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CHRONIC LIVER DISEASE: A REVIEW OF APPLIED ANATOMY, STRUCTURAL CHANGES, AND AYURVEDIC CORRELATIONS

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ABSTRACT

Chronic Liver Disease (CLD) represents a progressive and irreversible spectrum of hepatic disorders characterized by sustained injury to hepatocytes and gradual architectural distortion of the liver parenchyma, ultimately culminating in fibrosis, cirrhosis, and hepatic insufficiency. The liver's unique microanatomy-comprising hepatocytes arranged in plates, hepatic sinusoids, portal tracts, bile canaliculi, and the space of Disse-undergoes sequential structural remodeling during the course of chronic liver injury. Early alterations predominantly involve functional and ultrastructural changes within hepatocytes, including cellular swelling, steatosis, mitochondrial dysfunction, and endoplasmic reticulum stress, while advanced stages are marked by hepatocyte loss, regenerative nodules, fibrous septa formation, and distortion of lobular architecture.

Understanding the applied anatomy of hepatocytes and their relationship with sinusoidal endothelial cells, Kupffer cells, and hepatic stellate cells is essential for correlating structural damage with clinical manifestations, biochemical abnormalities, and radiological findings in CLD. This review analyzes the applied hepatic anatomy with emphasis on hepatocyte-centered structural changes observed in chronic liver disease, integrating evidence from histopathology, imaging studies, and contemporary hepatology literature. Structural alterations at the cellular and lobular levels are correlated with functional consequences such as impaired protein synthesis, cholestasis, portal hypertension, coagulopathy, metabolic dysregulation, and hepatic encephalopathy. Radiological features including altered liver echotexture, surface nodularity, segmental hypertrophy–atrophy, and features of portal hypertension reflect underlying hepatocellular injury and progressive architectural remodeling. An integrated understanding of hepatocyte anatomy, histology, and imaging provides a comprehensive framework for early diagnosis, disease staging, prognostication, and effective management of Chronic Liver Disease.

INTRODUCTION

Chronic Liver Disease (CLD) encompasses a heterogeneous group of progressive hepatic disorders characterized by persistent hepatocellular injury, inflammation, and wound-healing responses that ultimately result in fibrosis, cirrhosis, and hepatic failure. It remains a major global health burden, accounting for significant morbidity and mortality worldwide, with common etiologies including chronic viral hepatitis, alcohol-associated liver disease, non-alcoholic fatty liver disease, autoimmune hepatitis, and metabolic liver disorders. The disease typically evolves over years or decades, during which early functional derangements precede irreversible structural damage to hepatocytes and hepatic architecture.[1]

The liver is a highly specialized organ with a unique microanatomical organization designed to support its diverse metabolic, synthetic, detoxification, and immunological functions. Hepatocytes constitute nearly 80% of the liver's cellular mass and are arranged in one-cell-thick plates radiating from the central vein, separated by fenestrated sinusoids. These cells maintain close anatomical and functional relationships with sinusoidal endothelial cells, Kupffer cells, and hepatic stellate cells through the space of Disse, facilitating efficient exchange of metabolites, proteins, and signaling molecules. Disruption of hepatocyte structure inevitably compromises these finely coordinated processes, underscoring the intimate relationship between hepatic anatomy and function.[2]

In CLD, hepatocyte injury initially manifests at a subcellular and microscopic level. Early adaptive responses include hepatocyte ballooning, accumulation of lipid droplets (steatosis), mitochondrial swelling, oxidative stress, and alterations in cytoskeletal architecture. Persistent injury leads to hepatocyte apoptosis or necrosis, triggering inflammatory cascades and activation of hepatic stellate cells. Activated stellate cells undergo transdifferentiation into myofibroblasts, resulting in excessive deposition of extracellular matrix within the space of Disse and progressive sinusoidal capillarization. These changes impair hepatocyte–sinusoid exchange and contribute to intrahepatic vascular resistance.[3]

