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Summary

Exocrine pancreatic insufficiency: is there a pancreas-liver axis?

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UEG Week Barcelona 2026 | Presentation

Endoscopy | IBD

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The speaker discussed de novo fatty liver disease developing after pancreatic resection, distinct from classic metabolic dysfunction-associated steatotic liver disease, and potential strategies to interrupt its development.

KEY POINTS

- The speaker presented a case of a patient who developed hepatic steatosis 10 months after pancreaticoduodenectomy and neoadjuvant chemotherapy for pancreatic adenocarcinoma, without traditional metabolic risk factors.
- A Mayo Clinic retrospective review found that de novo fatty liver developed six months earlier in patients with malignant versus benign indications for pancreatic resection, with incidence nearly doubled to 20 percent, and preoperative BMI was the correlating risk factor.
- A Japanese randomized trial reported that high-dose pancreatic enzyme replacement therapy was associated with 20 percent incidence of de novo fatty liver compared to 64 percent with low-dose therapy.
- The speaker recommends considering interventions including preoperative optimization of metabolic risk factors and dietary enrichment with choline and protein, postoperative aggressive diabetes control, and use of pancreatic enzyme replacement therapy, though the speaker emphasized that evidence for enzyme therapy in this indication is not solidified.
- Pancreaticoduodenectomy was reported to carry higher risk of hepatic steatosis development than distal pancreatectomy, and meta-analysis identified female gender and malignant conditions as additional risk factors.
- It remains unknown whether this post-resection fatty liver affects cardiovascular or liver-related outcomes in a population with shortened lifespan due to underlying malignancy.

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