperforms KCHC in predicting mortality outcomes in pediatric ALF, offering a more reliable prognostic tool for early risk stratification. Its higher sensitivity and predictive accuracy support its potential integration into clinical practice. Future research should focus on validating PLIU Score in larger cohorts and exploring additional biomarkers to enhance its prognostic utility.

Keywords: Pediatric acute liver failure, PLIU score, King's college criteria, Prognostic model, Mortality prediction

INTRODUCTION

ALF in children is a life-threatening condition characterized by rapid hepatic dysfunction, coagulopathy, and encephalopathy in previously healthy individuals, often leading to multiorgan failure and high mortality if not promptly managed.[1,2] Despite advances in pediatric hepatology, ALF remains a complex clinical challenge with diverse etiologies, including viral infections such as Hepatitis A, B, and E, metabolic disorders like Wilson's disease, drug toxicity, particularly paracetamol overdose, and autoimmune hepatitis.[3,4] The incidence of pediatric

ALF varies globally, and in resource-limited settings like Bangladesh, where access to liver transplantation is severely restricted, early identification of high-risk cases is essential for optimizing outcomes.[5,6] Given the rapid progression of ALF and the critical time-sensitive nature of medical intervention, prognostic scoring systems play a pivotal role in assessing disease severity and guiding clinical decision-making.[7,8] One of the most widely used prognostic tools for ALF is the King's College Hospital (KCH) criteria, which relies on biochemical parameters such as bilirubin, prothrombin time (PT)/International Normalized Ratio (INR), and clinical markers of hepatic encephalopathy.[9]

Developed initially for adults and later adapted for pediatric patients, the KCH Criteria have been instrumental in stratifying patients for liver transplantation.[10] However, its application in pediatric ALF remains controversial, particularly in non-acetaminophen-induced cases, where its sensitivity has been reported to be as low as 61%, leading to the potential overuse of liver transplantation or delayed recognition of critical cases.[9,11]

A meta-analysis comparing KCHC with alternative models found that KCHC exhibited high specificity (83%) but low sensitivity (47%), making it a less reliable predictor of ALF outcomes in children with non-acetaminophen etiologies.[10] Given these limitations, there is a growing need for more accurate pediatric-specific prognostic tools. The PLIU score is a more recent and specialized scoring system designed to enhance the prediction accuracy of ALF outcomes in children. Unlike KCHC, which focuses predominantly on hepatic dysfunction, PLIU incorporates biochemical markers, hemodynamic parameters, and organ dysfunction indicators, providing a more comprehensive risk assessment for pediatric ALF patients.[12,13]

A multinational study evaluating LIU Score (a core component of PLIU Score) in 461 pediatric ALF cases found that it outperformed KCHC in predicting transplant-free survival, with a C-index of 0.81 compared to 0.61 for KCHC.[12] Similarly, a comparative study assessing LIU and PRISM scores reported higher predictive accuracy of LIU (AUC 0.73) compared to KCHC, reinforcing its superior prognostic utility in pediatric ALF.[13] Furthermore, evidence suggests that LIU Score dynamically reflects clinical deterioration, making it more sensitive in identifying high-risk patients earlier in disease progression.[14] Despite the promising predictive value of PLIU score, comparative research directly evaluating its performance against KCHC in pediatric ALF is limited, particularly in resource-constrained settings like Bangladesh. The scarcity of validated studies examining these models in a pediatric cohort from developing countries highlights a critical knowledge gap in ALF prognosis and risk stratification.[15] Given the high mortality associated with pediatric ALF and the restricted availability of liver transplantation in Bangladesh, a robust, pediatric-specific prognostic model is necessary to optimize clinical decision-making, allocate critical care resources effectively, and potentially reduce unnecessary liver transplant referrals.[16] This study aims to compare the predictive performance of the PLIU score and KCH criteria in determining outcomes among children with ALF admitted to a tertiary care hospital in Bangladesh.

