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"Srotorodha as a Contributing Factor in Insulin Resistance: A Critical Review"

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Abstract:
Background: Insulin resistance is increasingly recognized as a central, driving factor in the pathogenesis of type 2 diabetes mellitus, often detectable ten to twenty years before overt disease manifests. Despite its established predictive value, the precise mechanisms underlying its development remain incompletely understood. One proposed contributing mechanism involves obstruction within the body's microcirculatory channels, a phenomenon that finds a notable conceptual parallel in the Ayurvedic understanding of Srotorodha.

Aim: This review sets out to examine *Srotorodha*, a state of channel obstruction linked to the accumulation of *Ama* (improperly metabolized toxic byproducts), and to explore its possible association with the pathogenesis of insulin resistance.

Methodology: Classical Ayurvedic texts including the *Charak Samhita*, *Sushruta Samhita*, and *Ashtanga Hridaya* with their respective commentaries were reviewed alongside standard Ayurvedic textbooks to build an understanding of *Srotorodha* and *Ama*. In parallel, contemporary scientific literature was searched across databases including PubMed, the Cochrane Library, BioMed Central, IndMED, and Google Scholar to compile relevant findings on the physiology and mechanisms of insulin resistance. Data from both streams were compiled and synthesized into a narrative review.

Discussion: *Ama*, understood as undigested or improperly transformed metabolic material, accumulates within the body's channels and manifests clinically as *Srotorodha*, alongside associated features such as *Bala Bhramsha*, *Gaurava*, *Aalasya*, and *Apakti*. From a physiological standpoint, insulin resistance similarly involves impaired glucose and insulin delivery to target tissues, with microvascular blood flow and capillary recruitment playing a key regulatory role in glucose disposal. *Srotorodha* may parallel this defect by obstructing minute channels, thereby impairing the reception, absorption, and assimilation of insulin and glucose at the tissue level. Elevated blood glucose and consequent hyperinsulinemia may further worsen *Srotorodha*, perpetuating a self-reinforcing cycle of metabolic dysfunction.

Conclusion: *Srotorodha*, as a hallmark clinical sign of *Ama* accumulation, may represent a contributing factor in the development of insulin resistance. A treatment approach targeting *Srotorodha* at an early stage could offer a valuable therapeutic strategy, helping to interrupt this cycle and providing a foundation for novel, Ayurveda-informed approaches to preventing and managing type 2 diabetes.

***Index Terms*: Insulin resistance, Srotorodha, Ama, type 2 diabetes, Ayurveda, Srotovaigunya**

I. INTRODUCTION

Type 2 diabetes mellitus has emerged as one of the most pressing metabolic disorders of our time, and at the heart of its development lies a phenomenon known as insulin resistance. Far from being a mere accompanying feature, insulin resistance is now understood to be a central, driving component in the pathogenesis of this disease. Several independent lines of evidence converge to support this understanding. First, longitudinal observations have shown that insulin resistance can be detected in individuals as early as ten to twenty years before overt diabetes actually develops, suggesting it is not a late-stage consequence but an early warning sign. Second, cross-sectional studies conducted across diverse patient populations consistently reveal the presence of insulin resistance in those already diagnosed with type 2 diabetes, reinforcing its close association with the disease. Third, and perhaps most compellingly, prospective cohort studies have repeatedly identified insulin

resistance as one of the strongest and most reliable predictors of future diabetes onset in currently healthy individuals. Taken together, this body of evidence firmly establishes insulin resistance as a marker of significant predictive value, even though the exact biochemical and molecular mechanisms that give rise to it remain only partially understood, despite decades of dedicated research.

To understand insulin resistance more precisely, it is necessary to first appreciate the normal physiological role of insulin. Insulin is a hormone secreted by the beta cells of the pancreas, and its primary function is to regulate blood glucose levels by facilitating the uptake of glucose into insulin-sensitive tissues, most notably skeletal muscle, adipose (fat) tissue, and the liver. When insulin binds to its receptors on these tissues, it triggers a cascade of cellular events that allow glucose to move out of the bloodstream and into the cells, where it can be used for energy or stored for later use. This process serves the dual purpose of supplying the body with fuel while simultaneously keeping blood glucose levels within a healthy range. Insulin resistance, then, refers to a state in which these target tissues become less responsive to insulin's signal, meaning that even normal or elevated levels of insulin fail to produce an adequate glucose-lowering effect.

