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Review Article

A CRITICAL REVIEW OF *SAMPRAPTI* OF *MADHUMEHA* IN *SUTRASTHANA* 17 OF *CHARAKA SAMHITA* AND ITS CORRELATION WITH TYPE 2 DIABETES MELLITUS PATHOPHYSIOLOGY

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ABSTRACT

Type 2 diabetes mellitus is a major global health burden, and Ayurveda had described a comparable condition, *Madhumeha*, more than two thousand years earlier. *Charaka Samhita*, *Sutrasthana* 17, *Sloka* 78-81, gives an unusually compact account of *Madhumeha's* pathogenesis that has drawn less scholarly attention than the fuller description in *Nidanasthana*. **Objectives:** This review examines these four *Sloka* closely, compares them with the treatment of *Prameha* and *Madhumeha* in *Sushruta Samhita*, *Ashtanga Hridaya*, *Madhava Nidana* and *Sharangadhara Samhita*, and builds a stage-by-stage comparison between this classical pathological model and the current understanding of type 2 diabetes mellitus. **Methods:** A narrative review was conducted following SANRA quality criteria. Classical texts were read in standard published editions. PubMed, Scopus and the DHARA/AYUSH research portal were searched **Results:** The *Sloka* describe a sequence of dietary and behavioural excess, tissue vitiation, obstruction of *Vata's* channel, depletion of *Ojas*, a relapsing-remitting course, and complications on neglect. Each stage finds a reasonably specific modern counterpart, from sedentary and caloric risk factors through visceral adiposity to molecular blockade of insulin signalling, progressive beta-cell decline, and diabetic complications. **Conclusion:** The comparison is strongest at the level of *Avarana* and weakest for entities like *Ojas* and *Chinta*. It is best read as a structured interpretation rather than an established biochemical equivalence, useful for generating hypotheses for further clinical study.

INTRODUCTION

Today the largest burden of lifestyle disorder in India is Type2 Diabetes mellitus than any other nations in this world. This fact has reignited a long pending question; how our ancient physicians approached this disease centuries before the entity Insulin was discovered[1]. In classical Ayurvedic text most of the description are bunched under the heading of *Premeha* and *Madhumeha*, is considered as the closest terminology. *Madhumeha* is considered as the most serious among them. *Charaka Samhita* treats *Madhumeha* extensively within its *Nidanasthana*[2] and *Cikitsasthana*[3]. There is another approach to this condition, which is mentioned in a most unusual place in *Charaka Samhita* i.e., *Sutrasthana* chapter 17, the *Kiyanta Shiraseeya* chapter- a section whose primary concern lies with disorders of the *Siras* and *Hridya* (Head and Heart) rather than with *Prameha* itself. Within four *Sloka* s (78-81), Acharya *Charka*, describes entire cause to complications of a particular form of *Madhumeha*, which as per the text arise due to *Avarana* (Obstruction) of *Vata* rather than *Kshaya* (depletion) of *Dhatus*[4]. Many research papers interpret this description type to be the closest to Type 2 Diabetes mellitus[5].

Comparing the Ayurvedic diseases with the Modern conditions is not a new practice, and many times this leads to difference of opinions, which is also no uncommon[6,7]. Many correlations come forth due to close relationship in mechanism of the disease both in classical texts and modern pathophysiology. The classical mechanism of *Madhumeha*, mentioned in *Sutrasthana* chapter 17, has not got much of readers attention unlike the *Dhatu Kashya* mechanism

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mentioned elsewhere in the same Samhitha; Most probably due to the fact that the chapter is mainly deals with the diseases of *Siras* and *Hridya* (head and heart) rather than with *Prameha*. Hence the *Avarana* mechanism of *Madhumeha* got very little attention. The present review is done with an aim to understand this mechanism and see if there is any commonalty between this mechanism and modern pathophysiology of type 2 Diabetes mellitus.

Research Question

The review is based on the following Research Question-

"To what degree does the pathological sequence attributed to *Madhumeha* in *Charaka Samhita*, *Sutrasthana* 17/78-81, align- across current biomedical accounts of type 2 diabetes mellitus, and how much of that alignment reflects a genuine comparison as opposed to a merely superficial resemblance"?

AIM

Primary objective: To critically examine the pathogenesis (*Samprapti* (pathogenesis)) of *Madhumeha* mentioned in *Charaka Samhita*, *Sutrasthana* 17/78-81, and to build a structured, stage-wise comparison linking this classical pathological model to present-day biomedical understanding of type 2 diabetes mellitus.

