IAMJ

INTERNATIONAL
AYURVEDIC
MEDICAL JOURNAL

Review Article | ISSN: 2320-5091 | Impact Factor: 6.719

REVIEW ON MEDOVAHASROTAS AND ITS MOOLSTHANA WITH REFERENCE TO OBESITY INDUCED HYPERTENSION

Jinu K Mathew[1], **Jaishree H Mhaisekar**[2]

[1]PG Scholar, [2]Associate Professor
Dept. of Rachana Sharir, Government Ayurveda College, Nagpur, Maharashtra, India

Corresponding Author: jinukidangayil@gmail.com

https://doi.org/10.46607/iamj2409042021

(Published Online: April 2021)

Open Access

Article Received: 26/03/2021 - **Peer Reviewed:** 30/03/2021 - **Accepted for Publication:** 31/03/2021

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ABSTRACT

Excess weight gain, especially when associated with increased visceral adiposity, is a major cause of hypertension accounting 65-75% of total cases of hypertension. Increased renal tubular sodium reabsorption by the impairment in renal pressure natriuresis plays an important role in initiating obesity induced hypertension. The present study conceptually tries to substantiate the relation between *moolasthana* described for *medovahasrotas* with reference to the pathology of obesity induced hypertension. The *moola* explained for *medovahasrotas* are *Kati, Vrikka, Vapavahana* and *Mamsa* (Kidney, Suprarenal gland, Lumbosacral region, Superficial fat) which are the general regions of deposition of visceral fat or *meda*. The increased *meda* will hamper the renal pressure natriuresis in mainly three ways:

1)Physical compression of the kidneys by fat in and around kidney.
2)Increased activation of RAAS.
3)Increased SNS activity.

Thus, *medovaha srotodushti* further enhances *medodhatudushti* (Obesity or *Sthoulyata*) itself, which in turn destroys the *srotomoola*. Hence, evidence of inherent relation among *medovaha srotas* and its respective *srotomoola* is established.

Keywords: *Medovahasrotas, Srotomoola*, obesity induced hypertension

INTRODUCTION

Srotas are defined as the channels or pores which continuously carry out the function of secretion, transportation, and excretion in the body[1].They help in the transportation of *dosha* and *dhatu* in the body during equilibrium state. The concept of *srotas* has been described by Charak, Susruta, Vagbhat etc even though there is slight difference in their numbers. *Dhatu* in the body are present in two forms.1) *Poshya dhatu* (That getting nourishment) 2) *Poshak dhatu* (Nourishing). Among these *poshak dhatu* are present in *Srotas* and *poshya dhatu* being present in *kala* ,receives nourishment from *poshak dhatu*. Each *Srotas* carry only their respective *poshak dhatu* and thereby called upon by the name of *dhatu* itself. Hence, the *srotas* that transports the *medodhatu* throughout the body are called as *Medovaha Srotas*. Each *srotas* consist of two parts or ends, one from which the *srotas* are getting originated (*Moola*), another which the nutritive material is being transported to respective sites. It has been clearly described in *samhitas* about *srotomoola, srotodushti hetu, srotodushti lakshana* of each *srotas*. This study tries to elicit the relation between *medovaha srotas* and its *moolasthana* with due consideration with structural involvement and pathophysiology of Obesity Induced Hypertension which is a major problem faced among the developed and developing countries.

Aim and Objectives

- To study the concept of *medovaha srotas*.
- To evaluate the relation between *medovaha srotas* and its *srotomoola* by correlating the *medovaha srotodushti lakshana* with pathophysiology of obesity induced hypertension.

Materials and Method

This is a conceptual study. Detailed literature of *medovaha srotas* were collected from classical *ayurvedic* texts and modern pathophysiology of obesity induced hypertension from journals and modern texts.

Literature Review

The term *srotas* is derived from the root word '*susravana*' means to exude, to ooze, to filter. *Medas* is derived from '*Jhimida snehana*', means the substance which has *snigdatva* property. The word *moola* means root, it may be root of origin, root of manifestation, root of nutrition etc.

