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ETIOLOGY AND PROGNOSTIC PREDICTORS OF POOR OUTCOMES AND ACUTE LIVER INJURY TO FAILURE PROGRESSION IN ACUTE VIRAL HEPATITIS

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PURPOSE / OBJECTIVES

Acute viral hepatitis (AVH) is a major global health burden (~1.3 million deaths/year) with a spectrum from **AH → ALI → ALF.** In India, **HAV is emerging as the predominant etiology**, and early prediction of **ALI → ALF progression** remains a key clinical challenge. **Dynamic laboratory changes (48–72 hr Δ values)** may better predict progression and outcomes.

Primary Objective:

•Identify predictors of **ALI → ALF progression** using baseline and **72-hour Δ parameters**

Secondary Objective:

•Define **etiological spectrum** (HAV, HEV, HBV, others)
•Identify predictors of **poor outcome in ALF** (death / liver transplantation)
•Determine **ALI → ALF progression rates** (overall & etiology-wise)
•Describe **clinical outcomes**

MATERIAL & METHODS

- This is an **ambispective cohort study** conducted at the Department of Gastroenterology, Dr. S.N. Medical College, Jodhpur, from **January 2022 to January 2026**.
- Patients aged **≥14 years** with **acute viral hepatitis** confirmed by serology (HAV, HEV, HBV, HCV) were included. Those with **chronic liver disease or non-viral etiologies** were excluded.
- Clinical and laboratory data were collected at **baseline (Day 1)** and **72 hours (Day 3)**, and **Δ values** (Day 3 − Day 1) were calculated.
- Patients were grouped into **ALI (progressors vs non-progressors)** and **ALF (good vs poor outcome: death/transplant)** cohorts.
- Primary outcomes were **ALI→ALF progression**; secondary outcomes included etiology, **poor outcome in ALF** and clinical outcomes.
- Analysis included **logistic regression and ROC curves**, with $p < 0.05$ considered significant.

Key Message

- ***72-hour dynamic changes outperform baseline values in predicting outcomes in acute viral hepatitis.***
- ***A simple bedside model (ΔPT + ΔLactate + Lactate Day 1) accurately predicts ALI → ALF progression (AUROC 0.94; NPV 96.5%).***
- ***In established ALF, ΔPT alone is a robust predictor of poor outcome (AUROC 0.82).***
- ***Serial PT ± lactate monitoring provides a low-cost, high-yield bedside strategy for early risk stratification in resource-limited settings***

RESULTS

- A total of **366 patients** were enrolled: **AH 216 (59.0%)**, **ALI 113 (30.9%)**, and **ALF 59 (16.1%)**. Median age was **27 years**, with **60.4% males**. Composite adverse outcome (**death/OLT**) occurred in **40 patients (10.9%)**, exclusively in ALI and ALF. **HAV** was the predominant etiology (**51.9%**), followed by **HEV (25.4%)** and **HBV (16.1%)**; **HBV** was more frequent in ALF (**20.3%**).
- **ALI→ALF progression** occurred in **22/113 (19.5%)**. Among ALI patients, all **Δ variables** were significantly higher in progressors, while among Day 1 variables only **lactate** showed discrimination (**AUROC 0.691, p=0.006**) in univariate analysis.
- For prediction of **ALI→ALF progression**, **Multivariable Model (ΔPT + ΔLactate + Lactate Day 1)** demonstrated excellent discrimination (**AUROC 0.943; bootstrap 0.946 [0.890–0.989]**), with **sensitivity 86.4%**, **specificity 91.2%**, **PPV 70.4%**, and **NPV 96.5%**.
- Among **59 ALF patients**, poor outcome occurred in **40 (67.8%)**. **ΔPT** was the strongest predictor (**AUROC 0.818, p<0.001**) and the preferred model, with **bootstrap AUROC 0.816 (0.709–0.918)**, **sensitivity 77.5%**, **specificity 78.9%**, and **PPV 88.6%**. The optimal cutoff was **ΔPT ≥ +4.0 seconds**.

