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A Critical Review on Anatomical Changes in the Liver with Respect to Liver Cirrhosis; An Integrative Perspective

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Abstract

Liver cirrhosis represents the end-stage of chronic liver disease, characterized by severe structural disruption, intense fibrosis, and the formation of regenerative nodules. In Ayurveda, this advanced anatomical and metabolic collapse is best correlated with *Yakrit Kshaya* (wasting of the liver parenchyma) and *Yakrutdalyodara* (abdominal distension involving the liver). While modern medicine explains these changes through the lens of hepatic stellate cell activation and extracellular matrix deposition, Ayurvedic *Sharir Rachana* (anatomy) and *Roga Nidan* (pathology) provide a profound macroscopic and systemic framework to understand these structural changes. Classical texts describe the liver (*Yakrit*) as a *Shonitaja* (blood-derived) and *Mamsapinda* (fleshy mass) organ, structurally acting as the *Moola* (root) of the *Raktavaha Srotas* (blood-carrying channels). This review critically analyzes the gross and microscopic anatomical changes in liver cirrhosis and correlates them with Ayurvedic concepts such as *Srotorodha* (channel obstruction), *Dhatu Kshaya* (tissue depletion), and *Kurma Prateekasha* (tortoise-shell like nodularity). By bridging modern histology with ancient structural descriptions, this paper provides a comprehensive understanding of cirrhotic pathogenesis, establishing a unified anatomical perspective.

Keywords: Liver Cirrhosis, Yakrit Kshaya, Raktavaha Srotas, Hepatic Stellate Cells, Ayurveda.

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Introduction:

The liver, or *Yakrit* in Ayurveda, is the largest internal organ, serving as the central metabolic engine of the human body. Historically, the evolution of Ayurvedic thought regarding liver disorders (*Yakrit Vikara*) shifted from general abdominal syndromes in the Vedic period to highly specific anatomical and physiological descriptions in the *Samhita* period. Acharya Sushruta, the pioneer of ancient surgery and anatomy, categorized the liver as a *Matruja Avayava* (maternal inherited organ) and a *Shonitaja* (blood-born) structure situated on the right side of the *Koshtha* (abdomen).¹

Modern medicine defines liver cirrhosis as a late-stage hepatic fibrosis resulting in widespread distortion of normal hepatic architecture. It is characterized anatomically by the replacement of healthy hepatic tissue with fibrotic scar tissue and regenerative nodules, ultimately leading to portal hypertension and end-stage liver failure.² In Ayurveda, the continuum of chronic liver injury is mapped meticulously from *Yakrit Vridhhi* (hepatomegaly) to *Yakrutdalyodara* (ascites due to liver pathology), culminating in *Yakrit Kshaya* (cirrhosis or parenchymal wasting). Despite the separation of thousands of years, the gross structural changes noted by ancient Ayurvedic scholars directly mirror modern pathological findings. This article aims to critically review the anatomical alterations in liver cirrhosis, correlating modern histological and macroscopic findings with Ayurvedic *Sharir Rachana* principles.⁴

Materials and Methods

This conceptual review is based on an extensive literature search. The Ayurvedic anatomical and pathological frameworks were sourced from classical texts, primarily the *Brihatrayi* (*Charaka Samhita*, *Sushruta Samhita*, and *Ashtanga Hridaya*), alongside medieval texts like *Bhavaprakasha* and commentaries (e.g., Dalhana, Chakrapani). Specific lexical sources such as *Shabdakalpadruma* and *Vachaspatyam* were utilized for etymological derivation (*Nirukti*). Modern anatomical, histological, and pathological data were retrieved from standard medical textbooks (e.g., Robbins Basic Pathology, Gray's Anatomy) and peer-reviewed journals indexing terms such as "liver cirrhosis," "hepatic fibrosis," "portal hypertension," and "Yakrit Kshaya."

Critical Review: Modern and Ayurveda Correlation**1 Normal Anatomical Baseline: Yakrit Sharir vs. Modern Anatomy**

To understand the pathological changes, one must establish the normal anatomical baseline.