Secondary Objectives

1. To offer a line-by-line textual reading of *Sutrasthana* 17/78-81, locating the passage within the five-part diagnostic scheme - *Nidana* (etiology), *Purvarupa* (prodrome), *Rupa*, *Samprapti* and *Upadrava*- that classical Ayurvedic nosology expects of any complete disease account.
2. To set this account alongside the opinions on *Madhumeha* in *Sushruta Samhita*, *Ashtanga Hrdaya*, *Ashtanga Sangraha*, *Madhava Nidana* and *Sharngadhara Samhita*, noting what is peculiar to Charaka's obstructive model when compared with other classical texts.
3. To weigh the dietary and behavioural causes (*Nidana*) named in the text against risk factors for type 2 diabetes mellitus that are already established in the modern literature.
4. To assess the classical notion of *Avarana* (obstruction), the impeding of *Vata*'s movement, against what is known of the molecular biology of insulin resistance, including lipid-metabolite-driven interference with the IRS-1-PI3K-Akt-GLUT4 signalling pathway.
5. Tracing a path from reversible insulin resistance and prediabetes through to advanced, insulin-dependent disease, with respect to the *Dosas* (*Vata*, *Pitta* and *Kapha*).
6. To read the classical notion of *Ojas Kshaya* (depletion) against the modern account of gradual pancreatic beta-cell decompensation.
7. To align *Madhumeha*'s classical complications, *Upadrava* and *Piḍika*, with recognized microvascular, infective and neurological complications of diabetes mellitus

METHODOLOGY

This is a narrative review rather than a systematic one. Its design is guided by the SANRA (Scale for the Assessment of Narrative Review Articles). The research question is mentioned above, the search procedure is described below, priority is given to peer-reviewed and classical primary sources, and the discussion keeps points of strong correlations and is separated from the speculative points.

Primary Sanskrit material was consulted through standard published editions together with their principal commentaries: *Charaka Samhita* accompanied by Chakrapanidatta's Ayurveda-Dipika; *Sushruta Samhita* with Dalhana's Nibandhasangraha; the two texts of Vagbhata, *Ashtanga Hrdaya* and *Ashtanga Sangraha*; *Madhava Nidana* with the Madhukosha commentary; and *Sharngadhara Samhita*. Readings were cross-checked against the e-text maintained by the National Institute of Indian Medical Heritage.

The literature search covered PubMed/MEDLINE, Scopus and the DHARA/AYUSH Research Portal, Reference lists belonging to retrieved articles were then hand-searched for further relevant sources. A record qualified for inclusion if it addressed either the pathogenesis or the classical-textual description of *Prameha*/*Madhumeha*, or the pathophysiology of insulin resistance and type 2 diabetes, and appeared in a peer-reviewed journal or a recognised classical-text edition. Opinion pieces, non-peer-reviewed online material, and articles not bearing on mechanism were set aside, with one exception, where a non-indexed source was the only place a particular classical-textual claim could be confirmed, this has been noted explicitly at the relevant point.

RESULTS

Reading Sutrasthana 17/78-81

The *Kiyanta Shiraseeya* chapter, *Sutrasthana* 17, begins the *Roga Catushka*, a group of four chapters concerned with disease classification. The central them and subject for this chapter basically revolves around diseases of Head and Heart, along with the *Dhatu Kshaya* doctrine. In the context of *Dhatu kashya* which continues to *Ojas Ksahya* concept, did Charaka discuss about the entity of *Madhumeha*. The *Sloka* s from 78 till 81 describes the cause, mechanism and symptoms of *Madhumeha.*

The aetiology or the causes mentioned in *Sloka* 78 describe the excess use of *Guru* (heavy), *Amla*

(sour) and *Lavana* (salty) foods; Freshly harvested rice (grains) and newly prepared wine (alcoholic preparation); habit of sleeping during day along with sedentary lifestyle, being the major cause for the above condition. In the next *Sloka* he adds additional causes, such as- lack of exercise, lack of mental exertion (points towards the sedentary lifestyle) and failure to undergo *Rutu shodhana* (Seasonal cleansing). This aetiology is mentioned results in the vitiation (especially excess) of *Kapha, Pitta, Medhas and Mamsa.*in the *Sloka* 80 the pathological mechanism involving these excess factors is mentioned; it says that when the above factors are increased in quantity it obstructs the movement of *Vata* in the channels. This deranged *Vata* is no longer able to move freely, it in turn carries the *Ojas* downward towards the *Basti* region this sets in as the stubborn form of *Madahumeha.* The 81st *Sloka* characterizes the course of the disease as unstable, with signs belonging to all three *Doshas* appearing intermittently, subsiding, and then returning. A closing line, states that neglect of the condition at this point gives rise to seven varieties of *Piḍika*.

