



Understanding Prediabetes *Samprapti* In Terms Of *Shatkriyakala* From Ayurvedic Perspective

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ABSTRACT:

Introduction: Prediabetes is a condition that precedes diabetes. Blood glucose levels become higher than normal but are not classified as diabetes. It is often described as a 'grey area' between normal blood sugar and diabetes. Understanding the pathophysiology of prediabetes from an Ayurvedic perspective, in terms of *Shatkriyakala*, will help intervene in the progression of the disease at crucial time points and prove useful in preventing further complications.

Method: A review of related topics was conducted using various classical *Ayurvedic* texts and modern sources. The compiled data was analyzed for proper interpretation of the topic.

Discussion: Prediabetes is a disease entity that shares characteristics with *Prameha* in *Ayurvedic* classics. The aetiology, pathophysiology, and symptoms are similar. *Kapha* and *Meda* share similar qualitative features and functions, and both are vitiated by similar etiological factors. In *Prameha*, *Bahudrava Shleshma* affects the *Meda*, *Mamsa*, and *Kleda Dhatus*, altering their quality.

The pathogenesis of prediabetes can be elaborated in the same manner as *Prameha*. High-calorie diets, sedentary lifestyles, and lack of physical activities lead to obesity and dyslipidaemias. This is the *Sanchay* stage, resulting in the production of excess free fatty acids, which are directed to the liver. This represents the *Prakopa* of *Amaroopa Shleshma* and *Abaddha Meda*. In the *Prasar* stage, these fatty acids circulate throughout the body and occupy insulin receptors in the *Sthansamshraya* stage. This results in insulin resistance in the body tissues, leading to prediabetes.

KEYWORDS: Prediabetes, *Samprapti*, *Shatkriyakala*

INTRODUCTION:

Prediabetes is a condition defined by blood glucose levels that are higher than normal but below the threshold for diabetes. It is considered a high risk state, with a significant likelihood of progressing to diabetes. Approximately 5%–10% of individuals with prediabetes develop diabetes annually.¹ Prediabetes is a precursor to type 2 diabetes mellitus and metabolic syndrome, with obesity often being the root cause of these conditions. Insulin resistance plays a significant role in the pathogenesis of all these metabolic disorders.²

Many unique concepts are presented in *Ayurveda*, and *Shatkriyakala* is one of them. This concept, introduced by Acharya Sushruta,³ helps to understand how diseases develop through sequential stages. Providing a comprehensive framework for explaining the pathogenesis of disease.

Prediabetes and diabetes mellitus have been extensively discussed within the context of *Prameha/Madhumeha* by *Ayurveda* researchers due to their remarkable similarity. The causes of diabetes mellitus are comparable to the *Ayurvedic* disease entity *Prameha/Madhumeha*. *Ayurvedic* texts describe the pathogenesis of this disease in an extremely evolved manner, involving the three *Doshas* (with a

predominance of *Kapha*) and ten *Dushyas* (ranging from *Rasa* to *Oja*, with particular emphasis of *Meda*).⁴ The significance of *Meda* (adipose tissue) as the principal *Dushya* has been recently confirmed by modern medicine, where central obesity and dyslipidaemia are considered key components of the disease's fundamental matrix.⁵

Prediabetes exists in the body before diabetes develops. The concept of *Shatkriyakala* emphasizes the importance of intervention before a disease fully manifests. Understanding prediabetes through the lens of *Shatkriyakala* can help determine the exact stage of *Dosha* and *Dushya* in the disease's pathophysiology according to Ayurveda. This understanding will also be beneficial in determining the appropriate interventions to halt disease progression and prevent further complications such as diabetes mellitus and cardiovascular disease. Therefore, an attempt is made to understand the pathophysiology of prediabetes from an Ayurvedic perspective, using the concept of *Shatkriyakala*.

AIM AND OBJECTIVES:

To study the pathophysiology of prediabetes in terms of *Shatkriyakala* from an Ayurvedic perspective.

