



Review Article

A CRITICAL ANALYSIS OF *PLIHODARA* WSR TO PORTAL HYPERTENSION

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ABSTRACT

Plihodara is a distinct clinical condition described under *Udara Roga* in Ayurveda and is classified among the *Ashta Mahagada* signifying its chronic and challenging nature. It is primarily characterized by progressive enlargement of the spleen resulting from *Agnimandya*, *Ama* formation, vitiation of *Rasa* and *Rakta Dhatu*, and obstruction of *Raktavaha Srotas*. In contemporary medicine, portal hypertension is recognized as a major consequence of chronic liver disease and is characterized by persistent elevation of pressure within the portal venous system. This rise in pressure results primarily from increased resistance to blood flow within the liver along with hemodynamic alterations in the splanchnic circulation. Clinically, it presents with splenomegaly, ascites, development of portosystemic collateral vessels, hypersplenism, and multiple systemic complications. Owing to its progressive course and widespread physiological impact, portal hypertension significantly increases both morbidity and mortality. This review critically analyzes the *Nidana Panchaka*, *Samprapti*, *Dosha-Dushya* involvement, and *Srotodushti* of *Plihodara* based on classical texts including *Charaka Samhita*, *Sushruta Samhita*, and *Ashtanga Hridaya*, and systematically correlates them with the modern understanding of portal hypertension. The striking similarity in clinical presentation suggests a possible conceptual relation between *Plihodara* and portal hypertension. The conceptual parallels highlight the depth of Ayurvedic pathological understanding and provide a framework for integrative interpretation of hepatosplenic disorders.

INTRODUCTION

Plihodara is a distinct clinical entity described under *Udar roga*. *Udar Roga* is categorized among one of the eight disease *Asthamahagada* (disease is difficult to manage)^[1]. *Udar roga* denotes the generalised distension or enlargement of varied etiology^[2]. Classical ayurvedic text attribute the pathogenesis of *Udara Roga* primarily to impaired digestive fire (*Agni*) and the accumulation of improperly digested food (*Ama*) and waste product^[3]. As per Ayurvedic principle *Agni* plays important during the disease process "*Rogah sarve api mande agnau sutaramudarani tu*"^[4]. *Agni dosha* (digestive metabolic dysfunction) and *Mala*

vriddhi (increased waste) causes vitiation of *Prana* (vital force) *Agni* (digestive fire) and *Apana* (eliminative vital force) and obstruction of the upward and downward channels of circulation^[5].

From a modern perspective, a portal hypertension is a pathological condition characterized by increased pressure in the portal venous system, The condition is defined by an increased portal pressure gradient, which represents the difference between portal venous pressure and the pressure in the inferior vena cava or hepatic vein, most commonly associated with chronic liver disease^[6]. The striking similarity in clinical presentation and disease progression between portal hypertension suggests a possible conceptual and pathological correlation between the two conditions.

Exploring this correlation provides a meaningful framework for interpreting classical Ayurvedic descriptions in light of contemporary medical understanding. Hence, the present article aims to analyze *Plihodara* with reference to its *Samprapti*

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(Pathogenesis) and clinical features and to correlate them with portal hypertension, there by contributing to an integrative understanding of splenic and portal venous disorders.

MATERIAL AND METHOD

Classical Ayurvedic texts including *Charaka Samhita*, *Sushruta Samhita*, *Ashtang Hridaya*, and relevant commentaries along with contemporary medical literature on portal hypertension and its pathophysiology. The collected data were critically analyzed to evaluate *Samprapti*, *Dosha dushya* involvement, *Srotas* pathology and clinical features, and a conceptual correlation was established between ayurvedic and modern perspective.

RESULT

Nidan panchak of Plihodara

Nidan (Causes)

According to *Acharya Charaka* and *Vagbhata Plihodara* arises due to various dietary and lifestyle disorder. These include intake of excessively irritating food, travelling in excess (*Asitasyatisankhsobh*), over-exertion (*Atichesta*), excessive sexual activity (*Ativyavaya*), carrying heavy loads (*Bhardhavan*), repeated vomiting (*Vaman*) and debility caused by chronic illness (*Vyadhikarshane*).^[7,8] *Acharya Sushruta* emphasizes that habitual consumption of *Vidhai* (food causing burning) and *Abhishyandi* diet. Then *Asrk* (blood) and *Kapha* become vitiated greatly make for enlargement of the *Pliha* (spleen) gradually, in the left

side of the abdomen especially, and produce *Plihodara* (abdominal enlargement due to enlargement of spleen).^[9]

Purvaroopa (pre-monitory signs and symptoms)^[10]

