WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
SJIF Impact Factor 7.523
Volume 6, Issue 2, 337-356. Review Article ISSN 2277– 7105

# REVIEW OF KAPHAJ NANATMAJ VYADHI WITH SPECIAL REFERENCE TO INSULIN RESISTANCE SYNDROME

***Vaidya Meenakshi Rewdakar Kole**

Associate Professor, Kayachikitsa Department, R.A. Podar Medical (AYU.) College, Worli, Mumbai-18.

Article Received on
01 Dec. 2016,

Revised on 21 Dec. 2016,
Accepted on 11 Jan. 2017

DOI: 10.20959/wjpr20172-7742

***Corresponding Author**
**Vaidya Meenakshi Rewdakar Kole**
Associate Professor,
Kayachikitsa Department,
R.A. Podar Medical (AYU.)
College, Worli, Mumbai-18.

## ABSTRACT

The industrialisation, stress during the work, dietary habits, lack of exercise and various varieties included in the regular diet like fast food, freeze fruits, increased amount of aerated drinks, etc. result into the entity which we call as INSULIN RESISTANCE SYNDROME. Diabetes mellitus, Hypothyroidism, Poly cystic ovarian syndrome, Atherosclerosis, Hypertension, Central obesity and Dyslipidaemia together are included in Insulin resistance syndrome. Insulin is a digestive tatva which is very essential for metabolism at cellular level. The cells get resisted to insulin and insulin resistance occurs. The process of dhaatuparinamana process is described in Charaka Sutrasthana 28, the food which we ingest is separated into prasada and kitta parts, after the action of jatharagni and bhutagni, nourishes the functional factors of body i.e. tridoshas, saptadhaatus and their malas. In different strotasas, the agnis of all dhaatus i.e. dhaatvagni, acts on this processed food particles and nourishes themselves and latter dhaatus. Any disturbances in the above chayapchaya process results into diseases like galganda, prameha, dhamanipratichaya, sthoulya. Rasa dhatvagni mandya leads to formation of apachita rasa dhaatu nirmiti (galganda, sthaulya, rasa raktagat snehavruddhi), mala dushti-kapha (prameha, dhamanipratichaya) and updhaatu dushti- raja (raja vaishamya). These diseases are included in Kaphaj Nanatmaj Vyadhi. Various diseases are described by Acharya Charaka, Sushruta, Madhavacharya and Sharangdhar under the entity of Kaphaj Nanatmaj Vyadhi. They have enumerated them as 20, but they mostly differ in names. Lastly Charaka has said that Kaphaj Nanatmaj Vyadhi can be numerous.

**KEYWORDS:** Insulin Resistance syndrome, chayapchaya, dhatu parinamana process,

kaphaj nanatmaj vyadhi, prasada, kitta. rasa dhatvagni mandya.

**KAPHAJ NANATMAJ VYADHI AND INSULIN RESISTANCE SYNDROME**

The classical Ayurvedic texts have vividly described Kaphaj Nanatmaj Vyadhi which basically comprise the diseases due to over nutrition and defective tissue metabolism. Ayurvedic Aspect of Obesity and Lipid disorders have been vividly conceived in Ayurveda with context of Medoroga (Dyslipidaemia) and Prameha (Diabetes).

Ayurveda is primarily concerned about conservation of health rather than eradication of disease. It presumes that improper dietary habits and deranged functions of different sets of Agni at different levels of metabolism give rise to formation of Ama (reactive antigenic factor). This Ama further disturbs the metabolism i.e. dhatvagni parinamana process and may lead to variety of diseases. Since last few decades, the conventional system of medicine is focusing on the concept of metabolic syndrome, which seems very similar to the concept of Kaphaj Nanatmaj Vyadhi of Ayurveda.

In the pathogenesis of Insulin Resistance Syndrome, key factors are Central adiposity and Insulin resistance. There is a tendency of central adipose tissue to direct the FFAs directly to the liver. These FFAs causes release of several pro-inflammatory mediators (i.e. cytokines, adiponectin, leptin, CRP, etc.), from the liver which are then directed to the systemic circulation and gives rise to other consequences of Insulin Resistance Syndrome. Ayurveda believes that obesity depends chiefly upon the quality of Rasa dhatu (the first post digestive essence from food) which is source of nourishment for the entire body elements. It is also the source of deranged Medodhatu in terms of Medodhatu vridhi (adiposity) during the parinamana of former dhatus.

According to Ayurveda classics there are three sets of Agnivyapara (Metabolic process) viz.

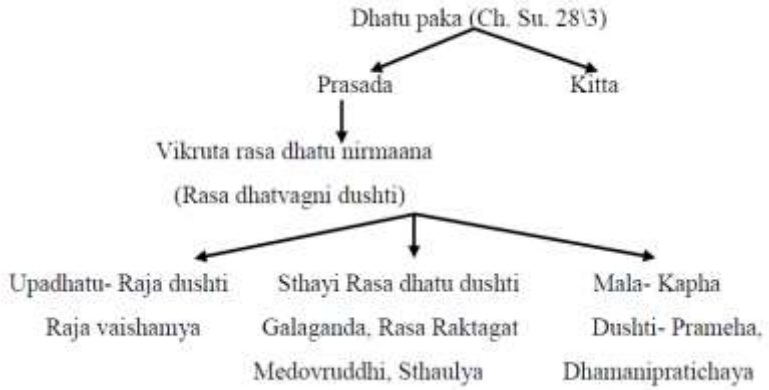
- Jatharagni level (Main Digestive factor) ⟶ Sthoola pachana
- Bhutagni level (Metabolism at gross level) ⟶ Sthoola and Sukshma pachana
- Dhatvagni level (Metabolism at tissue/cellular level) ⟶ Sukshma Pachana

All type of ingested foodundergoes Jatharagnipaka (Metabolic process) at the level of Amashaya (stomach) and Pachyamanashaya (small intestine). The nature of Jatharagnipaka (Metabolic process) is Sanghatbheda (disintegration of macromolecule of food into smaller

one) to make it fit for assimilation, digestion and absorption. During the action of Jatharagni on ingested food, the Ahara rasa is formed. This Ahara rasa is absorbed via Rasa and Raktavaha srotasa (Circulatory channels) and it reaches towards the liver. The same idea is also conceived in modern physiology and opines that both diet and drug after absorption through G.I tract primarily metabolize at the level of liver through its subsequent metabolic enzymes. The metabolic enzymes and associates of liver are comparable to Rasa dhatvagni of Ayurveda. This Rasa dhatvagni metabolizes the Ahara rasa in its respective strotasa and leads to formation of poshakansha of former Rakta dhatu, Updhatu Raja and Stanya, Sthoola ansha for Rasa dhatu itself and mala Kapha; further the parinamana of latter dhatu take place.

