

Microbiome: Implications for clinicians - gut-liver and gut-pancreas axis

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The gut-liver and gut-pancreas axes represent bidirectional communication systems where microbiome dysbiosis and barrier dysfunction drive chronic liver disease progression and predict acute pancreatitis severity and long-term complications.

KEY POINTS

- The speaker stated that gut barrier disruption and dysbiosis, with increased gram-negative bacteria and endotoxins, promote low-grade systemic inflammation, Kupffer cell activation, and hepatic fibrogenesis in all chronic liver disorders.
- In murine models, rifaximin reduced inflammation and fibrosis and improved gut barrier occludin expression, while metformin reduced hepatic lipid accumulation; GLP-1 receptor agonists decreased liver fat deposition and improved steatosis, ballooning, inflammation, and fibrosis in MASLD, the speaker reported.
- The speaker stated that faecal microbiota transplantation achieved safety and reduced encephalopathy recurrence in a trial using capsules and enema, with clinical response associated with engraftment; FMT improved HOMA index but did not reduce hepatic fat content in MASLD.
- In a multicenter European study of acute pancreatitis, the speaker reported that the initial emergency-room rectal microbiome showed highly significant differences in beta diversity between patients who developed mild versus moderately severe or severe disease and correlated with mortality and length of stay.
- A machine learning classifier using 13 differentially abundant microbial species predicted acute pancreatitis severity with diagnostic accuracy better than existing clinical scores, the speaker stated.
- The speaker reported that the initial admission microbiome predicted post-discharge diabetes within three years with an area under the curve above 90 percent, and oral microbiome prediction of post-discharge diabetes is currently under review.

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