Review Article

Crossroads of Health: Exploring the Interplay between the *Hridaya* and *Yakrita* in Health and Disease - A Comprehensive Narrative Review

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Abstract:

In Ayurveda system of medicine there are close relationships between each and every organs have been conceptualized according to *Acharya* Kashyapa theory of organogenesis. The human body in Ayurveda is viewed as an integrated system where every organ functions in harmony with others. The heart and liver share a close embryological, anatomical, physiological, pathological and pharmacological relationship. **Knowledge gap:** In Ayurveda, the correlation between the heart (*Hridaya*) and liver (*Yakrita*) remains an underexplored area. **Need of the study:** To summarize the Ayurvedic *Naidanik* approach towards cardio-hepatic diseases and to set a benchmark concept and its practical need. Studying how the Hridaya and Yakrita interact helps us see how disease in one can influence the other. **Materials and Methods:** The data for this self-narrative review is collected from *Brihitrayee* with respective commentary and indexed journals. **Discussion:** The liver and heart embryologically, physiologically, anatomically, pathologically, pharmacologically interconnected via Rasa and Rakta. The organokines, communicators for organ interaction are the *Dhatwansha* from respective organs participated in reciprocation. Mutual causation between heart and liver diseases, exemplifies as *Nidanarthakar Roga*.

Conclusion: *Rasa - Rakta Dhatu* with respective *Srotasa* are seem to be involved in *Hrid- Yakrit Roga.* Rigorous researches are to be undertaken to explore in deeper *Hrid-Yakrit* interrelations.

Keywords: *Hridroga*, *Yakritroga,* Ayurved biology, Heart-Liver Interactions, *Nidanarthakar Roga.*

Introduction:

The human body in Ayurveda is viewed as an integrated system where every organ functions in harmony with others. There are close relationships between each and every organ have been conceptualized according to *Acharya* Kashyapa theory of organogenesis[(1)]. So, the heart and liver interaction also conveyed.

The interplay between the heart and liver is intricately linked through the concepts of *Dhatu, Srotasa*, and impact of *Dosha*, *Agni* and *Ama*. Ayurvedic *Samhita* describe the heart is considered the seat of *Chetana*[(2)] (consciousness) and the controller of circulation[(3)], the liver is a central organ responsible for major digestive and metabolic activities particularly in *Raktavaha Srotasa* as the principal site of *Rakta Dhatu* formation. Any dysfunction in the liver such as in conditions like *Yakrit Vikara* can directly impact cardiac health, manifesting in imbalances of *Pitta Dosha* and *Rakta Dushti*. In present-day medicine, NAFLD illustrates this connection clearly, as it is strongly associated with greater cardiovascular risk. This interconnected view highlights the importance of maintaining harmony as well as preventing and treating the body holistically, addressing both liver and heart health for overall well-being.

Knowledge gap-

Classical Ayurvedic texts offer limited direct references linking the *Hridaya* and *Yakrita*, making it difficult to establish clear connections across their embryology, anatomy, physiology, pathology, and pharmacology as understood in modern medicine. Ayurveda typically addresses *Hridaya* and *Yakrita* separately, with little systematic understanding of their interconnection. There is also a lack of clinical research assessing both organs together from an Ayurvedic perspective, particularly in diseases involving multi-organ dysfunction, such as metabolic, hepatic, and cardiovascular conditions. Bridging these gaps requires further theoretical exploration of classical texts and clinical research integrating Ayurvedic principles with modern medical knowledge.

Need of the study-

This basic research is essential for understanding of embryological, anatomical, physiological and pathological aberrations occurred for improving prevention, diagnostic accuracy, developing targeted treatments, and enhancing overall patient care in *Hrid-Yakrit Vikara*. It is important to unravel the intricate connections between these vital organs within the broader framework of Ayurvedic integrative

biology and to set a benchmark concept for its practical need.