The premonitory symptoms include inability to appreciate the digestion and indigestion of the food, loss of appetite (*Kshunnasha*), delayed digestion of sweet, greasy and heavy for digesting foods (*Atisnigdha-Guru-Annam Pachyate Chirat*), foods that causes a burning sensation (*Bhuktam Vidahyate*), intolerance to overeating (*Sahate Natisauhityam*), slight edema in the feet (*Ishacchophashcha Padayo*), persistent decline in physical strength, breathing difficulty also with light exertion (*Alpe-Api-Vyayame Shwasamruchhati*), abdominal distension due to accumulation of fecal material as a result of dryness or *Udarvata* (abnormal upward course of *Vata*) abdominal pain, fullness, distension, or bursting-type abdominal pain at the region where the bladder joins the surrounding tissue, distension of the abdomen even after minor meal, the appearance of dilated veins, and the loss of normal skin folds (*Rajjanma Valinasha*).

Roopa (Common sign and symptoms)

The *Lakshana* of *Plihodara* described by *Acharya Charaka Sushruta* and *Vagbhata* show both concordance and variation in manifestation. A comparative analysis of these *Lakshana* is presented in table 1 below.^[11,12,13]

<i>Lakshana</i>	<i>Charaka</i>	<i>Sushruta</i>	<i>Vagbhata</i>
<i>Daurbalya</i> (weakness)	+	+	-
<i>Aruchi</i> (anorexia)	+	-	+
<i>Asayavairasya</i> (tastelessness)	+	-	+
<i>Avipaka</i> (indigestion)	+	-	-
<i>Agnimandya</i> (weak <i>Agni</i>)	+	+	-
<i>Manda Jvara/Jvara</i> (mild fever)	+	+	+
<i>Pipasa</i> (excessive thirst)	+	-	+
<i>Pandu</i> (anemia)	+	+	+
<i>Swasa</i> (dyspnoea)	+	-	+
<i>Kasa</i> (cough)	+	-	+
<i>Adhmana</i> (abdominal distension)	+	-	+
<i>Shool/Ruk</i> (pain)	+	-	+
<i>Varchamutragraha</i> (faeces and urinary retention)	+	-	-
<i>Chardi</i> (vomiting)	+	-	+
<i>Murcha</i> (syncope)	+	-	+
<i>Daha</i> (burning sensation)	-	-	+
<i>Moha</i> (altered consciousness)	-	-	+

<i>Angmarda</i> (body ache)	+	-	-
<i>Angsada</i> (fatigue)	+	-	-
<i>Karsya</i> (emaciation)	+	-	-
<i>Tamah pravesh</i> (transient loss of consciousness)	+	-	-
<i>Nila harit raji yukt udara</i> (abdomen with bluish greenish venous marking)	+	-	+

As shown in table 1, *Manda jawar*, *Pandu* are uniformly described by all three *Acharya*, indicating a common *Dosha* basis of the disease. *Acharya Charaka* provides a more extensive description including *Avipaka*, *Agnimandya*, *Varchmutragraha* and neurological features such as *Tamah Pravesh*.

Acharya Sushruta emphasize *Agnimandya*, *Pandu* and *Kshina Bala* reflecting a concise pathological approach. *Acharya vaghbhata* highlights advanced features like *Daha Moha Murcha* and *Udar Varna Vaivaryana*.

Samprapti (Pathogenesis)

Due to the etiological factors the spleen located in the left side gets displaced from its normal place and thus become enlarged or the blood increased (accumulated) through *Rasa* (plasma) *Rakta* (blood)

also causes enlargement of spleen^[14]. Enlarged spleen distends the abdomen causing *Pilohdara*.

Splenic enlargement takes place in two different ways, viz., by displacement (*Cyuta virdhi*) or by the increase in the quantity of blood (*Acyuta virddhi*), *Rakta* or blood increases in quantity because of the increase in the quantity of *Rasa* or chyle. Increase in blood can also take place because of the increase in the quantity of other tissue elements like *Mansa Dhatu* or muscle tissue.^[15]

Patient suffers from progressive enlargement of the spleen. Initially the enlarged spleen is palpable as a hard mass identical to the oval metal ball used by blacksmith. The surface of the spleen appears smooth similar to the dorsum of the tortoise. If left untreated the enlarging spleen encroach the whole abdomen including abode of *Agni*.



