

Review Article

Liver disorders in Unani and modern medicine: current concepts, etiopathogenesis, diagnosis and future perspectives: a narrative review

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ABSTRACT

Liver diseases are a major cause of morbidity and mortality worldwide, encompassing a wide spectrum of conditions ranging from acute infections to chronic liver disorders and malignancies. Owing to the liver's central role in metabolism, detoxification and immune regulation, hepatic diseases are complex and require a comprehensive understanding. While modern medicine explains these disorders through pathological and biochemical mechanisms, Unani medicine interprets them on the basis of humoral imbalance and the principles of Ilmul Amraz. A narrative review was conducted by systematically searching electronic databases, including PubMed, Scopus, DOAJ, Wiley Online Library, Springer Link, Science Direct, MDPI, NCBI, Google Scholar and Index Copernicus. Relevant peer-reviewed articles, review papers and classical Unani texts were identified using predefined keywords related to liver diseases, hepatic disorders, Unani medicine and integrative medicine. The retrieved literature was critically analyzed and compared. The review demonstrates that both Unani and modern medicine provide valuable perspectives on liver diseases. Modern medicine offers detailed insights into disease mechanisms, diagnosis and evidence-based therapeutic strategies, whereas Unani medicine emphasizes on humoral equilibrium, temperament (Mizāj). Several complementary concepts were correlated, suggesting potential areas for integrative approaches in the prevention and later on management of liver disorders. An integrated understanding of liver diseases through both Unani and modern medical perspectives may enhance clinical decision-making and patient care. Future interdisciplinary research is warranted to validate traditional concepts and explore evidence-based integrative strategies for improving liver disease management.

Keywords: Liver disease, Hepatic disorders, Liver pathology, Hepatic pathophysiology, Unani medicine

INTRODUCTION

The liver is a vital organ responsible for over 500 distinct functions, including the metabolism of carbohydrates, lipids and proteins; synthesis of plasma proteins such as albumin and clotting factors; bile production; storage of vitamins and minerals and detoxification of endogenous and exogenous substances.^{1,2} Disorders affecting the liver can disrupt these functions, resulting in systemic manifestations and life-threatening complications. Globally, liver diseases cause millions of deaths annually, with viral hepatitis, alcoholic liver disease (ALD), non-

alcoholic fatty liver disease (NAFLD), cirrhosis and hepatocellular carcinoma (HCC) as predominant contributors.³

In the Unani system of medicine, which is based on the principles of Greco-Arabic medicine, liver diseases are understood through the lens of Ilmul Amraz (the science of diseases), emphasizing the balance of the four humors (Akhlāt) i. e; blood (Dam), phlegm (Balgham), yellow bile (Ṣafra'), and black bile (Sawdā) and their influence on organ function and health.^{4,5} The liver (Kabid) holds a central role in this system, being the principal organ

responsible for the production and purification of blood and the maintenance of bodily temperaments (Mizāj).⁶

Allopathic medicine utilizes evidence-based diagnostic tools such as histopathological examination and liver function tests (LFTs) to classify and manage liver diseases. The integration of these two systems offers a promising avenue for enhanced therapeutic outcomes by combining holistic care with scientific precision.

The objectives of this review are: To review the Etiopathogenesis, classification, and clinical manifestations of major liver disorders from both modern medical and Unani perspectives, to compare the concepts of liver diseases, their etiopathogenesis, diagnosis in Unani medicine with contemporary biomedical evidence and to identify areas of convergence, current evidence, and future research opportunities for integrating Unani and modern approaches in the prevention and may be in the management of liver disorders.

LITERATURE SEARCH

Relevant literature was identified through a systematic search of multiple electronic databases, including PubMed, Scopus, DOAJ, Wiley Online Library, SpringerLink, ScienceDirect, MDPI, NCBI, Google Scholar, and Index Copernicus. The search strategy incorporated a combination of Medical Subject Headings (MeSH), free-text terms, and traditional Unani terminology. Keywords included liver disease, hepatic disorders, liver pathology, hepatic pathophysiology, Unani medicine, Amraz-e-Kabid, and integrative medicine, together with their related synonyms. In addition, classical Unani literature and relevant peer-reviewed publications were screened to ensure broad coverage of the available evidence. The retrieved literature was critically reviewed to provide a comprehensive comparison of the concepts, pathophysiology, diagnostic approaches, and therapeutic perspectives of liver disorders in both Unani and modern medicine.