Aim and Objective:

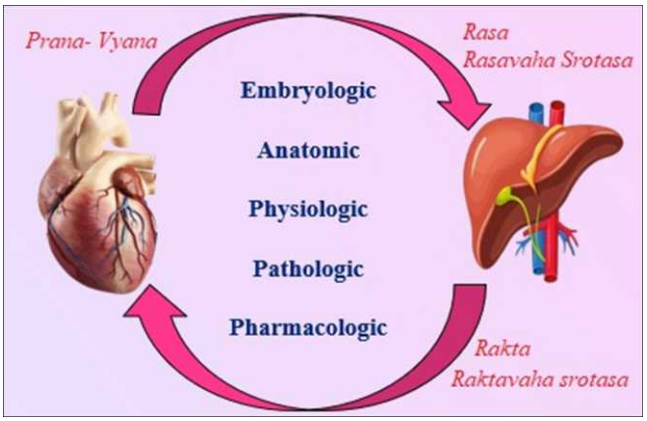
To explore and establish the thinkable Ayurveda perspective for *Hridaya* and *Yakrita* interconnection in conformity with modern medical science.

Materials and Methods:

The data for this self-narrative review is collected from *Brihitrayee* with respective commentary and thorough review of publicly available literature on heart-liver interactions. This involved gathering and synthesizing relevant information and insights from existing research articles related to the same from indexed journals with thoughtful self-narrative.

Discussion:

Figure: 1- The figure showing interplay between *Hridaya* and *Yakrita* at various levels



Embryological interactions-

Genetically, the *Hridaya* and *Yakrita* both are formed and derived from maternal expressions[4]. The *Garbha* (embryo) is formed from the *Sanyoga* (combination) of *Shukra* (sperm) and *Artava* (ovum)[5]. The *Shukra* (sperm) and *Artava* (ovum) are the *Garbha- Utpadak Bija*. During the embryological period, the *Anga-Pratyanga* are formed from the *Garbhautpadak Bija* or the *Bijabhaga*. If the *Bija* or *Bijabhaga* is *Dushita*, the *Anga* (body parts) which will be formed also be *Vikrita*. This clearly shows that not only the formation of *Sharira Avayava* (organs) depends on the characteristics of *Bija* or *Bijabhaga* but also the formation of disease in the later period of life[6]. According to Charaka, during fetal development in the third month, the *Hridaya*, the seat of consciousness (*Chetana*) becomes well developed[7]. As per the Kashyapa heart is derived from blood and liver is formed after bay from heart showing their inherent embryological signaling and origin[1].

As per *Acharya Sushruta Mruduni Matrujani*, This clearly signifies maternal predominance in the development of the Hridaya and Yakrita. *Garbhotpadaka bhava* described by Acharya Charaka and Sushruta clearly stated that the body's soft organs are derived from the maternal factors (*Matraj Bhava*)[4,8]. The *Hridaya* consists of *Mansa*, *Shuddha Rakta*, *Kapha*, and *Sarvadhatu Sara* (the essence of all tissues), or *Oja*, along with the mother's *Rasa*. On the other hand, the *Yakrita* is composed of *Shuddha Rakta* and the mother's *Rasa*.

As per the modern medicine perspective; The embryo's growing need for nutrients and oxygen makes the cardiovascular system the earliest to develop[9]. Although the liver and heart arise from different embryonic cell lineages, their early development occurs side-by-side and is guided by a coordinated sequence of inductive signals. Three signaling pathways Wnt, FGF, and BMP are known to play crucial roles in both heart and liver development. The anterior endoderm, during gastrulation, activates cardiogenic Wnt pathways most notably Wnt/β-catenin to regulate early cardiac development, influencing both heart tube formation and the growth of cardiac progenitor cells. Later in development, FGF signals from the cardiogenic mesoderm act on the nearby anterior endoderm, inducing the transcription of hepatic genes and supporting liver formation. Additional signals such as Bone Morphogenetic Proteins (BMP) signaling from septum transversum promotes liver bud growth and also provide further information required for the formation of liver progenitor cells and liver specification[10, 11]. This suggests that the heart is one of the first organs to develop and plays a pivotal role in liver organogenesis. The process aligns with the Kashyapa theory of organogenesis, which posits that the heart's early development is essential for the induction of other organs, including the liver[11,12].

Anatomical interactions-

According to *Acharya Sushruta*, the heart is situated in between the two breasts and occupying a position in the mediastinum. The *Hridaya* is a *Marma* which when hurt causes instantaneous death[8]. It is formed from the essence part of *Shonita* and *Kapha*.