- **Modern Perspective:** The liver is a highly vascular, solid, encapsulated organ situated in the right upper quadrant. Microscopically, it is composed of hepatic lobules featuring central veins, radiating hepatocytes, and sinusoids lined with endothelial cells and Kupffer cells. The Space of Disse houses Hepatic Stellate Cells (HSCs).⁵

- **Ayurvedic Perspective (Sharir Rachana):** *Yakrit* is described as a dark-colored mass (*Kalamansa*), a fleshy clump (*Mamsapinda*), and a great tendon-like container (*Mahasnayu/Karanda*). It is inherently tied to the vascular system, recognized by Acharya Charaka and Sushruta as the *Moola* (root) of the *Raktavaha Srotas*. Sushruta mentions *Raktadhara Kala* (blood-holding membrane), which is specifically found in the *Sira* (veins) and *Yakrit*, correlating structurally with the highly vascularized endothelial and sinusoidal networks of the modern liver.⁶

2 Gross Anatomical Changes in Cirrhosis

- **Modern Pathology:** In early disease, the liver may be enlarged (hepatomegaly) due to fatty infiltration or inflammation. As cirrhosis progresses, the liver parenchyma undergoes severe atrophy and necrosis. The organ shrinks, becomes dense, firm, and develops a distinctly nodular surface (micronodular or macronodular) due to fibrotic septa wrapping around regenerating hepatocytes.
- **Ayurvedic Correlation:** Ayurveda observes this transition vividly. The initial stage of hepatomegaly is termed *Yakrit Vriddhi*. As chronicity sets in (due to toxins like *Madya* / alcohol or *Meda* / fat), it transitions to *Yakrit Kshaya*. The term *Kshaya* beautifully captures the macroscopic "shrinking" and "wasting" of the functional organ. Classical texts note that during palpation of advanced *Yakrutodara*, the liver feels remarkably hard, described as *Kurma Prateekasha* (resembling the hard shell of a tortoise). This is a precise macroscopic description of the hard, nodular surface of a cirrhotic liver.⁶

3 Microscopic & Vascular Changes: Fibrogenesis and Srotorodha

- **Modern Pathology:** The core anatomical shift in cirrhosis occurs in the Space of Disse. Chronic inflammation causes Hepatic Stellate Cells (HSCs) to lose their Vitamin A droplets and transform into myofibroblasts. These cells produce excess Type I and III collagen (extracellular matrix), leading to sinusoidal capillarization. This fibrosis destroys the normal fenestrations, obstructing blood flow and leading to hepatocyte malnutrition and portal hypertension.
- **Ayurvedic Correlation:** Ayurveda views this through the lens of *Srotorodha* (channel obstruction) and *Raktavaha Srotas Dushti*. The *Teekshna* (piercing) and *Vidahi* (burning) properties of etiological factors (like alcohol) aggravate *Pitta* and dry up the *Sneha* (natural unctuousness) of the liver. This desiccation and fibrous scarring perfectly align with *Srotorodha*. Because the *Raktadhara Kala* (endothelial lining) is obstructed by fibrotic tissue, the natural flow of *Rakta* (blood) and *Rasa* (plasma) is impeded. The "starving" of hepatocytes due to sinusoidal capillarization is mirrored in the concept of *Dhatvagni Mandya* (failure of cellular metabolic fire), where the organ can no longer nourish itself.⁷

4 Extra-Hepatic Anatomical Manifestations

Cirrhosis is not confined to the liver; it drives severe systemic anatomical changes due to portal hypertension and metabolic failure.