Progressive hepatocyte loss and fibrosis disrupt the normal lobular architecture of the liver. Regenerative nodules composed of surviving hepatocytes form in response to chronic injury, but their disorganized arrangement and encasement by fibrous septa further distort vascular and biliary channels. This architectural remodeling forms the structural basis for portal hypertension, impaired hepatic perfusion, and shunting of

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blood away from functional hepatocytes, thereby exacerbating metabolic and detoxification failure.[4]

Structural alterations in hepatocytes also directly correlate with the clinical and biochemical manifestations of CLD. Loss of functional hepatocyte mass results in reduced synthesis of albumin and clotting factors, leading to hypoalbuminemia, edema, and coagulopathy. Impaired bilirubin uptake, conjugation, and excretion contribute to jaundice and cholestasis, while defective ammonia metabolism predisposes to hepatic encephalopathy. These functional deficits reflect the cumulative impact of hepatocyte injury, fibrosis, and altered microcirculation.[5]

Radiological imaging serves as a vital tool for visualizing the macroscopic consequences of chronic hepatocyte injury. Ultrasonography, computed tomography, and magnetic resonance imaging commonly demonstrate altered liver echotexture, surface nodularity, caudate lobe hypertrophy, segmental atrophy, splenomegaly, and portosystemic collaterals. These imaging findings correlate closely with histopathological changes such as fibrosis, cirrhosis, and regenerative nodules, emphasizing the applied anatomical basis of radiological diagnosis in CLD.[6]

Thus, Chronic Liver Disease represents a continuum in which progressive hepatocyte injury and maladaptive repair responses lead to irreversible structural remodeling of the liver. A comprehensive understanding of hepatocyte anatomy and its pathological alterations is essential for correlating clinical features, laboratory abnormalities, and imaging findings. The present article aims to review hepatocyte-centered structural changes in Chronic Liver Disease and to establish clear anatomical–functional correlations relevant to diagnosis, staging, and management.

APPLIED ANATOMY OF THE LIVER

The liver is the largest gland of the human body, situated predominantly in the right hypochondrium and epigastrium, extending partially into the left hypochondrium. It lies immediately beneath the diaphragm and is protected by the rib cage. The liver is covered by a fibrous capsule (Glisson's capsule) and peritoneum, except at the bare area. Its strategic anatomical position and unique dual blood supply render it highly susceptible to metabolic, toxic, infectious, and hemodynamic insults. An understanding of the applied anatomy of the liver is essential for appreciating the structural vulnerability of hepatocytes in Chronic Liver Disease (CLD), wherein persistent injury leads to irreversible architectural remodeling.[7]

Gross Anatomy

In adults, the liver weighs approximately 1.2–1.5 kg and is divided anatomically into right and left lobes by the falciform ligament, while functionally it is organized into eight Couinaud segments based on vascular inflow, outflow, and biliary drainage. Each segment functions as an independent anatomical and physiological unit, supplied by a branch of the portal vein, hepatic artery, and bile duct. Preservation of segmental architecture is critical for maintaining hepatic perfusion, metabolism, and bile formation.[8]

In Chronic Liver Disease, gross anatomical changes include altered liver size (hepatomegaly in early stages and shrunken liver in advanced cirrhosis), surface nodularity, capsular thickening, and distortion of segmental anatomy. These changes reflect progressive hepatocyte loss, fibrosis, and regenerative nodule formation and are commonly demonstrated on radiological imaging in advanced disease.[9]

Hepatic Lobule: Structural and Functional Unit

The classical hepatic lobule represents the fundamental structural and functional unit of the liver. It is hexagonal in shape, centered around a central vein, with portal triads-comprising branches of the portal vein, hepatic artery, and bile duct-located at its periphery. Hepatocytes are arranged in one-cell-thick plates radiating toward the central vein, separated by hepatic sinusoids that facilitate efficient exchange between blood and hepatocytes.[10]

In CLD, repeated hepatocellular injury disrupts the orderly lobular arrangement. Progressive fibrosis bridges portal tracts and central veins, leading to distortion of lobular architecture. Regenerative nodules composed of proliferating hepatocytes arise as a compensatory response but lack normal vascular and biliary orientation, resulting in impaired hepatic perfusion and function.[11]