Medovaha Srotomoola

Vrikka, Vapavahana - Charaka
Vrikka, Kati - Susruta
Vrikka, Mamsa - Vagbhata[2]

Vrikka - Koshtanga formed by the *sara* of *rakta* and *medodhatu*. There are two *vrikka*. The term *vrikka* may include the kidney along with the suprarenal glands.

Vapavahana –It is also a *koshtanga*. Chakrapani interpreted as *Tailavartika*. Dr. Ghanekar commented it as omentum, where maximum *meda* is stored.

Kati –It denotes to the flanks or lumbosacral region where fat accumulates.

Mamsa –It is not yet interpreted satisfactorily, yet superficial fat over muscle can be considered.

Medovaha Srotodushti Hetu

Avyayama, divaswapna, atisnigda-ahaar sevana and *atisevana of varuni madhya* are described as *hetu* for *medovaha sroto dushti* by Charaka in *srotovimana adhyaya*[3] which means lack of exercise, daytime sleep, eating too much oily food, and excess intake of alcohol especially wines vitiate the *medovaha srotas*.

Medovaha Srotodushti Lakshana

Srotodushti lakshana is described by Charak where as Susruta being *salyapradhana* described *srotoviddha lakshana*. Anyhow Dalhana in the commentary clarifies this difference. According to Charaka *dushtilakshana* includes premonitory symptoms explained for *prameha*, the eight *doshas* explained for *atisthoola* i.e. decrease of life span, enthusiasm, infertility, bad odour of body, excessive sweating, excessive appetite and thirst etc[4]. Susruta describes symptoms of injury to *medovaha strotomoola* as excessive sweating, oily appearance of body, dryness of palate, obesity, oedema, and thirst.[5]

The term *medoroga* was first used by *Acharya* Madhava. It is used as a broad term which include obesity and related metabolic syndrome. Adhamalla further tried to distinguish between two types of *medoroga* 1) Obesity with its clinical features (*sthoulyata*) 2) Lipid disorders where *meda* acts as an

etiological factor in the genesis of other secondary diseases.

Obesity Induced Hypertension

Excess weight gain, especially when associated with increased visceral adiposity is a major cause of hypertension, accounting for 65-75% of the risk of human primary hypertension. Studies in diverse population of the world have shown that there exist a linear relationship between BMI and systolic and diastolic blood pressure[6,7]. Clinical studies indicate that maintenance of a BMI<25 kg/m^2 is effective in primary prevention of hypertension[8,9].

The impact of obesity on BP mainly depends on a) How long the person has been overweight b) The distribution of fat on the body. It worsens the condition if excess adiposity is maintained over several years. Also, apart from mere raised BMI of subcutaneous fat, it is the visceral or retroperitoneal fat which makes vulnerable to hypertension[10].

Impaired Renal Pressure Natriuresis

Impaired renal pressure natriuresis is the basic cause of hypertension in obese subjects. Mainly, three major factors are responsible for the pathology.

1) Physical compression over the kidneys due to increased visceral, retroperitoneal and renal sinus fat.

2) RAAS (Renin-Angiotensin-Aldosterone System) activation

3) SNS (Sympathetic Nervous System) activation, especially renal sympathetic nerve activity.

In addition to this, CKD (Chronic Kidney Disease) over a much longer time amplify the effects in obese persons.

Increasing Blood Pressure by Physical Compression

Increased visceral and retroperitoneal fat may increase blood pressure by physically compressing the kidneys. In patients with visceral obesity, intraabdominal pressure rises in proportion to sagittal abdominal diameter reaching high levels as high as 35-40 mmHg[11].These high pressures compress the renal veins, lymph vessels, ureters and renal parenchyma. Also, retroperitoneal fat often encapsulates the kidney, attach tightly to the renal capsule and invades the renal sinuses, causing additional effects on intrarenal pressure[12].In addition to compressing the kidneys, retroperitoneal and renal sinus fat may cause inflammation and expansion of renal medullary extracellular matrix that could further impair renal function. The compression on kidney by visceral fat increases the interstitial fluid pressure which will compress the thin loops of Henle and vasa recta, reducing renal tubular flow and medullary blood flow, thereby increasing sodium reabsorption in the loop of Henle. Increased sodium reabsorption will contribute to renal vasodilation, glomerular hyperfiltration and renin secretion eventually resulting in hypertension[13].

