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A Review of Pathogenesis of Hypertension: Ayurvedic approach

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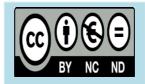
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ABSTRACT: -

Hypertension has become a global burden, except for genetic multifactorial influences, it is due to faulty & stressful life style. Till now the actual etiopathology of hypertension is unknown, due to lack of knowledge of actual etiopathogenesis, we are unable to do complete cure of hypertension and that's why the mortality and morbidity shows a considerable %. It is the most important and single factor of cardiovascular disorder, coronary disorder, CHF and CRF. In Ayurveda there is no straight literature present about HTN or *Uchha raktachapa* but on the basis of symptoms it can be correlate with the *Vata vyadhi* named *raktgata vata* (hypertension) which is explained in Ayurveda. In this article we try to explain its etiopathogenesis (*Samprapti*) in ayurvedic terms which is made on the basis of involved *dosha*, *dhatu* (body tissues), *Srotasa* (channels) and other factors. It can be considered as *Vata* dominant *Tridoshaja vyadhi* (Disease caused by *Tridosha*) & *Ama* (collection of toxins) is involved in its pathogenesis. So, it can be effectively treated by *Tridosha* pacifying, *Agnivardhaka chikitsa* (enhancing digestive strength).

Key words- Ayurveda, Hypertension, Raktagatavata, pathogenesis of raktagatavata



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Review Article.

INTRODUCTION-

Ayurveda is ancient science of medicine in the world which originate in Vedic era. In ancient time majority of disease were treated with traditional drugs described in Ayurveda but with time entry of modern medicine has overshadow the traditional medicine. The incidence of life style disorders are still alarmingly increasing; there is dire need to search for actual pathogenesis for an effective and safe magical remedy because of lack of current therapies which can provide complete cure and treat patient without adverse effect.¹

Symptoms of *Raktagatvata* can be correlate with *Hypertension*.¹The disease *Raktagata vata* is one of the *Nanatmaja vata vyadhi*² (specific diseases caused by individual *Doshas*) i.e., *Raktagata vata* (HTN) disease occurs due to vitiation of *Vata dosha* specially.

Previous scholars of Ayurveda coined different terms for HTN such as *Dhamani pratichaya*, Siragatavata, Rudhirmada, Raktagatavata, Raktapradoshaja vikara, Raktachap adhikya, Bhrama and Avratavata.³The Nidans (Causative factor) described by Charak in the context of Vata vyadhi vitiates only the Vata dosha and not, the factors involved in other disease like *Dushya*, *Ssrotas*(channels) etc.⁴ This vitiated vata dosha when flows with Rakta dhatu (Blood tissue), which is in normal state and other factors like *Raktavaha srotas* (channels which circulates blood) etc., leads to disease Raktagata vata (HTN). All factors except vata, develop Vishamaavastha (Abnormal state) due to vitiated Vata dosha but not due to *Hetu* (causative factor).⁵

Following symptoms are produced due to vitiated *Vata dosha* in *Rakta dhatu* (Blood tissue)-

- Acc. to Acharya Charak and Acharya Vaghbhat
 Throbbing pain, Burning sensation, Discoloration, emaciation, Anorexia.^{7,8}
- Acc. to Yogratnakar- Burning sensation in feet, Blister formation, Increased Pulsation, Blood discharge.⁹

As per Ayurvedic concepts, there are many other factors which are involve to develop the disease Raktagata vata like Udana, Vyaan and Praana vata, Saadhak and Paachaka pitta, Avalambak kapha, Rasa (plasma), Rakta (blood), Meda dhatu (fat), Pranavaha (channels carrying air), Mutravaha (channels carrying urine), Swedavaha (channels carrying sweat), *Medavaha* (channels carrying fat tissue).^{2,3}In modern medicine HTN is a Cardiovascular disorder which is defined as the high blood pressure. When the systolic blood pressure remains elevated above 150mmHg and diastolic blood pressure remains elevated above 90mmHg. It is considered as HTN. About in 90% patients there is no known cause for hypertension and this is very important to be alert.

Most cases of HTN does not exhibit any symptoms i.e. why it is k/a silent killer, but in some cases, people feels-¹⁰

- Giddiness
- Palpitation
- Excessive sweating
- Fatigue
- Exertion
- Dyspnoea
- Headache
- Ins<mark>omnia</mark>
- Irritation
- Tinnitus
- Numbness
- Pain in calf region
- Loss of memory

HTN is clinically classified into Stage-1 and Stage-2 HTN by the National Institute of Health, US-¹¹

| | Systolic BP(mmHg) | Diastolic BP(mmHg) |
|-------------|-------------------|--------------------|
| Normal | <120 | <80 |
| Pre HTN | 120-139 | 80-89 |
| Stage-1 HTN | 140-159 | 90-99 |
| Stage-2 HTN | >160 | >100 |

HTN is also classified into-¹¹

1.PRIMARY HTN- when the underlying cause of HTN is absent.