Elsewhere in the Samhita, the cause, mechanism, symptoms and complications of *Prameha* are each given a full chapter of their own; here, that same material is compressed into four *Sloka s* within a chapter concerned with an entirely different subject. Later authors, typically read this passage as Charaka's account of the *Avarana* route to *Madhumeha*, separate from the *Dhatus Kashaya*-based route described more fully in *Nidanasthana* 4, where *Vata*, once the other two doshas have subsided, itself begins drawing *Ojas*, *Majja* and *Lasika* into the urinary tract[2]. Whether the underlying mechanism is obstruction or depletion turns out to matter considerably for how well each map onto a particular modern diabetic phenotype, these points are discussed in the later sections.

Before we go to the comparisons of mechanism of the *Madhumeha* and its co relation with Type 2 Diabetes mellitus, it's wise to look briefly what the other classical and contemporary authors have to say about the mechanism of this disease. Sushruta's *Nidanasthana* takes up explaining *Prameha* at length, presenting it as a disorder of over-nourishment in which *Ama* (immature) *Vata*, *Pitta* and *Kapha* combine with *Medas* and come to rest at the neck of the bladder- a mechanism similar to Charaka's own[8]. Where Sushruta differs is in the rigour of his prognosis: the ten *Kapha*-dominant forms of *Prameha* are held to be curable, the six *Pitta*-dominant forms manageable, and the four *Vata*-dominant forms, which he terms *Kshaudrameha* rather than *Madhumeha*, largely beyond treatment.

Vagbhata's two works, *Ashtanga Hrdaya* and *Ashtanga Sangraha*, explains the mechanism very similar to that of Charaka and Sushruta rather than to extend their thinking substantially[9,10]. Their principal contribution lies in naming, more clearly than either of the earlier texts manages on its own, the two distinct routes to *Madhumeha*- one arising from generalised tissue depletion, the other from obstruction. *Madhava Nidana*, composed considerably later, adds little to the original mechanism of disease, but establishes the six-part template- cause, prodrome, symptoms, mechanism, complication and prognosis- that subsequent Ayurvedic nosological writing continues[11]. Which we greatly follow today also.

Sharngadhara Samhita belongs to a different genre altogether, a formulary rather than a nosological treatise, and offers nothing directly on the causation of *Madhumeha*[12]. Its relevance here is with the fact that, Sharangadhara is credited with the earliest formal treatment of pulse diagnosis, within which *Prameha* is assigned a distinctive "beaded" pulse quality, and his appendix describes *Tanḍava roga* (a chorea-like involuntary movement disorder), during the search one case-based analysis has linked this to the non-ketotic hyperglycaemic chorea occasionally observed in poorly controlled diabetes[13].

Table 1: Classical texts and their contribution to the *Prameha/Madhumeha*

Text	Location	Contribution	Term for *Madhumeha*
Charaka Samhita	Su.17 (this passage); Ni.4; Ci.6	Two mechanisms distinguished: obstruction and depletion	*Madhumeha*
Sushruta Samhita	*Nidanasthana*	Over-nourishment framing; prognosis graded by *Dosha*	*Kshaudrameha*
Ashtanga Hrdaya/ Sangraha	*Nidanasthana*	Synthesis of Charaka and Sushruta; both mechanisms named explicitly	*Prameha* / *Madhumeha*
Madhava Nidana	*Prameha* chapter	Six-part disease-description template	*Prameha* / *Madhumeha*
Sharngadhara Samhita	Pulse chapter; appendix	Pulse diagnosis; chorea named as a complication	not a nosology text

Etiological Factors and Their Modern Comparison

Now we have reached a junction where we need to compare the concept mentioned in *Charka* with what is the mechanism described in modern science. This comparison will give us the strongest link which are similar in

both the mechanism and also will highlight the weak links in the mechanism, these could not be explained with much of the available evidences.

