



Review Article

ROLE OF ABHRAKADI YOGAM IN THE MANAGEMENT OF TYPE II DIABETES MELLITUS

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ABSTRACT

Diabetes Mellitus is a metabolic disorder predominantly affecting carbohydrate metabolism, characterized by high blood sugar levels (hyperglycemia). It occurs either due to insufficient secretion of insulin by the pancreas or when cells don't respond to the insulin being produced. Type 2 diabetes is the most prevalent form of diabetes accounting for about 90% of the total diabetic population. Maintaining a healthy diet, engaging in regular physical activity, and prioritizing mental well-being are the key factors to maintaining proper bodily metabolism. Sedentary lifestyles and hereditary factors primarily contribute to the development of type 2 diabetes by causing metabolic imbalances. In Ayurveda, descriptions of *Prameha* particularly *Madhumeha* show close similarities with type 2 diabetes mellitus. According to Ayurveda, all diseases stem from improper functioning of *Agni*, often interpreted as metabolic errors seen in diabetes. *Madhumeha* is a condition characterized by vitiation of *Tridosha* and *10 Dushya*. Correcting the *Agni* and restoring the normalcy of *Dosha* and *Dhatu* is necessary for managing Diabetes. Here a literary review is done in evaluating the potential mode of action of *Abhrakadi Yogam* mentioned in *Yogaratanakara*, *Prameha Chikitsa* having *Deepana*, *Srotosodhana*, *Vatanulomana*, *Rasayana*, *Pramehaghna* properties in the management of Type 2 diabetes mellitus.

INTRODUCTION

Diabetes mellitus is recognized as one of the most significant health challenges worldwide. According to the World Health Organization (WHO), diabetes is a chronic metabolic disease characterized by elevated levels of blood glucose, also known as hyperglycemia^[1]. Today, diabetes poses a substantial burden on the healthcare and socioeconomic sectors globally^[2]. In 2019, estimations showed that India had approximately 77 million individuals with diabetes, and this number is expected to rise to over 134 million by 2045^[3].

Diabetes Mellitus is characterized by relative or absolute insulin deficiency due to β -cell dysfunction or impairment in insulin action i.e., insulin resistance, either or both of which may be present with two forms of diabetes namely Type1 diabetes mellitus (T1DM) and Type2 diabetes mellitus (T2DM). Type 1 diabetes is mainly characterized by insulin deficiency caused by autoimmune destruction of β -cells and Type 2 largely results from the body's ineffective use of insulin also referred to as insulin resistance (IR).

Type 2 Diabetes Mellitus

Type 2 Diabetes Mellitus (T2DM) is the commonest form of diabetes, accounting for approximately 90% of the total Diabetic population. It can occur at any age but predominantly affects older individuals, usually above the age of 40, as well as obese adolescent children^[4]. T2DM is primarily an acquired condition arising from factors such as physical inactivity, consumption of a high-fat diet, and a sedentary lifestyle. Obesity and hypertension are major risk factors for T2DM, while genetic inclination may also contribute to it. Classical clinical presentations in patients with T2DM include

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symptoms like polyuria, polydipsia, dryness of mouth, fatigue, tingling sensation, numbness in the extremities, blurred vision, insomnia etc. Prolonged duration and inadequate glucose control can lead to severe damage to the heart, blood vessels, eyes, kidneys, nerves, teeth, and also increase the risk of infections. Overtime diabetes and poor glucose control can lead to serious damage to heart, eyes, kidneys and nerves, risk of developing heart attack, permanent vision loss, amputation^[5], etc also cause developing infections.