It is the seat of *Pranavaha Dhamani*. *Pleeha* (spleen) and *Phuphusa* (lungs) are situated just inferior to the left aspect of the *Hridaya*, whereas *Yakrita* and *Kloma* (mediastinum) are located inferior to its right aspect described in *Garbha Vyakarana Sharira* (embryology)[2]. Heart resembles just like a lotus bud. *Hridaya* is the site of *Chetana* and the seat of *Oja*[13]. It is one of the *Trimarma*[14], type of *Sira Marma* and *Sadyapranahara Marma*[15].

Yakrita is situated below *Hridaya* but on the right side of the body. Along with *Pleeha*, *Yakrita* can be considered as *Shonitashaya* for the formation of *Rakta*[16]. It is one of the *Mulasthan* for *Raktavaha Srotas*. It is one of the *Koshthanga*[17] and is the main site of

Ranjak Pitta[18]. The *Raktadhara Kala* resides over the *Sira*, *Yakrita* and *Pleeha* and the circulation of *Rakta* occurs[19].

The heart is positioned in the left chest, whereas the liver occupies the right upper quadrant of the abdomen. This placement results in the heart being superior to the liver. The heart plays a central role in the circulation of many organs by supplying oxygen-rich blood and returning oxygen-poor blood to the lungs. Because the liver has high metabolic demands, it receives nearly one-fourth of the cardiac output through two vessels- the hepatic artery and the portal vein- showing its strong dependence on cardiac function. Its venous outflow drains directly into the hepatic veins and the inferior vena cava, both lacking valves, which allows any rise in right-sided heart pressure to be transmitted straight to the liver. Consequently, both acute and chronic cardiac disorders can precipitate corresponding hepatic dysfunction, and liver disease may similarly influence cardiac status[20]. This mutual dependence highlights the strong connection between the two organs.

Physiological interactions-

Hridaya alone holds the special status of being the *Srotomula* for both *Pra□avaha* and *Rasavaha Srotasa*, two of the most essential bodily pathways[4, 21]. *Acharya* Shushruta designates *Hridaya* as a *Sadhyapra□ahara Marma*[15], and *Acharya Charaka* includes it among the *Trimarma* as well as the *Dasha-vishe□hayatana*. This site is regarded as a principal *Pra□ayatana*[22]. By its constitution, the *Hridaya* is a form of *Sira Marma*, being the source from which *Rasa, Rakta*, and *Ojas* are supplied to the entire body. Structurally, Hridaya is considered a type of Sira Marma, as it distributes Rasa, Rakta, and Ojas throughout the body including *Yakrita* through *Sira*[23]. *Hridaya* which is the *Sthana* of *Rasa* circulates the *Rasa* with the help of *Vyan Vayu* through its *Dhamani* to the entire body and nourishes the other *Dhatu*[24, 25]. *Hridaya* is said to be the seat of *Rakta*[26]. *Hridaya* it-self being a muscular organ derives its nutrition from *Rasa* and *Rakta* and vital energy from *Oja*[14].

Acharya have described the root of *Raktavaha Srotas* is *Yakrita* and *Pleeha* and *Raktavahi Dhamini* [21] (blood vessels) which carry the *Rakta Dhatu* to *Hridaya* through *Saman Vayu*[27, 28]. *Rakta* being *Prana* (life) of individual, it has been given a supreme importance in health and disease.

After the process of *Ahara Pachana*, the *Prasada-bhuta Rasa* is then transported to the *Yak□ita*, where it undergoes *Ragak□ita* (coloration or transformation). In this process, the *Rasa* is converted into *Rakta Dhatu*, while the *Kitta-Amsha* is transformed into *Mala* and *Mutra*[27,28].

Acharya Charaka has described that the *Teja* (metabolic energy) component of *Rasa* combines with the *U□hma* of *Pitta* specifically, *Ranjaka Pitta* to impart the red color and qualities necessary for the formation of *Rakta Dhatu*. Supporting this, *Acharya* Dalhaṇa, in his commentary on the Sushruta Samhita, explains that *Teja-Rupa Ranjaka Pitta* interacts with *Avyapanna* (non-pathological), *Vikra-rahita Rasa Dhatu* to produce healthy *Rakta*[27,28]. This is a Prana of every tissue of the body. So, in this context *Hridaya* and *Yakrita* are connected via *Rasa-Rakta* and respective *Srotasa*.