The formed nutrient pool at the site of liver, reaches towards the heart through Rasa and Raktavaha srotasa. Now these forms of metabolic components of diet are transported all over the body. After seeing the above concept, it is clear that in Adiposity, due to hypo-functioning of Dhatvagni, there is excessive formation of Apachita Rasa with excess guru, manda and snigdha guna (adipose tissue) which ultimately leads to Snehavruddhi (adiposity). Later in dhatu parinamana by this Apachita Rasa of latter dhatu, Meda dhatu which is of similar properties to that of Kapha dosha, is produced leading to more complications like Sthaulya.

**Process of Pathogenesis of Insulin Resistance Syndrome through Ayurveda**




Due to excessive indulgence in Adhyashana, Madhura, Shita, Snigdha ahara, vishamashana and lack of physical exercise, Avyavaya, Divasvapna, etc. (sedentary life styles) may be accompanied with or without Beejadosha, there is aggravation of all the three doshas but especially Kapha dosha. This leads to Agnimandya at Jatharagni and Dhatu levels. Thus the rasa dhatvagnimandya leads to formation of vitiated Kapha dosha, Vitiated Updhatu, Vitiated Rasa dhatu itself. This form of Kapha have physical similarity with Sama Rasa and

Medadhatu, which gets accumulated over immovable parts of the body in undifferentiated manner.

- **Prakopa**

The increase of vitiated Kapha dosha in quantity and quality is responsible for the disturbance in the functions of Agni at different level in the body especially at the level of Bhutagni, Rasa dhatvagni and Medodhatvagni. The deranged functions of Agni may lead to formation of Ama. Because Kapha, Rasa and Meda are of same nature i.e. they are ashrayi of Kapha dosha. This type of Kapha dosha along with Apachita Rasa (Guru, snigdha, Manda guna) causes strotorodha i.e. blockage of micro channels. This blockage of micro channels can be compared with the downstream signalling of the Insulin receptors due to excess formation of Free Fatty Acids i.e. FFA.

- **Prasara**

If a person continues to consume above hetu, there is more rasa dhatvagnimandya and leads to formation of more vitiated updhatu, mala and rasa dhatu itself. The excess FFA preformed and newly formed (mainly from the visceral adipose tissue) circulates all over the body.

- **Sthana Samshraya**

The above formed vitiated elements in dhatu parinamana process gets localized at different places in the body. FFAs which are directed to the liver stimulate, release of different pro inflammatory mediators. In due course of time these inflammatory mediators play a key role in the pathogenesis of atherosclerosis. Most of the FFAs occupy the insulin receptors by molecular mimicry, leading to Insulin resistance. Beside this, Insulin resistance causes an imbalance between production of NO and secretion of endothelin-I, leading to decrease blood flow and activation of sympathetic system which may lead to develop Hypertension.

- **Vyakta**

The vitiated updhatu Raja leads to Raja vaishamya, vitiated kapha dosha leads to Prameha and Dhamanipratichaya and vitiated Rasa dhatu itself causes Galaganda, Rasaraktagat Snehavruddhi and Sthaulya. If the whole process still continues, it causes downstream signalling of the insulin receptors due to occupancy by the circulating FFAs, which causes Insulin resistance and the condition known as Hyperinsulinemia. Initially this stage represents as postprandial hyperglycaemia, then fasting hyperglycaemia and finally as the Hyperglycaemia or Type 2 DM. FFAs which are directed to the liver are associated with

increased production of Apo-B containing triglyceride. In the presence of hypertriglyceridemia, a decrease in the cholesterol content of HDL is a consequence of reduced cholesteryl ester of the lipoprotein core in combination with cholesteryl ester transfer protein mediated alteration in triglyceride, making the particle small and dense i.e. increase in LDL concentration. Small dense LDLs are thought to be more atherogenic. They may be toxic to the endothelium, and they are able to transmitthrough the endothelial basement membrane and adhere to glycosaminoglycan and results in atherosclerosis and Hypertension.

- **Bheda**

The complications of Insulin Resistance syndrome such as Atherosclerosis, Cardio vascular diseases, Poly cystic ovarian syndrome (PCOS), Hypertension etc. can be considered as the Bheda stage.

Kaphaj Nanatmaj Vyadhi mentioned in ayurvedic texts and diseases included under Insulin Resistance syndrome can be correlated as:

- Dhamani Upalepa\ Dhamani pratichaya (Ch. Su.)- Atherosclerosis
- Atisthaulya (Ch. Su.)- Obesity
- Galaganda (Ch. Su.)- Hypothyroidism
- Bahumutrata (Sha. Pu.), Alasya, Nidradhikya, Staimitya, Gurugatrata- Diabetes Mellitus
- Rasa Raktagat Vruddhi (Ch.)- Dyslipidaemia
- Raja Vaishamya- PCOS