Eligibility criteria

Studies were included if they met the following criteria: Published in peer-reviewed journals or as an official report. Focused only on liver disorders from either modern medicine, Unani medicine, or an integrative perspective. Discussed the pathophysiology, classification, clinical manifestations of liver diseases. Full-text articles available for review. Included original research articles, systematic reviews, narrative reviews, meta-analyses, clinical guidelines and relevant classical Unani literature.

Studies were excluded if they are: Articles not related to liver disorders or lacking relevance to the objectives of the review. Conference abstracts, editorials, letters to the editor, commentaries and dissertations without sufficient methodological details. Articles for which the full text was unavailable. Studies with inadequate methodological

quality or insufficient information to support the review objectives.

DISCUSSION

Liver diseases in Unani medicine

Classification of liver diseases in Unani medicine

According to Unani medicine liver is origin of natural Quwā (natural power).

Liver is main source of nutrition synthesis. Masariqa (Mesenteric vein) absorb Kailoos (chylous) from intestine and transport to liver.^{7,8}

Liver has five types of Quwā (faculty/powers), these are Quwwat Jādhiba (absorptive faculty), Quwwat Māsika (retentive faculty), Quwwat Hādima (digestive faculty), Quwwat Dāfi'a (expulsive faculty). If one or more than one Quwwat becomes weakens, then disease will occur.^{9,10}

Waja'al-Kabid (Painful disorders of liver)

Liver pain is felt in the membrane of liver not in the structure of liver.¹¹

Etiopathogenesis

It occurs either due to Sū'-i-Mizāj (Dys temperament) in liver or due to distension in liver wall by gaseous accumulation or due to inflammation or any injury or surgery or due to the formation of any stone within the liver. It is caused by hepatic insufficiency that results in weakness of digestion by the liver thereby causing excessive production of vapors.¹¹⁻¹⁴

A'zam Khān says that, Waja'al-Kabid is mainly caused by Sū'-i-Mizāj Ḥārr (morbid hot temperament) and Sū'-i-Mizāj Bārid (morbid cold temperament) of liver.¹²

Du'f al-Kabid (Hepatic insufficiency)

A disease in which all the four faculties of liver i.e. Quwwat Hādima (digestive faculty), Quwwat Māsika (retentive faculty), Quwwat Jādhiba (absorptive faculty), Quwwat Dāfi'a (expulsive faculty) or some of these faculties become weak.¹³⁻¹⁵

Etiopathogenesis

It occurs either due to the Dys temperament of liver or whole body, diseases in the nearby organs (e. g; spleen, pancreas, uterus, stomach, intestine, lungs), major disease of liver itself.

Digestive and retentive power are mainly weakened due to Sū'-i-Mizāj Bārid (morbid cold temperament) and Sū'-i-Mizāj Raṭb (morbid wet temperament) while retentive

power weakens due to Sū'-i-Mizāj Raṭb ((morbid wet temperament) and expulsive power weakens due to Sū'-i-Mizāj Yabis (morbid dry temperament).^{13,16}

Sudād al-Kabid (Obstructive liver disorders)

Sudād al-Kabid (obstructions of liver) is a disease of the liver due to the accumulation or retention of abnormal humours (Akhlāt), characterized by obstruction of sinusoids/parenchyma and vessels in which chyle absorbs and metabolize.^{15,17}

Etiopathogenesis

It occurs due to Sū'-i-Mizāj Hārr (morbid hot temperament) and Sū'-i-Mizāj Bārid (morbid cold temperament) of liver, which weakened the digestive, distinctive and expulsive power of liver. It may also occur due to strong absorptive power. This condition may also arise due to excessive intake of Qābiḍ Ghidhā' (thick and viscous diets).¹⁵ Akhlāt Ghalīz (thick humour), which may stick in the vessels and obstruct their lumen and leads to the formation of Sudād.¹⁷

Sigher al-Kabid (Cirrhosis of liver/decreased size of liver)

According to WHO, it is a morbid state characterized by heaviness after intake of food in moderate quantity, weak digestion and gradual weight loss. According to Hippocrates those who have small liver and develop continuous indigestion so that patient may develop Ikhtināq (hysteria).¹⁶

Etiopathogenesis

It may be acute or chronic. Acute condition mostly occurs in young women (esp. between mid-phase of pregnancy). It may occur due to chronic alcoholism, syphilis anxiety fever intake of third-degree drugs for long duration etc.