Action of Insulin

Insulin action is initiated in response to the glucose signaling. Glucose, a macronutrient predominantly present in poly and oligosaccharides undergoes enzymatic hydrolysis to monomers. It is then absorbed in the intestine through an active transport mediated by transporter SGLT1 during the postprandial state and enters the blood stream through facilitated diffusion by glucose transporter GLUT2^[14]. In postprandial the glucose peaks due to hepatic glucose production. A balance is maintained between glucose entering and leaving the extracellular fluid (glucose homeostasis). Glucagon hormone is responsible for increase of glucose in circulation by means of gluconeogenesis and glycogenolysis on response to dietary intake. When blood glucose is elevated, Insulin binds to the plasma membrane receptors on the target cells and stimulates β -cells to secrete insulin for cellular glucose uptake by means of insulin mediated glucose transporter (GLUT4) and further release of energy by means of glycolysis in the cytoplasm. Upon binding, tyrosine kinase enzyme (RTKs) is activated for phosphorylation. Tyrosine kinase phosphorylates tyrosine residues on cytoplasmic side. Then phosphorylates insulin receptor substrates IR-1 and 2. This phosphorylation event is a crucial step in the insulin signaling pathway. These phosphorylated tyrosine residues serve as docking sites for proteins with phosphoinositide 3-kinase (PI3K) activity. Upon activation of PI3K, then activates downstream signaling molecules, known as protein kinase B (PKB). The PKB signaling pathway is essential for controlling cell growth, proliferation, enhanced cell survival by inhibiting apoptosis and modulation of cellular metabolism. It then triggers a cascade of intracellular signaling events such as stimulation of glycolysis, inhibition of gluconeogenesis, activation of glycogen synthesis, inactivation of glycogenolysis, inhibition of- lipolysis, ketogenesis and protein breakdown, anabolism (protein synthesis) and finally leading to decrease of blood sugar level by promoting its utilization and storage, uptake of glucose by muscle and adipose tissue, and increasing

quantities of enzymes like glucokinase, phosphofructokinase and pyruvate kinase.

Pathophysiology

The pathophysiology of the condition is complex and involves multiple factors. Disturbances in the blood glucose regulation lead to pathophysiology of Type 2 DM and other metabolic diseases. The condition is mainly characterized by hyperglycemia. The action of Insulin gets reversed. In T2DM, in the initial phases, fasting insulin is increased but response of β - cell to glucose is diminished also called, insulin resistance. In these patients, high insulin levels are seen, Leptin in adipose tissue is also expressed. The metabolic changes are mainly the result of a low insulin-glucagon ratio. In obesity, insulin sensitivity, as well as the modulation of β -cell function, decreases^[15] and the number of receptors is decreased on the insulin responsive cells (muscle cells, fat cells, and liver cells). Overeating cause increased insulin production but decreased synthesis of receptors. Due to reduced glucose uptake, hyperglycemia occurs.

When blood glucose level becomes beyond renal threshold, glucose is excreted into urine. Glucose, which is the key fuel for cellular energy production, and when its uptake is impaired, a state of energy deprivation or energy crisis at the cellular level is encountered. Features of deranged carbohydrate, protein and fat metabolism are seen in patients. Due to osmotic effect, more water accompanies the glucose leading to polyuria. This obligatory water loss, combined with hyperosmolarity, tends to deplete intracellular water, triggering the osmoreceptors of the thirst centres of the brain to compensate the loss. So intense thirst (polydipsia) appears and patient takes more water. The deficiency and ineffective utilization of glucose leads to breakdown of fat and protein, which tend to induce a negative energy balance and lead to loss of weight. To compensate the loss of glucose and protein, patient will take more food (polyphagia) and also to meet energy crisis. Insulin resistance and hyperglycemia can disrupt lipid metabolism, leading to increased levels of circulating free fatty acids (FFA) and triglycerides. This can contribute to fatty liver consequently and the development of dyslipidemia, a risk factor for cardiovascular disease. There is an excess of acetyl CoA, which cannot be oxidised by TCA cycle, due to limited availability of oxaloacetate as a result of gluconeogenesis. This further cause ketogenesis, resulting hyperlipidaemia. There is an increased breakdown of proteins and amino acids for providing substrates for gluconeogenesis when protein metabolism is impaired. This causes muscle wasting in the patients. The absence of anabolic effect of insulin enhances the muscle wasting^[17].

Ayurvedic View

In Ayurveda, various *Acharyas* has contributed descriptions on the disease *Prameha* which can be correlated to diabetes as in both conditions, similar pathology is observed. *Madhumeha* bears resemblance in etiopathogenesis and clinical features with Type 2 Diabetes mellitus. The patient with diabetes voids excessive sweet urine which can be compared to 'Madhusama Mutra' and *Prabhuta Avila Mutrata*.