RAAS (Renin Angiotensin Aldosterone System)

Obese subjects, especially those with visceral obesity, often have mild to moderate increase in plasma renin activity, angiotensinogen, ACE activity, Angiotensin 2, and aldosterone[14]. RAAS activation occurs despite NaCl retention, Volume expansion and Hypertension which typically suppress renin secretion and Angiotensin 2 formation. Abnormal activation of RAAS system will result in NaCl reabsorption and there is increase of ECF and eventually elevation of blood pressure. Abnormal activation of RAAS may contribute to glomerular injury and loss associated with obesity not only by increasing blood pressure but also through intrarenal effects. Thus, constriction of efferent arterioles by Angiotensin 2 modulates the rise in glomerular hydrostatic pressure caused by arterial hypertension.

SNS activation

Obesity generally decreases parasympathetic tone and increases sympathetic activity. These changes in autonomic activity are associated with increased heart rate, reduced baroreflex sensitivity as well as hypertension[15,16]. Although cardiac SNS activity may not be elevated, RSNA (Renal Sympathetic Nerve Activity) and MSNA (Muscle Sympathetic Nerve Activity) are generally increased in obese compared to lean subjects[17,18]. Increased RSNA stimulates renin secretion and renal sodium reabsorption which in turn contribute in developing obesity hypertension. Visceral obesity elicits greater SNS activation than does subcutaneous obesity. Several mediators of SNS activation in obesity have been suggested including:

1) Impaired baroreceptor reflexes

2) Hyperinsulinemia 3) Angiotensin II
4) Cytokines released from adipocytes such as leptin, tumor necrosis factor and interleukin-6
5) CNS propriomelanocortin pathway.

DISCUSSIONS

It is well clear that direct correlation of *medovahasrotomoola* with probable modern anatomical structures like kidney, lumbosacral region, superficial fascia is possible since because of the variable amount of deposition of fat (meda) in these areas. But the greatness of the work of ancient Acharyas are so fascinating that, even the pathophysiology also can be well explained by relating these structures. The increase in blood pressure in the body are normally achieved by combined effects of RAAS mechanism and SNS activity. The activation of RAAS and SNS will take place when there occurs a fall in blood pressure. This normal physiology will get hampered in obese people ,especially in those having visceral adiposity. In impaired renal pressure natriuresis, the above normal physiological process will deviate from normalcy. The principal cause of this impairment is the deposition of visceral fat i.e. *meda* over the body. Excess deposition of *meda* is nothing but the *medodhatu dushti* which eventually hamper the respective *srotomoola* itself. Hence, *samprapti* can be summarised as

Medovahasrotodushtihetu ⇨ *Medovahasrotodushti* ⇨ *Medodhatudushti* ⇨ *Medovahasrotomoola dushti*

When impairment in renal pressure natriuresis persists over a long period of time, will leads to Chronic Kidney Disease in which pedal oedema is a classic presentation. Susruta includes *Sthulasophata* as a *lakshana* of *medovaha srotomoola vidha/dushti.* Thus, it can be concluded that there is inherent relation of *medovaha srotas* with its *moolasthana*, and any dearrangement of its normal function eventually lead to the destruction of *srotomoola.* So, the line of treatment of obesity induced hypertension should be made by considering the *srotomoola dushti* and by avoiding *medovaha srotodushti hetu* apart from antihypertensive drugs.

CONCLUSION

There exists an inherent relation between *medovahasrotas* and its *srotomoola*. Any dearrangements in any of the two, will hamper the other and resulting in malfunctioning of the system.

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Source of Support: Nil
Conflict of Interest: None Declared

How to cite this URL: Jinu K Mathew & Jaishree H Mhaisekar:Review on Medovahasrotas and it's Moolsthana with Reference to Obesity Induced Hypertension. International Ayurvedic Medical Journal {online} 2021 {cited April, 2021} Available from: http://www.iamj.in/posts/images/upload/833_837.pdf