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Summary

REDUCED PLASMIN GENERATION IN CIRRHOSIS IS ASSOCIATED WITH DISEASE PROGRESSION, 90-DAY MORTALITY IN ACUTE-ON-CHRONIC LIVER FAILURE, AND PORTAL VEIN THROMBOSIS

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Plasmin generation progressively declines across cirrhosis severity and is independently associated with disease progression, 90-day mortality in ACLF, and portal vein thrombosis, but not with non-portal hypertensive bleeding.

KEY POINTS

- In 228 cirrhosis patients, plasmin generation peak decreased from 67.2 nM in compensated disease to 23.2 nM in ACLF, and endogenous plasmin potential declined from 229 to 72 nM·min ($p<0.001$), indicating progressive hypofibrinolysis with advancing disease.
- Lower plasmin generation was independently associated with first decompensation in compensated patients (aHR 0.89-0.97), further decompensation in acute decompensation (aHR 0.98-0.99), and 90-day liver-related mortality in ACLF (aHR 0.97-0.99), all p□0.023.
- Lower plasmin generation independently predicted portal vein thrombosis (7% incidence, aHR 0.52-0.55, p□0.048) after adjusting for inflammatory burden, but showed no association with non-portal hypertensive bleeding (20.9% incidence).
- Sepsis-associated ACLF exhibited further marked reductions in plasmin generation beyond what was explained by ACLF severity alone, suggesting sepsis induces additional hypofibrinolysis.
- Thrombin-to-plasmin generation ratios progressively increased across disease stages, indicating an increasing imbalance between coagulation and fibrinolysis that reached its highest level in ACLF.

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