An excess of heavy, fatty food is understood today to produce the caloric surplus behind visceral and ectopic fat deposition[14]. Freshly harvested grain and newly made drink can reasonably be understood as high-glycaemic index products, which are capable in generating the repeated glucose and insulin surges associated with such foods. Sedentary comfort and the lack of exercise correspond closely to one of the most firmly established risk factors in the diabetes literature; a study comparing sedentary with physically active adults found fat-cell insulin sensitivity to be roughly tenfold lower among sedentary subjects, independent of body weight[15]. Sour and salty foods fit less neatly, bearing more directly on cardiovascular and renal risk than on glycaemic control as such. Weakest of all from a modern standpoint are the lack of mental exertion and of seasonal cleansing - the former connected only loosely to overall energy expenditure pointing towards a sedentary lifestyle, the latter do not have any clear biomedical parallel.

Table 2: Etiological items in Su. 17/78-79 and their modern correlates

Classical item	Modern correlate
Heavy, oily, sour, salty food in excess	Chronic caloric surplus; ectopic and visceral lipid deposition
Freshly harvested grain, newly made drink	High glycaemic-load carbohydrate; repeated glucose/insulin spikes
Excess sleep, sedentary comfort	Sedentary behaviour as an independent driver of adipose/muscle insulin resistance
Lack of exercise	Loss of exercise-driven, insulin-independent glucose uptake and oxidative capacity
Lack of mental effort	Weak/inferential link to overall energy expenditure
Neglect of seasonal cleansing	No clear modern equivalent

Avarana and Insulin Resistance at the Molecular Level

Now is the time to do the real comparison of both the mechanism at the molecular level. How closely these two concept blends in or otherwise. *Charaka's* claim is that *Kapha* and *Medas* accumulate and obstruct the channel through which *Vata* travels; *Vata* itself is not diminished, only its passage is blocked. A close resemblance to this in the modern physiology is described as- Fat accumulating within muscle raises diacylglycerol and ceramide, lipid species that activate protein kinase C and related enzymes; these enzymes add phosphate groups to serine residues on insulin receptor substrate-1, interfering with the tyrosine phosphorylation normally needed to carry the insulin signal forward to the enzymes governing glucose-transporter movement to the cell surface[16]. Biopsies from people with metabolic syndrome bear this out directly: excess serine phosphorylation appears at specific sites on insulin receptor substrate-1, even while insulin receptor numbers and total glucose-transporter levels remain essentially normal, blunting the muscle's response to insulin[17]. The analogies mentioned here are form various research publications found in indexed journals.

The signal itself, *Vata* or insulin, remains present in normal or even elevated amounts. What fails is not the signal but its movement along a functioning pathway. The substance responsible for the blockage, whether framed as *Kapha-Medas* or as diacylglycerol and ceramide, arises from the very same dietary and behavioural causes that set the disease process in motion. None of this should be read as claiming that *Kapha* and *Medas* simply are diacylglycerol and ceramide- they remain broad physiological categories rather than specific molecules- but the underlying logic of the classical account, *Avarana* (obstruction) rather than deficiency, matches what modern biochemistry has independently established for at least the muscle component of insulin resistance.

Dhatus (Tissue) Involvement and the Staging of Disease

As per Charka ten *Deha Datus* (body tissue) are involved in the pathogenesis of *Premeha*. These are- *Medas*, *Mamsa*, *Rasa*, *Rakta*, *Vasa*, *Majja*, *Lasika*, *Shukra*, *Ambu* and *Ojas*[2]. *Medas* and *Mamsa* receives most sustained attention in the text, and it is precisely these two tissues that modern medicine identifies as the starting point of metabolic disease- adipose tissue as the source of excess circulating fatty acids, muscle as the principal site both of glucose uptake and of the signalling block already described. *Rakta* corelates well onto glycated blood proteins such as HbA1c, and *Vasa* onto fatty deposits within liver and pancreas. The remaining tissues admit less specific parallels, such as the *Shukra* related to late complication like erectile disfunction mentioned as the late complications in diabetes etc.

The most clinically significant comparison which is also very useful in planning of treatment is the

staging of the disease. Charaka and Sushruta describe *Prameha*, if left untreated advances through three *Dosha*-dominant stages- *Kaphaja* curable; *Pittaja* manageable though not curable; and *Vataja* where *Madhumeha* is among its four subtypes is considered to be incurable. At this final stage the underlying mechanism itself shifts, from *Avarana* (obstruction) toward active *Dhatu Kshaya* (depletion), as *Vata* begins drawing *Ojas*, *Majja* and *Lasika* into the *Basti* [6,18].