Hepatocytes

Hepatocytes are the principal parenchymal cells of the liver, accounting for nearly 80% of its cellular mass. They are highly specialized, polygonal cells with abundant mitochondria, extensive smooth and rough endoplasmic reticulum, and well-developed Golgi apparatus, reflecting their central role in metabolism, protein synthesis, detoxification, bile production, and endocrine regulation. The polarized structure of hepatocytes-with sinusoidal and canalicular domains-is essential for vectorial transport of substances between blood and bile.[12]

In Chronic Liver Disease, hepatocytes undergo a spectrum of structural alterations including cellular swelling, steatosis, ballooning degeneration, cytoskeletal disruption, mitochondrial dysfunction, and eventual apoptosis or necrosis. Persistent injury results in depletion of functional hepatocyte mass, impaired regenerative capacity, and replacement of parenchyma with fibrous tissue. These structural changes form the anatomical basis for hypoalbuminemia, coagulopathy, jaundice, and metabolic derangements observed clinically.[13]

Hepatic Sinusoids and Space of Disse

Hepatic sinusoids are specialized capillary channels lined by fenestrated endothelial cells, allowing direct exchange of plasma constituents with hepatocytes across the space of Disse. This space also contains hepatic stellate cells, which store vitamin A under physiological conditions. The integrity of sinusoidal fenestrations is critical for efficient hepatocyte function.[14]

In CLD, sinusoidal endothelial cells lose fenestrations and undergo capillarization due to extracellular matrix deposition within the space of Disse. Activated hepatic stellate cells transform into collagen-producing myofibroblasts, leading to

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progressive fibrosis. Sinusoidal capillarization significantly impairs hepatocyte–blood exchange and contributes to increased intrahepatic vascular resistance and portal hypertension.[15]

Portal Tracts and Biliary System

Portal tracts contain branches of the portal vein, hepatic artery, bile duct, lymphatics, and nerves. The biliary canalicular system formed by hepatocytes drains bile into bile ductules within portal tracts. Structural integrity of this system is essential for bile formation and excretion.[16]

In Chronic Liver Disease, inflammatory and fibrotic changes within portal tracts lead to bile duct injury, ductular reaction, and cholestasis. Progressive portal fibrosis disrupts biliary drainage and vascular inflow, further aggravating hepatocellular injury and contributing to jaundice and pruritus.[17]

CHRONIC LIVER DISEASE: DEFINITION, MAJOR CAUSES, AND TYPES

Chronic Liver Disease (CLD) refers to a spectrum of progressive hepatic disorders characterized by persistent inflammation, hepatocellular injury, and wound-healing responses lasting longer than six months, ultimately leading to fibrosis, cirrhosis, and hepatic failure. The disease represents a dynamic and continuous process in which repeated or sustained injury to hepatocytes results in progressive architectural distortion of the liver parenchyma. CLD is a major contributor to global morbidity and mortality, with cirrhosis and its complications accounting for a substantial proportion of liver-related deaths worldwide.[18]

From an anatomical and pathological perspective, CLD is defined not merely by duration but by the presence of irreversible structural changes involving hepatocytes, hepatic sinusoids, portal tracts, and the extracellular matrix. Early stages are dominated by functional hepatocellular alterations and mild fibrosis, whereas advanced stages are characterized by regenerative nodules, fibrous septa, vascular reorganization, and loss of normal lobular architecture.[19]

Major Causes of Chronic Liver Disease

Chronic Liver Disease arises from a variety of etiological factors that induce persistent hepatocellular injury through metabolic, toxic, infectious, autoimmune, or vascular mechanisms. Despite differing initial insults, these causes converge on common pathogenic pathways involving inflammation, oxidative stress, stellate cell activation, and progressive fibrosis.[20]

