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REVIEW ARTICLE

A Conceptual Study on *Kloma* w.s.r. to *Jalodar*

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ABSTRACT

Kloma is an abdominal organ described in various *Ayurvedic samhitas* under the heading of *koshthanga* which is responsible for thirst regulation (*pipasa).* References of *kloma* in various *Ayurvedic samhitas* are scattered and exact anatomical consideration of *kloma* is still controversial till date. On carefully reviewing the *Ayurvedic classics* as *Charak samhita* specially in refrence to *samprapti* (pathogenesis) of *jalodar* it is clear that *jalodara* occurs by vitiation of *kloma* or *klomavahi nadis* (structures or *srotas* related to *kloma*). Thus if we consider *klomavahi nadi* as portal vein which is responsible for ascites as in portal hypertension, then explanation of mechanism of ascites can be co-related in both *Ayurvedic* texts and contemporary science.

Key words: Ascites, *Klomavahi Nadi, Kloma, Jalodar*.

INTRODUCTION

Description of *kloma* in various *Ayurvedic samhitas* is different as it is said that *kloma* is the *moola* of *udakvaha srotasa*. [1-3] The term *kloma* apart from *srotas mool* has come in different places in classic texts (*samhitas*) as in *Charak samhita* it is also described under the heading of *koshthang,* [4] but exact anatomical correlation of *kloma* is still controversial. In *Charaka Samhita chikitsa sthana, kloma* has been mentioned in context with *jalodar*. [5] Here while describing the pathogenesis of *jalodar* (ascites), it is said that due to the etiological factors like consuming large amount of water after taking fatty substances or by persons with *mandagni* (metabolic factors) or by weak/lean persons, harm is caused to *jathragni* (impaired metabolism) which leads to vitiation in the *vata dosha* in *kloma* and this interferes/obstructs the *srotas* in/related to *kloma*, leading to disturbance in natural flow of *kapha* and *jala* (watery part) which increases the *jala* in *udar* (abdominal cavity) and ultimately formation of *jalodar*. Here *kloma* is a subject of discussion as pathology in this part or *srotas* related to this part (*klomavahi nadi*) is causing *jalodar.*

In modern science *jalodar* can be related to ascites. Ascites is caused in many diseases, the commonest cause being liver cirrhosis in which ascites develops due to portal hypertension.

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Background

Description of *kloma* in various *Ayurvedic samhitas* is different as it is said that *kloma* is the *moola* of *udakvaha srotasa*. [1-3] The term *kloma* apart from *srotas mool* has come in different places in classic texts (*samhitas*) as in *Charak samhita* it is also described under the heading of *koshthang,* [4] but exact anatomical correlation of *kloma* is still controversial. In *Charaka Samhita chikitsa sthana, kloma* has been mentioned in context with *jalodar*. [5] Here while describing the pathogenesis of *jalodar* (ascites), it is said that due to the etiological factors like consuming large amount of water after taking fatty substances or by persons with *mandagni* (metabolic factors) or by weak/lean persons, harm is caused to *jathragni* (impaired metabolism) which leads to vitiation in the *vata dosha* in *kloma* and this interferes/obstructs the *srotas* in/related to *kloma*, leading to disturbance in natural flow of *kapha* and *jala* (watery part) which increases the *jala* in *udar* (abdominal cavity) and ultimately formation of *jalodar*. Here *kloma* is a subject of discussion as pathology in this part or *srotas* related to this part (*klomavahi nadi*) is causing *jalodar.*

In modern science *jalodar* is related to ascites and ascites is caused in many diseases, the commonest cause being liver cirrhosis in which ascites develops due to portal hypertension.

Anatomical considerations

Anatomical consideration of *kloma* has been taken differently by different *acharayas,* on different parameters like position *koshthang, aakriti, vidhhalakshan* and *pipasa*. Of all these, pancreas fulfills the maximum parameters.

Acharya Ghanekar claims *kloma* as gall bladder, *Gananathsen* described it as pancreas and some scholars tried to correlate the *kloma* with various body parts like pancreas, right lung and gall bladder. [6-8]

In reference to pathogenesis of *jalodar* described by *Acharya Charaka*, direct involvement of pancreas is not reported, however structure nearest to pancreas which is directly involved in formation of *jalodar* (ascites) is portal vein (portal hypertension).