MATERIAL AND METHOD:

Ayurvedic classical textbooks such as *Charaka Samhita*, *Sushruta Samhita*, *Astanga Hridayam*, and various modern textbooks have been analyzed to collect material related to this topic. Other research papers were also reviewed to gain additional knowledge on the subject. The collected material was compiled and analyzed to extract the essence of the topic.

REVIEW OF LITERATURE:

Prediabetes is an intermediate state of hyperglycaemia in which glycaemic measures (such as glucose level and HbA1c) are elevated beyond normal levels but do not reach the diagnostic thresholds for diabetes mellitus.

Current diagnostic criteria for prediabetes according to the American Diabetes Association include:⁶

- Fasting plasma glucose (impaired fasting glucose): 100-125 mg/dl and/or
- Plasma glucose concentration after 2 hours post 75 g OGTT (impaired glucose tolerance): 140-199 mg/dl and/or
- HbA1c: 5.7-6.4%

As prediabetes is a precursor to diabetes, the pathology is interrelated. Normal glucose homeostasis is controlled by three interrelated processes: gluconeogenesis (glucose production occurring in the liver), uptake and utilization of glucose by the peripheral tissues, and insulin secretion by the pancreatic islet cells. Prediabetes begins with an accumulation of excess free fatty acids in adipose tissue, as well as non-adipose tissues such as the liver, muscle, and pancreas.⁷

Insulin resistance plays a significant role in the pathophysiology and development of prediabetes and further diabetes. Different mechanisms of insulin resistance and insulin secretion are established in various subtypes of prediabetes. In prediabetes, patients with isolated impaired fasting plasma glucose exhibit considerable hepatic insulin resistance, while those with isolated impaired glucose tolerance show significant peripheral insulin resistance.⁸

One theory suggests that obesity is a major cause of increased insulin resistance. Intra-abdominal "central" adipose tissue is metabolically active and releases large quantities of free fatty acids (FFA); additionally, the clearance of FFA may be reduced.⁹ Once plasma FFA levels are elevated, they inhibit insulin's antilipolytic action, which further increases the rate of FFA release into the circulation.¹⁰ This may induce insulin resistance because FFAs compete with glucose as a fuel supply for oxidation in peripheral tissues such as muscle. Furthermore, adipose tissue releases several hormones (including various peptides called 'adipokines' due to their structural similarity to immunological 'cytokines') that act on specific receptors to influence insulin sensitivity in other tissues. Since the venous drainage of visceral adipose tissue is into the portal vein, central obesity may particularly impact insulin sensitivity in the liver, adversely affecting gluconeogenesis and hepatic lipid metabolism.

CONCEPT OF SHATKRIYAKALA:³

The concept of *Shatkriyakala* in Ayurveda refers to the time of treatment or opportunity in the process of disease manifestation. *Shatkriyakala* (six stages) includes *Sanchaya* (stage of accumulation), in which there is a gradual accumulation of *Dosha* in their respective seats. *Prakopa* (stage of aggravation) is when the accumulated *Dosha* becomes excited and aggravated. *Prasara* (stage of spreading) involves the aggravated *Dosha* leaving their original place and spreading to other parts of the body through different *Srotas*. In *Sthanasamshraya* (stage of localization), vitiated *Dosha* moves to different places and becomes localized at the site of *Khavaigunya*, i.e., derangement in *Dhatu* or an organ. *Vyakti* (stage of manifestation) is when symptoms of a specific disease appear, and *Bheda* (stage of complication) is characterized by all diagnostic features of the manifested disease, with or without complications.

PRAMEHA:

Prameha/ Madhumeha, a clinical entity mentioned in *Ayurvedic* classics, bears resemblance to diabetes. *Prameha* is a *Tridoshaj Vyadhi* involving *Kapha* as the predominant *Dosha*. *Kapha* is the first *Dosha* mentioned in *Dosha Dushya Sangraha* of *Prameha*. While explaining the *Samprapti* and types, *Kaphaj Prameha* is discussed initially, indicating the importance of *Kapha* in the pathogenesis of *Prameha*. There are twenty types of *Prameha* (ten *Kaphaj*) caused by the three *Doshas* and ten *Dushyas*. In the initial stage of pathogenesis, excess *Kapha* vitiates *Meda* and *Kleda*, leading to *Kaphaja Prameha*. However, as the disease progresses, the dominance of the *Dosha* varies.