In chronic condition which occurs either due to the persistent pressure on the site of liver, ascites, cardiomegaly, chronic peritonitis, malnutrition, fasting in case of stomach cancer or poor people. All these conditions may hamper the blood supply towards the liver which leads to the decrease nutrition supply towards the liver, which decreases the liver in size.¹⁶

This condition may also develop due to defect in genetic makeup during intrauterine life.¹⁸

Waram-e-Kabid (Inflammatory disorders of liver)

Inflammation in the liver may occur by different humors, including sanguineous (Damawī), bilious (Safrāwī), phlegmatic (Balghamī), and melancholic (Sawdāwī). It can further present as a soft swelling (Rikhw), hard swelling (Sulb), flatulent condition (Nafkh), or as inflammation accompanied by gaseous distension (Rīhī).^{11,15,19-21}

Etiopathogenesis

It occurs due to Sū'-i-Mizāj Hārr (morbid hot temperament) and Sū'-i-Mizāj Bārid (morbid cold temperament) which alter the digestive and expulsive power of liver, gastric weakness leads to the formation of abnormal chyle in the stomach acts as a causative factor for hepatic inflammation, obstruction nearby the gallbladder restrict the bile flow from liver to gallbladder which increases the heat of blood and increases the accumulation of bile within the liver causing the inflammation of liver. Along with these factors alcoholism, any injury may also cause the disease.^{11,14,15}

Dubayla al-Kabid (Liver abscess)

When large abscess is develops in liver, it is called as Dubayla. Sometime numerous small abscesses are formed, which merge with one another and form Dubayla (liver abscess). Abscess is formed when the morbid matter (Mādda) accumulates beneath the membrane covering the liver specifically between the liver's surface and the peritoneal membrane it is referred to as Dubayla'. Every inflammation of the liver that results in the formation of pus is termed Dubayla'.^{13,16}

Etiopathogenesis

It mainly occurs by Waram-i-Hārr (hot inflammation), which is caused by chronic alcoholism, irregularity in food intake, injury at the site of inflammation, bilious calculus within the liver, any infection etc.¹⁵⁻¹⁷

Istisqā' (dropsy)

Istisqā' is a morbid state in which matter of cold temperament gets accumulated in body. when this morbid matter is accumulated in internal or external organs which causes inflammation in the organ. This type is known as Istisqā' Laḥmī (Anasarca), but when matter is accumulated within body cavity like peritoneal cavity, then it is known as Istisqā' Ziqqī (ascites) and Istisqā' Tabli (abdominal distension due to accumulation of gaseous matter).^{13,14}

Etiopathogenesis

The specific cause of Istisqā' are either confined to the liver or involve another organ together with it. The main cause is weakness of the liver. Due to weakness liver is unable to properly absorb the water. Consequently, the organs cannot digest it naturally. Therefore, part of the water accumulates in the abdomen, while another part remains undigested within the organs. This causes inflammation in the stomach and body.^{13,16}

Yarqān (Yellowish or blackish discoloration of skin)

A morbid state usually occurring due to abnormal condition of liver, gallbladder or spleen.¹³

It is of two types. When there is yellowish discoloration of body and eyes, then it is known as Yaqān Aşfar, while blackish discoloration of body occurs in Yaqān Aswad.^{11,12,14,20}

Etiopathogenesis

Yaqān Aşfar develops either due to excessive formation of Şafrā' by organ or any obstruction in the path or intake of such foods which increases the formation of Şafrā' or Dystemperament of liver or weak evacuation power of liver or bite of poisonous animal/bacteria or due to the incomplete evacuation of Şafrā' from liver and gall bladder. Yaqān Aswad is caused by Sū'-i-Mizāj Ḥārr (morbid hot temperament) of liver, due to intense heat the blood convert into Malen (Sawdā) and Sū'-i-Mizāj Bārid (morbid cold temperament) of liver, due to which the blood becomes thick and blackish, which alter the body color.^{12,13,15}