By assessing sensitivity, specificity, PPV, and NPV of both scoring systems, this research will provide essential insights into their clinical applicability and accuracy in pediatric ALF prognosis. Given the limitations of KCHC and the emerging evidence supporting the superior predictive accuracy of PLIU, this study has the potential to inform future guidelines and improve risk assessment strategies for pediatric ALF patients in Bangladesh and other low-resource settings.[10,13] If validated, PLIU Score may serve as a more reliable and dynamic tool for early identification of high-risk pediatric ALF cases, ultimately improving patient outcomes and reducing ALF-related mortality.

METHODS

This cross-sectional comparative study was conducted at the Department of Pediatric Gastroenterology and Nutrition, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh, over 18 months (January 2020-June 2021). The study aimed to compare the prognostic accuracy of the PLIU Score and KCH Criteria in predicting outcomes in children with ALF. A total of 28 pediatric ALF patients were included based on the criteria of biochemical liver injury, coagulopathy (INR >1.5 with encephalopathy or INR >2.0 without encephalopathy), and no pre-existing chronic liver disease. Patients above 18 years or with incomplete records were excluded. Data on demographics, clinical features, and laboratory parameters (bilirubin, prothrombin time, INR, ammonia, ALT, and albumin) were collected at admission, day 3, and day 5.

The PLIU Score was calculated using. LIU = (3.507×peak total bilirubin) + (45.51×peak prothrombin time) + (0.254×peak ammonia). The KCH criteria were applied separately for acetaminophen-induced and non-acetaminophen-induced ALF. The primary outcome was survival without liver transplantation or death, and both scoring systems were assessed for sensitivity, specificity PPV, and NPV.

Statistical analysis was performed using SPSS (Version 25.0, Chicago, Illinois). The receiver operating characteristic (ROC) curve was used to compare the discriminatory ability of the scoring systems. A p value <0.05 was considered statistically significant. The study received IRB approval from BSMMU, and written informed consent was obtained from caregivers. Data confidentiality was strictly maintained.

RESULTS

A total of 28 pediatric patients with ALF were included in the study. The majority of participants (53.57%) were aged 6–10 years, followed by 25% aged 2–5 years and 21.43% aged 11–17 years. Males accounted for 60.71% of the cohort, while females constituted 39.29%. Among clinical findings, jaundice was universally present in all participants (100%), while anemia was observed in 75% of cases. Hepatomegaly was a common feature, affecting 92.86% of patients, whereas splenomegaly (3.57%), ascites (14.29%), and coagulopathy (10.71%) were relatively less frequent. Kayser-Fleischer (KF) rings, a marker for Wilson's disease, were detected in 35.71% of patients. Regarding clinical history, 32.40% of patients had a history of consanguinity, and 17.86% reported a family history of liver disease-related mortality. Blood and blood product transfusion history was noted in 7.14% of cases, while 21.43% had a history of consuming street food. A small proportion (10.71%) reported previous episodes of abdominal distension. Hepatitis B vaccination coverage was high (82.14%) among the participants. Notably, altered sleep patterns or drowsiness, suggestive of hepatic encephalopathy, were present in 96.40% of cases.

Table 1: Demographic and clinical profile distribution of the participants (n=28).

Variables	N	%
Age (in years)		
2-5	7	25
6-10	15	53.57
11-17	6	21.43
Gender		
Male	17	60.71
Female	11	39.29
Physical examination findings		
Anemia	21	75
Jaundice	28	100
Coagulopathy	3	10.71
Hepatomegaly	26	92.86
Splenomegaly	1	3.57
Ascites	4	14.29
KF Ring	10	35.71
Clinical history		
History of consanguinity	9	32.40
History of death of any family members from liver disease	5	17.86
History of blood and blood product transfusion	2	7.14
History of taking street food	6	21.43
History of abdominal distension	3	10.71
History of hepatitis B vaccination	23	82.14
History of alteration of sleep pattern, drowsiness	27	96.40

Table 2: Etiology of acute liver failure among the participants (n=28).