Acharyas categorized *Madhumeha* under *Santharpanajanya Vikara* due to its primary association with a sedentary lifestyle and obesity. *Madhumeha* is one among the 20 types of *Prameha* and is a variety of *Vatika Prameha*. *Acharya Charaka* classified *Prameha* into *Avaranajanya* and *Dhatukshayajanya Madhumeha*^[6]. The former type is a *Kapha-Pitta Pradhana Tridosha Vikara* and the latter is a *Vata Dosha* predominant condition. The initial phases of T2DM can be correlated to *Avaranajanya Madhumeha* due to the obstruction of *Vata Dosha* by the *Avaraka Kapha* and *Pitta*. Due to over indulgence of *Guru, Snigda, Amla, Lavana Rasayukta Ahara, Asyasugha* and avoidance of *Vyayama* leads to excessive increase of *Kapha, Pitta, Meda* and *Mamsa*. They in turn cause obstruction to the normal pathway of *Vata* i.e., *Vata* gets *Avritha* by vitiated *Dosha* and *Dushya*. In the *Nidana Sthana*, *Acharya* described *Madhumeha* as a *Vatika* type of *Prameha*, and if not treated it progresses to manifestation of *Upadrava*.

The main culprit in the pathogenesis involves 10 *Dushyas* namely *Bahudrava Sleshma, Bahuabadha Medas, Mamsa, Sariraja Kleda, Sukra, Rakta, Vasa, Majja, Lasika* and *Rasa*^[7]. In the initial phases, *Kapha Dosha* being the first *Dosha* affected, losses its *Sthiratva* i.e., the *Drava Guna* is increased due to *Dhatvagnimandya* and *Pitta Vridhi*. *Bahudrava Sleshma* then vitiate its *Ashraya Dhatu Medas* along with the *Kleda* to *Bahuabadha Medas* and vitiates all other

Dushya. All the *Kledayukta Dushya* enters the *Mutravaha Srotas*, vitiate it and gets lodged in *Basti*, which is a *Apana Vata Sthana* and gets further afflicted by *Viguna Apana Vata* and is excreted. *Avaranajanya Madumeha* is *Krichra Sadhya* and *Dhatukshayaja* type is *Asadhya*.

As it is a *Medopradoshaja Roga*, it shows the features of *Medo Dushti Lakshanas, Poorvarupa* like *Karapadayo Suptadaha, Mukhaatalukanta Sosha, Alasya*, etc. As the name suggest, the patient excretes urine that possesses the taste and colour resembling honey i.e., characterized by a combination of *Kashaya-Madhura Rasa* and *Ruksha Guna*. Due to vitiation of *Kleda*, patients void *Prabhuta Mutra*, further causing *Trishna*, and *Asyashosha*. In the later phases, *Vata Dosha* is predominant and *Ojas* is also eliminated through urine. It makes the person *Krusha* which is manifested with *Prameha Upadrava*. Progressively many complications like neuropathy, retinopathy, nephropathy, impotency, etc occur.

Acharya Susruta mentions *Tikta, Katu, Kashayanurasa, Sara Guna, Katu Vipaka, Ushna Veerya, Shoshana* and *Chedana* in the treatment of *Madhumeha*. In our classics, numerous herbomineral preparations are mentioned in the management of *Madhumeha*. *Abhrakadi Yogam* mentioned in *Yogaratanakara*, is one such herbomineral formulation with the above-mentioned properties and is taken here for evaluating the potential mode of action in T2DM.

Abhrakadi Yogam^[16]

Abhrakadi Yogam, mentioned in *Yogaratanakara, Uttarahda, Prameha Chikitsa* is a herbomineral formulation. It contains 5 ingredients namely, *Abhraka Bhasma, Hareetaki, Vibheetaki, Amalaki* and *Haridra*. The formulation is a *Sukshma Choorna Kalpana* and has indication in all types of *Prameha*.

