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Summary

Differential diagnosis and medical workup of fulminant liver failure

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Surgery | Hepatobiliary

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The speaker provided a systematic approach to diagnosing and managing acute liver failure, emphasizing early etiologic identification to enable specific treatments that can avoid liver transplantation.

KEY POINTS

- Acute liver failure is defined by coagulopathy plus encephalopathy, but the speaker recommends referral to specialists before both develop because survival can be very short once the combination is present.
- Hyperacute liver failure (days 1-7) typically from paracetamol or ischaemia shows very high aminotransferases and potential for survival even with severe encephalopathy, while acute/subacute liver failure (from day 7) from immunological causes shows lower aminotransferases but significant mortality risk even with low-grade encephalopathy.
- Parenchymal collapse in acute liver failure can mimic cirrhosis on imaging, and cirrhosis is not detectable on imaging in approximately 20% of cases; liver biopsy's most important role is excluding causes that preclude transplantation such as malignancy or lymphoma.
- N-acetylcysteine should be initiated as early as possible (ideally within 12 hours) for paracetamol toxicity, and meta-analyses have shown beneficial effect of NAC in all causes of acute liver failure independent of aetiology.
- For autoimmune hepatitis, the speaker stated that steroids can be very effective even in severe acute liver failure and should be given early (the speaker uses 1 mg/kg intravenously), but if steroids don't work by day 3 based on INR and bilirubin changes, they should be stopped to avoid increasing post-transplant infection risk.
- Wilson's disease diagnosis relies on clinical picture including haemolysis and low alkaline phosphatase rather than copper or ceruloplasmin levels; the speaker stated penicillamine can help even in severe cases and reported good experience with plasma exchange.

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