**Summary of Samprapti factors**

Dosha: Kapha

Dusya: Rasa, Rakta (due to sahacharya) Agni: Rasa dhatvagnimandya

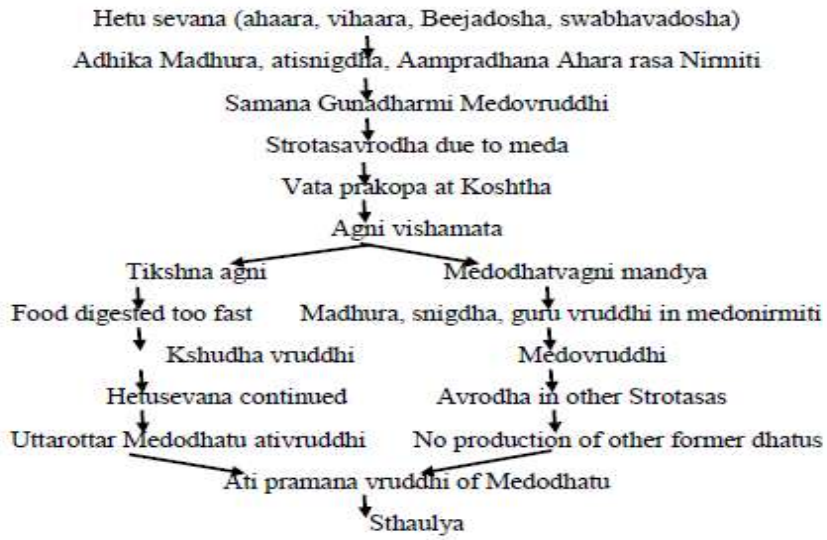
Srotasas: Rasavaha

Srotodushti: Sanga, Vimarga gamana, Atipravritti Adhisthana: Sarva sarira Sukshma Agni Udbhavasthana: Amashaya, Grahaniand Laghvantra.

According to modern physiology the “First step of a hormone’s action is to bind a specific receptor at the target cell”. Cell that lack receptors for the hormones, do not respond. Receptors for some hormones are located in target cell membrane, where as other hormone receptors are located in the cytoplasm or nucleus. When this hormone combines with its receptors, this usually initiates a cascade of reaction in the cell, with each stage becoming more powerfully activated.

- A type of hormone will act on a particular tissue. The target tissues that are affected by a hormone are those that contain its specific receptors. Similarly each Dhatvagni has a specific action at its own dhatu. Though all the Agneyansa (Minute part of metabolic fire) are in circulation, a specific Agneyansas will have action on a specific dhatu (Tissue).
- Insulin is an important hormone which is crucial for normal metabolism of carbohydrates, fats and proteins. The role of Rasadhatvagni is to convert the received food material to micro forms to nourish all the latter dhatus to perform their functions. Similarly insulin activity is associated with energy abundance from energy giving food in the diet and it plays an important role in storing the excess energy.
- Without insulin no cell membrane permits the glucose molecule to transport inside the cell. Insulin increase glucose transport and usage by most of the cells of the body in the same way that it affects glucose transport and usage in the muscle. Hence Insulin resistance can be compared with Rasadhatvagnimandata (defective metabolism) which ultimately leads to Hyperglycaemia, Hypertriglyceridemia and other components of Insulin Resistance Syndrome.

**STHAULYA**




Sthaulya is a disease primarily of dosha Kapha and dushya Rasa and Medo dhatu.

Above mentioned hetu are primarily of snigdha, Madhura, Guru properties which leads to overload for digestion on agni. This results into formation of Amapradhan Ahara rasa. This guru, atimadhura and snigdha Ahara rasa causes accumulation of medo dhatu which has similar properties. The accumulation of Medo dhatu causes strotovarodha in the body leading to Vata prakopa in koshtha. The vitiated vayu in koshtha causes Agni vaishamya. Thus the

Tikshnagni in digests the food at a faster rate, further leading to kshudha vruddhi. If still the hetu sevana is continued there is further vitiated increment of medodhatu. This finally leads to disease Sthaulya.

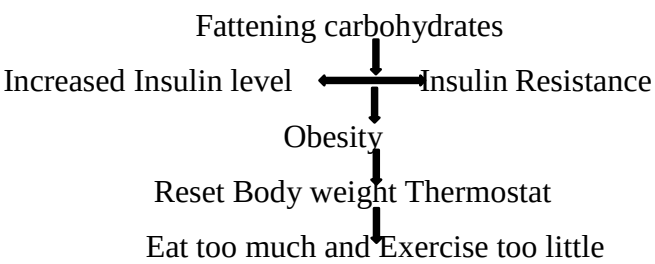
qÉåSxÉÉuÉÚ¨ÉqÉÉaÉïiuÉÉiÉç mÉÑwrÉÇìiÉ AlrÉå lÉ kÉÉiÉuÉ:| qÉåSxiÉÑ cÉÏrÉiÉå iÉxqÉÉiÉç AzÉ£ü: xÉuÉïiMüqÉïxÉÑ||
pÉÉ. mÉë. qÉkrÉqÉ ZÉÇQû

On the other hand there is Medodhatvagnimandya which leads to formation of apachit meda dhatu with more guru, snigdha and madhura properties. This medovruddhi leads to avrodha of other strotasas in body and there is no formation of other dhatu rather than apachita meda dhatu. Hence there is more vruddhi of medodhatu leading to the disease Sthaulya.

- Insulin also promotes fat synthesis and storage. Rasadhatvagnimandya further leads to formation of vitiated Kapha doaha and similar vitiated factors like Snehavruddhi. Thus Insulin resistance also has the role of Medodhatvagni mandata on which medodhatu vridhi i.e. Adiposity are seen. Insulin activates Lipo-protein lipase in the capillary walls of the adipose tissue, which splits the triglyceride in to the fatty acids.
- The number of receptors in a target cell (Pachakansha in dhatu) usually does not remain constant from day to day or even from minute to minute. The receptors dhatus themselves are often inactivated or destroyed during the course of their function and at other time they are reactivated or new ones are formed by their respective Dhatvagni.
- Hence the Dhatvagni are the chemical or hormonal factors essential for changing the sequences of bases at DNA up to formation of functional protein for the purpose of synthesis of its tissue (anabolic) and enzymatic factors essential to yield energy for tissue functions (catabolism).

**Pathogenesis of Obesity**

Hormonal Obesity Theory:




Elevated FFA seen in Obesity contributes to impaired glucose utilization in the skeletal

muscle by increased hepatic synthesis of glucose and by impaired beta cell functions. FFA impairs insulin mediated glucose uptake and accumulate as TG in both skeletal and cardiac muscle, whereas increased glucose production and TG accumulation are seen in liver.