Cause of liver failure	N	%
Indeterminate	5	17.86
Wilson disease	8	28.57
HAV	10	35.71
HEV	4	14.29
Autoimmune hepatitis	1	3.57

Table 3: Cross-tabulation of etiology and clinical outcome of studied patients (n=28).

Etiology	Survival without liver transplant (n=18)		Death (n=10)	
	N	%	N	%
Autoimmune hepatitis	5	27.78	0	0.00
HAV	3	16.67	1	10.00

Continued.

Etiology	Survival without liver transplant (n=18)		Death (n=10)	
	N	%	N	%
HEV	1	5.56	0	0.00
Indeterminate	4	22.22	4	40.00
Wilson disease	5	27.78	5	50.00

Table 4: Laboratory parameters at admission among the participants (n=28).

Laboratory parameters	N	%
Hb (gm/dl)		
6-9	12	42.86
>9	16	57.14
Platelet count (thousands/cumm)		
Low (<150)	4	14.29
Normal (150-350)	23	82.14
Raised (>350)	1	3.57
Prothrombin time (second)		
10-49	7	25.00
50-100	18	64.29
>100	3	10.71
Serum ALT (IU/l)		
Normal (<65)	3	10.71
Raised (>65)	25	89.29
Serum bilirubin (mg/dl)		
5.9-8.8	8	28.57
8.9-11.7	6	21.43
11.8-17.4	4	14.29
>17.4	10	35.71
Serum albumin (g/dl)		
<2	1	3.57
2.5-3.3	2	7.14
3.4-4.4	24	85.71
>4.5	1	3.57

Table 5: Comparison of laboratory parameters between survivors and non-survivors (n=28).

Variable	Survival without liver transplant (n=18)		Death (n=10)		P value
	N	%	N	%	
Features of coagulopathy					
Present	3	16.67%	0	0.00	0.533ns
Absent	15	83.33%	10	100.00	
Prothrombin time (second)					
10-49	6	33.33%	1	10.00	0.481ns
50-100	11	61.11%	7	70.00	
>100	1	5.56%	2	20.00	
Serum ALT (U/l)					
Normal	2	11.11%	1	10.00	1.000ns
Raised	16	88.89%	9	90.00	
Serum bilirubin (mg/dl)					
5.9-8.8	8	44.44%	0	0.00	0.039 s
8.9-11.7	3	16.67%	3	30.00	
11.8-17.4	1	5.56%	3	30.00	
>17.4	6	33.33%	4	40.00	

ns: non significant, s:significant

Table 6: Comparison of KCHC and PLIU score in predicting outcomes of pediatric acute liver failure.

Variable	Survival without liver transplant (n=18)		Death (n=10)		P value
	N	%	N	%	
KCHC criteria					
Criteria met	14	77.78	4	40.00	f 0.09 ns
Criteria not met	4	22.22	6	60.00	
LIU Score					
LIU score (≤ 233)	12	66.67	2	20.00	f 0.04 s
LIU score (> 233)	6	33.33	8	80.00	

ns: non significant, s:significant

Table 7: ROC curve analysis summary (PLIU score).

	Cut of value	Sensitivity	Specificity	Area under the ROC curve	95% CI lower bound	95% CI upper bound
LIU score	233	70	33	0.75	0.543	0.957

The analysis of etiological factors among the 28 pediatric ALF patients revealed that hepatitis A virus (HAV) was the most common cause, accounting for 35.71% of cases. Wilson's disease was identified in 28.57% of patients, highlighting its significant contribution to pediatric ALF. Indeterminate cases, where no definitive cause could be established despite diagnostic evaluation, constituted 17.86% of the cohort. Hepatitis E virus (HEV) was responsible for 14.29% of cases, while autoimmune hepatitis was the least common etiology, detected in only 3.57% of patients. The clinical outcome analysis of the 28 pediatric ALF patients revealed that the majority of patients (64.29%, n=18) survived without requiring liver transplantation, while 35.71% (n=10) succumbed to the disease (Figure 1). The cross-tabulation of etiology and clinical outcome among the 28 pediatric ALF patients revealed notable variations in survival rates across different causes. Among the 18 patients who survived without liver transplantation, the highest survival rates were observed in autoimmune hepatitis (27.78%) and Wilson's disease (27.78%), followed by indeterminate cases (22.22%), HAV-induced ALF (16.67%), and HEV-induced ALF (5.56%).