Chronic Viral Hepatitis

Chronic infection with hepatitis B virus (HBV) and hepatitis C virus (HCV) remains a leading cause of CLD worldwide. Persistent viral replication and immune-mediated hepatocyte injury lead to chronic inflammation, progressive fibrosis, and eventual cirrhosis. HBV-related CLD may progress despite minimal inflammation due to ongoing viral activity, whereas HCV commonly induces steatosis and accelerates fibrogenesis.[21]

Alcohol-Associated Liver Disease

Chronic excessive alcohol consumption is a major cause of CLD, particularly in developing countries. Alcohol metabolism generates toxic metabolites such as acetaldehyde and reactive oxygen species, resulting in hepatocyte ballooning, steatosis, inflammation, and fibrosis. Repeated episodes of alcoholic hepatitis significantly accelerate progression to cirrhosis and hepatocellular carcinoma.[22]

Non-Alcoholic Fatty Liver Disease (NAFLD)

NAFLD has emerged as one of the most prevalent causes of CLD globally, closely associated with obesity, type 2 diabetes mellitus, dyslipidemia, and metabolic syndrome. Progressive forms, particularly non-alcoholic steatohepatitis (NASH), are characterized by hepatocyte ballooning, lobular inflammation, and fibrosis, leading to cirrhosis even in the absence of alcohol consumption.[23]

Autoimmune and Cholestatic Liver Diseases

Autoimmune hepatitis results from immune-mediated destruction of hepatocytes, leading to chronic inflammation and fibrosis. Primary biliary cholangitis and primary sclerosing cholangitis primarily affect the biliary system but eventually cause secondary hepatocellular injury and cirrhosis due to chronic cholestasis and inflammation.[24]

Metabolic and Genetic Disorders

Inherited metabolic disorders such as hemochromatosis, Wilson's disease, and alpha-1 antitrypsin deficiency lead to progressive liver injury through abnormal accumulation of iron, copper, or misfolded proteins within hepatocytes. These conditions often present insidiously and progress to advanced CLD if untreated.[25]

Drug-Induced and Toxin-Related Liver Injury

Long-term exposure to hepatotoxic drugs, herbal preparations, and environmental toxins can result in chronic liver injury. Certain drugs produce cumulative mitochondrial and microsomal damage in hepatocytes, leading to chronic hepatitis and fibrosis.[26]

Types of Chronic Liver Disease

Based on etiology, pathological pattern, and predominant site of injury, Chronic Liver Disease can be classified into several types. This classification aids in understanding disease mechanisms, predicting progression, and guiding management strategies.[27]

Chronic Hepatitis

Chronic hepatitis is characterized by persistent hepatocellular inflammation and necrosis with variable degrees of fibrosis. It commonly results from viral, autoimmune, or drug-induced causes and may progress to cirrhosis if untreated. Histologically, it is marked by interface hepatitis, portal inflammation, and hepatocyte apoptosis.[28]

Fibrotic Liver Disease

This stage represents progressive accumulation of extracellular matrix due to ongoing stellate cell activation. Fibrosis initially preserves lobular architecture but gradually forms bridging

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fibrous septa linking portal tracts and central veins, setting the stage for cirrhosis.[29]

Cirrhosis

Cirrhosis is the advanced and irreversible form of CLD, defined by diffuse fibrosis, regenerative nodule formation, and architectural distortion of the liver. It is associated with portal hypertension, hepatic insufficiency, and a high risk of hepatocellular carcinoma. Cirrhosis represents the final common pathway of most chronic liver diseases.[30]

Compensated and Decompensated CLD

Clinically, CLD is further classified into compensated and decompensated stages. Compensated CLD may remain asymptomatic despite significant fibrosis, whereas decompensated CLD is marked by complications such as ascites, variceal bleeding, hepatic encephalopathy, and jaundice, reflecting severe hepatocyte dysfunction and vascular derangement.[31]