Table 1: Ingredients of Abhrakadi Yogam^[8,9,10,11,12]

Sl No.	Ingredients	Botanical Name/ English Name	Family	Part Used
1	<i>Abhraka Bhasma</i>	Incinerated ash of Biotite	-	-
2	<i>Hareetaki</i>	<i>Terminalia chebula</i>	Combretaceae	Fruit
3	<i>Vibheetaki</i>	<i>Terminalia bellerica</i>	Combretaceae	Fruit
4	<i>Amalaki</i>	<i>Embllica officinalis</i>	Phyllanthaceae	Fruit
5	<i>Haridra</i>	<i>Curcuma longa</i>	Zingiberaceae	Rhizome

Table 2: Pharmacological properties

S.No	Ingredients	Rasa	Guna	Virya	Vipaka	Karma
1	Abhraka bhasma	Madhura	Snigda	Sheeta	Madhura	Yogavahi, Medhya, Deepana, Rasayana, Tridosahara
2	Hareetaki	Lavana varjita Pancharasa	Laghu, ruksha	Ushna	Madhura	Cakshushya, Deepana, Pachana, Hridya, Medhya, Rasayana, Anulomana, Balya, Rechana, Tridosahara
3	Vibheetaki	Kashaya	Ruksha laghu	Ushna	Madhura	Deepana, Pachana, Anulomana, Kaphapittajit, Bhedaka, Tridosahara
4	Amalaki	Amla, Kashaya, Tikta, Katu, Madhura	Ruksha Laghu	Sheeta	Madhura	Rasayana, Anulomana, Dahaprasamana, Hridya, Trodosahara
5	Haridra	Tikta, katu	Ruksha laghu	Ushna	Katu	Pramehanasaka, Vishaghna, Kaphapittanut

DISCUSSION

Type 2 diabetes mellitus is characterized by its progressive nature, much like the progression observed in *Prameha*. At different stages of *Dosa Kopa*, the condition in *Prameha* advances, indicating a gradual development and worsening of the disease. In T2DM, the impaired peripheral utilization of glucose by the target cells due to the insulin resistance may be interpreted as an *Avarana* type of pathology. *Vata Dosha* subsequently gets vitiated in the presence of *Avarana*. In *Madhumeha*, *Vata* is being obstructed by vitiated *Kapha*, *Pitta*, *Medas* and other *Dushya*. Thus, proper metabolism or the *Dhatu Parinama* through *Rasadi Dhatuvaha Srotas* gets impaired. The disturbed metabolic state suggest defect in the *Dhatvagni*. The impaired *Medodhatvagni* can be compared with inhibition of PKB signaling. So, digestion, tissue building and other cellular functions will not take place properly. The energy crisis at the cellular level indicates the deprivation of *Dhatu Poshana* due to disruption of *Dhatu Parinama*.

The main principle in the management of hyperglycemia is the compensation of disturbed metabolic process i.e., to normalise blood glucose with hypoglycemic medications. In Ayurveda, *Madhumeha* occur due to either qualitative or quantitative defect in *Dhatvagni*. Thus, correcting *Agni* is necessary. As the *Dhatu Poshana* in *Uttarottara Dhatu* are hampered, *Srothoshodhana* is necessary.

Abhrakadi Yogam contains ingredients with predominance of *Kashaya*, *Amala*, *Madhura Rasa*, *Laghu*, *Rooksha Guna*, *Ushna Veerya*, and is *Akapha* (which does not increase *Kapha Dosha*). Due to its *Laghu Ruksha Guna*, it is *Kaphahara* and *Vata Vardhaka*, the *Laghu Guna* does *Srothoshodhana* by removing the accumulated *Medas* through its *Lekhana*

nature. Thus, normalising the *Samana Vata Gati*, *Kapha Shamana* and improves digestion. The *Ruksha Guna* helps in *Kleda Shoshana*, so it may help in reducing polyuria by the *Sthambhana* nature. The *Ushna Veerya* helps in *Vata Kapha Shamana*, *Deepana* and *Pachana*. The *Madhura Vipaka* helps in *Pitta Shamana*, *Dhatu Poshana* and cures *Vibandha*. All the ingredients are *Tridosahara* and are beneficial in *Avaranajanya Madhumeha* which is a *Kapha Pitta Pradhana Tridoshika Vikara*. The formulation predominantly contains *Deepana*, *Pachana*, *Anulomana*, *Rasayana* properties. Thus, the formulation helps in maintaining the *Sthiratva* of *Dhatu* without increasing *Kapha*. It has *Pramehaghna* property which helps in *Vikara Prasamana*. *Madhu* (honey) is mentioned as *Anupana*. *Madhu* is having *Lekhana* property of honey helps *Kleda Shoshana*, it has *Medo Kshayakara* action. It helps in enhancing the *Pramehaghna* property of *Abhrakadi Yogam* through its *Yogavahi* property.