Inter-organ communication is regulated through organokines- protein molecules synthesized and released by specific tissues. Rather than acting merely as indicators of tissue activity, these organ-derived factors exert paracrine, endocrine, or dual modes of action. The heart produces a group of signaling proteins known as cardiomyokines, while the liver releases hepatokines. Together, these molecules play a vital role in coordinating functional interactions between the heart and liver[12].

a) Cardiomyokines Link the Heart to Liver[12]

Apart from producing natriuretic peptides (NPs), cardiomyocytes release cardiomyokines can act locally through autocrine or paracrine pathways, or exert endocrine effects on distant organs, including the vasculature, kidneys, skeletal muscle, bone, adipose tissue, and the liver. They modulate hepatic inflammation and lipid metabolism and non-secretary cardiac genes influence liver metabolism. Atrial natriuretic peptide (ANP) also plays a protective role in the liver by reducing the activity of pro-inflammatory transcription factors and limiting necrotic and apoptotic damage.

b) Hepatokines Link the Liver to Heart[12]

Hepatocytes release hepatokines, which influence metabolic regulation locally and throughout the body using autocrine, paracrine, and endocrine signaling routes. Their effects extend to remote organs such as skeletal muscles, adipose depots, pancreatic tissue, blood vessels, and cardiac tissue.

Pathological interactions-

Being *Mula Sthana* of *Rasavaha Srotasa*, the *Prasadbhuta Ahara Rasa* (nutrients) is circulated from the *Hridaya* through *Dhamani* to nourish various *Avayava*, *Dhatu*, and *Upadhatu*[29]. When the *Dosha* become aggravated, they vitiate the *Rasa Dhatu*, which circulates through the *Hridaya*, impairing its physiology and resulting in *Hridroga*[30]. According to the *Dhatu*

PoshanNyaya,whentheHridayabecomesdiseased,itsfunction of distributing nutrition is hindered, leading to impaired formationandfunctioningofsubsequent*Dhatu, Upadhatu*and *Avayava*including*Yakrita*.Thesuccessive*Dhatu*suchas*Rakta, Mamsa*,and*Meda*arealsovitiated,initiatingthedevelopmentof various diseases[31]. Inadequate formation of *Rakta Dhatu*, primarily formed in the *Yakrita*, leads to dysfunction of *Yakrita* and further vitiation of the *Raktavaha Srotasa*, resulting in insufficientnourishmenttothe*Hridaya*.

The*Hridaya*develops from the essence of*Rakta Dhatu* during intrauterine life[2]. *Rakta* is considered the *Jeevanam* of an individual; hence, any alteration in its quality or quantity significantlyaffectsthefunctionalandstructuralintegrityofthe *Hridaya*[32]. As per Sushruta, the *Hridaya* is a *Sira Marma*[15]. Depletion of *Rakta* causes *Sira Shaithilya*, which affects the *Hridaya* as it is composed mainly of *Sira*[33]. Moreover, the vitiationof*RaktaSara*alsoadverselyimpacts*Hridaya*function.

Beingthe*Mula Sthana*of*Raktavaha Srotasa, Yakrita*produces *Rakta* with the help of *Ranjaka Pitta* and circulates it through *Raktavaha Srotas*a. When the *Yakrita* and its constituents are vitiated,theformationof*ShuddhaRakta*ishampered,leadingto thecirculationof*AshuddhaRakta*[34]. Thisresultsin*VyanaVayu* vitiation in the *Hridaya*, disrupting its natural *Sankocha* (contraction) and *Vikasa* (expansion) functions, eventually manifestingas*Hridroga*.The*Hridaya*isalsoregardedasthesite of *Ojas.* Any disequilibrium in the *Hridaya* can lead to *Oja Kshaya*,whichmayprovefatal[14].