2.SECONDRY HTN- when BP raised due to some underlying disorders such as-

- Cardiovascular disorder
- Endocrine disorder
- Renal disorder
- Neurogenic disorder
- During Pregnancy

Both are further classified into *Benign and Malignant*.

HTN causes major effects on three main organs-Heart and its vessels, Nervous system and kidneys. It manifests as-

- Renal failure
- Lt. ventricular failure
- MI
- Cerebral hemorrhage
- Retinal hemorrhage.

AIMS AND OBJECTIVE-

- To understand the concept of blood pressure regulation & HTN according to ayurvedic and modern view
- To understand the role to *tridosha* in the development of HTN.
- To generate a hypothesis for a etiopathogenesis of HTN.

MATERIAL AND METHODS-

In this part, literary review of *raktagata vata* (HTN) is collected from the Ayurvedic samhitas,

journals & articles on the google & from State Ayurvedic college Varanasi library. Similarly, modern review of HTN is collected from modern pathology, medicine books.

OBSERVATION AND DISCUSSION-

In general, normal blood pressure is regulated by 2 hemodynamic forces- cardiac output and total peripheral vascular resistance. Cardiac output is determined by stroke volume and heart rate; stroke volume is related to myocardial contractility and to the size of the vascular compartment. Peripheral resistance is determined by functional and autonomic changes in small arteries and arterioles. Factors which alter these two factors result in HTN.¹²

With this background knowledge, we next turn to the mechanism involved in the development of HTN-¹⁰⁻¹²

- 1- *Intravascular volume* the initial elevation of blood pressure in response to vascular volume expansion. Blood volume and blood pressure is increase in response to a high NaCl intake, decreased capacity of kidney to excrete sodium, increased activity of the Renin-angiotensin system.
- 2- Autonomic nervous system- in the regulation of BP fallowing factors-Adrenergic reflexes, Adrenergic function, Norepinephrine, Epinephrine, Dopamine, a no. of various receptors like- alpha, beta, baroreceptors are involved. Circulating catecholamine concentrations, increased sympathetic outflow,

behavioral or physiological stress and change in blood volume, autonomic neuropathy, impaired baroreflexes are affect BP regulation mechanism and results in HTN.

- 3- *Renin-Angiotensin-Aldosterone* this system regulates the BP via vasoconstriction and sodium retention. Angiotensin II is the primary factor for secretion of aldosterone, stimulates vascular smooth muscle cell & myocytic growth, play a role in the pathogenesis of atherosclerosis. Mineralocorticoid receptors activation induces structural and functional alterations in heart, kidney, and blood vessels. Increased activity of the renin-angiotensinaldosterone axis is associated with HTN.
- 4- Vascular mechanismvascular radius &compliance of resistance arteries are important determinants of arterial pressure. In hypertensive patients structural, functional, mechanical changes may reduce the lumen diameter of small arteries and arterioles. Factors like remodeling (alteration in the vessel wall), stiffness of vessel, altered ion transport by vascular smooth muscle, impaired vascular endothelial function are altered vessel volume and resistance and results in HTN.

On the basis of the cause of above mechanism, HTN is classified into 11 -

- Primary- the cause of HTN is unknown but a number of factors are related to its development. These are under;
- Genetic factors
- Racial and environmental factors
- Risk factors- age, sex, atherosclerosis, smoking, alcohol intake etc.
- Salt sensitivity/ Ca ion intake
- Cell membrane defect

- 2. *Secondary* mechanism underlying secondary HTN with identifiable cause. Based on the etiology, these are described under-
- Renal hypertension
- Endocrine hypertension
- Coarctation of aorta
- Neurogenic

According to Ayurveda the *Nidana* (causative factor) *of raktagatavata* (HTN) as described in the context of *vatavyadhi* (disease caused by vata) by Acharya Charak, are^{2,3}-

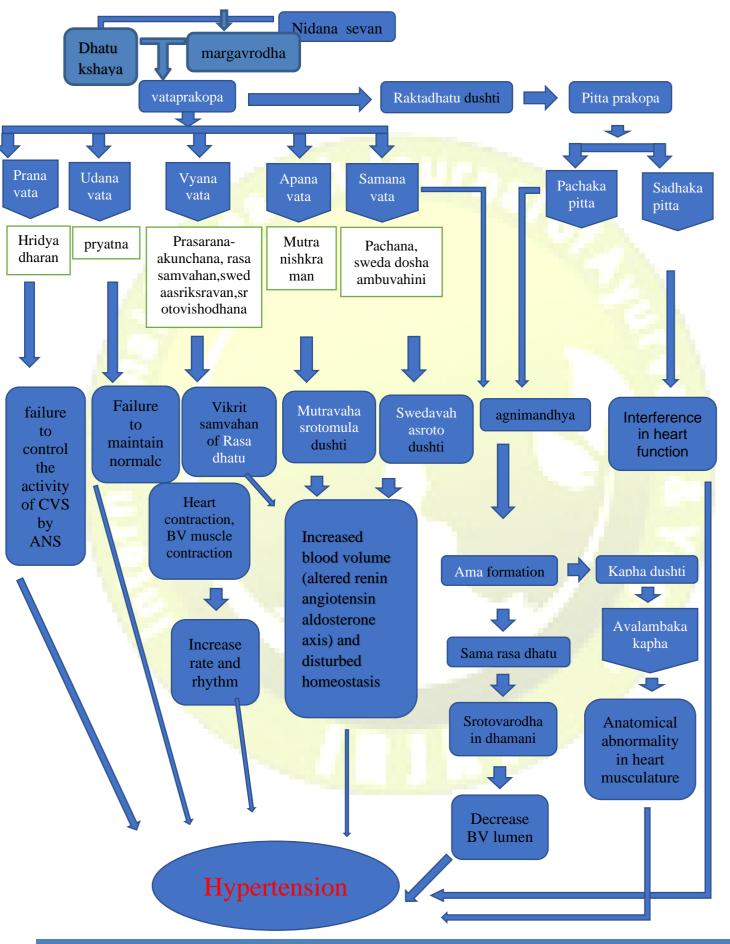
Aharaj Nidana (Food related factors)- *Ruksha* (dry), *sheeta* (cold), *alpa* (less quantity), *laghu anna* (light food), *ama bhojan* (undigested food).