There are three main factors in the pathogenesis of obesity:

1. Excessive lipid deposition
2. Diminished lipid mobilization
3. Diminished lipid utilization

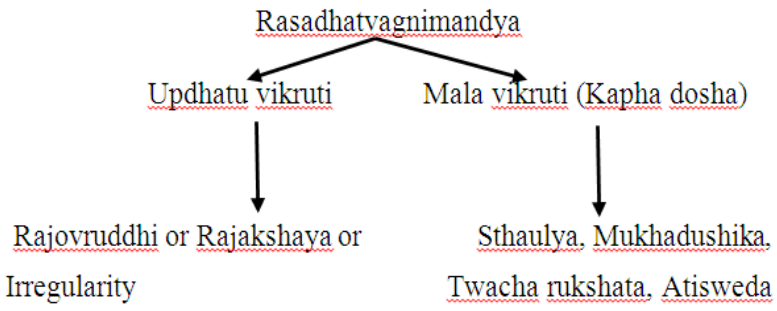
Obesity is strongly associated with Insulin Resistance:

a) Resistance to action of Insulin impairs glucose utilization and hence hyperglycaemia.
b) There is increased hepatic synthesis of glucose.
c) Hyperglycaemia in obesity results into higher levels of free fatty acids and cytokines affect the peripheral tissue sensitivity to respond to Insulin.

**RAJAVAISHAMYA**

**The Ritukala phase**

This phase is influenced mainly by Kapha. Level of Kapha increases (Kaphachaya) at the end of Rajakala. During Ritukala Kapha's level reaches at its peak. This is called as Kapha Prakopa. Level of Pitta starts increasing in latter half of Ritukala. This stage is called as Pittachaya. Vata is at its normal level during the whole phase. According to Aurveda for regeneration and growth Kapha dosha is essential. Ritukala is a period resembling proliferative phase. Kapha always acts through Rasa. Rasa is described as plasma, including the interstitial fluid and lymph. The role of plasma in proliferation of endometrium is obvious so, role of Kapha through Rasa can easily be understood. Hence when Kapha dosha gets vitiated it causes disturbances in the menstrual cycle and ovulation.

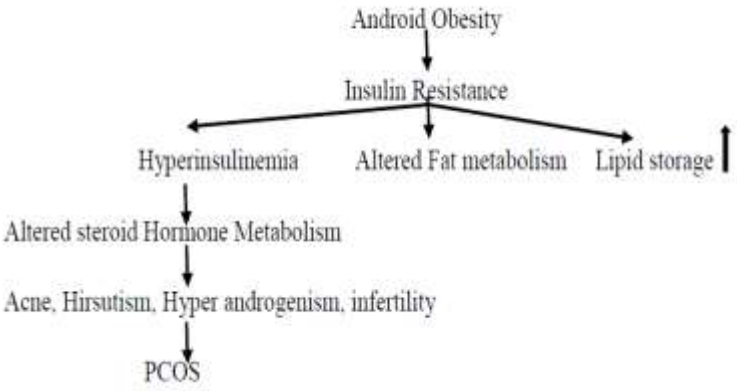



Hetu sevana leads to Rasa dhatvagnimandya. During the Dhatuparinamana process, Updhatu

of Rasa dhatu i.e. Raja gets vitiated along with Mala Kapha. Vitiated Raja leads to either Raja kshaya or vruddhi or irregularity. Vikruta Kapha dosha due to strotorodha causes sthaulya, Mukhadushika, Twakrukshata etc.

## POLY CYSTIC OVARIAN SYNDROME

### Pathogenesis




In an Obese or Non- obese patient, Insulin Resistance leads to Hyperinsulinemia. This increases GnRH pulse frequency, LH over FSH dominance, increased ovarian androgen production and decreased follicular maturation. This altered sex hormone metabolism causes signs and symptoms of PCOS. Insulin Resistance also leads to increased lipid storage which again leads to android obesity. Thus the vicious cycle goes on.

## GALAGANDA

### Samprapti

Dosha: Kapha Dushya: Rasa, Meda

Agni: Jatharagni, Dhatvagni

Ama: Jatharagni mandya Janita, Dhatvagnimandya Janita Srotasa: Rasavaha Srotasa and Medovaha Srotasa Srotodusti: Sanga, Vimarga– gamana, Atipravrutti Adhisthana: Galganda granthi

Udbhavasthana: Amashaya, Rasavaha strotasa Prasara: Rasayanis

Rogamarga: Bahya Vyaktisthana: Sarva sharira


Etiological factors aggravate Kaphadosha resulting Jatharagni Mandya and Dhatvagni Mandya. Rasa dhatvagnimandya is a major feature of the disease. This rasa dhatvagnimandya results into Manda gati of chayapachaya process i.e. slowed metabolism in the body and also leads to formation of apachita rasa dhatu vruddhi of Manda, guru, snigdha properties which causes strotorodha and leads to formation of apachit Kapha dosha of similar properties of Rasa dhatu. This type of kapha dushti causes the symptoms like Bharavruddhi, twacha rukshata, mandabuddhita etc.

In hypothyroidism, hormonal disturbances makes many metabolic disturbances and decrease in basal metabolic rate which results into the disease. The part of the Jatharagni, which works on the Dhatu level, its exacerbation and diminution causes Dhatu Kshaya and vikrut Dhatu Vriddhi respectively.