<i>Dosha</i>	<i>Kapha Pradhan Tridoshaj</i>
<i>Dushya</i>	<i>Rasa Rakta</i>
<i>Agni</i>	<i>Jatharagni Mandangni</i>
<i>Adhisthan</i>	<i>Pliha</i>
<i>Srotas</i>	<i>Rasa and Raktavaha</i>
<i>Srotodushti</i>	<i>Sanga and Vridhi</i>
<i>Rogmarga</i>	<i>Abyantra</i>
<i>Sadhya Asadhyta</i>	<i>Krichsadhya</i>

Portal hypertension

Portal hypertension is caused by a combination of two simultaneously occurring hemodynamics processes: (1) increased intrahepatic resistance to the passage of blood flow through the liver due to cirrhosis and regenerative nodules, and (2) increased splanchnic blood flow secondary to vasodilatation within the splanchnic vascular bed. The portal venous system normally drains blood from the stomach, intestines, spleen, pancreas, and gallbladder, and the portal vein is formed by the confluence of the superior mesenteric and splenic veins. Deoxygenated blood from the small bowel drains into the superior mesenteric vein along with the blood from the head of the pancreas, the ascending colon, and part of the transverse colon. Conversely, the splenic vein drains the spleen and the pancreas and is joined by the inferior mesenteric vein, which brings blood from the transverse and descending colon as well as from the superior two-thirds of the rectum.^[16]

The primary reason for the increased vascular resistance is the dysregulation of liver sinusoidal endothelial cells and hepatic stellate cells, or from dynamic changes (i.e., vasoconstriction). Structural changes are due to the alteration of hepatic microcirculation. Such alterations result from the activation of hepatic stellate cells, leading to the development of fibrosis, regenerative nodules, vascular occlusion, and angiogenesis. The increased production of endothelial vasoconstrictors (e.g., endothelin) and decreased release of vasodilators (e.g., nitric oxide) within the liver leads to sinusoidal constriction, thereby increasing hepatic resistance. Portal hypertension stemming from this is augmented and perpetuated by the increased blood flow within the splanchnic circulation. This increased blood flow is attributed to the increased release of splanchnic vasodilators, notably nitric oxide, resulting from increased shear stress and a reduced effective arterial volume. The splanchnic arterial dilatation plays a significant role in the worsening and progression of portal hypertension. Thus, portal hypertension results from increased resistance to portal venous flow and increased portal blood flow due to splanchnic vasodilation.^[17]

DISCUSSION

Portal hypertension is characterised by prolonged elevation of the portal venous pressure (normally 2-5mmHg). Patients developing clinical features or complications of portal hypertension usually have portal venous pressure above 12mmHg.^[18]

Congestive splenomegaly is common in patients with portal hypertension. Clinical features

include the presence of an enlarged spleen on physical examination and the development of thrombocytopenia and leukopenia in patients who have cirrhosis. Some patients will have fairly significant left-sided and left upper quadrant abdominal pain related to an enlarged and engorged spleen.^[19] Patients represent with ascites along with peripheral edema.

Collateral vessels may be visible on the anterior abdominal wall and occasionally several radiate from the umbilicus to form a caput medusae. In portal hypertension, a local hyperdynamic state occurs around the spleen. Splenomegaly is associated with a poor prognosis in cirrhosis and is caused by spleen congestion and by enlargement and hyperactivation of splenic lymphoid tissue.^[20]

Plihodara is explained as a long-standing condition arising from obstruction of the *Raktavaha Srotas*, resulting in disturbed blood circulation and venous stasis. This pathological concept shows close resemblance to portal hypertension, wherein elevated resistance to portal venous flow produces widespread systemic effects.

The description of *Pliha vridhhi* in *Plihodara* corresponds to congestive enlargement of the spleen observed in portal hypertension, commonly accompanied by anaemia and generalized weakness due to hypersplenic activity. The classical finding of *Nila-Harita Raji Yukta Udara* can be conceptually correlated with caput medusae, reflecting the formation of collateral venous channels as a consequence of vascular obstruction. Symptoms such as *Udara Shula* and *Adhmana* are attributed to *Vata* aggravation along with *Udakavaha Srotas* dysfunction, which parallels abdominal discomfort and fluid accumulation seen in portal hypertension. In progressive stages, manifestations like *Moha*, *Murccha*, and *Tamah pravesha* suggest involvement of *Manovaha Srotas* and can be interpreted as hepatic encephalopathy. In Ayurveda, untreated *Plihodara* progresses to *Jalodara*, characterized by fluid accumulation in the abdomen. Similarly, in modern medicine, persistent portal hypertension leads to the development of ascites. Thus, the transition from *Plihodara* to *Jalodara* parallels the progression of portal hypertension to ascites. Hence, *Plihodara* can be critically viewed as a chronic hepatosplenic disorder marked by circulatory impairment, abnormal fluid retention, and neuropsychiatric features.

CONCLUSION

Plihodara is a well described disease entity in Ayurveda that exhibits remarkable clinical similarity with portal hypertension. The symptomatology of

Plihodara ranging from splenomegaly, digestive impairment, abdominal distension, and discolouration of abdominal walls to systemic features like debility and emaciation closely parallel the manifestations of portal hypertension. This correlation highlights the depth of Ayurvedic pathological understanding and provides a strong conceptual framework of integrative interpretation. Recognising this correlation may help in better clinical understanding, early diagnosis and integrative management approaches for splenic and portal venous disorders.

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