This staging of the disease explained in classical texts resembles modern natural history of type 2 diabetes reasonably closely (Table 4). The *Kaphaja* stage resembles early insulin resistance and prediabetes, largely reversible through lifestyle change; the *Pittaja* stage resembles established, pharmacologically controlled diabetes, where control is achievable even as underlying beta-cell decline generally continues (probably the stage of insulin resistance); and the *Vataja* stage, can be considered as stage of depletion rather than excess, resembles advanced insulin-dependent diabetes with significant loss of beta-cell reserve. What gives this comparison weight is that the shift in mechanism the classical texts describe at the *Pitta*-to-*Vata* transition corresponds to an actual transition modern diabetology also recognises, from compensated resistance to decompensated, beta-cell-limited disease.

Table 3: *Dosha*-wise staged progression of *Prameha* and its modern correlate

Stage	Prognosis	Classical mechanism	Modern correlate
Kaphaja Prameha	Curable	Tissue excess; treatments for *Dosha* and tissue coincide	Early insulin resistance/ prediabetes
Pittaja Prameha	Manageable	Faster onset; *Medas* more deeply involved	Medically controlled type 2 diabetes
Vataja Prameha (incl. *Madhumeha*)	Incurable	Mechanism shifts to depletion of *Ojas*, *Majja*, *Lasika*	Advanced, insulin-requiring diabetes

Role of *Ojas* Depletion and Beta-Cell Decompensation

The passage where *Charaka* mentions, that the vitiated *Vata* carries the *Ojas* to the bladder, is quit significate, it must not be just considered as glucosuria vaguely. If we read between the lines and infer what *Charaka* might wanted to convey, this stage may be considered as the stage of transition which is progressive (if it's not managed properly). A closer analysis to the concept of two-stage decline of beta-cell function now recognised over the course of type 2 diabetes, an early compensatory stage in which beta-cells increase their insulin output to counter peripheral resistance[19], it matches the classical mention of *Ojas* being drawn upon yet still replenished - consistent with the intermittent symptoms described in *Shloka* 81. This is followed by a later stage in which sustained exposure to high glucose and free fatty acids damages the beta-cells directly, so that their function deteriorates steadily regardless of treatment[20]. This indicates the transition from resistance to absolute depletion of insulin, the mechanism commonly seen today with association of Obesity. This two-stage disease pathology fits both the wording of the *Shloka* mentioned in *Charka* and the classification of fully established *Madhumeha* as incurable, which makes far more sense as a description of largely irreversible beta-cell loss than as a description of sugar appearing in the urine, a finding that is in principle reversible. *Ojas* nonetheless remains a considerably broader construct than beta-cell reserve alone, and this correlation should not be extended beyond the specific point.

Complications: *Upadrava* and *Pidika*

The general complications of *Prameha*, *Charaka* lists thirst, diarrhoea, fever, a burning sensation, weakness, loss of appetite and indigestion, noting that carbuncles tend to appear once the disease reaches its chronic stage[2]. The passage in *Sutrasthana* 17 mentions that if the disease process is neglected, to give rise to seven distinct forms of *Piḍika*. A modern correlation to the complications is mentioned in Table 5. the symptoms like- burning sensation, body ache, weakness and tremor- can be corelated to diabetic peripheral neuropathy; excessive urinary discharge as nephropathy; and necrotizing carbuncles as diabetic foot ulceration and soft-tissue infection, consistent with the well-documented impairment of immune-cell function under sustained hyperglycaemia[21]. *Sharangadhara's* account of *Taṇḍava roga* adds a further, less frequently cited complication corresponding to hyperglycaemic chorea[13].

Table 4: Classical *Upadrava/Pidika* and their modern diabetic complication correlates

Classical term	Modern correlate
Burning sensation, body ache, weakness, tremor	Diabetic peripheral neuropathy
Excessive urinary discharge	Diabetic nephropathy
Thirst	Polydipsia from osmotic diuresis

Necrotising carbuncles (*Alaji*, *Vidradhi*)	Diabetic foot ulcer, cellulitis, soft-tissue infection
Fever	Recurrent infection from impaired immune function
Taṇḍava roga (Sharangadhara's appendix)	Non-ketotic hyperglycaemia-induced chorea

DISCUSSION

To understand the comparison mentioned above we need to go stage by stage. The causes named in the *Shloka* match established risk factors reasonably well. The obstruction described as *Avarana* has a close counterpart in the biochemistry of IRS-1 serine phosphorylation. The tissues singled out as most affected are the same tissues modern medicine treats as central. The three-stage progression tracks a genuine shift in mechanism rather than an arbitrary severity scale. The complications, checked against a dedicated modern analysis, correspond organ by organ to recognised diabetic sequelae.