The predominant *Dosha* involved in *Hridroga* is *Vata* particularly*Vyana*and*PranaVayu*.Theprimary*Dhatu*involved inthepathogenesisof*Hrid-YakritRoga*andtheir*Upadrava*are *Rasa*and*Rakta*.Improperformationof*Rasa*anddysfunctionof *Rasagni* leads to *Rakta Dhatu* vitiation, which subsequently disturbs the other *Dhatu, Upadhatu*, and *Mala*. From an embryological perspective,*Rakta*isacrucialcomponentinthe formationofboth*Yakrita*and*Hridaya*.Theinterconnectedroles of*Yakrita*and*Hridaya*indiseasemanifestationcanbeattributed to the vitiation of *Rasa, Rakta, Rasavaha*, and *Raktavaha Dhamani.*

The mutual causation between heart and liver diseases, termed *Nidanarthakar Roga*. *Nidanarthakaratva*, is pivotal in understanding disease pathogenesis[35]. *Vyadhi Sankara*, elucidated by *Acharya* Chakrapani, denotes the intricate interplay of diseases resulting in compounded clinical symptoms. Accordingly, Coexistence of *Hrid-Yakrit Roga* significantly impacts treatment strategies, clinical presentation, and outcomes[35]. *Upadrava* (complications) is secondary disease that manifest in association with the primary disease in the body. *Hrid-Yakrit Vikara* exemplifies this interdependency as mutual *Upadrava*[36, 37]. Sushruta, Madhavnidana identify *Bhrama, Klama, Sada*, and *Shosha* as specific *Upadrava* of *Hridroga*. However, contemporary contexts reveal evolving complications, including *Yakrita Vikara.*

Cardiovascular diseases (CVD) and liver disorders influence one another, making their assessment clinically significant. However, distinguishing how each condition contributes to a patient's presentation can be challenging, as both share overlapping risk factors and underlying pathological mechanisms.

1. LiverdiseasesaffectingtheHeart[12,38]

Cirrhoticcardiomyopathy:

CirrhoticCardiomyopathy(CCM)ispresentswithbothsystolic and diastolic dysfunction, electromechanical abnormalities, and impaired ventricular ejection fraction due to decreased myocardial contractility and inadequate heart rate response duringexertion.Centralhypovolemiainhepaticcirrhosisleads to sympathetic nervous system activation, resulting in a hyper dynamic circulatory state with elevated heart rate and cardiac output.Thepathophysiologyinvolvesproinflammatorystates causing cardiomyocyte apoptosis, alteration in myosin isoforms(fromαtotheweakerβsubtype),anddownregulationof β-adrenergicreceptorfunctioncontributingtomyocardialhypo responsiveness. Prolonged QT intervals potentially linked to chronicnoradrenalinexposureandchronotropicincompetence areattributedtoelevatedbilesalts,andincreaseduricacidlevels. As liver failure advances, systemic vasodilatation progresses and cardiac systolic reserve diminishes, ultimately reducing effectivecirculatoryvolume.

Livertransplantation:

Cardiovascular complications following liver transplantation can manifest as Takotsubo cardiomyopathy (TTC) or Stress-induced cardiomyopathy marked by hypo or dyskinesia in the apical, lateral, and posterior walls of the left ventricle. Elevated levels of circulating catecholamines increase the heart's vulnerabilitytovasospasminmultipleepicardialvessels.

Non-alcoholicFattyLiverDisease:

Non-alcoholic fatty liver disease (NAFLD) contributes to increased risk of heart-related complications, such as coronary artery disease, structural changes in the myocardium, cardiac arrhythmias, and left ventricular diastolic dysfunction. The heightened lipogenesis is associated with a greater risk of cardiovascular issues. Considered a chronic, subclinical inflammatory disorder, NAFLD leads to the release of pro-inflammatory cytokines, including elevated leptin levels, decreased cardio-protective adiponectin, and various chemokines, along with other inflammatory markers. Serum levels of IL-6, IL-1, TNF-α, and C-reactive protein (CRP) have been linked to cardiovascular events in NAFLD. Additionally, increased oxidative stress and elevated production of

prothrombotic factors-specifically factors VII, IX, XI, and XII-can further exacerbate cardiovascular risk and contribute to atherosclerosis.

2. Heart Diseases Affecting the Liver[12,39,40]

Hypoxic hepatitis:

An abrupt decrease in cardiac output in acute heart failure causes reduction in blood flow to the liver results in severe hepatic hypoxia and cell death, despite compensatory mechanisms attempting to maintain function, ultimately leading to hypoxic hepatitis.

Cardiac cirrhosis:

In chronic heart failure, persistent passive congestion in the liver, known as congestive hepatopathy, leads to sinusoidal hypertension and centrilobular fibrosis, ultimately resulting in a condition referred to as "cardiac cirrhosis."