After reviewing various *Ayurvedic texts*, the *Samprapti* of *Prameha* can be understood as follows:

Kapha Kara Ahara Vihara leads to *Jatharagnimandya*, resulting in *Asamyak Ahara Parinaman*, which produces *Ama Rasa*. *Kapha* is a *Mala* of *Rasa Dhatu*. The *Sharirastha Kapha* is continuously nourished by this *Kapha* during the *Parinaman* of the *Ahara Rasa* into *Rasa Dhatu* by the action of *Rasadhatvagni*. However, as the *Ahara Rasa* is in the form of *Ama*, it generates *Saam Kapha*. This vitiated *Kapha* loses its quality due to the presence of *Ama*. This leads to an increase in *Dravaguna* (*Bahudravata*) of *Sleshma*, thereby causing further *Dhatu Dushti*. *Jatharagnimandya* is accompanied by *Bhootagni Mandya*, especially that of *Apyagni*. Both *Agnimandya*s lead to improper formation of *Kleda*, resulting in insufficient *Dhatvagni* to act on *Kleda*. *Kleda* is a liquid substance produced in the body during digestion; it travels along with *Rasa* all over the body, helping in the nourishment of *Dhatu* and collecting *Dhatu Malas*. Thus, *Jatharagni*, *Dhatvagni*, and *Bhootagni Mandyata* lead to *Dushti* in the process of digestion, resulting in an increase in *Kleda* in most of the *Kapha Pradhan Dhatu*s. *Meda* hence loses its quality, resulting in “*Bahuta*” and “*Abaddhata*.” Since *Meda* and *Kapha* are similar in their properties and *Meda Dhatu* is the *Ashray Sthan* of *Kapha Dosha*, the *Mishribhavan* of *Kapha* and *Meda Dhatu* occurs, causing *Meda Dusthi*.

The *Dosha Dushya Sammurchana* of *Prameha* begins with the *Samsarga* of *Shareera Kleda/Mamsa* by *Dushta Meda*. When *Kleda* is involved, it directly affects the *Mutra* and *Sweda*. In normal physiology, *Mutra* and *Sweda* maintain the balance of *Kleda* in the body. *Sweda* holds *Kleda* in the body, and *Mutra* excretes it according to the body's condition and requirement. If *Kleda* is vitiated, it directly affects the physiology of *Mutra* and *Sweda*, disrupting the assemblage of bodily elements and causing *Shaithilya*. Thus, the symptoms manifest due to *Kleda* vitiation includes *Prabhutamutrata*, *Swedavruddhi*, and *Daurgandhya*. The vitiated *Kleda* is eliminated from the body through *Mutra*.

Vitiation of the *Mamsa Dhatu* provides an affable atmosphere for the manifestation of putrefied carbuncles (*Pidika*) like *Sharavika* and *Kacchapika* in the muscle.

Table 1: Stages in Prameha Samprapti

Sr.No.	Stages in Samprapti	Reason	Details
1	<i>Kapha Sanchaya</i>	<i>Kapha Kara Ahara Vihara</i>	<i>Anubandha</i> of <i>Nidan</i> , <i>Dosha</i> , <i>Dushya</i> causes <i>Sanchaya</i>
2	<i>Kshipra Prakopa</i> of <i>Shleshma</i>	<i>Prak Atibhuyastvat</i>	As <i>Kapha</i> and <i>Meda</i> are already accumulated, they immediately cause vitiation.
3	<i>Kshipra Visrupthi</i> all over the body	<i>Shareera Shaithilyat</i>	Spreads all over the body due to its laxity
4	Initial combination of <i>Vikrut Kapha</i> with <i>Meda</i>	<i>Medasaha Bahu Abaddhatvat</i> and <i>Samana Gunatvat</i>	<i>Kapha</i> initially mixes with <i>Meda</i> because the fat is abundant, non-compact, and shares similar properties with <i>Kapha</i> .
5	<i>Dushana</i> of <i>Meda</i> by <i>Prakupita Shleshma</i>	<i>Vikrutatvat</i>	<i>Mishribhavan</i> of <i>Kapha</i> and <i>Meda</i> <i>Dhatu</i> (<i>Dosha Dushya Sammurchana</i>).
6	<i>Samsarga</i> of <i>Shareera Kleda</i> - <i>Mamsa</i> by <i>Dushta Meda</i>	<i>Kleda-Mamsayoho Atipramanabhivruddhatvat</i>	Vitiated <i>Kapha</i> and <i>Meda</i> affect <i>Kleda</i> and <i>Mamsa</i>