Conversely, mortality was highest among patients with Wilson's disease (50%), followed by indeterminate etiology cases (40%), while HAV-related ALF accounted for 10% of deaths. No deaths were recorded in cases of autoimmune hepatitis or HEV-associated ALF. The laboratory assessment of the 28 pediatric ALF patients at admission revealed significant variations in hematological and biochemical parameters. Hemoglobin (Hb) levels were above 9 g/dl in 57.14% of patients, while 42.86% had Hb levels between 6–9 g/dl, indicating mild to moderate anemia in a considerable proportion of the cohort. Platelet counts were within the normal range (150–350 thousand/cumm) in 82.14% of patients, whereas 14.29% had thrombocytopenia (<150 thousand/cumm) and 3.57% had elevated platelet counts (>350 thousand/cumm). Regarding coagulation parameters, prolonged PT was observed in most patients, with 64.29% having PT between 50–100 seconds, while 10.71% had PT exceeding 100 seconds, suggesting severe coagulopathy in these cases. ALT levels were elevated (>65 IU/l) in 89.29% of patients, reflecting significant hepatic injury. Serum bilirubin levels varied widely, with 35.71% of patients having markedly elevated levels (>17.4 mg/dl), while 28.57% had values between 5.9–8.8 mg/dl and 21.43% between 8.9–11.7 mg/dl. Serum albumin levels were within the normal range (3.4–4.4 g/dl) in 85.71% of cases, but 7.14% had borderline low levels (2.5–3.3 g/dl), and 3.57% had severe hypoalbuminemia (<2 g/dl).

A comparative analysis of laboratory parameters between survivors and non-survivors among the 28 pediatric ALF patients revealed significant differences in serum bilirubin levels, while other parameters showed no statistically significant variations. Among the 18 patients who survived without liver transplantation, 16.67% had features of coagulopathy, while none of the non-survivors exhibited coagulopathy (p=0.533, not significant). PT was prolonged in both groups, with the majority of patients (61.11% of survivors and 70% of non-survivors) having PT values between 50–100 seconds, and 20% of non-survivors presenting with severe coagulopathy (PT >100 seconds). However, the difference in PT distribution between the two groups was not statistically significant (p=0.481). Serum ALT levels were elevated in almost all patients, with no significant difference between survivors (88.89%) and non-survivors (90.00%) (p=1.000, not significant). Serum bilirubin levels, however, demonstrated a statistically significant difference (p=0.039), indicating that higher bilirubin levels were more common among non-survivors. While 44.44% of survivors had bilirubin levels between 5.9–8.8 mg/dl, none of the non-survivors fell within this range. In contrast, 40% of non-survivors had bilirubin levels exceeding 17.4 mg/dl, compared to 33.33% of survivors. Additionally, 30% of non-survivors had bilirubin levels between 8.9–11.7 mg/dl and 11.8–17.4 mg/dl, while only

16.67% and 5.56% of survivors fell within these respective ranges.