- **Collateral Circulation (Caput Medusae):** Modern anatomy notes that restricted portal flow forces blood into portosystemic anastomoses, causing dilated veins on the anterior abdominal wall (Caput Medusae). Acharya Charaka documented this exact physical sign in *Yakrutdalyodara*, describing the appearance of prominent, discolored vascular networks on the abdomen as **Nilā (blue), Harita (green), and Haridra (yellow) Raji** (lines/veins).⁸
- **Ascites (Jalodara):** Modern medicine attributes ascites to portal hypertension and hypoalbuminemia. Ayurveda explains that when *Rasa* (plasma/nutrient fluid) cannot properly enter the hardened *Yakrit* due to *Srotorodha*, it becomes morbid and accumulates in the *Twak-Mamsa Antara* (space between skin and muscle in the abdominal cavity), leading to massive distension (*Udara*).⁹
- **Muscle Wasting (Sarcopenia):** Cirrhotic patients exhibit severe muscle wasting. In *Sharir Rachana* and pathology, this is classified as **Anuloma and Pratiloma Dhatu Kshaya**. Because the liver (*Yakrit*) fails to process *Rasa* into *Rakta*, subsequent tissues like *Mamsa* (muscle) starve (Anuloma). Furthermore, the body consumes its own *Mamsa* to survive,

leading to extreme emaciation (*Karshya*) of the limbs despite a distended abdomen.

Discussion

The comparative analysis of liver cirrhosis reveals that Ayurvedic scholars possessed a deeply structural and functional understanding of liver pathology, well before the advent of microscopy. The nomenclature used in Ayurveda is heavily grounded in anatomical reality. For instance, the transition from *Yakrit Vriddhi* to *Yakrit Kshaya* perfectly encapsulates the natural history of alcoholic and non-alcoholic fatty liver diseases progressing into end-stage fibrotic shrinkage.

The identification of the liver as the *Moola* of *Raktavaha Srotas* demonstrates an early understanding of the portal venous system and the hepatic sinusoidal network. When *Srotorodha* (fibrosis) occurs, it is logical that the back-pressure results in *Pleeha Vriddhi* (splenomegaly) and visible abdominal veins (*Nilā/Harita Raji*).

Furthermore, modern histopathology emphasizes the failure of synthetic functions in cirrhosis—most notably the drop in albumin and coagulation factors. In Ayurvedic anatomy, the liver is the seat of *Ranjaka Pitta*, the fiery essence responsible for coloring *Rasa* into *Rakta*. When *Yakrit Kshaya* occurs, *Ranjaka Pitta* fails. The modern finding of impaired coagulation directly correlates with the Ayurvedic observation of blood leakage (e.g., *Sarakta Chhardi* / hematemesis seen in advanced *Kumbhakamala* or cirrhotic failure). The

hypoalbuminemia correlates with the severe loss of *Rasa-Rakta Saara*, leading directly to the profound *Daurbalya* (weakness) and *Ksheenabala* (loss of strength) seen in these patients.¹⁰

From a therapeutic (*Chikitsa*) anatomical standpoint, because the physical structure of the liver is heavily scarred (*Kurma Prateekasha*), Ayurveda categorizes advanced *Yakrutodara* and *Yakrit Kshaya* as *Krichrasadhya* (difficult to cure) or *Asadhya* (incurable). Treatment protocols like *Virechana* (therapeutic purgation) aim to decompress the *Raktavaha Srotas*, reduce portal pressure, and eliminate morbid *Pitta* before irreversible fibrotic *Kshaya* takes full effect.

Conclusion

Liver cirrhosis is a profound structural collapse of the hepatic architecture, moving from inflammation and enlargement to fibrotic scarring, nodular regeneration, and parenchymal wasting. This critical review establishes that the modern anatomical and histological understandings of cirrhosis are remarkably congruent with Ayurvedic *Sharir Rachana* and pathological concepts. The modern descriptions of fibrogenesis, portal hypertension, and sarcopenia find their exact classical counterparts in *Srotorodha*, *Nila/Harita Raji*, and *Dhatu Kshaya*. Understanding *Yakrit Kshaya* as an anatomical destruction of the *Raktavaha Srotas* not only validates the structural intelligence of classical Ayurveda but also provides a holistic, integrative

framework for modern researchers and clinicians managing chronic liver diseases.

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