While explaining the *Samprapti* of *Prameha*, it is necessary to understand the concept of *Vikara Vighata Bhava* and *Abhava*. This concept is mentioned by *Acharya Charak* in *Prameha Nidan*.¹¹ The factors responsible for the development of *Samprapti* of *Roga* are *Nidan*, *Dosha*, and *Dushya*. The intensity of the disease, its occurrence, and its non-occurrence are all influenced by the amalgamation of these three factors.

When *Nidan*, *Dosha*, and *Dushya* differ in terms of *Dravyata*, *Gunata*, *Karmata*, etc., it can lead to a delay or absence of disease manifestation. When *Nidanadi* factors exhibit similarity in terms of *Dravyata*, *Gunata*, and *Karmata*, they facilitate the manifestation of a disease. Conversely, if there is no association between these *Nidanadi* factors, the process of disease manifestation will be delayed. A rapid and vigorous association among *Nidanadi* factors will lead to early disease manifestation, characterized by the presence of all signs and symptoms.

Kala or time also plays a role in disease manifestation. *Sannikrutha Nidana* can result in an instant vitiation of *Dosha*, leading to the immediate manifestation of the disease. *Bala* or strength of the *Nidanadi* factors also determines the nature of disease manifestation. Out of these three factors, if both *Nidan* and *Dosha* are strong enough, they will manifest the disease in a full-blown form. Conversely, if the *Dhātu* is strong enough or there is no *Dhātu Vaigunya*, the disease pathology will not progress.

If there is an absence of association between these *Nidanadi* factors, or if the association of *Nidanadi* factors takes a considerable amount of time, it leads to a delayed manifestation of the disease. If these factors lack sufficient strength, the disease will manifest in a milder manner, with fewer signs and symptoms. Additionally, the presence of *Vyadhi Virodha Bhava* (i.e., *Balavruddhikar Bhava* like *Satmya Sampat*, *Sharir Sampat*, etc and *Dhatu Sarata*, *Vyadhiksham Sharir*, etc.) may prevent the disease from manifesting or result in a milder form of the disease.

A *Kapha Pradhan* individual is more prone to *Prameha*. If such a person indulges in *Kapha Prakopa Ahar* and *Vihar*, it immediately vitiates the *Kapha Dosha* (*Drava Guna*), causing *Rasa*, *Kleda*, *Meda*, and *Mamsa Dhatu Dushti*. In *Drava Roopa Dhatu*, this *Vikruti* is in the form of increased *Dravata*, while in the case of *Ghan Dhatu*, the *Vikruti* is in the form of *Shaithilya*.

Thus, disease manifestation depends on how exogenous factors like *Ahara*, *Vihara*, and *Manasika Nidana* influence the endogenous factors (*Dosha*). The intensity, quality, and quantity of exogenous causative factors, as well as the body's internal condition (healthy, vitiated *Dosha*, or weak status *Dhatu*), determine how severe the disease will develop in the person's body.

DISCUSSION:

Shatkriyakalatmak Samprapti of Prediabetes:

Prediabetes can be understood in terms of *Prameha Samprapti*, as the etiological factors, *Dosha*, and *Dushya* involved in the *Samprapti* are the same. *Prediabetes Shatkriyakala* can be interpreted in terms of *Prameha Samprapti*.