Viharaja Nidana (Life style related factors)-Vyavaya (sexual indulgence), Atiprajagaran (remaining awake at night), Visham upachar (inappropriate therapeutic measures), dosha & asrik srava (discharge of dosha and blood), langhan (fasting), *plavan* (swimming), excessive walking & exercise, vichestta (inappropriate physical activities), divaswapna (sleep during day time), vegsandharana (suppression of natural urges), abhighata (injury), Gaj, Ushtra, Ashwa, shighra yana apatasanata (riding over elephant, camel, horse or fast moving vehicle).

Mansika Nidana (Psychological factors)- achinta, shoka, krodha, bhaya (excessive worry, grief, anger, fear), Dhatu kshya (depletion of tissues).

Samprapti (Pathogenesis) -

In Ayurveda pathology of any disease is explained in terms of *dosha*, *dushya*, *srotas* (*channels*), *mala(waste)* in terms of their, *Kshaya* (decrease) and *Vriddhi* (increase) and *prakopa* (aggravation). *Avarana* (covering), *Ama* (undigested food) and other factors are also involved. A hypothetical pathogenesis can be drawn as fallows-



Roopa^{2,3,4} –

The symptoms produced due to vitiation of *dosha*, *dushya* and other factors, are given below-

Prana vayu - Ruja (pain), headache, Bhrama(Giddiness)

Vyana vayu - Spandana(Palpitation)

Udana vayu - Weak memory

Vata dosha - Santapa (irritation), krashta (Weakness), stambha (Stiffness), Sparshanash

(Numbness) Insomnia, Tinnitus, Pain in calf region *Pitta dosha – paad daha* (Burning in feet), *raaga/vivarnata* (Discoloration), Haemorrhage, Excessive sweating, Insomnia, *santapa* (Burning sensation), Giddiness

Ama - shotha (Oedema), klama (Tiredness without exertion)

Sama rasa dhatu - aruchi (Anorexia)

Rakta pradoshaja vikara - arunshi (Boils over body)

Swedvaha srotodushti - excessive sweating

The other factors do not produce symptoms directly but they involved in pathogenesis like- Samana Vata and Pachaka Pitta prakopa are responsible for agnimandhya (dyspepsia) and producing Ama. Due to agnimandhya (dyspepsia) transformation of food to dhatu is hampered so that excessive Meda Dhatu (bad cholesterol) is form and deposited in blood vessel.⁴ The seat of *Sadhaka Pitta* is *Hridya* (heart) but its functions Budhhi (intellect), Medha (power of retention of knowledge) etc. are related to *Mana*(mind). So, it shows the control of mind over heart along with *Prana* and *Vyana Vata.⁵Kapha* is responsible for smooth circulation and musculature of heart and BV. By study of a etiology, pathophysiology, by observing the sign & symptoms of HTN & by correlating these with the features of vata, pitta & kapha in their natural as well as in vitiated states, the possible doshaja vitiation seems to be vata predominant tridoshaja vikara.⁶

CONCLUSION-

Establishment of definitive pathogenesis is the aim of this study. The disease raktagatavata (HTN) is manifest by the aggravation of vata, pitta & kapha *dosha*, the possible *doshaja* vitiation seems to be *vata* predominant *tridoshaja vikara. Rasa* (Plasma) *Rakta* dhatu(blood tissue) are two *dushya* and which are directly involved and *mamsa* (muscles) and *meda dhatu* (Fat) involved indirectly⁷. Rasavaha (channels carrying plasma), raktavaha (channels carrying blood), and mutravaha srotas (channels carrying urine), are mainly involved in its pathogenesis, mamsavaha(channels carrying muscle tissue), medvaha, (channels carrying fat), swedvaha(channels carrying sweat), and manovaha (channels *carrying* srotas *thoughts/emotions*), play a complimentary role in its pathogenesis. At the time of treatment design for HTN, the above factors should be addressed properly with special consideration of strength of disease.

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