According to Gray's anatomy the portal system includes all the vein draining the abdominal part of digestive tube (excluding the lower anal canal but including the pre-terminal esophagus), the spleen, pancreas, and gall bladder. [9] Blood is conveyed from the viscera by the portal vein to liver, where it ramifies like an artery and ends in the sinusoids from which the blood again converges to reach inferior vena cava via the hepatic veins. The blood, therefore, passes through two sets of "exchange vessels"-the capillaries of the digestive tube, spleen, pancreas, and gall bladder and hepatic sinusoids

In adults, the portal vein and its tributaries have no valves. But in the fetus and even for a short period postnatally, valves are demonstrable in the tributaries, usually atrophying but occasionally persisting in degenerated form.

The portal vein is the principal afferent blood vessel of liver. In adults, it measures about eight centimeter long, beginning at the second lumbar vertebra level at the junction of the superior mesenteric and splenic veins, anterior to the inferior vena cava and posterior to the neck of the pancreas. It inclines slightly to the right as it ascends behind the superior part of duodenum, CBD and gastro-duodenal artery where it is directly anterior to the inferior vena cava. It then enters the right border of lesser omentum, ascending anteriorly to the epiploic foramen to the right end of the porta hepatis. The main portal branches accompany the corresponding branches of the hepatic artery into the liver. In lesser omentum, it is posterior to both the common bile duct and hepatic artery- the former being to the right and surrounded by the hepatic nerve and plexus, and accompanied by many lymph vessels and some lymph nodes.

Ascites and portal hypertension

Ascites is accumulation of serous fluid within the peritoneal cavity. [10] It forms because of conditions directly involving the peritoneum (infection, malignancy), or diseases remote from the peritoneum (liver disease, heart failure, hypoproteinemia). Cirrhosis is the commonest cause of ascites.

The mechanism of ascites formation in cirrhosis is complex but portal (sinusoidal) hypertension, [11] and renal retention of sodium are universal. Portal hypertension is the initial mechanism that determines leakage of ascites into the peritoneal space. Sinusoidal (portal) hypertension results from hepatic venous outflow block secondary to regenerative nodules and fibrosis.

DISCUSSION

In *jalodar*, *Acharya Charaka* has said that the dysfunction in *kloma* causes the disease, whereas in contemporary science portal hypertension is said to be the direct cause of ascites. Ascites is the commonest complication of cirrhosis. Increased hepatic sinusoidal pressure is an essential prerequisite for the development of ascites. Three interrelated pathophysiological processes contribute to the development of ascites. These include systemic arteriolar vasodilatation; activation of Na and H_2O retention; and sinusoidal portal hypertension. Due to this, the increased sinusoidal pressures leads to increased fluid movement from sinusoids to the space, thereby increasing hepatic and thoracic duct lymph flow which can be as much as 24 times the normal. Both increased outflow resistance and portal venous inflow contribute to sinusoidal hypertension and formation of splanchnic lymph. When lymph production exceeds the capacity of lymphatics to return it to circulation, the excess lymph spills out into the peritoneal cavity. This is initially reabsorbed via microscopic pores on the peritoneal space of the diaphragm that communicates with supra-diaphragamatic lymphatics. When lymph formation exceeds its reabsorption, clinically evident ascites occurs.

Kloma is anatomically correlated with pancreas by some school of thoughts, but dysfunction of pancreas causes ascites only in acute conditions like pancreatitis and that too not to a bigger extent. Thus in this context, term *kloma* can be elaborated to vessels (*srotas* around *kloma*) in which portal system is the one which causes ascites in course of development of portal hypertension.

CONCLUSION

Kloma as described by *Acharya Charaka* in reference to *jalodar* that after intake of *sneha padartha* (fatty diet) or *durbal and krisha purusha* (weak persons) who is already suffering from *mandagni* (reduced metabolism), if he indulges with excessive intake of water leads to further vitiation of *jatharagni* and because of this, the already vitiated *vayu* along with vitiated *kapha* in *kloma nalika* mixes with the *udak mishrit kapha* leads to excessive accumulation of water in *udar* resulting in *jalodar*.[12] According to this, if we consider *kloma* as pancreas which has been already told by various scholars, [7, 13] then structure nearby it which is responsible for the formation of ascites is the portal vein. Thus, vitiation of structures or *srotas* nearby *kloma* causing *jalodar* can be compared to the portal vein causing ascites.

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