Every comparison has its own limitations, there are both agreement and disagreement points in a review, if only the agreement points are taken for drawing a conclusion it will create a misleading picture. Many authors compare *Madhumeha* to diabetes mellitus, based on the terminology. In reality it reflects an incomplete understanding of diabetes's own pathogenesis, a strong and defendable correlation should be based on the comparison of the mechanism, rather than just observations like both the conditions can produce sweet tasting urine[5]. Many authors use the terms *Premeha* and *Madhumeha* as synonyms and sometimes *Premeha* is loosely denoted as *Madhumeha* in general, rather than specific, incurable subset mentioned in classical texts. This review was aimed at drawing a comparison between the concept mentioned in *Charka Sutrasthana* 17 especially understanding the mechanism of *Avarana* (Obstruction) in manifestation of *Madhumeha*.

Further the point of comparison was the route or the process of the disease and how much similarity are there between the concept which are mentioned in the classical texts. Based on the molecular model which is mentioned in the result section, it was observed that the *Avarana* Mechanism mentioned in *Sutrasthana* 17 is closely related to Type 2 Diabetes mellitus pathology, related to the metabolic conditions like obesity. Whereas the *Dhatu Kashaya* route mentioned elsewhere in the Charka, is more or less comparable to the insulin deficiency state leading to diabetes. In the present scenario it is quite evident that a long-standing Type 2 Diabetes which might have started as insulin resistance model can change to more serious insulin deficit model, the same pathology have been mentioned in *Charka* where it is mentioned that the symptoms are variable until the involvement of *Ojas* takes place; it means the stage before the *Ojas* involvement can be the stage of resistance and stage after the involvement of *Ojas* can be the state of Insulin deficiency.

The other limitation of the study is based on the methodology. Much of the published work linking specific *Dosha-dushya* categories to specific laboratory markers, including studies relating *Prakrti* (an individual's constitutional type) to HOMA-IR or inflammatory markers, comes from small, single-centre, observational designs rather than adequately powered prospective studies[22]. Such work is useful evidence that constitutional typing correlates statistically with metabolic risk; but it does not establish that a *Dosha* category is, at a molecular level, identical to a biomedical pathway. The comparison developed here is strongest, at *Avarana* level and weakest exactly where it is most holistic, at *Ojas* and *Cinta* (mental exertion), and it should be read as a means of generating testable hypotheses rather than an established equivalence.

This study has its own limitations, as it is a narrative review in design, no risk of bias instrument was applied, to all the biomedical studies referred. The data collection strategy is described in the methodology section. The biggest challenge is translating Sanskrit technical vocabulary always involves choices another reader might make differently. Future work might look in to measure IRS-1 phosphorylation status or HOMA-IR in patients classified by *Prameha* subtype rather than by *Prakrti* alone, test whether interventions aimed specifically at the *Avarana* mechanism alter insulin-signalling markers, and clarify further the textual relationship between the obstruction- and depletion-based accounts within *Charaka Samhita* itself.

CONCLUSION

A research work cannot be concluded, as it paves way for further research and study on the subject, or sometimes will act as a pathfinder for some other studies. The present review is just a beginning on understanding the mechanism mentioned in *Charaka Samhita Sutrasthana* 17, a chapter dedicated to the disease concerning the head and heart along with *Kashaya* of *Dhatus* in the body. In this chapter *Charka* has given the most specific account of metabolic condition arising due to excessive use of diet and inactivity leading to the vitiation of *Dosas* and *dhatus* which further obstruct the pathway of *Vata* leading to imbalance in *Ojas*, this chronological order fluctuates in the initial stages before settling done into a fixed pattern. It also reflects to the point that of these indicators are neglected then can cause severe

complication, indicated in the mention of *Piḍika.* When the *Sloka* s were mapped and read stage by stage and compared to the modern endocrinology, the disease sequence aligned at many points with precision like-with visceral-fat-driven insulin resistance, its molecular basis in lipid-driven interference with IRS-1 signalling, the staged decline from reversible prediabetes to irreversible beta-cell loss, and the organ-specific complications of established type 2 diabetes. This comparison holds good at the points where clear evidences are available and its weak at point where the analogy is generalised.

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