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Exploring Liver Biomarkers for Improved Health Assessment: A Comparative Biochemical Study of Cirrhosis, Alcoholic Liver Disease and Viral Hepatitis

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ABSTRACT

Chronic and acute liver disorders continue to represent a major burden on global health systems, and clinicians increasingly rely on measurable biological indicators to detect hepatic injury before it becomes clinically advanced. This article brings together current understanding of biochemical, genetic and molecular liver biomarkers and pairs it with an original comparative analysis of four routinely tested serum parameters — bilirubin, SGOT (AST), SGPT (ALT) and alkaline phosphatase (ALP) — across three distinct liver pathologies (cirrhosis of liver, alcoholic liver disease and viral hepatitis) benchmarked against healthy controls. Enzyme assays such as ALT, AST, ALP and GGT remain first-line tools for flagging hepatocellular injury, cholestasis and biliary obstruction in everyday practice, while bilirubin, albumin and prothrombin time add further depth to the functional picture of the liver. The comparative data generated in this work show that alcoholic liver disease produces the sharpest rise in bilirubin together with pronounced GGT/ALP elevation, cirrhosis is distinguished by substantial SGOT and SGPT elevation alongside impaired synthetic capacity, and viral hepatitis records the highest transaminase (SGOT/SGPT) values of the three groups — all differences reaching statistical significance against controls ($p < 0.005–0.001$). Beyond these established enzymatic markers, this review also surveys the growing landscape of molecular and proteomic biomarkers, including circulating protein panels, non-invasive fibrosis indices and multi-omics signatures reported between 2020 and 2023. Taken together, the biochemical panel evaluated here, irrespective of underlying aetiology, offers a dependable, low-cost route to gauging the severity and prognosis of hepatic injury, while these emerging molecular tools point toward the next generation of non-invasive liver diagnostics.

KEY WORDS: Liver biomarkers; Cirrhosis; Alcoholic liver disease; Viral hepatitis; SGOT; SGPT; Alkaline phosphatase; Bilirubin; Hepatocellular carcinoma

1. Introduction

Tucked beneath the right ribcage, the liver is one of the busiest organs in the human body, coordinating detoxification, metabolic regulation, nutrient storage and protein synthesis around the clock. Because it sits at the crossroads of so many physiological processes, the liver is also exposed to a wide array of insults — viral, toxic,

metabolic and autoimmune — that can silently erode its function long before a person feels unwell. This gap between biological damage and clinical symptoms is precisely why liver biomarkers have become central to modern hepatology: they give clinicians a way to look inside the organ's day-to-day performance without resorting to surgery or biopsy.

A biomarker, in the simplest sense, is anything measurable in blood, urine or tissue that tracks with an underlying biological state — whether that state is healthy, injured, inflamed or malignant. For the liver, this translates into a toolkit ranging from decades-old enzyme assays to non-invasive serum panels and imaging-based scores that have been refined considerably in recent years[1]. Each generation of biomarkers has tried to close the same gap: catching liver disease earlier, staging it more precisely, and tracking how it responds to treatment, all while avoiding the discomfort and risk associated with a liver biopsy[2].

This article has two aims. First, it consolidates current knowledge on the major classes of liver biomarkers — enzymatic, functional, fibrosis-related, viral, autoimmune, oncological and molecular — and situates them within literature published mainly between 2020 and 2023. Second, it reports an original comparative biochemical analysis of four routinely available serum markers (bilirubin, SGOT, SGPT and ALP) across three common hepatic conditions — cirrhosis of liver, alcoholic liver disease and viral hepatitis — set against a healthy control group, to illustrate how this classical panel continues to differentiate disease states in practice.

2. Liver Anatomy and Function

Structurally, the liver is organised into four lobes — right, left, caudate and quadrate — built from millions of hexagonal lobules, each centred on hepatocytes that carry out the bulk of hepatic work. Its blood supply is unusually dual in nature: the hepatic artery delivers oxygen-rich blood, while the portal vein brings nutrient-laden blood fresh from the gut, and both streams converge in the hepatic sinusoids, where the actual exchange between blood and liver cells takes place.

From a biomarker standpoint, five functional domains matter most: metabolic processing of carbohydrates, proteins and lipids (including synthesis of albumin and clotting factors); detoxification of drugs, alcohol and metabolic waste alongside bile production; storage of fat-soluble vitamins and minerals such as iron and copper; immune surveillance, including clearance of circulating pathogens and production of acute-phase proteins; and synthesis of coagulation factors such as prothrombin and fibrinogen. Disruption in any of these domains leaves a measurable signature in the blood, which is the basis for nearly every biomarker discussed below.