One of the clinically challenging aspects of insulin resistance is that it typically develops silently, without producing any noticeable symptoms in its early stages. It is often only after insulin resistance has progressed far enough to cause secondary metabolic disturbances most notably a rise in blood glucose levels that patients begin to experience recognizable symptoms. These may include persistent lethargy or tiredness, increased hunger, and a form of cognitive sluggishness sometimes described as "brain fog." Alongside these symptoms, several other clinical signs are frequently observed in individuals with insulin resistance, including central or abdominal weight gain (commonly referred to as belly fat), elevated blood pressure, and abnormal cholesterol levels. While the underlying causes of insulin resistance are multifactorial, one contributing mechanism that has drawn attention is the obstruction of microcirculatory channels within the body, which is thought to impair the delivery of insulin and glucose to target tissues at the cellular level.

This concept of microchannel obstruction finds a striking parallel in classical Ayurvedic physiology, where it is described under the term Srotorodha. In Ayurvedic understanding, Srotorodha refers to blockage or obstruction within the srotas the minute and macro channels through which nutrients, wastes, and bodily fluids are transported and exchanged. Importantly, Srotorodha is not considered an isolated phenomenon; rather, it is regarded as one of the principal indicators of Ama accumulation within the body's channel systems. Ama is a foundational concept in Ayurvedic pathology, referring to the toxic, undigested, or improperly metabolized byproducts of food that result from impaired digestive fire (Agni). Because this material has not been properly broken down, the body is unable to utilize it as a nutrient; instead, it behaves as a foreign, clogging substance within the system. When Ama is allowed to persist and accumulate over time, it progressively obstructs the normal flow within both the minute and major channels of the body, giving rise to a pathological state known as Srotovaigunya a condition of channel dysfunction that Ayurvedic texts describe as the fertile ground from which various disease processes can originate or take shape.

The presence of Ama in the body is rarely limited to Srotorodha alone; it typically manifests alongside a constellation of other clinical features that together help identify its presence. These include Bala Bhramsha (a noticeable loss of physical strength and vitality), Gaurava (a subjective sensation of bodily heaviness), Anila Mudhata (disturbed or sluggish movement of Vata Dosha), Aalasya (persistent laziness or lack of initiative), Apakti (incomplete or faulty digestion), Nishthiva (excessive salivation or drooling), Mala Sanga (obstruction in the elimination of waste products such as Purisha, or stool), Aruchi (loss of appetite or aversion to food), and Klama (a form of exhaustion or fatigue). Recognizing this cluster of symptoms allows the Ayurvedic physician to identify Ama as an underlying causative factor even before it manifests as overt disease.

Given this framework, it becomes reasonable to propose that Srotorodhaunderstood as a hallmark clinical sign of Ama accumulation may itself be one of the contributing factors in the pathogenesis of insulin resistance. If this correlation holds true, it carries meaningful clinical implications: a treatment strategy specifically directed at resolving Srotorodha could offer physicians a valuable therapeutic avenue. By identifying and addressing Ama as the root cause, clinicians may be better equipped to intervene early, potentially protecting patients from progressing further along this detrimental metabolic pathway toward type 2 diabetes.

Aim and Objectives

This review sets out to examine *Srotorodha* and explore its possible association with insulin resistance.

Methodology

To build an understanding of *Srotorodha* as described in the human body, classical Ayurvedic texts were consulted, including the *Charak Samhita* with the Chakrapani commentary, the *Sushruta Samhita* with the Dalhana's Nibandhasamgraha commentary, and the *Ashtanga Hridaya* with the Arunadatta commentary,

alongside other standard Ayurvedic textbooks. In parallel, a search of contemporary scientific literature was carried out to understand the nature of insulin resistance. Databases and repositories including PubMed, Medknow, the Directory of Open Access Journals, the Cochrane Library, BioMed Central, IndMED, Free Medical Journals, and Google Scholar were systematically searched to identify studies relevant to both modern medical and Ayurvedic prognostic understanding. A range of articles were screened for relevance, and the resulting data were compiled, interpreted, and synthesized into this narrative review.