Congestive Hepatopathy:

It refers to liver congestion caused by impaired hepatic venous outflow, typically due to right-sided heart failure. This condition leads to elevated pressures in the hepatic veins, reduced hepatic blood flow, and lower arterial oxygen levels. Valvular heart diseases, cardiomyopathy, left heart failure, and constrictive pericardial disease can all contribute to the development of congestive hepatopathy. Chronic congestion results in the dilation of hepatic sinusoids, which causes the leakage of red blood cells and protein-rich fluid into the perisinusoidal space of Disse. This process is associated with steatosis, atrophy, and/or necrosis in the centrilobular region of the liver parenchyma.

3. Genetic Associations between Liver Disease and Cardiovascular Disease:[41]

The presence of the PNPLA3 I148M variant has been found to significantly increase the risk of early-onset coronary artery disease (CAD) in individuals with type 2 diabetes (T2DM). Those with the homozygous PNPLA3 I148M genotype face an even higher risk of cardiovascular disease, in the context of NAFLD. On the other hand, the TM6SF2 E167K variant, which is linked to hepatic steatosis, fibrosis, and the development of hepatocellular carcinoma (HCC), appears to offer a protective effect against cardiovascular disease by lowering total cholesterol, LDL cholesterol, and serum triglyceride levels. The PEMT rs12936587 variant, however, has been associated with a higher susceptibility to CAD.

While certain genes strongly linked to NAFLD do not appear to directly contribute to an increased risk of coronary artery disease, the TM6SF2 E167K variant seems to provide a protective effect against cardiovascular disease, and the PNPLA3 I148M variant does not have a significant impact on CVD. In contrast, associations have been observed with the TRIB1 and PEMT genes.

Pharmacological interactions-

Currently, there is no conclusive evidence on how cardiac herbs affect liver health or vice versa. However, most herbs are metabolized in the liver. These medicines interact through *Rasa-Rakta* pathways, influencing *Tridosha* and *Agni*, strengthening *Avayava*, promoting *Srotasa Shodhana*, and through *Rasayana* action (rejuvenation). According to Ayurvedic principles, the heart is characterized by a *Sheeta* nature, whereas the liver is inherently *Ushna* in nature[42], indicating a reciprocal *Prakriti* (functional constitution). Consequently, the *Guna* (pharmacodynamic properties) of herbal interventions for cardiac and hepatic disorders tend to be contrasting. Inappropriate use or disregard of established Ayurvedic guidelines for administration may lead to unintended effects on either organ system.

Hridya Mahakashaya, a group of cardio protective herbs in Ayurveda abundant in Vitamin C, phytochemicals like alkaloids, polyphenols, glycosides, triterpenoids, tannins, flavonoids, and beta-carotene. These constituents exhibit antioxidant, anti-inflammatory, immunomodulatory, antidiabetic, anxiolytic, and antimicrobial properties, which help reduce oxidative stress and inflammation, major contributors to CVD and liver diseases. In Ayurvedic terms, the *Amla rasa* of these herbs aid *Deepana*, *Pachana*, and *Vatanulomana*, helping normalize the vitiated *Dosha* and interrupt the pathogenesis of *Hridroga*[43,44].

Initial studies indicate that Arjunolic Acid (AA), a bioactive compound derived from the bark of Terminalia arjuna, could offer potential benefits in treating NAFLD. Extracts from T. arjuna bark at a dose of 500 mg/kg have demonstrated notable anti-lipidemic, hepatoprotective, and renoprotective effects in animal models of obesity and metabolic disorders induced by a high-fat diet[45,46].

In cardiovascular therapeutics, common Ayurvedic herbs such as *Ashwagandha* (Withania somnifera), *Brahmi* (Bacopa monnieri), *Guggulu* (Commiphora mukul) and *Gokshur* (Tribulus terrestris) are frequently used which have been associated with hepatotoxicity when used inappropriately or without supervision, potentially leading to hepatocellular, cholestatic, or mixed liver injury patterns[47].

Heart Disease Affects Liver Pharmacokinetics:[48]

1. Reduced Liver Blood Flow:

Heart failure reduces cardiac output and peripheral perfusion, potentially decreasing liver blood flow and impacting drug clearance. This affects drug pharmacokinetics (PK) and pharmacodynamics (PD). Regardless of etiology, chronic heart failure can modify drug absorption, distribution, metabolism, and elimination.