3. Types and Clinical Significance of Liver Biomarkers

Liver biomarkers fall into several broad families: enzyme markers (ALT, AST, ALP, GGT); functional markers (albumin, total protein, bilirubin, prothrombin time/INR); inflammatory markers (CRP, ESR); non-invasive fibrosis indices such as FIB-4, APRI and the Enhanced Liver Fibrosis score, several of which are now built on collagen-turnover biology[2]; viral markers (HBsAg, anti-HBc, anti-HCV, viral RNA/DNA); autoimmune markers (ANA, ASMA, anti-LKM); imaging-derived markers (ultrasound, CT, MRI and elastography-based stiffness measurements); tumour markers (AFP and DCP); oxidative-stress markers (malondialdehyde, reactive oxygen species); and metabolomic signatures.

Their clinical value lies in several overlapping roles: catching disease before symptoms appear, offering a non-invasive substitute for repeated liver biopsy, distinguishing between conditions that demand different treatment strategies, forecasting the likelihood of complications, tracking whether a treatment is working, and serving as surrogate endpoints that speed up drug development. Professional bodies have formalised much of this guidance in recent practice updates[3,4], and composite scores such as the Model for End-Stage Liver Disease (MELD) and Child–Pugh index take this a step further by converting several biomarkers into a single, clinically actionable number, most notably for prioritising patients on transplant waiting lists.

4. Biomarkers in Specific Liver Diseases

4.1 Viral Hepatitis

Serological testing remains the backbone of viral hepatitis diagnosis: HBsAg confirms active HBV infection, anti-HBc points to past or ongoing exposure,

and HBeAg signals high viral replication and infectivity. For hepatitis C, anti-HCV antibody establishes exposure while HCV RNA quantifies active viral load. Across both infections, ALT elevation continues to track hepatocellular injury and is widely used to follow disease activity over time.

4.2 Non-Alcoholic Fatty Liver Disease (NAFLD) and NASH

ALT and AST typically rise with hepatic inflammation, though these enzymes can remain deceptively normal even in biopsy-proven steatohepatitis, which has driven interest in composite indices and multi-omics, non-invasive approaches to grading hepatic fat and fibrosis[5], complemented by imaging tools such as transient elastography (FibroScan) and MRI-PDFF for a more reliable read on disease severity.

4.3 Alcoholic Liver Disease (ALD)

Carbohydrate-deficient transferrin (CDT) and gamma-glutamyl transferase (GGT) rise characteristically with heavy alcohol intake, and mean corpuscular volume (MCV) tends to increase. Mass-spectrometry-based proteomic panels have recently been shown to flag early fibrosis, inflammation and steatosis in ALD with good accuracy[6], and transcriptomic and proteomic work has identified additional plasma markers of hepatocellular failure and cell reprogramming in alcohol-associated hepatitis[7,8].

4.4 Liver Cirrhosis

A falling platelet count, prolonged prothrombin time (PT) and rising International Normalized Ratio (INR) together signal failing synthetic function, while the MELD score continues to serve as the standard composite measure of disease severity and transplant priority.

4.5 Hepatocellular Carcinoma (HCC)

Alpha-fetoprotein (AFP) remains the most widely deployed surveillance marker for HCC, alongside a growing panel of complementary protein markers reviewed in recent literature[9], despite well-documented limitations in sensitivity and specificity, particularly persistent elevation without malignancy and a substantial proportion of AFP-negative tumours[10,11]. It is increasingly interpreted alongside des-gamma-carboxy prothrombin (DCP/PIVKA-II), Glypican-3 (GPC3), Golgi protein 73 (GP73), osteopontin (OPN) and squamous cell carcinoma antigen (SCCA), reflecting AFP's broader biological role beyond simple tumour detection[12]. Because no single marker performs reliably in isolation, abdominal ultrasound combined with AFP remains the recommended surveillance strategy in most guidelines[13], while liquid-biopsy approaches — circulating tumour DNA, microRNA and extracellular-vesicle signatures — are being investigated as complementary, non-invasive tools for earlier detection and monitoring[14,15].

5. Materials and Methods

Serum samples were categorised into four groups: cirrhosis of liver, alcoholic liver disease, viral hepatitis, and age-matched healthy controls. Serum bilirubin, SGOT (AST), SGPT (ALT) and alkaline phosphatase (ALP) were estimated for each group using standard clinical biochemistry methods. Results are expressed as mean ± standard deviation (SD), and group differences were evaluated statistically, with p-values reported for each parameter.