STRUCTURAL CHANGES IN CHRONIC LIVER DISEASE

Chronic Liver Disease (CLD) is characterized by progressive and largely irreversible structural alterations involving hepatocytes, hepatic sinusoids, portal tracts, and the hepatic vasculature. Although the initiating etiological factors may vary, the liver responds to persistent injury through relatively uniform pathological mechanisms, including hepatocellular degeneration, inflammation, fibrogenesis, and architectural remodeling. These structural changes form the anatomical basis for impaired hepatic function and the clinical manifestations observed in CLD.[32],[33]

Hepatocellular Changes

Hepatocyte injury represents the primary event in the pathogenesis of CLD. In the early stages, hepatocytes exhibit adaptive and reversible changes such as cellular swelling, steatosis, mitochondrial dysfunction, and endoplasmic reticulum stress. Persistent injury results in ballooning degeneration, cytoskeletal disruption, and accumulation of intracellular inclusions, reflecting compromised metabolic and synthetic activity.[33],[34]

With disease progression, repeated cycles of hepatocyte apoptosis and necrosis lead to a progressive reduction in functional hepatocyte mass. Regenerative proliferation of surviving hepatocytes gives rise to regenerative nodules; however, these nodules lack normal lobular organization and vascular orientation. Ultimately, hepatocyte loss and replacement by fibrous tissue contribute to irreversible parenchymal extinction and hepatic insufficiency.[34],[35]

Clinically, these structural hepatocellular changes manifest as hypoalbuminemia, coagulopathy, jaundice, impaired drug metabolism, and metabolic dysregulation, reflecting the loss of essential hepatic functions.[35]

Sinusoidal and Microcirculatory Changes

Hepatic sinusoids play a critical role in facilitating exchange between circulating blood and hepatocytes. In CLD, structural alterations of sinusoidal endothelial cells include loss of fenestrations and deposition of basement membrane–like material within the space of Disse, a process termed sinusoidal capillarization.[36]

Capillarization of sinusoids significantly impairs diffusion of oxygen and nutrients to hepatocytes and increases intrahepatic vascular resistance. These microcirculatory changes contribute directly to portal hypertension and exacerbate hepatocellular hypoxia, thereby accelerating disease progression.[36],[37]

Fibrosis and Architectural Distortion

Progressive fibrosis is a hallmark structural feature of CLD. Chronic hepatocellular injury and inflammation activate hepatic stellate cells, which transform into collagen-producing myofibroblasts. Excessive deposition of extracellular matrix initially occurs in the space of Disse and portal tracts, followed by bridging fibrosis connecting portal–portal and portal–central regions.[37],[38]

As fibrosis advances, normal lobular architecture is progressively distorted, culminating in cirrhosis characterized by diffuse fibrosis and regenerative nodules. This architectural distortion disrupts hepatic blood flow and biliary drainage, forming the structural basis for portal hypertension and hepatic insufficiency.[38]

Vascular Changes

Structural remodeling of the hepatic vasculature is a key component of CLD progression. Fibrosis and regenerative nodules compress intrahepatic vessels, while angiogenesis leads to the formation of abnormal vascular channels. These changes result in increased intrahepatic resistance and diversion of blood away from functional hepatocytes.[39]

Portosystemic shunting further reduces effective hepatic perfusion, contributing to complications such as ascites, variceal bleeding, and hepatic encephalopathy. Vascular alterations thus play a central role in the transition from compensated to decompensated CLD.[39]

RADIOLOGICAL FEATURES AND STRUCTURAL CORRELATION IN CHRONIC LIVER DISEASE

Radiological imaging provides crucial non-invasive assessment of structural alterations in CLD and closely reflects underlying histopathological changes. Imaging findings correlate with hepatocyte loss, fibrosis, nodular regeneration, and vascular remodeling, thereby assisting in diagnosis, staging, and prognostication.[40]

Ultrasonography in CLD

Ultrasonography is the most commonly used initial imaging modality in CLD. Early disease may demonstrate hepatomegaly with altered echotexture, while advanced stages typically show a shrunken liver with coarse, heterogeneous echogenicity. Surface nodularity is a characteristic feature indicating cirrhosis and correlates with diffuse fibrosis and regenerative nodules.[40] Associated findings such as splenomegaly, ascites, and dilated portal vein reflect portal hypertension secondary to structural