**Signs and Symptoms**

As mentioned earlier, hypothyroidism has signs and symptoms of many systems. For the study of the signs and symptoms in the light of Ayurvedic principles, the relationship of Dosha and Dushya in each sign and symptom of Hypothyroidism is as follows:

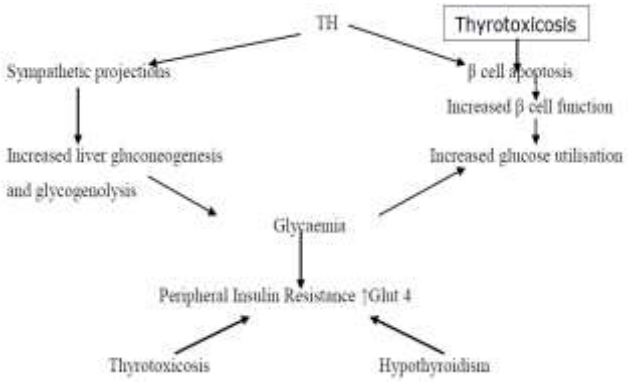
1. Heaviness in body: Kapha vrudhi (Ch. Su. 17/56 and A. H. Su. 11/7) Kaphaj Nanatmaj Vyadhi(Ch.Su.20/17).
2. Puffiness of body features: Kapha Vridhi (Ch.Su.18\22 and A.H. Su. 12\53).
3. Agnimandya: Kapha vridhi (A.H. Su. 11/7) Pitta Kshaya (A.H. Su. 11/16).
4. Dry, Coarse skin/Hair: Vata Vrudhi (Ch Su. 20/11 Kapha vrudhi (Ch. Su. 17/56).
5. Anaemia (Nakhadinam shauklyam): Kapha vrudhi (Ch. Su. 17/56).
6. Hoarseness of voice: Kapha vrudhi (Sharangdhara).
7. Sluggishness: Kapha Vrudhi (A.H. Su. 11/7).
8. Cold intolerance: Kapha Vrudhi (Ch.Su.17, A.H.Su.11, Su.Su. 15).

9. Forgetfulness/ Brain fog: Kupita Kapha (Ch. Su.12/12 and Sharangdhar).
10. Sleepiness- Kapha Vrudhi (A.H.Su. 11/7).

Clinical picture shows the dominance of Kapha Dosha. Majority of the Nanatmaja Roga of Kapha Dosha can be included as a signs and symptoms of Hypothyroidism i.e. Tandra, Atinidra, Stamitya, Gurgatrata, Aalashya, Balasaka, Apachana, Hridayolepa, Galganda, Atisthula, Shitagni, Svetavbhasta. (Ch. Su. 20/11). The conditions accompanied with any of the above symptoms should be diagnosed as a Kapha-disorder.

**HYPOTHYROIDISM**

**Pathogenesis**




The effects of T4 and T3 have a large impact on glucose homeostasis. This concept was acknowledged by Nobel Prize winner Dr Bernardo Alberto Houssay in his lecture in 1947 "The blood sugar and the production and consumption of glucose are kept within normal bounds, therefore there is an equilibrium between the glands of internal secretions which reduce the blood sugar (pancreas) and those which raise it (antero hypophysis, adrenals, thyroid, etc.)".

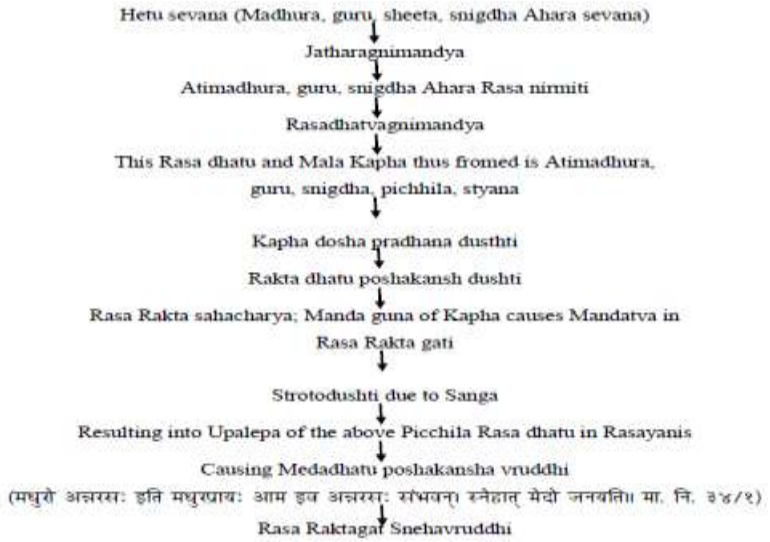
Thyroid hormones exert both insulin agonistic and antagonistic actions in different organs. However, this occurs in a fine balance necessary for normal glucose metabolism. Deficit or excess of thyroid hormones can break this equilibrium leading to alterations of carbohydrate metabolism. Thyroid receptor-mediated effects on gene transcription and translation are key in the regulation of glucose metabolism.

According to the results of studies with complementary DNA (cDNA) microarray analysis in mouse liver, this organ is a major target of thyroid hormones.(56) Studies performed in adipocytes and skeletal muscle of rats made hypothyroid have shown that these tissues are

less responsive to insulin with regards to glucose metabolism.Several genes involved in gluconeogenesis, glycogen metabolism and insulin signalling that are regulated by thyroid hormones have been identified. Thyroid hormones have insulin antagonistic effects at the liver that lead to an increased glucose hepatic output, via an enhanced rate of gluconeogenesis and glycogenolysis. With regards to lipid metabolism, both lipogenesis and lipolysis are stimulated by T3. However, in the context of insulin resistance, the conversion of glucose into fatty acids together with non-suppressed gluconeogenesis is simply perpetuating the hyperinsulinemic state.

## RASA-RAKTAGAT SNEHAVRUDDHI

### Samprapti




Madhura, snigdha, guru, sheeta guna Ahara sevana causes Jatharagnimandya. It should always be kept in mind that once Jatharagni is impaired, the Bhutagni and Dhatwagni would also get further impaired. This Jatharagnimandya leads to formation of Ahara Rasa of Ati guru, snigdha, madhura guna. Ahara Rasa of such gunas impairs the Rasa dhatvagni and this Rasa dhatvagnimandya leads to formation of similar gunayukta Rasa dhatu and Mala Kapha. This formed vitiated Rasa dhatu and Kapha dosha are of pichhila, snigdha, guru and styana gunas. The Prasada Ahara Rasa is transported by Rasa – Rakta to all other dhatus.