Sanchaya: In *Prameha*, vitiated *Kapha Dosha* first affects *Sharirastha Rasa* and *Meda Dhatu* due to their *Dravya*, *Guna*, and *Karma* similarity. They get vitiated more or less by the same etiological factors. This can also be understood by the *Ashraya Ashrayi Bhava*. Thus, excessive indulgence in *Nidan Sevan* of *Guru*, *Snigdha Aahar*, and *Avyayamadi Vihar* leads to *Kapha Dosha Sanchaya*, i.e., *Pramanataha* and *Gunataha Vruddhi* of *Malarupi Shleshma*. Increased visceral fat deposition can be considered as a stage of accumulation. Contemporary medicine also acknowledges that a high-calorie diet, sedentary habits, and stressors play an important role in the etiopathogenesis of prediabetes. Additionally, positive family history, overweight, and obesity are basic risk factors for transitory dysglycaemia.

Prakopa: If *Kaphakar Aahar Vihar* continues, it further vitiates *Kapha* and *Meda* without any resistance due to their similar properties with *Kapha* and *Meda*. This type of *Anukulatva* may be seen in individuals with *Kapha Prakriti* who have a genetic predisposition to *Prameha*. The *Kshipra Prakopa* of *Kapha* is due to its association with already accumulated *Kapha* and the circulation of *Dosha* from its site (*Prak Atibhuyastvat*). The abnormal visceral fat deposition leads to the breakdown of fat, resulting in the excessive formation of Free Fatty Acids (FFA). These FFAs are directed to the liver, causing hepatic glucose overproduction, release of various pro-inflammatory mediators, and initiating the process of inflammation. This can be correlated with *Aam Rupa Kapha Prakopa* and *Abaddha Meda*.

Prasar: In this stage, the provoked *Kapha* spreads all over the body because of *Sharir Shaithilya*, which is one of the predisposing factors for *Nidana* towards the *Dosha*. Rapid dissemination (*Kshipra Visrupthi*) of *Kapha* all over the body occurs due to *Shareera Shaithilyat*. Newly formed FFAs (mainly from the visceral adipose tissue) circulate throughout the body. This can be considered the stage of spread.

Sthan Samshraya: The initial combination of the provoked *Kapha* and *Bahu Abadha-Meda* due to their similar properties occurs in the *Sthansamshraya* stage. Most of the FFAs occupy the insulin receptors by molecular mimicry; this can be compared with the stage of localization. At this stage, indistinct symptoms appear.

Vyakti: The provoked *Kapha* (*Vikruta*), after combining (*Samsarga*) with *Bahu-Abadha-Meda*, causes its vitiation, resulting in *Prameha*. Due to the occupancy of circulating FFAs on insulin receptors, downstream signaling of the insulin receptors takes place, leading to insulin resistance and a condition known as hyperinsulinemia. This stage can be considered the stage of disease manifestation.

Bheda: When provoked *Kapha* and *Dushta Meda* amalgamate with *Kleda* and *Mamsa*, they produce *Prameha* with the main clinical features of *Prabhutamutrata* and *Avilmutrata*. The manifestation of *Upadrava* or complications of prediabetes, such as metabolic disturbances, leads to the development of not only Type 2 diabetes but also metabolic syndrome and cardiovascular diseases (CVD). This can be considered the *Bheda* stage of prediabetes.

CONCLUSION:

Prediabetes is a precursor condition that occurs in the body before the onset of full blown diabetes. Insulin resistance, beta-cell dysfunction, increased lipolysis, inflammation, poor incretin action, and hepatic glucose overproduction are pathophysiological stages of prediabetes that are similar to those of diabetes. By comprehending *Prameha Samprapti* in terms of *Shatkriyakala*, the *Samprapti* of prediabetes can be explained. *Shatkriyakala* provides a crucial window of opportunities to intervene and treat the condition before overt symptoms arise. Treatment and preventive strategies should be considered after determining the *Nidan*, *Dosha*, and *Dushya* components in relation to *Anubandha*, *Bala*, and *Kala* in prediabetes.

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