Hareetaki, by its *Anulomana* property helps in correcting the *Viguna Apanavayu* and *Malapaka*. So, the constipation can be corrected. Also, with the *Brihmana* and *Balya* properties, muscle wasting and emaciation can be managed. It is proven to have antioxidant, hypoglycaemic and hypolipidemic activity. In a streptozotocin induced in vivo study, the chloroform extract of *Terminalia chebula* seeds produced dose-dependent reduction in blood glucose and had significant Reno protective activity and marked inhibition of glycosylation^[18].

Vibheetaki having *Bhedana* property expels the *Badha Mala* through *Guda Marga*. The aqueous extract of *Terminalia bellerica* has proved to enhance insulin-stimulated glucose uptake in adipocytes in an invitro study. At higher concentration, the extract inhibited

protein glycation. Thus, *Vibheetaki* has proved to be a potent drug with anti-diabetic activity^[19].

Amalaki is a *Pramathi dravya* which helps in *Dosha Sanchaya*, removes the *Srotho Sanga* and thus helps in correcting *Dhatvagni* and *Dhatu Parinama*. The *Sheeta Virya* and *Dahaprasamana* property helps in *Pittashamana* thus beneficial in burning sensation of limbs or neuropathy. *Emblica officinalis* has been proved to have anti-diabetic, anticholestremic, antilipidemic and antioxidant activity which helps in clearing *Srotas* and regulation the insulin function. In *invivo* studies, the methanolic extract *Phyllanthus emblica* extracted Quercetin, a flavonoid proved to inhibit glycogenolysis, reduce hepatic glucose production and lower blood glucose.^[20]

Haridra is having *Ushna Veerya*, *Katu Vipaka* thus helps in *Medohara* and *Amapachaka*. *Haridra* with its *Prabhava* is *Pramehaghna*. It has actions in *Raktavaha Srotas*. The curcumin has been proved to have antidiabetic, antioxidant, antihyperlipidemic activity. Many *in vivo* studies on curcumin have proven to reduce plasma glucose, increase plasma insulin levels, increase GLUT-2 transporters^[21].

Abhrakabhasma or biotite ash is a mineral drug used. It is a proven antihyperglycemic, hepato-protective and immune modulator effect which can act as *Rasayana*, it improves the *Dhatu Sarata* and *Nadi Balya*. In an *invivo* anti-hyperglycemiaic activity proved to induce insulin secretion from pancreatic beta cells, reducing oxidative stress induced free radicals. Due to its nano-size are more specifically acid-modified moieties and can cross lining of GI tract to ensure bioavailability also act as nerve tonic. It is also hepato-protective, nervin stimulant

The *Madhura Rasa* worsens the condition of hyperglycemia thus the antagonistic *Rasa* such as *Katu*, *Tikta* and *Kashayanurasa* were indicated in Type 2 DM. *Kashaya Rasa* with its *Sthambhana* property is useful in controlling excessive urination. *Katu* and *Tikta Rasa* have *Deepana* and *Pachana* properties. Thus, it increases the *Pachakagni* and consequently corrects the metabolism. In emaciated patients, *Laghu*, *Mridu*, *Snigda*, *Sara*, *Slakshna* and *Santarpana* properties can be used. *Ushna Virya* helps in this condition. *Madhura Virya* drugs can be selected but it should not vitiate *Kapha* and should not increase *Madhuryata* in body thus *Amlaki*, *Hareetaki* are beneficial.

CONCLUSION

The *Abhrakadi Yogam* is a herbomineral formulation mentioned in *Yoga Ratnakara*, *Prameha Chikitsa*. Management of T2DM before the onset of serious complications is mandatory. The ingredients of *Abhrakadi yogam* is having *Deepana*, *Pachana*, *Vatanulomana*, *Pramehaghna*, *Rasayana*, *Balya*, etc

properties and proven antidiabetic, antilipidemic, antioxidant properties along with honey used as adjuvant helps in reducing the blood sugar. Thus, the formulation is able to break the *Samprapti* there by relieving the symptoms.

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