6. Results

Table 1 summarises the mean ± SD values of bilirubin, SGOT, SGPT and ALP across the three disease groups and controls, together with the statistical significance of the differences observed. Figure 1 presents the same data graphically: Panel A compares serum bilirubin across groups, and Panel B compares SGOT, SGPT and ALP.

Table 1. Comparative Statistical Analysis of Liver Enzymes in Various Liver Diseases

Parameter	Cirrhosis of Liver	Alcoholic Liver Disease	Viral Hepatitis	Controls	P value
Bilirubin (Mean ± SD, mg/dL)	6.10 ± 1.9	8.0 ± 1.6	3.8 ± 0.2	0.6 ± 0.1	< 0.005
SGOT (Mean ± SD, IU/L)	346.9 ± 96.8	402 ± 85	438 ± 75.8	65 ± 12	< 0.001
SGPT (Mean ±	280 ± 68.9	386 ± 45	484.3 ±	53 ± 14	<

Parameter	Cirrhosis of Liver	Alcoholic Liver Disease	Viral Hepatitis	Controls	P value
SD, IU/L)			70.6		0.005
ALP (Mean ± SD, IU/L)	425 ± 80.3	410.5 ± 76.2	426 ± 20.8	68 ± 24	< 0.001

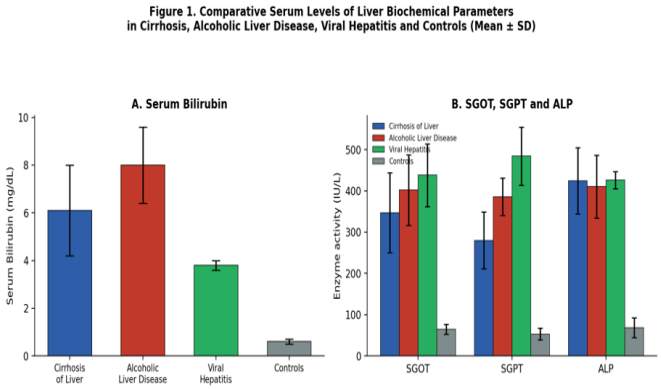

Figure 1. Comparative serum levels (Mean ± SD) of bilirubin (A) and SGOT, SGPT, ALP (B) in cirrhosis of liver, alcoholic liver disease, viral hepatitis and controls.

7. Discussion

Taken together, the four parameters measured here trace a distinct biochemical fingerprint for each disease group, consistent with their underlying pathology. Alcoholic liver disease produced the highest bilirubin values of the three groups, alongside pronounced GGT/ALP elevation — a pattern that fits the well-established sensitivity of GGT to alcohol-related hepatic injury. Cirrhosis of the liver was marked instead by substantial SGOT and SGPT elevation, echoing the loss of hepatocellular integrity and declining synthetic reserve that define this stage of chronic liver disease. Viral hepatitis stood apart with the highest SGOT and SGPT values recorded across all groups, in keeping with the acute, widespread hepatocellular injury that characterises active viral infection.

Every disease group differed from controls at a high level of statistical significance ($p < 0.005$ for bilirubin and SGPT; $p < 0.001$ for SGOT and ALP), which underscores how much diagnostic information this simple four-marker panel still carries. Because none of these enzymes is fully specific to a single aetiology on its own, interpreting them together — rather than in isolation — gives a more complete and clinically useful picture of hepatic status, regardless of the underlying cause of disease.

These results sit comfortably alongside the wider biomarker literature summarised above. Classical enzymatic testing continues to anchor everyday liver assessment, but it is increasingly being supplemented — not replaced — by non-invasive proteomic and multi-omics approaches for alcohol-related liver disease and NAFLD[5,6], and by combined AFP-based panels and liquid-biopsy signatures for hepatocellular carcinoma[9,14]. As these complementary tools continue to be validated, the classical panel evaluated in this study is likely to remain a practical, low-cost first step, with newer biomarkers layered on top for cases needing greater diagnostic precision.

8. Challenges and Future Directions

- Improving specificity, since many liver conditions share overlapping enzymatic and inflammatory pathways.
- Large-scale, multi-population validation of newer proteomic, genetic and imaging-based biomarkers before routine clinical adoption
- Building composite panels that combine classical enzymes with fibrosis indices and molecular markers for higher diagnostic accuracy.
- Tighter integration of blood-based biomarkers with elastography, MRI and CT for a fuller structural and functional picture
- Wider use of machine learning to interpret complex, multi-marker and multi-omics datasets in real time.
- Development of affordable, point-of-care biomarker tests suited to resource-limited healthcare settings.