2. HepaticCongestion:

Backup of blood in the liver due to right-sided heart failure can cause congestion, reducing hepatic enzyme activity and impactingdrugmetabolism.

3. DrugrelatedLiverInjury:

This may result from direct hepatocellular effects or altered hepatic perfusion, particularly relevant in cardiovascular conditions.

LiverDiseaseAffectsHeartPharmacokinetics:[49]

The liver's central role in drug metabolism is significantly impactedbyhepaticdiseasessuchascirrhosis.

1. PharmacokineticChanges:

Liver impairment and associated co morbidities can alter cytochrome P450 activity, typically reducing drug clearance and increasing serum drug levels. Liver diseases such as cirrhosis, Non alcoholic steato hepatitis, viral hepatitis, and cholestatic liver diseases also affect drug pharmacokinetics by modifying transporter expression and function, influencing transporter-mediateddrug-druginteractions.

2. Hypertension:

In cirrhosis, diminished hepatic clearance can result in higher drug concentrations, increasing the likelihood of adverse effects. Angiotensin converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), and calcium channel blockers (CCBs) can be used with caution in compensated cirrhosis.

3. Hyperlipidaemia:

Statins in decompensated cirrhosis may cause toxicity and rhabdomyolysis[50].

4. ProteinBinding:

In cirrhosis, hepatic albumin production is reduced by 60–80%, leading to hypoalbuminemia. This reduction increases the unbound fraction of highly albumin-bound drugs, raising the riskofdrugtoxicity.

5. ChangesinVolumeofDistribution:

In liver disease, the volume of distribution of cardiovascular drugs such as CCB, ACE inhibitors, and ARBs can be altered, impacting drug disposition. In alcoholic liver disease (ALD), antipsychotics like haloperidol and quetiapine may cause QT prolongation and arrhythmias. Additionally, in ALD, compromised hepatic metabolism may be further impaired by drugslikepropranolol,whichcanreducehepaticbloodflowand exacerbatemetabolicdysfunction[51].

Diagnosticapproach:

*Dosha-Vata*dominant*Pitta*

Dushya-Rasa,Rakta

*Srotasa-Rasavaha*and*Raktavaha*

Mala-Kapha,Pitta

Samprapti-Atiprvritti,Sanga,Siragranthi,Vimarggaman

VyadhiMarga-Madhyam

Hetu-NidanarthakarVyadhi-eitherofthemmayberesponsible forother

VyadhiSankara-bothconditionsmaycoexist

Upadrava-oneentitycanbecomplicatedbyother

Arishta-presenceofoneentitymaybebadprognosticindicator forother

Summary table showing Heart- Liver Mutual Interactions

Interrelations	Ayurveda Concepts	Modern Concepts
Embryological	*Raktadhatu - Mrudunit Matrujani*	BMP, Wnt, FGF signalling
Physiological	*Rasa - Rakta Dhatu and Srotasa, Vyana Vayu, Samana Vayu*	Cardiomyokines and Hepatokines
Anatomical	*Rasavaha - Raktavaha Srotasa*	Hepatic artery and vein, Portal vein, IVC
Pathological	*Vitiated Rasa - Rakta Dhatu and Srotasa, Vyana, Samana Vayu, Nidanarthakara Vyadhi, Vyadhi Sankar, Upadrav*	Cirrhotic Cardiomyopathy, NFLD, Hypoxic Hepatitis, Congestive Hepatopathy, Cardiac Cirrhosis
Pharmacological	*Rasa - Rakta Pathways, Agni Srotasa Shodhana Hepatotoxicity*	Pharmacokinetics and Pharmacodynamics alteration in diseases

Conclusion:

The liver and heart embryologically, physiologically, anatomically, pathologically, pharmacologically interconnected via *Rasa* and *Rakta*. The organokines, communicators for organ interaction are the *Dhatwansha* from respective organs participated in reciprocation which under the influence of *Prakrut Vyana* and *Samana Vatadosha* accomplish their functions. *Vata* is the *Pradhan Dosha* and *Rasa - Rakta Dhatu* are seem to be the prime *Dhatu* involved. *Hrid-Yakrit Roga* are not only limited to mutual pathogenesis but these affect the whole body progressively. Furthermore, rigorous researches are to be undertaken to explore in deeper *Hrid-Yakrit* interrelations.

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Conflict of Interest: Nil

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