Literature Review

Defining *Ama*

Ayurvedic *Acharyas* have offered several complementary definitions of *Ama* across different *Samhitas*:

1. When *Agni* (digestive fire) functions improperly, the first *Dhatu*, *Rasa* (chyle), fails to undergo proper digestion. The resulting *Anna Rasa* putrefies as it remains retained in the *Amashaya* (stomach), and this putrefied product is termed *Ama*.
2. Through improper dietary or lifestyle habits (*Nidana Sevana*), *Agni* becomes vitiated and is unable to digest food adequately. This undigested food subsequently ferments and is converted into a toxic substance.
3. Food that has not undergone *Vipaka* (complete metabolic transformation into *Ahararasa*) develops a foul odor (*Durgandha*) and a sticky consistency (*Picchila*), producing *Gatrasadana* (bodily heaviness/debility); this substance is termed *Ama*.

Effects of *Ama*

According to Acharya Vagbhata, when the *Tridosha*, the seven *Dhatus*, and the *Malas* become afflicted by *Ama*, they are collectively described as *Sama*, and any resulting disease is likewise classified as a *Sama* disorder. *Ama* has the capacity to vitiate the *Doshas*, *Dhatus*, and *Malas*, thereby generating disease. *Sama Dosha* is capable of spreading throughout all *Rogamarga* (disease pathways) and can migrate between *Shakha* (peripheral channels) and *Koshtha* (the gastrointestinal core) in either direction. It circulates through the body alongside *Rasa Dhatu* and tends to settle at sites of *Khavaigunya* (structural or functional weakness), where it gives rise to disease.

Clinical Features (*Lakshanas*) of *Ama*

1. *Srotorodha* — obstruction within the channels
2. *Bala Bhramsha* — a sense of weakness
3. *Gaurava* — a sense of heaviness
4. *Aalasya* — laziness
5. *Anila Mudhata* — impaired functioning of *Vata Dosha*
6. *Apakti* — indigestion
7. *Nishthivana* — excessive salivation
8. *Mala Sanga* — constipation
9. *Aruchi* — loss of taste/appetite
10. *Klama* — lethargy

Insulin Resistance

Physiological role of insulin. Insulin functions as the principal hormone governing cellular energy supply and macronutrient balance, orchestrating the anabolic processes characteristic of the fed state. It is essential for enabling glucose to enter insulin-dependent tissues, particularly skeletal muscle and adipose tissue. When exogenous energy is abundant, insulin signaling suppresses the breakdown of stored fat in adipose tissue while promoting its synthesis. Within muscle cells, insulin-driven glucose uptake supports glycogen synthesis and storage, and favors carbohydrates rather than fatty acids or amino acids—as the immediate fuel source for muscular contraction. In this way, insulin promotes both glycogen and lipid synthesis in muscle while simultaneously suppressing lipolysis and the use of muscle-derived amino acids for gluconeogenesis. When amino acid supply is sufficient, insulin exerts a broadly anabolic effect on muscle tissue.

Mechanism of insulin action and its regulation. At the systemic level, insulin's actions are shaped by its interplay with other hormones. Although insulin is the dominant regulator of metabolism during the fed state, it functions alongside growth hormone and IGF-1; growth hormone secretion, triggered in part by insulin itself, helps guard against insulin-induced hypoglycemia. Other counter-regulatory hormones glucagon, glucocorticoids, and catecholamines become more active during fasting states, with glucagon in particular promoting glycogen breakdown, gluconeogenesis, and ketogenesis. Rising glucose levels trigger the initial phase of glucose-stimulated insulin release from the secretory granules of pancreatic beta cells. This glucose sensing occurs via the enzyme glucokinase, which phosphorylates glucose into glucose-6-phosphate (G6P), generating ATP in the process.

Mechanism underlying insulin resistance. When insulin-mediated glucose uptake and utilization become defective, the storage of glucose as glycogen in muscle and liver tissue declines correspondingly. As insulin resistance progressively worsens, the pancreas becomes increasingly unable to compensate through greater insulin output. Over time, as beta-cell function falters and can no longer overcome the underlying resistance, chronic hyperglycemia often termed glucotoxicity sets in, which in turn further impairs insulin action. Glycemic control deteriorates further due to the glucotoxic effects on skeletal muscle, compounded by worsening dysregulation of lipid metabolism, particularly involving free fatty acids (FFAs). As the pancreas attempts to overcome insulin resistance by raising insulin secretion, individuals become hyperinsulinemic, a state that produces exaggerated responses in tissues that retain insulin sensitivity. However, resistance is not confined to muscle and liver alone various cellular functions across the body can exhibit insulin resistance. Notably, insulin resistance in adipocytes drives increased lipolysis, releasing fatty acids and triggering downstream consequences such as dyslipidemia and vascular abnormalities linked to elevated circulating free fatty acids. High FFA levels further compound insulin resistance by promoting hepatic glucose output while simultaneously reducing glucose uptake in skeletal muscle.