Diagnostic methods (Unani medicine)

Shape (Shakl)

A longitudinal swelling, indicates the inflammation of abdominal muscles over the liver.²²

Site

If swelling is in the lower part of the right chest indicates inflammation of the liver.²²

Color

Blackish or whitish color can suggest the presence of liver illness.²²

Lams (touch and palpate)

If margin of liver on touch feels hot and soft, then the temperament is Ḥārr Raṭb otherwise Ḥārr Yābis when there is dryness. if feel cold on touch along with softness then the temperament is Bārid Raṭb otherwise Bārid Yābis. if hardness feels, then it indicates the Waram sulb.¹²

Pain

If there is a feeling of heaviness along with tension, it indicates the presence of obstruction or inflammation. If there is only tension, it suggests the presence of flatulence in the liver. If heaviness is present without pricking pain, the morbid matter is located within the parenchyma of liver, indicating inflammation or obstruction. If heaviness is accompanied by pricking pain, the morbid matter is situated near the membrane or capsule covering the liver.¹²

Veinous prominence

Thick, soft and prominent veins of liver indicate that the normal temperament of liver is Ḥārr Raṭb. If veins along

with prominence and thickness it is hard in nature, then the temperament is Ḥārr Yābis. If veins are dull and weak then the temperament is Bārid Yābis, but if softness is there then the temperament will become Bārid Raṭb.¹²

Some of the symptoms show the internal states of the organs, such as shortness of fingers, which denotes smallness of the liver.^{23,24}

The diagnosis is holistic, considering systemic symptoms, temperamental changes, and environmental factors.

Liver diseases in modern medicine: pathophysiological and biochemical perspectives

Pathological classification

Allopathic medicine classifies liver diseases based on their etiology and morphological changes, enabling accurate diagnosis for targeted treatment.²⁵

Viral hepatitis

Caused by infection with hepatitis viruses (HAV, HBV, HCV, HDV, and HEV), which primarily target hepatocytes and trigger inflammatory responses within the liver. Persistent infection, particularly with HBV and HCV, results in chronic hepatic inflammation, progressive fibrosis, and cirrhosis. Long-standing disease also markedly increases the risk of developing HCC.²⁶

ALD

It results from prolonged excessive alcohol consumption and is characterized by progressive liver injury. It commonly evolves from hepatic steatosis to alcoholic hepatitis, fibrosis, and eventually cirrhosis. Histological features include fatty change, Mallory-Denk bodies, inflammatory cell infiltration, and varying degrees of fibrosis.²⁷

NAFLD

It is closely associated with metabolic syndrome and is considered its hepatic manifestation. Insulin resistance plays a central role in the development of hepatic steatosis by promoting lipid accumulation and impairing fatty acid metabolism. Obesity, type 2 diabetes mellitus, dyslipidaemia, and hypertension are major risk factors that contribute to disease progression. If left untreated, NAFLD may advance to steatohepatitis, fibrosis, cirrhosis.²⁸

Cirrhosis

Liver fibrosis represents a wound-healing response to persistent hepatic injury and is characterized by excessive accumulation of extracellular matrix proteins. Continuous inflammation activates hepatic stellate cells, which are the principal mediators of fibrogenesis. Progressive fibrosis

disrupts normal liver architecture, eventually leading to cirrhosis, portal hypertension, and impaired hepatic function. Early recognition and targeted therapeutic strategies are essential to prevent irreversible liver damage.²⁹

HCC

It can develop in both cirrhotic and non-cirrhotic livers, although cirrhosis remains the predominant predisposing condition. The underlying liver pathology influences the tumour microenvironment, disease progression, and clinical outcomes. Therefore, evaluation of the history of liver is essential for accurate diagnosis, prognosis, and therapeutic decision-making.³⁰

Biochemical assessment

Diagnosis and monitoring rely heavily on serum biomarkers:

Alanine aminotransferase and aspartate aminotransferase

Aminotransferases, particularly ALT, are widely employed as biochemical indicators for the detection, evaluation, and monitoring of chronic liver disorders. Owing to its high sensitivity, ALT serves as an important marker of hepatocellular injury and plays a significant role in assessing disease severity, predicting clinical outcomes, and guiding therapeutic decisions in conditions such as metabolic dysfunction-associated steatotic liver disease (MASLD) and chronic viral hepatitis.³¹