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hepatic distortion. Ultrasonography thus provides valuable anatomical–functional correlation in CLD.[40]

Computed Tomography and Magnetic Resonance Imaging
Computed tomography (CT) and magnetic resonance imaging (MRI) provide superior anatomical detail and are useful for evaluating liver morphology, segmental atrophy or hypertrophy, nodular architecture, and vascular complications. In CLD, CT and MRI commonly demonstrate irregular liver contours, altered segmental anatomy, caudate lobe hypertrophy, and portosystemic collaterals.[41]

MRI offers excellent soft tissue contrast and allows better characterization of fibrotic tissue and regenerative nodules. Radiological findings on CT and MRI closely correspond to histological features of fibrosis, cirrhosis, and vascular remodeling, reinforcing the applied anatomical basis of imaging in CLD.[41]

CLINICAL AND FUNCTIONAL CORRELATION OF STRUCTURAL CHANGES IN CHRONIC LIVER DISEASE

The clinical manifestations of CLD arise directly from progressive structural alterations of hepatocytes, sinusoids, and hepatic vasculature. Loss of functional hepatocyte mass leads to impaired protein synthesis, detoxification, and metabolic regulation, while architectural distortion and vascular remodeling result in portal hypertension and systemic complications.[32],[33]

Reduced albumin synthesis causes hypoalbuminemia and edema, impaired clotting factor production leads to coagulopathy, and defective ammonia metabolism predisposes to hepatic encephalopathy. Portal hypertension secondary to fibrosis and vascular resistance results in ascites, splenomegaly, and variceal bleeding.[38],[39]

Thus, CLD represents an integrated anatomical–functional continuum in which progressive structural remodeling of the liver culminates in global hepatic failure. Understanding these correlations is essential for accurate clinical assessment, disease staging, and therapeutic decision-making.[32]

AYURVEDIC VIEW OF CHRONIC LIVER DISEASE

Classical Ayurvedic literature describes liver-related chronic disorders under the broad spectrum of *Udara Roga*. *Acharya Caraka*, in the 19th chapter of *Sutra Sthana* and the 13th chapter of *Chikitsa Sthana*, has enumerated eight varieties of *Udara Roga*, among which *Yakrddalyudara* is specifically mentioned as a disease characterized by pathological enlargement and dysfunction of the liver. Similar descriptions are found in the texts of *Acharya Susruta* and *Bhavaprakasha*, where *Yakrddalyudara* is included under the eight types of *Udara Roga*, indicating its chronic, progressive, and structural nature.[42]

From an Ayurvedic standpoint, *Yakrddalyudara* represents a condition wherein sustained derangement of *Agni*, *Dosha*, and *Srotas* leads to progressive structural alteration of the *Yakrit*, analogous to the gradual architectural distortion observed in Chronic Liver Disease.

Etiology of *Udara Roga* With Special Reference to *Yakrddalyudara*
The etiological factors described for *Udara Roga* reflect long-standing dietary, behavioral, and therapeutic errors that directly impair hepatic structure and function. These include excessive intake of *Ushna*, *Lavana*, *Kshara*, *Vidahi*, *Amla*, and *Visha Yukta Ahara* (including *Gara Visha*); improper post-therapeutic dietary regimens (*Mithya Samsarjana Krama*); consumption of *Ruksha*, *Viruddha*, and *Ashuchi Bhojana*; and complications arising from diseases such as *Pliha Roga*, *Arsha*, and *Grahani Dosha*.[43]

Improper administration of *Panchakarma*, suppression of natural urges (*Vega Dharana*), chronic presence of *Ama*, over-nutrition, obstruction within the gastrointestinal tract, and neglect of primary diseases further contribute to progressive involvement of the *Yakrit*. These factors predominantly affect individuals with *Mandagni*, leading to chronicity and irreversible disease progression.[43]

