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CONCEPT IN PATHOGENESIS OF *JALODARA* WITH SPECIAL REFERENCE TO PORTAL HYPERTENSION

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ABSTRACT
Jalodara is one of the eight types of *udar roga* described in Ayurveda. It is mentioned in all three *brihatrae* texts (*Charaka Samhita*, *Susruta Samhita* and *Astang Hridaya*).[1,2,6] *Jalodara / Dakodar* is formed due to accumulation of *jala* (body fluid) in *udara* (abdomen). *Mandagni* is claimed to be the main factor behind its pathogenesis.[5] In all the three texts there is involvement of *udak vaha srotas* or *salil srotas* or *ambu srotas* with respect to *jalodara* pathology. *Udak vaha srotas* has its *moola* (base) which are *talu and kloma*. *Kloma* is a controversial subject in ayurveda. Some authors compare it to pancreas. It is mentioned that *dusti* (defect) of *udak vaha srotas* or *kloma* is leading to *jalodara*. So if we compare *kloma* to pancreas and look for the anatomical relations of pancreas then we will see that there is portal vein forming behind neck of pancreas. In modern science it is proved that pathology in portal vein which is portal hypertension is the cause for development of ascites. So here if for the term *kloma*, the related and associated structures to pancreas are taken then by studying the Portal vein, *jalodar*a is well explained.

KEYWORDS: *Jalodara, udak vahi srotas, kloma*, pancreas, portal vein, portal hypertension, ascites.

INTRODUCTION

The *udar roga* is classified into the eight great diseases (*asta maha gada*) in ayurveda. *Mandagni* has the key role to play in its development. There are eight types of *udar roga* (abdominal disorders) mentioned in texts namely- *vataodara, pittodara, kaphodara, sannipatodara, pleehodara, baddhodara, kshatodara and udakodara or jalodara*. *Jalodara* is basically considered to be the disease in which there is filling of *jaliya ansh / jal* (body fluid) inside *udara* (abdomen). It is understood to be a difficult to treat disease. In modern part it is ascites which is basically free fluid inside peritoneal cavity in abdomen. It is most common feature in liver diseases and is a sequelae of portal hypertension in liver cirrhosis.

Synonyms of *Jalodara*- *udakodara, dakodara, jaatodaka, ajaatodaka*.

MATERIAL AND METHODS- Comparative literary study of Jalodar (ascites) in both ayurvedic and modern view.

Pathogenesis of *Jalodara* in ayurvedic classics

Apart from common etiological factors of *udar roga*, there are also specific cause of *jalodara* mentioned which are excessive amount of water intake after heavy fatty diet or to the persons having *mandagni*. These etiological factors cause destruction of further *agni (jathragni)* followed by vitiation of *vata* in relation to *kloma* and obstruction due to vitiated *kapha*. These cause collection of watery part inside abdomen and formation of *jalodara*. *Jalodara* is a difficult to treat disease and it is considered to be a sequelae of every other type of *udar roga*[4] when it becomes incurable.

Ascites & Portal Hypertension in modern literature

Ascites refers to the collection of excess fluid in the peritoneal cavity. It usually becomes clinically detectable when at least 500ml have accumulated. Many liters may collect and cause massive abdominal distension. It is frequently seen in liver cirrhosis.[7]

It is generally a serous fluid having as much as 3gm/dl of protein (largely albumin) as well as the same concentration of solutes such as glucose, sodium and potassium as in the blood.

Ascites is considered to be a clinical consequence of portal hypertension, other are the formation of portosystemic venous shunts, congestive splenomegaly and hepatic encephalopathy. Increased resistance to portal flow in portal hypertension may develop from prehepatic, intrahepatic, and posthepatic causes. The

dominant intrahepatic cause is cirrhosis, accounting for the most of the cases of portal hypertension. Portal hypertension in cirrhosis results from increased resistance to portal blood flow at the level of the sinusoids and compression of central veins by periventricular fibrosis and expanded parenchymal nodules. Anastomoses between the arterial and portal systems in the fibrous bands also contribute to portal hypertension by imposing arterial pressure on the normally low pressure portal venous system.