Alkaline phosphatase and gamma-glutamyl transferase

Elevated serum alkaline phosphatase (ALP) levels can occur in a wide range of primary and secondary liver disorders. This increase is primarily attributed to enhanced enzyme synthesis triggered by cholestatic processes.³²

GGT is a highly sensitive biochemical marker that is elevated in the majority of patients with liver disease, irrespective of the underlying etiology. However, markedly increased enzyme levels are more frequently observed in individuals with cholestatic liver disorders.³³

Serum bilirubin

Bilirubin is a routinely measured biochemical parameter in the evaluation of patients with suspected liver disease and other systemic disorders.

Although it is an established indicator of hepatobiliary dysfunction, bilirubin alone lacks sufficient sensitivity and specificity to accurately assess liver function.

Consequently, its interpretation should be integrated with the patient's clinical history, the magnitude of bilirubin elevation, and accompanying biochemical abnormalities to facilitate an accurate diagnosis.³⁴

Serum albumin and prothrombin time/international normalized ratio

In patients with liver disease, prolonged prothrombin time (PT) and an elevated international normalized ratio (INR) are valuable indicators of impaired hepatic synthetic capacity, particularly when coagulation abnormalities are present.³⁵

Alpha-fetoprotein

Persistent elevation of alpha-fetoprotein (AFP) is frequently observed in patients with chronic liver disease, particularly those with cirrhosis, even in the absence of radiological evidence of HCC. Consequently, AFP lacks sufficient specificity for HCC surveillance and should not be used as a standalone screening biomarker.³⁶

Molecular and cellular mechanisms of liver diseases

Liver diseases develop through a complex interaction of infectious agents, metabolic disturbances, immune-mediated injury, genetic susceptibility, and environmental factors. Regardless of the underlying cause, persistent hepatic injury activates a series of molecular and cellular events that progressively impair liver structure and function. Chronic inflammation, oxidative stress, dysregulated cell signaling, and extracellular matrix deposition collectively contribute to fibrosis and cirrhosis.³⁷

Viral replication and immune-mediated hepatocyte injury

Chronic viral infections are major contributors to progressive liver disease. Viral replication within hepatocytes initiates both innate and adaptive immune responses. Cytotoxic CD8⁺ T lymphocytes, natural killer (NK) cells, macrophages, and inflammatory cytokines including tumor necrosis factor- α (TNF- α), interferon- γ (IFN- γ), and interleukin-6 (IL-6) participate in eliminating infected hepatocytes. Although these immune responses help control viral infection, persistent immune activation results in repeated hepatocellular injury, apoptosis, and necrosis. Continuous cycles of liver cell destruction and regeneration promote chronic inflammation, fibrogenesis.³⁷

Oxidative stress and mitochondrial dysfunction

Oxidative stress plays a central role in the pathogenesis of numerous liver disorders, including metabolic dysfunction-associated steatotic liver disease (MASLD), alcoholic liver disease (ALD), drug-induced liver injury, and chronic viral hepatitis. Excessive production of reactive oxygen species (ROS) results from mitochondrial dysfunction, cytochrome P450-mediated metabolism, lipid overload, alcohol metabolism, and exposure to toxins. When antioxidant defense mechanisms become overwhelmed, ROS induce lipid peroxidation, protein oxidation, DNA damage, and mitochondrial injury. These

molecular alterations impair hepatocyte viability, stimulate inflammatory signaling pathways such as nuclear factor-kappa B (NF- κ B), and accelerate disease progression toward fibrosis and cirrhosis.³⁷

Activation of hepatic stellate cells and fibrogenesis

Hepatic stellate cells (HSCs) are the principal mediators of liver fibrosis. Under physiological conditions, HSCs remain quiescent and serve as the primary storage site for vitamin A within the liver. Following chronic hepatic injury, inflammatory cytokines, transforming growth factor- β (TGF- β), platelet-derived growth factor (PDGF), and oxidative stress activate HSCs, transforming them into proliferative, myofibroblast-like cells. Activated HSCs produce excessive amounts of extracellular matrix proteins, including collagen types I and III, fibronectin, and laminin. Progressive accumulation of these matrix components disrupts the normal hepatic architecture, leading to fibrous septa formation, regenerative nodules, portal hypertension, and ultimately cirrhosis. If the underlying injury persists, fibrosis may progress to irreversible end-stage liver disease.³⁷