Grades

Contribution of Blood Flow to Glucose Metabolism

- Glucose metabolism and blood flow are intrinsically coupled processes — a change in one inevitably influences the other.
- Just as shifts in metabolic activity can alter blood flow, changes in blood flow can themselves drive metabolic shifts.
- Heightened metabolic demand is well documented to recruit additional blood flow, ensuring the delivery of necessary substrates to tissues.
- Changes in insulin-mediated capillary recruitment correlate positively with changes in insulin-stimulated glucose disposal.
- Findings from both animal and human studies indicate that capillary recruitment and blood flow within skeletal muscle play a significant physiological role in enhancing the delivery of insulin and glucose to metabolically active target tissues.
- Insulin influences glucose uptake through two complementary pathways:
    - A **direct effect** — increasing glucose uptake within skeletal muscle.
    - A **substantial indirect effect** — promoting glucose disposal through increased blood flow.

Discussion

Ayurvedic perspective on Srotorodha:

- The body's internal transport network serves as the functional platform through which various biological factors carry out their activities.
- *Srotorodha* facilitates interaction between different body tissues at the site of an underlying defect caused by *Ama*.
- Obstruction of the body's minute channels the state termed *Srotorodha* can manifest as a range of disorders, including obesity, diabetes, cardiovascular disease, and numerous other conditions.
- *Ama* is understood to give rise to *Srotorodha*, which in turn leads to *Sroto Dushti* (channel vitiation) and, subsequently, to irregular tissue transformation.
- Disease manifestation in the body can thus be traced back to the progression of these underlying obstructive processes.

Contemporary physiological correlation:

- The body relies on a variety of transforming substances — enzymes, hormones, catalytic agents, and others to carry out normal metabolic functions.
- When these substances fail to operate correctly, abnormal metabolites are generated that the body cannot properly utilize.
- These metabolites subsequently accumulate across different physiological systems, disrupting normal function.
- Within the Ayurvedic framework, such accumulated material can be understood as *Ama*.
- *Ama* is not a single, fixed entity but a broad, generalized concept encompassing many types of malformed or improperly processed substances in the body, all implicated in the production of various diseases.
- The buildup of metabolic byproducts and waste that go unutilized or unexcreted can likewise be classified as *Ama*.

- *Srotorodha*, in this context, serves as a key clinical indicator that *Ama* is present within the body's channels.

Pathological link to insulin resistance:

- *Srotorodha* obstructs the minute channels of the body, disrupting the nourishment of surrounding tissues.
- This obstruction interferes with the normal processes of reception, absorption, and assimilation carried out by receptors located within these channels.
- Over time, internally generated impurities and metabolic toxins may accumulate further, contributing to degenerative tissue changes.
- The reduced ability of insulin-sensitive cells to respond to insulin may itself stem from *Srotorodha*, which restricts insulin's entry into the relevant channels and simultaneously hampers glucose uptake from the bloodstream.
- As blood glucose levels rise, the pancreas is signaled to increase insulin output in an effort to restore normal levels, resulting in hyperinsulinemia.
- This elevated circulation of glucose and insulin promotes further accumulation of internal metabolic and cellular waste, intensifying *Srotorodha* to an even greater degree.
- Addressing *Srotorodha* early, through a targeted treatment approach, may help individuals interrupt this self-perpetuating cycle and limit further disease progression.

Conclusion

1. A comprehensive understanding of *Ama* in all its dimensions is essential for both preventive and curative approaches to disease.
2. Although the formation of *Ama* occurs to some degree as a continuous physiological process, considerable care must be taken to prevent its excessive accumulation.
3. When *Ama* accumulates and stagnates within the body, it gives rise to *Srotorodha*, which disrupts physiological homeostasis and increases an individual's susceptibility to disease.
4. Identifying and understanding the relationship between the various factors that modulate insulin sensitivity and insulin resistance viewed through an Ayurvedic lens represents an important prerequisite for the development of novel, more targeted treatment strategies for this condition.

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