The Vitiated Rasa dhatu also affects the latter Rakta dhatu poshakansha. Rasa and Rakta are present in sahacharya in Rasayanis and they have a proper flow which is disturbed by the above styana, guru kapha.

The strotodushti is of Sanga type. The vitiated ati snigdha, guru Rasa dhatu accumulates in the Rasayanis alongwith Rakta and causes Upalepa of Rasayanis i.e. Rasa Raktavahi Sira. Later there is Meda dhatu poshaknsha vruddhi which causes more vruddhi of styana, picchila, snigdha guna of Kapha and Rasa dhatu due to Ashrayashrayee bhava.

It is described that during the pachana of any Madhura, guru shleshmala ahara, firstly sneha yukta ama or Ahara Rasa is formed and latter apachit Meda dhatu vruddhi occurs.

Dosha: Kapha
Dushya: Rasa, Rakta and Meda dhatu poshakansha Agni: Jatharagni and Rasadhatvagnimandya Strotasa: Rasavah Strotasa.
Strotodushti: Sanga, Margavarodha(Ch.Su. 21/3-4) Adhishthana: Sarva sharira (rasa rakta vahini) Udbhavasthana: Amashaya, Grahani.
Roga marga: Bahya

**DYSLIPIDAEMIA**

**Pathogenesis**

In general free fatty acid flux to the liver is associated with increased production of ApoB-containing, triglyceride-rich, very low-density lipoproteins (VLDLs). The effect of insulin on this process is complex, but hypertriglyceridemia is an excellent marker of the insulin resistant condition. Not only Hypertriglyceridemia is a feature of Insulin Resistance Syndrome, but the patients also have elevated levels of lipoproteins.

The other major lipoprotein disturbance is a reduction in HDL cholesterol. In the presence of hypertriglyceridemia, a decrease in the cholesterol content of HDL is a consequence of reduced cholesteryl ester content of the lipoprotein core in combination with cholesteryl transfer protein- mediated alterations in triglyceride that make the particle small and dense. This change in lipoprotein composition also results in increased clearance of HDL from the circulation.

The increase in adipocyte mass and accompanying decreased insulin sensitivity associated with obesity have multiple effects on lipid metabolism. More free fatty acids are delivered from expanded and insulin resistant adipose tissue to the liver, where they are re- esterified in hepatocytes to form TGs, which are packaged into VLDLs for secretion into circulation. In addition, increased insulin levels promote fatty acid synthesis in the liver.

Insulin Resistance also leads to reduced transcription of LPL (Lipoprotein Lipase) in skeletal muscles and adipose tissue as well as increased production of LPL inhibitor apoC-III by the liver.

**DHAMANIPRATICHAYA**

**Samprapti**

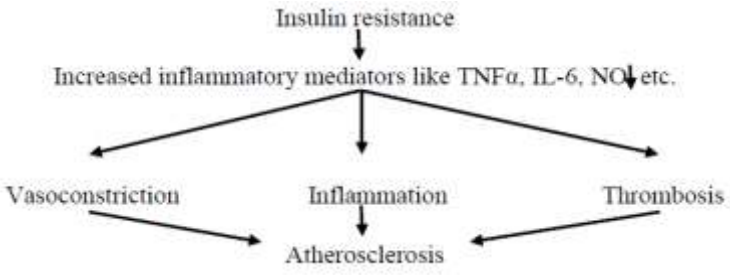

Rasa Raktagat snehavruddhi causes accumulation of styana, pichhila, snigdha Kapha in Dhamani which leads to Dhamani pratichaya. The food, which has reached the Amashya after undergoing digestion, is absorbed and distributed to all Ashayas in the body through Dhamanis.

When Medo Dhatvagni is impaired, the homologues nutrients present in Poshaka Medo Dhatu will be in excess in circulation and this can be referred to the conditions such as Dyslipidaemia. This is because the Poshaka Medo Dhatu cannot be assimilated into Sthayi Medo Dhatu by Medo Dhatwagni. The cause for excess Poshaka Medo Dhatu in circulation is not only the Medo Dhatwagnimandya, but there may be decrease in Jatharagni and Rasadhatvagni also.

Thus more styana Rasa dhatu is formed due to Rasa and Medodhatvagnimandya gets accumulated over walls of Dhamani and leads to Dhamanipratichaya.

**ATHEROSCLEROSIS**

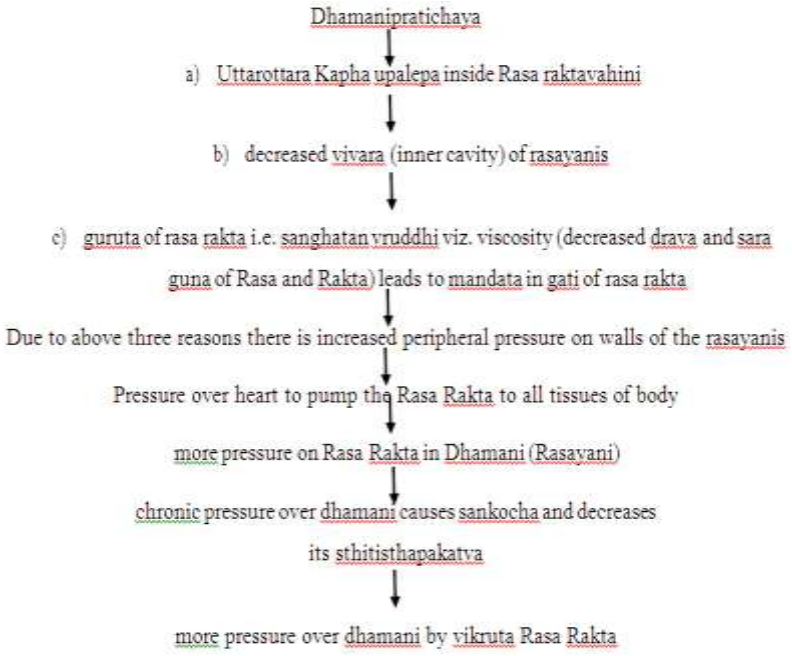
**Pathophysiology**



Hyperinsulinemia along with obesity and dyslipidaemia stimulates the synthesis of inflammatory mediators by the adipose tissue. There is increased inflammatory mediators like TNFα, IL-6, CRP and decreased NO in the vessels. This causes altered vascular reactivity, impaired fibrinolysis and inflammation which further leads to Atherosclerosis.