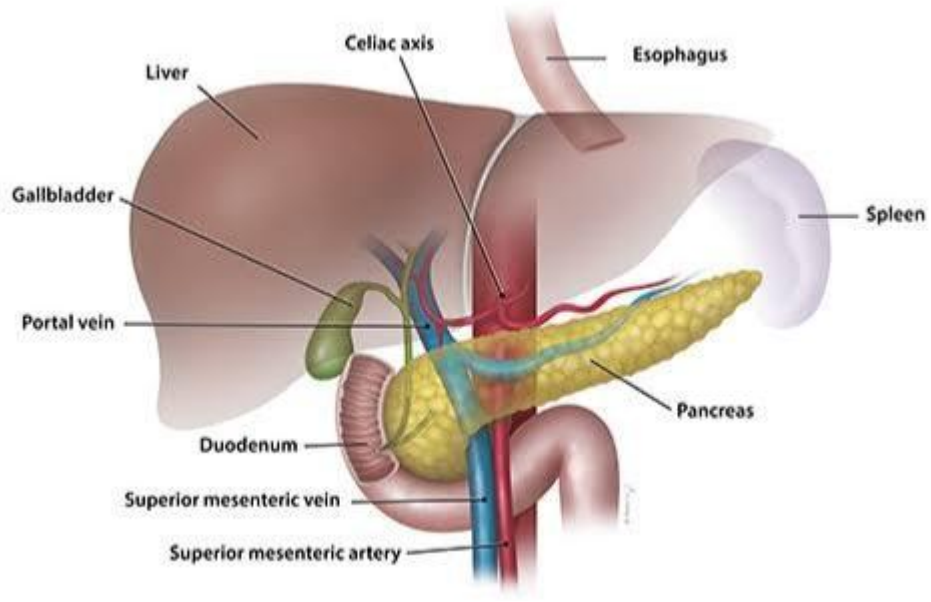
The pathogenesis of ascites is complex. Involving one or more of the following mechanism-

- Sinusoidal hypertension- alters starling forces and drives fluid into the space of disse, which is then removed by hepatic lymphatics; this movement of fluid is also promoted by hypoalbuminemia.
- Leakage of hepatic lymph into the peritoneal cavity
- Renal retention of sodium and water due to secondary hyperaldosteronism.

DISCUSSION

In the ayurvedic view we see that *udak vahi srota* and *kloma* comes frequently while describing the pathogenesis of *jalodara*. *Udak vahi srotas* defect is occurring due to the the obstruction by *kapha* which is causing dysfunction in *kloma* and related structures. *Kloma* although a controversial but very important topic in ayurveda has been called as the *mool* (basis) of *udak vaha srotas* by *Charaka* and *Susruta* both. Its *viddha lakshana* (traumatic symptoms) is *pipasa* (thirst) and *marana* (death). *Kloma* according to some authors has been compared ton pancreas. Pancreas[8] or *Agnashaya* is a J-shaped abdominal organ which lies retroperitoneally, overlying & transversely crossing the bodies of the L1 & L2 vertebrae (at transpyloric plane) on the posterior abdominal wall. It lies posterior to the stomach between the duodenum on the right side and spleen on the left side. It is a exocrine as well as a endocrine gland. There is no direct relation between the pancreatic pathophysiology in relation to ascites in liver cirrhosis. So *kloma* mentioned in ayurvedic texts in relation to *jalodara* is to be broadly studied with the relations and structures around pancreas.

Gayadasa in reference to pathogenesis of *jalodara* has mentioned *dusti* (defect) of *salilvaha (udak vaha) srotas* with *kloma moola.*[3] In anatomical relations of pancreas there is a important structure called portal vein which begins behind the neck of pancreas by joining of superior mesenteric vein with splenic vein. This portal vein[9] is a large vein which collects blood form alimentary tract and conveys it to the liver. It 8cm long and begins at the level of L2 vertebra behind the neck of the pancreas. It runs upwards and little to the right, first behind the neck of the pancreas, next behind the first part of the duodenum and lastly to the right free margin of the lesser omentum. It ends at the right end of the porta hepatis by dividing into right and left branches which enter the liver.



Now as we know that in development of ascites there is role of portal hypertension and portal hypertension will develop in the portal vein which begins behind to the neck of pancreas. Thus when the term kloma mentioned in the jalodara pathogenesis was assessed considering the factors of obstruction due to kapha and development of ascites as per modern view, the structure which should be considered here is the portal vein.

CONCLUSION

Jalodara (Ascites) is the type of *udar roga* which is difficult to treat and is also found in other types of *udar roga* as they progress. Its pathogenesis is related to the vitiation of *vata* and *kapha* with obstructive defect in *udak vaha srotas* and pathology related to *kloma*. In relation to *kloma* is the *kloma nadi* or the portal vein which begins behind neck of pancreas and ends up to

liver. Liver cirrhosis causes portal hypertension due to increased obstruction to blood flow at liver sinusoidal level. This causes leakage of hepatic lymph into peritoneal cavity. It increases and gets exerbated by other factors such as hypoalbuminemia and hyperaldosteronism. These all causes ascites.

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