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NONINVASIVE ASSESSMENT OF LIVER FIBROSIS: THE ROLE OF ELASTOGRAPHY

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Abstract: Cirrhosis, irrespective of the underlying etiology, is associated with significant morbidity and mortality. The development of portal hypertension often precipitates life-threatening sequelae, profoundly impacting the quality of life of patients and their carers[1]. Despite recent advancements in diagnostic modalities and the design of rigorous clinical trials targeting PH and its complications, the management of patients with decompensated cirrhosis continues to pose significant challenges within the field of hepatology[2,3].A better understanding of the evolving decompensation pathways in cirrhosis, particularly the distinction between AD and NAD, offers important clinical and pathophysiological insights. NAD has emerged as a distinct clinical entity characterized by low severity, the absence of systemic inflammation, and a more indolent trajectory compared to AD. Unlike classical AD events, such as acute variceal bleeding, NAD is associated with reduced urgency for hospitalization, therefore allowing for a more nuanced risk stratification and resource allocation[3].

Key words: Liver elastography, liver stiffness, extracellular matrix, collagen types I and III, liver fibrosis, alcoholic liver disease.

Moreover, NAD may represent a transitional phenotype toward recompensation, a concept gaining recognition in the recent literature but not yet well-defined in clinical practice. Biomarker profiles in NAD, including elevated markers of hepatocyte cell death without significant inflammation, suggest novel mechanistic pathways and therapeutic targets, particularly in modulating programmed cell death[2].

Ultimately, recognizing NAD as a pathophysiologically and prognostically distinct subset of cirrhosis underscores the need to better understand its progression dynamics and integrate this understanding into future clinical pathways, research, and management.

As interventional radiology and hepatology evolve, the integration of advanced imaging techniques with biomarkers, genetic profiling, and artificial intelligence will enable clinicians to adopt a more nuanced approach to the management of PH and its complications. By offering a more precise evaluation of vascular anatomy, fibrosis stage, and patient-specific risk factors, these tools will facilitate more precise and individualized therapeutic regimens, ensuring that the most appropriate intervention—whether it be TIPS or other novel interventional radiology procedures such as RTO and its different forms, ATO, or an alternative therapy—can be selected for each individual patient.

Beyond interventional radiology techniques, biomarkers and genetic profiling are introducing another dimension to the management of PH. The use of biomarkers, such as procollagen III peptide (PIIINP), hyaluronic acid, tissue inhibitor of metalloproteinases 1 (TIMP1), procollagen type III N-terminal propeptide (PRO-C3), interleukin 6 (IL-6), urinary neutrophil gelatinase-associated lipocalin (NGAL) or copeptin, likely offer valuable prognostic insight, helping to identify those at higher risk of developing important clinical endpoints in decompensated cirrhosis in the future.

Genetic profiling is particularly promising in the era of personalized medicine. Genetic mutations in the PNPLA3, TM6SF2, or MBOAT7 genes are known to influence liver fibrosis progression and may also guide more personalized treatment approaches and monitoring strategies. mRNA therapy is an emerging therapeutic approach for diseases, which has been at the forefront of the novel COVID-19 vaccines and can be targeted to the liver to promote hepatocyte regeneration and correct underlying genetic disorders caused by a loss-of-function

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phenotype. Combining mRNA therapy and CRISPR/Cas9 may further leverage the advantages of both methods to treat rare liver diseases.

The extent to which modulating the gut microbiota impacts the natural history of decompensated cirrhosis remains unclear. Yet, microbiome-based therapeutics, including prebiotics, probiotics, synbiotics, postbiotics, antibiotics, bacteriophages, antibodies to specific species, selected consortium products, and fecal microbiota transplant, hold promise to ameliorate the progression of liver disease and may also lead to the discovery of novel treatments and targeted biomarkers.

Furthermore, artificial intelligence and derived technologies can offer promising avenues for diagnosis, prognostic predictions, stratifying patients, and personalizing treatment plans. Recently, the Dieta app to gauge stool AI characteristics was accepted and increased the insight into the lactulose dose and Bristol stool scale in cirrhosis[8].

The interruption of the mechanisms that initiate and perpetuate PH remains the ideal strategy to counter the complications associated with cirrhosis. Promising agents mitigating increased intrahepatic vascular resistance, such as statins, PPAR agonists, GLP-1 agonists, SGLT2 inhibitors, sGC activators and stimulators, ribaroxaban, enoxaparin, and dual or pan-FXR receptor agonists, have the potential to alter the clinical course of advanced chronic liver disease, complementing the traditional etiologic approach. However, the full translational potential of these therapies still requires further validation through ongoing studies.

Antifibrotic treatments are likely to be developed in the next decade, on the basis of a better understanding of the pathogenesis of fibrosis. In the future, patients with cirrhosis are likely to be treated with a targeted anti-inflammatory agent that can reduce portal pressure and simultaneously serves as an antifibrotic or fibrinolytic agent.