Among the 18 survivors, 77.78% met the KCHC criteria, while 22.22% did not meet the criteria. In contrast, among the 10 non-survivors, only 40% met the KCHC criteria, while 60% did not, indicating that KCHC failed to predict mortality in a substantial proportion of fatal cases. However, the difference between survivors and non-survivors in terms of KCHC applicability was not statistically significant (p=0.09), suggesting limited sensitivity of KCHC in predicting ALF outcomes in pediatric patients. In contrast, the PLIU score demonstrated statistically significant predictive value (p=0.04). A LIU Score ≤ 233 was observed in 66.67% of survivors, whereas only 20% of non-survivors fell within this range. Conversely, 80% of non-survivors had an LIU Score >233, compared to 33.33% of survivors, indicating a strong correlation between higher LIU Scores and increased mortality risk. The predictive performance comparison between KCHC and PLIU Score demonstrated that PLIU Score outperformed KCHC across all prognostic parameters (Figure 2). The sensitivity of PLIU Score (80%) was significantly higher than that of KCHC (40%), indicating that PLIU was more effective in correctly identifying patients at risk of mortality. Similarly, specificity was higher for PLIU (66.7%) compared to KCHC (22.2%), suggesting better discriminatory power in differentiating between survivors and non-survivors. The PPV, which reflects the probability that patients identified as high risk actually succumb to ALF, was 57.1% for PLIU compared to 22.2% for KCHC, reinforcing the greater reliability of PLIU in predicting mortality. The NPV, indicating the likelihood that patients classified as low risk survive, was also higher for PLIU (85.7%) compared to KCHC (40%), confirming its superior accuracy in identifying patients likely to recover. Overall, diagnostic accuracy was significantly higher for PLIU score (71.4%) than for KCHC (28%), suggesting that PLIU Score is a more robust prognostic tool for pediatric ALF than KCHC. These findings highlight the limitations of KCHC in predicting outcomes and the potential role of PLIU Score as a superior alternative for risk stratification in pediatric ALF cases.

The receiver operating characteristic (ROC) curve analysis was conducted to evaluate the predictive performance of the PLIU Score in determining mortality outcomes among pediatric ALF patients. The area under the ROC curve (AUC) was 0.75, indicating a fair to good discriminative ability of the PLIU Score in differentiating between survivors and non-survivors. A cutoff value of 233 was identified as the optimal threshold for mortality prediction. At this cutoff, the sensitivity was 70%, meaning that the PLIU Score correctly identified 70% of patients who did not survive. However, the specificity was relatively low (33%), indicating that a portion of surviving patients also fell within the high-risk range based on the PLIU Score. The 95% confidence interval (CI) for the AUC ranged from 0.543 to 0.957, reinforcing the statistical reliability of the model despite a moderate level of specificity. These findings suggest that while the PLIU Score is a useful tool for mortality risk assessment in pediatric ALF, additional clinical markers may be required to improve its specificity for predicting survival outcomes.

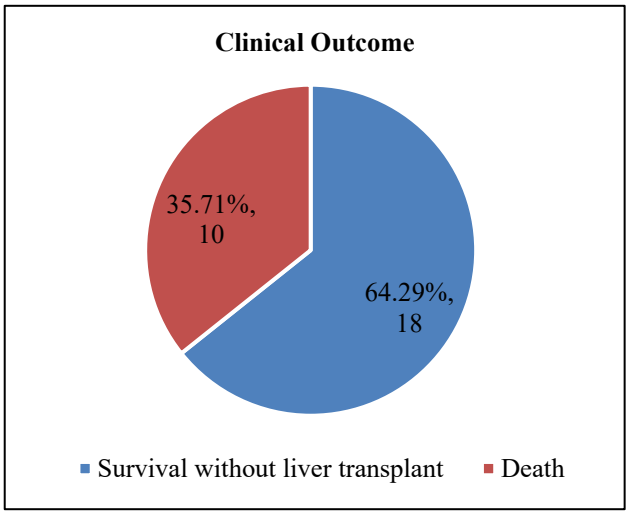

Figure 1: Clinical outcome distribution of the participants (n=28).