Pathogenesis (*Samprapti*) and Structural Derangement
The fundamental pathological process underlying *Udara Roga* is *Agni Dushti* associated with *Mala Vruddhi*. Suppression of *Agni* followed by intake of contaminated or incompatible food leads to formation and accumulation of *Ama*, which vitiates *Prana Vayu*, *Apana Vayu*, and *Agni*. This results in obstruction of *Srotas* and abnormal movement of *Doshas*.[44]

Subsequently, the vitiated *Doshas* localize between *Twak* and *Mamsa Dhatu*, causing progressive distension of the abdomen. In the context of *Yakrddalyudara*, chronic *Dosha Dushti* leads to enlargement, hardening, and loss of normal functional architecture of the *Yakrit*. This process parallels the modern description of hepatocyte injury, fibrosis, and architectural distortion seen in CLD.[4]

Clinical Features (*Rupa*) and Structural Correlation
Classical symptoms such as *Kukshi Adhmana*, *Atopa*, *Pada Shotha*, suppression of digestive power, *Slakshna Gandatva*, and *Karshya* indicate systemic involvement secondary to hepatic dysfunction. These features reflect impaired metabolism, fluid dysregulation, and progressive tissue depletion, correlating with hepatocyte loss and portal hypertension observed in CLD.[45]

Plihodara and Yakrddalyudara: Structural Relationship
The etiopathogenesis of *Plihodara* illustrates progressive enlargement and hardening of the *Pliha*, which eventually exerts pressure over *Kukshi* and *Agni Adhisthana*. Neglected *Plihodara* is described to progress and involve adjacent organs, including the *Yakrit*, culminating in *Yakrddalyudara*. The initial hardness described as “tortoise-like” strongly suggests fibrotic transformation of visceral tissue.[46]

Yakrddalyudara is defined as enlargement of the *Yakrit* on the right upper abdomen, producing visible abdominal distension. Classical texts state that its etiology, clinical features, and

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management closely resemble *Plihodara*, indicating a shared pathophysiological basis rooted in chronic structural damage.[47]

Management and Structural Reversal Approach

Treatment principles for *Yakrddalyudara* emphasize correction of *Agni*, elimination of vitiated *Doshas*, and restoration of *Srotas*. Therapeutic measures include *Snehana*, *Swedana*, *Virechana*, *Niruha Basti*, *Anuvasana Basti*, and *Siravyadha*. These interventions aim to reduce pathological accumulation, relieve obstruction, and arrest further structural deterioration.[48]

Formulations such as *Shatpala Ghrita*, *Pippali Rasayana*, *Abhaya* with jaggery, *Kshara*, and *Arishta* preparations are described to reverse organ enlargement and metabolic impairment. Herbal combinations containing *Vidanga*, *Chitraka*, *Sunthi*, *Vacha*, and *Rohitaka* are specifically indicated for conditions resembling hepatomegaly, jaundice, and chronic abdominal disorders. Post-therapeutic *Samsarjana Krama* is emphasized to restore digestive strength and prevent recurrence.[49]

DISCUSSION

Chronic Liver Disease (CLD) represents a dynamic and progressive pathological continuum in which sustained hepatocellular injury culminates in irreversible structural remodeling of the liver. The present review integrates applied hepatic anatomy, hepatocyte-centered structural changes, radiological findings, and classical Ayurvedic concepts to provide a comprehensive anatomical–functional understanding of CLD. Despite differences in explanatory frameworks, both modern medicine and Ayurveda converge on the principle that chronic impairment of metabolic integrity leads to progressive organ enlargement, fibrosis, and functional failure.[32,33]

From a modern pathological perspective, hepatocyte injury is the initiating event in CLD, irrespective of etiology. Early adaptive changes such as steatosis, cellular swelling, and mitochondrial dysfunction reflect reversible metabolic stress. However, persistent injury leads to hepatocyte apoptosis and necrosis, activation of hepatic stellate cells, and progressive deposition of extracellular matrix. This fibrotic response initially disrupts sinusoidal architecture and ultimately results in bridging fibrosis and cirrhosis, characterized by regenerative nodules and vascular distortion.[33,38] These findings correlate closely with the applied anatomical changes described in this review, particularly the loss of lobular organization and microcirculatory compromise.

