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Summary

Rebalanced, not fixed: Understanding bleeding risk and haemostatic adaptations in cirrhosis

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The speaker presented that cirrhosis creates a rebalanced but fragile haemostatic state through simultaneous loss of pro- and anticoagulant factors, and that bleeding risk depends on clinical context rather than laboratory values like INR or platelet count.

KEY POINTS

- The speaker stated that in compensated cirrhosis, haemostatic factors and anti-haemostatic factors both fall, creating a new equilibrium that is not stable and has limited reserve capacity.
- PT/INR and platelet count are poor indicators of bleeding risk because they measure only components of haemostasis, not the global haemostatic state; INR reflects liver synthetic function better than bleeding risk, according to the speaker.
- The speaker reported that infection, sepsis, AKI, ACLF, and procedural tissue injury can rapidly shift the haemostatic balance toward bleeding or thrombosis; these factors change the state itself rather than simply adding risk.
- For variceal haemorrhage, the speaker stated that vascular and portal pressure factors dominate over coagulation failure in most patients.
- The speaker recommended treating the underlying cause first, supporting haemostatic failure selectively when clinically relevant, and using blood products sparingly because volume overload can be as dangerous as haemorrhage itself.
- The speaker emphasised that management should correct the haemostatic failure that matters clinically, not normalise every abnormal laboratory value.

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