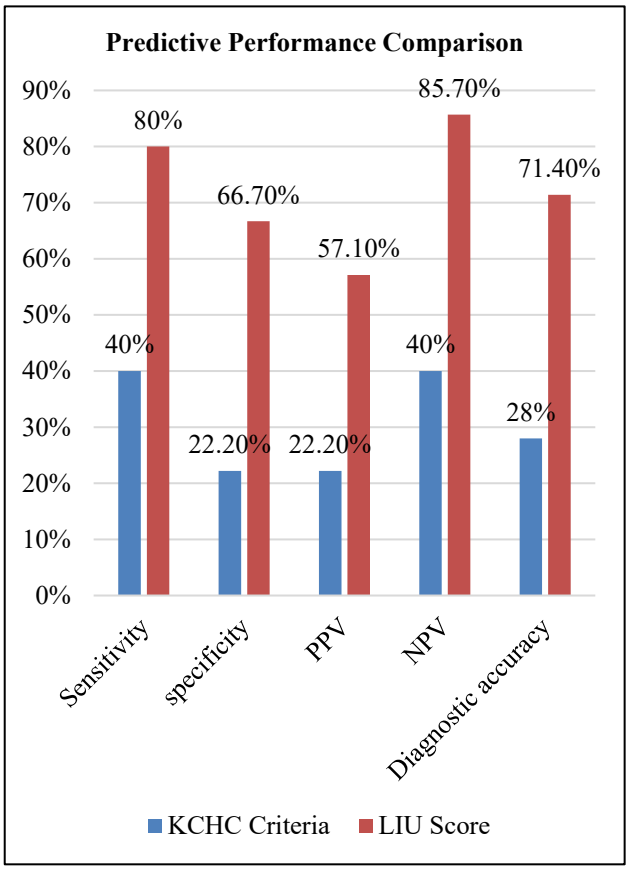

Figure 2: Predictive performance comparison (KCH vs. PLIU) (n=28).

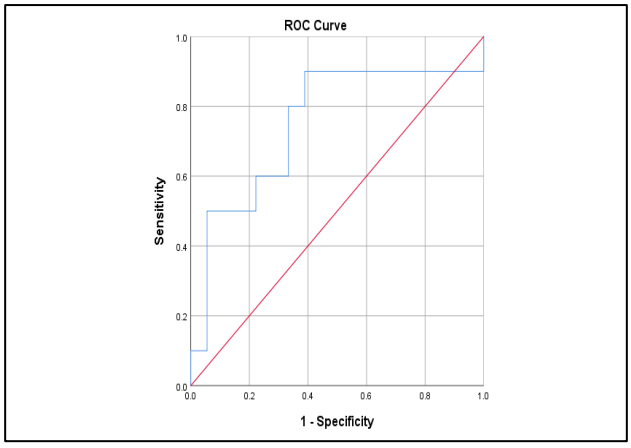

Figure 3: Receiver-operator characteristic (ROC) curve of LIU score for prediction of mortality outcome in studied patient.

DISCUSSION

The demographic and clinical profile of our study participants revealed that pediatric ALF predominantly affected children aged 6–10 years (53.57%), with a male predominance (60.71%). These findings align with previous studies, which have also reported that ALF is more common in male children and frequently presents in the younger pediatric population.[17] Jaundice was universally present in our cohort (100% of patients), while hepatomegaly (92.86%) and anemia (75%) were also frequently observed. These findings are consistent with prior studies that have identified jaundice, hepatomegaly, and anemia as hallmark clinical features of pediatric ALF.[18] Additionally, KF rings were detected in 35.71% of cases, indicating a significant prevalence of Wilson's disease, which corroborates findings from studies emphasizing Wilson's disease as a major metabolic cause of ALF.[19] Notably, 32.40% of our patients had a history of consanguinity, supporting prior research indicating a strong genetic predisposition in ALF cases linked to inherited metabolic disorders.[15] The etiological distribution of ALF in our study was dominated by Hepatitis A virus (HAV) infection (35.71%), followed by Wilson's disease (28.57%) and indeterminate ALF (17.86%). These findings align with global epidemiological data, which report HAV as a leading cause of pediatric ALF in regions with high viral hepatitis prevalence.[20] Additionally, Wilson's disease was a significant contributor to ALF in our cohort, with a 50% mortality rate, consistent with other studies that have documented poor prognosis in Wilson's disease-associated ALF cases.[21] Indeterminate ALF accounted for 17.86% of cases, slightly lower than global reports indicating 20–50% of ALF cases remain of unknown etiology, reinforcing the need for advanced diagnostic tools for better etiological identification.[22] The study revealed that 64.29% of pediatric ALF patients survived without requiring liver transplantation, while 35.71% succumbed to the disease. Mortality was highest in Wilson's disease (50%) and indeterminate ALF cases (40%), consistent with studies demonstrating high fatality rates in metabolic and idiopathic ALF cases.[23] Importantly, no deaths were observed in patients with autoimmune hepatitis or HEV-related ALF, supporting previous findings that autoimmune ALF has a more favorable prognosis with corticosteroid therapy.[24]