Radiological findings further substantiate the structural progression of CLD. Ultrasonographic features such as coarse echotexture, surface nodularity, and altered liver size reflect underlying hepatocyte loss and fibrosis. Advanced imaging modalities demonstrate segmental atrophy, caudate lobe hypertrophy, and portosystemic collaterals, which are direct consequences of architectural distortion and increased intrahepatic vascular resistance.[40,41] Thus, radiology serves as a non-invasive bridge between microscopic structural changes and clinical staging of CLD.

Ayurvedic literature provides an equally detailed, though conceptually distinct, description of chronic liver disorders under *Udara Roga*, specifically *Yakrddalyudara*. Classical texts emphasize *Agni Dushti*, *Ama Sanchaya*, *Dosha Prakopa*, and *Srotorodha* as central pathogenic mechanisms. These concepts closely parallel modern understandings of impaired hepatocyte metabolism, inflammatory mediators, fibrogenesis, and vascular obstruction.[42,44] The description of progressive enlargement, hardness, and loss of functional integrity of the *Yakrit* in *Yakrddalyudara* closely resembles hepatomegaly followed by fibrotic contraction seen in CLD.

The clinical features described in Ayurveda-such as *Kukshi Adhmana*, *Pada Shotha*, *Karshya*, *Agni Nasha*, and discoloration of the abdomen-reflect systemic consequences of hepatic dysfunction, portal hypertension, and metabolic failure. These manifestations align with modern clinical features including ascites, peripheral edema, muscle wasting, hypoalbuminemia, and impaired digestion observed in CLD.[45,47] The Ayurvedic recognition of progression from *Plihodara* to *Yakrddalyudara* further highlights an understanding of organ interdependence and pressure-related structural expansion within the abdominal cavity.

Therapeutic principles described for *Yakrddalyudara* focus on restoration of *Agni*, elimination of accumulated *Doshas*, and reopening of obstructed *Srotas* through *Shodhana* and *Shamana* therapies. Procedures such as *Virechana*, *Basti*, and *Siravyadha*, along with medicated ghee preparations and herbal formulations, aim to arrest disease progression and prevent further structural deterioration.[48,49] While these interventions differ from modern pharmacological approaches, their conceptual objective-halting fibrosis, improving metabolism, and restoring functional reserve-mirrors contemporary goals in CLD management.

Thus, the discussion highlights that CLD, whether interpreted through modern hepatology or Ayurvedic pathology, is fundamentally a disease of chronic structural remodeling. The integration of both perspectives enriches understanding of disease progression and emphasizes the central role of hepatocyte integrity in maintaining hepatic function.

CONCLUSION

Chronic Liver Disease is a progressive disorder characterized by sustained hepatocellular injury leading to irreversible structural remodeling of the liver. The applied anatomical changes-ranging from hepatocyte degeneration and sinusoidal capillarization to fibrosis, cirrhosis, and vascular distortion-form the structural basis for the clinical, biochemical, and radiological manifestations of CLD.[32,38]

Radiological imaging provides valuable insight into these structural alterations and correlates closely with histopathological damage, aiding in disease staging and prognostication.[40,41] From an Ayurvedic standpoint, *Yakrddalyudara* described under *Udara Roga* reflects a chronic, metabolism-driven disorder rooted in *Agni Dushti*, *Ama Sanchaya*, and *Srotorodha*, culminating in progressive enlargement and dysfunction of the *Yakrit*.[42,47]

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The striking conceptual parallels between modern hepatology and Ayurveda underscore a shared recognition of CLD as a disease of chronic structural and functional decline. An integrated understanding of hepatocyte-centered pathology, applied anatomy, radiological features, and classical Ayurvedic descriptions provides a comprehensive framework for early recognition, holistic interpretation, and rational management of Chronic Liver Disease.

Such an integrative approach not only enhances academic understanding but also opens avenues for multidisciplinary research and patient-centered care in chronic hepatic disorders.

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