RESULTS

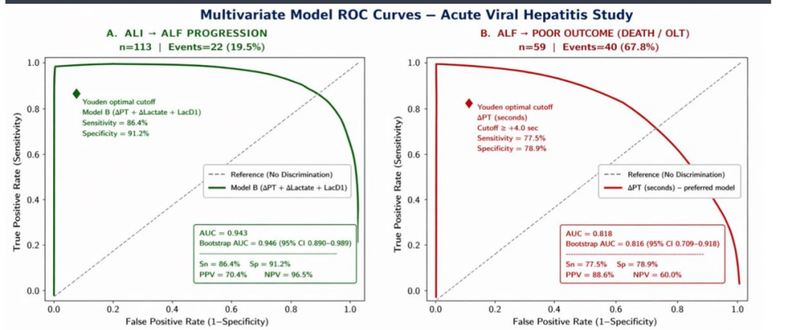

Figure. *Multivariate ROC curves for preferred predictive models in acute viral hepatitis. Panel A — Multivariable Model (ΔPT + ΔLactate + Lactate Day 1) for ALI-to-ALF progression (n=113, events=22, 19.5%): AUC=0.943; Panel B — ΔPT (single predictor model) for ALF poor outcome (n=59, events=40, 67.8%): AUC=0.818; Δ = 72-hour change from Day 1 admission value. Filled diamonds (◆) indicate Youden-optimal cutpoints. All models use Day 1 and 72-hour delta variables exclusively.*

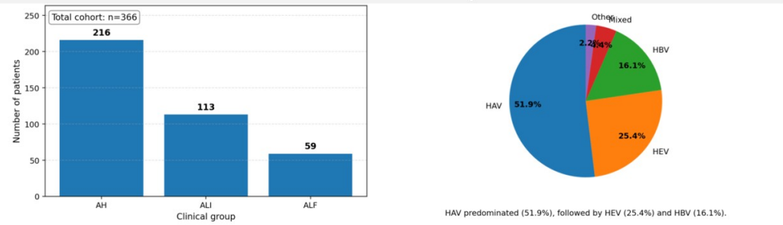

Figure. *Panel A - Baseline Cohort distribution; Panel B – Etiological distribution*

Variable	AUROC	p-value
ΔMELD	0.938	<0.001
ΔPT	0.934	<0.001
ΔINR	0.927	<0.001
ΔLactate	0.845	<0.001
ΔCreatinine	0.732	<0.001
Lactate D1	0.691	0.006

Predictor	OR (95% CI)	p-value
ΔPT	1.129 (1.049–1.216)	0.001
ΔLactate	5.540 (1.662–18.461)	0.005
Lactate D1	4.961 (0.999–24.638)	0.050

Figure. *Panel A – Key univariate predictors of ALI to ALF Progression; Panel B – Multivariate bedside model*

SUMMARY / CONCLUSION

- In this prospective cohort of **366 patients** with acute viral hepatitis, **HAV** was the predominant etiology, while **HBV** was proportionally enriched in ALF. Nearly **one-fifth of ALI patients progressed to ALF**, and **poor outcome occurred in over two-thirds of ALF patients**, highlighting the substantial clinical burden of severe AVH.
- Baseline admission values alone showed limited prognostic utility. In contrast, **72-hour dynamic changes** provided superior risk stratification across the disease spectrum, indicating that **trajectory-based assessment** better reflects ongoing hepatic injury than single time-point evaluation.
- For ALI patients, a bedside model using **ΔPT, ΔLactate, and Lactate Day 1** accurately predicted progression to ALF with excellent discrimination and high negative predictive value, allowing early identification of patients requiring closer monitoring and timely escalation of care.
- In established ALF, **ΔPT alone** emerged as the most practical predictor of poor outcome.
- Serial monitoring of **PT**, with lactate where available, offers a simple, clinically applicable strategy for **early triage, transplant referral prioritization, and decision-making in resource-limited settings**.

REFERENCES

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