Genetic and epigenetic alterations in hepatic carcinogenesis

The progression from chronic liver disease to HCC is driven by the accumulation of genetic mutations and epigenetic modifications. Chronic inflammation and oxidative DNA damage promote mutations in tumour suppressor genes and oncogenes involved in regulating cell proliferation, apoptosis, and DNA repair. Epigenetic mechanisms, including aberrant DNA methylation, histone modifications, and dysregulated microRNA expression, further alter gene expression without changing the DNA sequence. In addition, persistent activation of signaling pathways such as Wnt/ β -catenin, PI3K/Akt/mTOR, JAK/STAT, and MAPK contributes to uncontrolled cellular proliferation, angiogenesis, and resistance to apoptosis. These molecular alterations collectively facilitate hepatocarcinogenesis and tumour progression.³⁷

Inflammatory cytokines and cellular signalling pathways

Persistent hepatic inflammation is sustained by a complex network of cytokines, chemokines, and intracellular signaling pathways. Kupffer cells, infiltrating macrophages, neutrophils, and lymphocytes release pro-inflammatory mediators including TNF- α , IL-1 β , IL-6, and chemokines that recruit additional immune cells to the liver. Activation of NF- κ B, JAK/STAT, and inflammasome pathways amplifies inflammatory responses and perpetuates hepatocyte injury. Simultaneously, transforming growth factor- β (TGF- β) signaling promotes extracellular matrix synthesis and fibrosis. These interconnected signaling networks determine the balance between tissue repair and progressive liver damage.³⁷

Cell death, regeneration and disease progression

Hepatocyte death occurs through multiple mechanisms, including apoptosis, necrosis, necroptosis, pyroptosis, and ferroptosis, depending on the nature and severity of hepatic injury. Loss of hepatocytes stimulates regenerative responses mediated by growth factors such as hepatocyte growth factor (HGF) and epidermal growth factor (EGF). However, repeated cycles of injury and regeneration increase genomic instability and create a microenvironment conducive to fibrosis and carcinogenesis. Failure of regenerative mechanisms eventually results in hepatic decompensation and liver failure.³⁷

Diagnostic imaging and biopsy (modern medicine)

Continuous advancements in hepatic imaging have significantly enhanced the evaluation of liver diseases by improving the accuracy of lesion detection, characterization, and follow-up. Modern imaging modalities, including ultrasonography (US), computed tomography (CT), and magnetic resonance imaging (MRI), offer complementary diagnostic information for both focal and diffuse hepatic abnormalities. The incorporation of contrast-enhanced techniques, quantitative imaging methods, and artificial intelligence has further refined disease staging and lesion assessment. In addition, novel imaging biomarkers enable non-invasive evaluation of hepatic steatosis, fibrosis, and therapeutic response. Collectively, these innovations are transforming clinical decision-making by supporting precision medicine while reducing reliance on invasive diagnostic procedures.³⁸

Integrative perspectives and future directions

The diagnostic approaches of Unani and modern medicine offer complementary perspectives on liver disorders. Modern medicine employs biochemical tests, imaging techniques, and histopathological evaluation for accurate diagnosis, whereas Unani medicine emphasizes assessment of Mizaj (temperament), Akhlat (humoral balance), pulse examination, and clinical observation. Integrating these approaches may provide a more comprehensive understanding of liver health. Future studies should explore correlations between traditional Unani diagnostic parameters and modern clinical findings to strengthen evidence-based integrative diagnostic models for liver diseases.

Such integrative frameworks require scientific validation, standardization of diagnostic criteria, and interdisciplinary collaboration.

CONCLUSION

Liver diseases represent a major global health concern and pose complex clinical challenges that require a multidimensional approach for effective management.

Unani Medicine, through the principles of *Ilmul Amraz*, provides valuable insights into disease by emphasizing humoral balance. In contrast, modern allopathic medicine offers advanced pathological, biochemical and diagnostic understanding. Integrating these two systems may enhance patient outcomes and promote a more comprehensive, preventive and patient-centered approach to liver health and disease management.

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