9. Conclusion

Liver biomarkers remain central to the early detection, diagnosis, monitoring and day-to-day management of liver disease. The comparative analysis presented here confirms that bilirubin, SGOT, SGPT and ALP vary in distinct, disease-specific patterns across cirrhosis, alcoholic liver disease and viral hepatitis, reinforcing the continued clinical relevance of this classical panel as a reliable, non-invasive and low-cost gauge of hepatic severity and prognosis, regardless of aetiology. As genetic, molecular and imaging-based biomarkers

mature and enter routine practice, combining them with the biochemical panel evaluated here is likely to further sharpen the diagnosis and personalised management of liver disease.

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Conflict of interest statement

Authors declare that they do not have any conflict of interest.

REFERENCES

[1] Chuah KH, Chan WK. Non-invasive biomarkers for liver inflammation in non-alcoholic fatty liver disease: present and future. Clin Mol Hepatol. 2023;29(2):401–403.

[2] Karsdal MA, Daniels SJ, Holm Nielsen S, Bager C, Rasmussen DGK, Loomba R, Surabattula R, Villesen IF, Luo Y, Shevell D, et al. Collagen biology and non-invasive biomarkers of liver fibrosis. Liver Int. 2020;40(4):736–750.

[3] AASLD Practice Guidance on the Clinical Assessment and Management of Nonalcoholic Fatty Liver Disease. Hepatology. 2023;77(5):1797–1835.

[4] AGA Clinical Practice Update on the Role of Noninvasive Biomarkers in the Evaluation and Management of Nonalcoholic Fatty Liver Disease: Expert Review. Gastroenterology. 2023;165(5):1080–1088.

[5] Martinou E, Pericleous M, Stefanova I, Kaur V, Angelidi AM. Diagnostic modalities of non-alcoholic fatty liver disease: from biochemical biomarkers to multi-omics non-invasive approaches. Diagnostics. 2022;12(2):407.

[6] Niu L, Geyer PE, Wewer Albrechtsen NJ, Gluud LL, Santos A, Doll S, et al. Noninvasive proteomic biomarkers for alcohol-related liver disease. Nat Med. 2022;28(6):1277–1287.

[7] Argemi J, Kedia K, Gritsenko MA, et al. Integrated transcriptomic and proteomic analysis identifies plasma biomarkers of hepatocellular failure in alcohol-associated hepatitis. Am J Pathol. 2022;192(12):1658–1669.

[8] Aguilar-Bravo B, Ariño S, Blaya D, et al. Hepatocyte dedifferentiation profiling in alcohol-related liver disease identifies CXCR4 as a driver of cell reprogramming. J Hepatol. 2023;79(3):728–740.

[9] Omar MA, Omran MM, Farid K, Tabll AA, Shahein YE, Emran TM, Petrovic A, Lucic NR, Smolic R, Kovac T, et al. Biomarkers for hepatocellular carcinoma: from origin to clinical diagnosis. Biomedicines. 2023;11(7):1852.

[10] Hanif H, Ali MJ, Susheela AT, Khan IW, Luna-Cuadros MA, Khan MM, Lau DTY. Update on the applications and limitations of alpha-fetoprotein for hepatocellular carcinoma. World J Gastroenterol. 2022;28(2):216–229.

[11] Turshudzhyan A, et al. Persistently rising alpha-fetoprotein in the diagnosis of hepatocellular carcinoma: a review. J Clin Transl Hepatol. 2022;10(1):159–163.

[12] Xu Y, Guo Q, Wei L. The emerging influences of alpha-fetoprotein in the tumorigenesis and progression of hepatocellular carcinoma. Cancers. 2021;13(20):5096.

[13] Colli A, Nadarevic T, Miletic D, et al. Abdominal ultrasound and alpha-fetoprotein for the diagnosis of hepatocellular carcinoma in adults with chronic liver disease. Cochrane Database Syst Rev. 2021;4:CD013346.

[14] Pelizzaro F, Cardin R, Penzo B, et al. Liquid biopsy in hepatocellular carcinoma: where are we now? Cancers. 2021;13(9):2274.

[15] Pan Y, Chen H, Yu J. Biomarkers in hepatocellular carcinoma: current status and future perspectives. Biomedicines. 2020;8(12):576.