Laboratory findings at admission revealed that 42.86% of patients had hemoglobin levels between 6–9 g/dl, indicating mild to moderate anemia, a common feature in pediatric ALF (Ocak, 2023). Prolonged prothrombin time (PT) >50 seconds was observed in 64.29% of cases, and severe coagulopathy (PT >100 seconds) in 10.71%, findings that align with global data showing PT prolongation as a hallmark of ALF-related coagulopathy.[25] Serum ALT was elevated in 89.29% of cases, consistent with findings that ALT elevation is a primary indicator of hepatocellular injury in ALF.[26] Furthermore, hyperbilirubinemia (>17.4 mg/dl) was present in 35.71% of cases, significantly correlating with mortality (p=0.039), a result supported by studies identifying bilirubin elevation as a key predictor of poor outcomes in ALF.[27]

Comparing laboratory parameters between survivors and non-survivors, we observed that severe PT prolongation (>100 sec) was more frequent in non-survivors (20%) than in survivors (5.56%), though the difference was not statistically significant (p=0.481). Additionally, hyperbilirubinemia was significantly associated with mortality (p=0.039), with all non-survivors having bilirubin levels above 8.9 mg/dl, aligning with global data demonstrating a strong correlation between high bilirubin levels and ALF fatality (28). Our study further assessed the predictive value of KCHC and PLIU Score. Authors found that KCHC criteria were met in 77.78% of survivors but only in 40% of non-survivors, demonstrating no significant association with mortality (p=0.09). This aligns with studies suggesting KCHC has lower sensitivity in pediatric ALF, particularly in non-acetaminophen cases.[9] Conversely, PLIU score >233 was significantly associated with mortality (p=0.04), observed in 80% of non-survivors but only 33.33% of survivors, reinforcing its superior prognostic accuracy compared to KCHC.[25] When evaluating predictive performance, PLIU Score demonstrated higher sensitivity (80%) compared to KCHC (40%), indicating greater effectiveness in identifying high-risk patients. Specificity was also higher for PLIU (66.7%) vs. KCHC (22.2%), confirming its better ability to distinguish survivors from non-survivors.

Additionally, PLIU had a higher positive predictive value (57.1%) and negative predictive value (85.7%) compared to KCHC (PPV 22.2%, NPV 40%), highlighting its superior reliability in ALF prognosis.[29] The overall diagnostic accuracy of PLIU (71.4%) was significantly better than KCHC (28%), supporting previous studies recommending the use of alternative scoring systems over KCHC in pediatric ALF.[10] Finally, the ROC curve analysis for PLIU score in study revealed an AUC of

0.75, indicating good discriminative ability in predicting mortality. The optimal cutoff value of 233 had 70% sensitivity and 33% specificity, suggesting that PLIU Score is a more reliable predictor of ALF outcomes compared to KCHC.[13] This is consistent with other research that has found PLIU to be a superior prognostic model for pediatric ALF.[30]

Limitations

The study was conducted in a single hospital with a small sample size. So, the results may not represent the whole community.

CONCLUSION

This study reinforces that PLIU Score is a more accurate prognostic tool than KCHC in predicting mortality in pediatric ALF. Its higher sensitivity, specificity, and diagnostic accuracy make it a better alternative for early risk stratification. Given that Wilson's disease and indeterminate ALF cases exhibit higher mortality, early risk identification using PLIU Score could facilitate improved management and survival outcomes. Future research should focus on refining PLIU Score further and integrating it into clinical practice for enhanced pediatric ALF prognosis.

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