## RAKTADAABA VRUDDHI

### Samprapti




In further pathogenesis of insulin resistance syndrome, there is more accumulation of Kapha on the inner core of rasayani. This leads to decrease in its diameter further causing Mandata in the gati of Rasa rakta. Also there is increase in the guruta of Rasa Rakta leading to the slowdown of flow of Rasa Rakta through these channels due to decreased inner cavity of rasayanis. This causes increased peripheral pressure exerted by Rasa Rakta over walls of rasayanis. But in order to supply Rasa Rakta to all tissues of the body, heart has to take extra effort and thus increases pressure over heart and ultimately on rasayanis. This chronicity leads to sankocha of Dhamani (rasayani) and decreases its sthitisthapakatva. Thus leads to increased pressure over Rasa rakta vahini (dhamani).

## HYPERTENSION

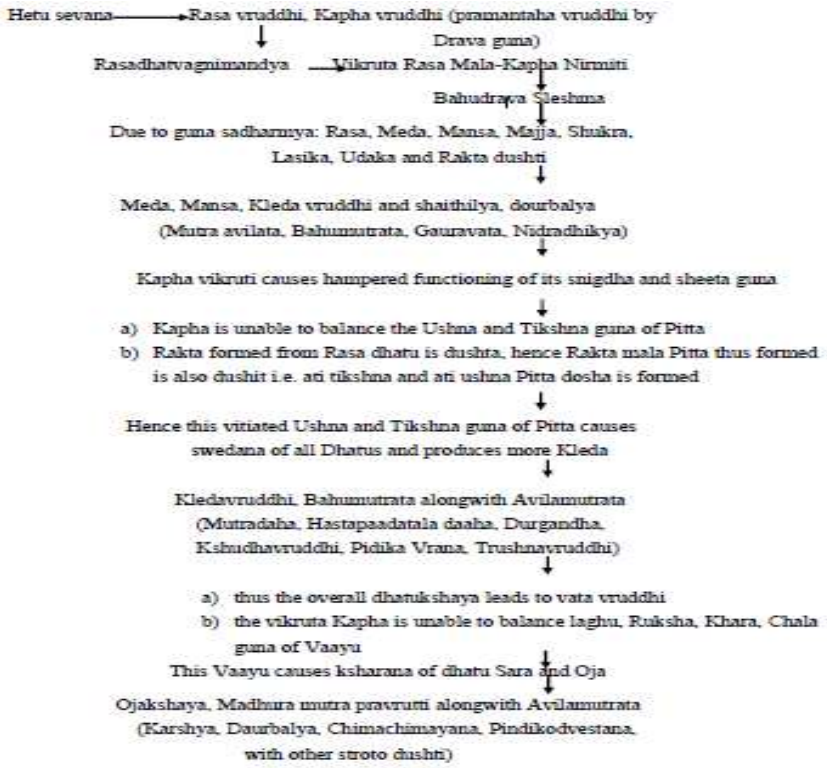
### Pathogenesis

The relationship between insulin resistance syndrome and hypertension is well established. Paradoxically, under normal physiologic conditions, insulin is a vasodilator with secondary

effects on sodium reabsorption in the kidney. However in the setting of insulin resistance, the vasodilatory effect of insulin is lost but the renal effect on sodium reabsorption is preserved. Insulin resistance is characterized by pathway specific impairment in phosphatidylinositol-3-kinase signalling. In the endothelium this impairment may cause an imbalance between the production of nitric oxide and secretion of endothelin 1, with a consequent decrease in blood flow.

**PRAMEHA**

**Samprapti**




- **Sanchaya**

The excessive indulgence in Nidana Sevana of Kapha vruddhikar ahara like Kledapradhana, Guru, Snigdhadi guna and Avyayamadi Vihara leads to Rasa dhatvagnimandya further leading to Kapha Dosha Sanchaya and vikruta Rasavruddhi. It is important to mention that the Kapha Dosha, which gets Sanchita here is having the quality Bahudravatva vividly supported by Charaka. In Prakritavastha, the Kapha remains in Badhdha form i.e. solid or binded form, but due to Nidana Sevana its Prakrita Badhdha form changes to Dravatva form and that too in excess amount i.e. Bahudravatva.

- **Prakopa**

These three factors Nidana, Dosha and Dushya get combined together in such a precise way that they lead to Prakopa of Bahudrava Kapha rapidly and Prameha in future. It indicates the Anukulatva between Hetusevana, Dosha and Dushya. Kaphakara Ahara Vihara vitiates Kapha Dosha and Rasa dhatu without any resistance due to similar properties. The Bahudrava Kapha Dosha is prone to develop Prameha and as it is already present in excess quantity from the beginning, hence it gets aggravated rapidly when the Anukula Nidanas are continued. This kapha also vitiates the elments of body having similar guna like Rasa, Meda, Mansa, Majja, Shukra, Lasika, Udaka and Rakta.

- **Prasara**

In this stage, the provoked Kapha gets spread all over the body owing to Sharira Shaithilya, Daurbalya.

- **Sthana Sanshraya**

The provoked Kapha has affinity towards Bahu-Abaddha Meda due to their similar properties and gets lodged there. The provoked Kapha (Vikrita) after combining with Bahu Abadhdha Meda causes its vitiation, the other important Dushyas are Sharira Kleda and Mamsa, which are already increased in large quantity, prior to vitiation of Kapha. The provoked Kapha with vitiated Meda gets combined with Sharira Kleda or Mansa or both. This is an important stage because the prodromal symptoms of the disease are manifested in this stage.