In the next decades, we are likely to witness the broader adoption of rapid non-invasive liver diagnostic assessments and validated, safe, and reproducible non-invasive techniques for monitoring PH. Replacing invasive and limited-in-availability hepatic venous pressure gradient (HVPG) measurements, these tools will not only revolutionize the management of PH, but they will serve as effective surrogates for diagnosing, staging PH, and predicting patient outcomes.

Effective artificial liver support remains a major unmet need in the management of advanced liver disease, as liver transplantation continues to be the only definitive curative treatment currently available. Notably, advances in regenerative medicine, or cell-derived therapies and bioartificial liver support, are expected to mark major breakthroughs in the future, offering the potential to reduce the high demand for liver transplantation. Extracorporeal liver support systems like Prometheus and the Molecular Adsorbent Recirculating System (MARS) are effective for improving short-term biochemical and hemodynamic parameters in patients with liver failure, which could be crucial as a bridge to liver transplantation. Although these systems offer temporary clinical improvements, robust evidence for a consistent long-term survival benefit remains inconclusive. Their impact on overall survival appears to be influenced by patient selection and the severity of liver and multi-organ dysfunction. Other novel interventions, such as recombinant alkaline phosphatase and liver dialysis devices such as DIALIVE—a liver dialysis device that aims to exchange dysfunctional albumin and remove DAMPs and PAMPs—show promise in mitigating inflammatory damage. Advances in immunotherapy and molecular-targeted agents also offer hope for cirrhosis-related HCC[4].

This shift toward precision medicine in hepatology promises to enhance outcomes, reduce complications, and provide a more cost-effective management strategy for patients with chronic liver disease. Ultimately, it is anticipated that there will be a more integrated and multimodal approach to PH management and shifting away from a “one-size-fits-all” paradigm, with TIPS and innovative techniques working synergistically to improve patient outcomes and enhance their quality of life.

From a morphological perspective, increased hepatic stiffness is primarily associated with excessive deposition of extracellular matrix components, particularly type I and type III collagen, accompanied by alterations in sinusoidal architecture and lobular restructuring. Elastography

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serves as a reliable noninvasive alternative to liver biopsy and demonstrates a strong correlation with established histological scoring systems, including METAVIR and Ishak.

Despite the presence of so-called “natural hypocoagulation” in chronic liver disease, patients remain susceptible to a broad spectrum of spontaneous or unprovoked venous thrombotic events [3,5]. Alongside instrumental methods, biochemical markers play a significant role in the assessment of hepatic function. Gamma-glutamyl transferase (GGT) reflects hepatic enzymatic activity; however, elevated levels may also be observed in biliary tract disorders, cardiovascular diseases, and during the use of certain medications. Alcohol intake induces GGT gene expression, and increased serum GGT activity is detected in approximately 75% of individuals with chronic alcohol abuse, with a reported sensitivity of 60–90% and specificity of 50–72% [2].

Aspartate aminotransferase (AST) and alanine aminotransferase (ALT) are intracellular enzymes involved in amino acid metabolism. Their elevation in serum indicates hepatocellular injury and disruption of cellular integrity. In most acute and chronic liver diseases, including steatosis, the AST/ALT ratio is ≤1, whereas in alcoholic hepatitis this ratio frequently exceeds 2 [6]. Hyperbilirubinemia represents an important indicator of hepatic insufficiency, and with increasing severity of alcoholic liver disease, serum bilirubin concentrations above 50 μmol/L are commonly observed [7].

Various elastography techniques—such as transient elastography (FibroScan), shear-wave elastography, and point shear-wave elastography—have demonstrated high diagnostic accuracy in the evaluation of liver fibrosis. In addition, assessment of stiffness heterogeneity enables visualization of focal lesions and uneven fibrotic patterns, which are characteristic of alcoholic liver disease, viral hepatitis, and nonalcoholic steatohepatitis.

The incorporation of elastography into routine clinical practice has markedly improved the early detection and longitudinal monitoring of chronic liver diseases. This method allows timely identification of subclinical fibrosis, supports clinical decision-making, and provides a noninvasive means of assessing therapeutic response. In patients with alcohol-related liver disease, early elastographic evaluation facilitates detection of fibrotic changes prior to clinical decompensation, thereby creating opportunities for early intervention and lifestyle modification.

Furthermore, elastography plays a crucial role in pre-transplant assessment by reducing reliance on invasive biopsy procedures and enabling accurate risk stratification in patients with advanced fibrosis or cirrhosis. Overall, ultrasound elastography represents a significant advancement in hepatology, integrating morphological and functional liver assessment. By quantitatively measuring tissue stiffness, it offers a reproducible, noninvasive, and precise tool for diagnosing and staging liver fibrosis of various etiologies, including alcohol-induced liver injury, and has become an essential component of modern hepatological diagnostics.

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