- **Vyakta**

In this stage of Vyaktavasta, there occur two types of manifestation:

- Puti Mamsa Pidika due to Mamsa Dhatu vitiation– The vitiated Kapha and Meda combines with Mamsa Dhatu leading to Puti Mamsa pidika.
- Mutravaha Srotodushti due to Sharira Kleda Dushti– If vitiated Kapha and Meda come in contact with Sahrira Kleda, then it changes in Mutra, the vitiated Kapha impedes the openings of Mutravaha Srotasa, which are already filled with vitiated Meda and Kleda, thus producing the disease Prameha.

The above two manifestations of Kleda and Mamsa Dushti will occur simultaneously or in two stages. In spite of so many Purvarupas, only two Lakshanas have been mentioned in the classics i.e; Prabhuta Mutrata and Avila Mutrata. Prabhuta Mutrata occurs as a result of Vriddhi Swarupa Kleda Dushti and Avila Mutrata is one of the signs of Kleda Dushti which

is due to dushit Kapha and also dhatunissarana i.e. dhatu get excreted through urine.

- **Bheda**

In this stage various complications of the disease manifest and the disease progresses towards Asadhyadta i.e. the disease becomes incurable. The Prameha disease attains Sthairya (stability) and Asadhya (incurability) status. Also in this stage the dominancy of doshas is expressed and the symptoms of the dominant dosha can be seen in patients. There are different types of prameha under three doshas. There are 10 types of prameha under Kapha dosha depending on which guna of Kapha is vitiated. Similarly there are 6 types of Prameha due to Pitta dushti and 4 types of Prameha due to Vata dushti.

**DIABETES MELLITUS**

**Type 2 D M**

Type 2 Diabetes mellitus has a complex etiology that develops in response to genetic and environmental influences, with insulin resistance & abnormal insulin secretion central to its development.

**Pathophysiology**

Postprandial elevations in serum glucose levels stimulate insulin synthesis and release from pancreatic ß cells. Insulin secreted into the systemic circulation binds to receptors in target organs (skeletal muscle, adipose tissue, liver). Insulin binding initiates a cascade of intracellular signal transduction pathways that inhibits glucose production in the liver, suppresses lipolysis in adipose tissue and stimulates glucose uptake into target cells (muscle and fat) by mechanisms such as the translocation of vesicles that contain glucose transporters to the plasma membrane.

Type 2 DM is characterized by three pathophysiological abnormalities: impaired insulin secretion, peripheral insulin resistance and excessive hepatic glucose production. Obesity, particularly visceral or central is common in type 2DM.

Type 2 diabetes is a metabolic disorder that results from complex interactions of multiple factors and is characterized by 2 major defects i.e. decreased secretion of insulin by the pancreas and resistance to the action of insulin in various tissues (muscle, liver and adipose), which results in impaired glucose uptake. The increase in lipolysis by adipose cells that are resistant to insulin and the subsequent increased levels of circulating free fatty acids (FFA)

also contribute to the pathogenesis of diabetes by impairing ß-cell function, impairing glucose uptake in skeletal muscles and promoting glucose release from the liver. In addition to its role as a source of excess circulating free fatty acids, adipose tissue has emerged in the last decade as an endocrine organ. Adipose tissue is a source of a number of hormones (adipo-cytokines or "adipokines") that appear to regulate insulin sensitivity (e.g., adiponectin, resistin), as well as appetite regulation (e.g., leptin), inflammation (e.g., tumour necrosis factor, interleukin-6) and coagulability (e.g., plasminogen activator inhibitor-1).

The initial response of the pancreatic ß cell to insulin resistance is to increase insulin secretion. Elevated insulin levels can be detected before the development of frank diabetes.

As the disease progresses, pancreatic insulin production and secretion decreases, which leads to progressive hyperglycemia.

**Insulin resistance**

Insulin resistance is a condition in which the insulin receptors of the cell no longer respond well to the hormone so blood sugar levels do not effectively drop and blood levels of both insulin and glucose stay high. With the blood glucose levels still remaining high, the body will again signal for insulin to be produced but it doesn't work. The most common cause of insulin resistance is the frequent consumption of sweet and starchy simple carbohydrates (such as candy, sodas, chips, fries, donuts, cookies, breads, pasta, pastries, bagels, juices and even "energy" bars) that keep blood sugar levels elevated. This puts a constant demand on the pancreas to provide insulin. In time, because the body has the amazing capacity to adapt, the cells simply lose their sensitivity to insulin. It has been identified that insulin resistance can be a precursor to seriously degenerative diseases such as diabetes, heart disease and obesity. In Type 2DM, insulin resistance in the liver arises from the failure of hyperinsulinemia to suppress gluconeogenesis, which results in fasting hyperglycemia and decreased glucose storage by the liver in the post prandial state.

**ACKNOWLEDGEMENT**

I express my deepest gratitude to Vd Neelam Sali and Vd Amit Hivrale, PG students of Kaya chikitsa department, R.A.Podar Medical (Ayu.) College, worli, Mumbai for their support and ideas during the study. I also express gratitude to Mr.Siddhesh Nar, technician of Kaya chikitsa department.

**REFERENCES**

1. "Effect of thyroid status on insulin action in rat adipocytes and skeletal muscle," M.
2. P. Czech, C. C. Malbon, K. Kerman, W. Gitomer and P. F. Pilch, Journal of Clinical Investigation, 1980; 66: 574–582.
3. Ayurvediya Prasuti Tantra evam Striroga, Prof Premavati Tiwari, Chaukhambha Orientalia, Edition 2014; Part 1, chapter 2.
4. Harrison's Principle of Internal Medicine, Dennis L. Kasper, Anthony S. Fauci, Stephen L. Hauser, Dan L. Lango, J. Larry Jameson, Joseph Loscalzo, Mc Graw Hill Education, 19th edition, 2015; Chapter 417.
5. Sushrut Samhita, Dalhan Teeka, Vd. Yadavji Trikamji, Chaukhamba Surbharati Reprint 2012.
6. Bhavaprakasha Nighantu, Dr. K. C. Chunekar, Chaukhambha Bharati, Reprint, 2010.
7. Charaka Samhita Vol. 1 and 2, Dr. Brahmananda Tripathi, Chaukhambha Surbharati Reprint, 2007.