He, who understands Vidya and Avidya both together, attains to the nature of immortals through Vidya (Knowledge of ritualistic philosophy), having conquered death by Avidya (Ritualistic).

-Ishavasyopanishad
# Chapter II Disease Profile

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BACKGROUND OF PUBLISHED PAPERS:

Type II diabetes mellitus, also known as non-insulin-dependent diabetes mellitus (NIDDM), is the predominant form of diabetes. In many patients, the initial diagnosis of type II diabetes is delayed perhaps by as much as 10 years because symptoms are often absent or very mild during its early stages. Type II diabetes is particularly common among the elderly and in many minority populations. The risk for type II diabetes increases with obesity, a family history of diabetes, and in women, history of gestational diabetes. It is believed that physical health is associated with the seven favorable habits: sleeping 7–8 h, eating breakfast almost every day, avoiding eating between meals, maintaining a desirable weight with respect to height, participating in active sports, limiting alcohol intake, and avoiding smoking cigarettes. To reduce the burden of diabetes among people, it is imperative to increase current efforts in diabetes prevention, quality diabetes care, and patient education. New initiatives may also be required, such as aggressive campaigns to decrease the likelihood of developing diabetes, especially among youth. The development of culturally sensitive programs to facilitate weight reduction among people with diabetes, using a balanced diet and increased physical activity, is also a high public health priority.

Diabetes is clearly a growing public health threat. This update is consistent with earlier prediction of the epidemic nature of diabetes. Specifically for diabetes, much of the impact of the continuing increase in obesity will be manifested in future years because of the substantial delay between the onset of obesity and the subsequent development of diabetes. Public health strategies to limit this increase and address its impact are urgently needed.

Many patients with diabetes take complementary therapies or nutrition supplements and conventional medicines concomitantly. A recent article reported that patients with diabetes are 1.6 times more likely than patients without diabetes to use complementary and alternative medicines. There are wide variations in prevalence rate of diabetes mellitus in various parts of our country. Survey of large number of people from rural as well as urban population of India, reported that prevalence of diabetes and impaired fasting glucose (IFG) is lower in rural population compared to the urban.
The prevalence rate (percent) of diabetes mellitus for persons above the age of 25 years was 3.77%. The prevalence in males was 4.58% and in females it was 2.66%. Impaired fasting glucose was 2.82% in male and 2.78% in female. The maximum prevalence was observed in the age group of 56 to 65 in both males and females. Education, income, and occupation were associated with increased diabetes risk in unadjusted models. In baseline models adjusted for demographics, respondents with <12 years of education had 50% excess risk compared with those with more education, income, and occupation were no longer significantly associated with increased risk. Further adjustment minimized the significance of all associations. Time-dependent effects were consistently elevated for low education and male blue-collar occupation, but non-significant after full adjustment. Socioeconomic disadvantage, especially with low educational attainment, is a significant predictor of incident Type 2 diabetes, although associations were largely eliminated after covariate adjustment. Obesity and overweight appear to mediate these associations. The comparison of employment and income of working-age people with and without diabetes was assessed. Diabetic individuals without complications had incomes similar to those of non-diabetic individuals. A striking increase in the prevalence of obesity, as well as diabetes, was reported between the second and third National Heath and Nutrition Examination Surveys. Despite these difficulties, several recent studies have demonstrated the potential for moderate, sustained weight loss to substantially reduce the risk for incidence of type 2 diabetes. Cigarette smoking is an independent and modifiable risk factor for type 2 diabetes. Smoking cessation is associated with weight gain and a subsequent increase in risk of diabetes, but in the long term, the benefits of giving up smoking outweigh the adverse effects of early weight gain. An age-standardized (35–79 years) prevalence of known and newly detected diabetes was 20% (17–24%) in Europeans, 22% (18–26%) in African-Caribbeans, and 33% (25–41%) in Pakistanis. Marked changes in prevalence represent only small shifts in glucose distributions. Regression models showed that greater waist girth, lower height, and older age were independently related to plasma glucose levels, as was physical activity. Substituting BMI and waist-to-hip ratio revealed their powerful contribution.

Many patients with diabetes may take complementary therapies or nutrition supplements and conventional medicines concomitantly. Some of these combinations...
may lead to potentially harmful interactions\textsuperscript{11}. It is believed that greater attention needs to be paid to the broader systems of environment and culture and their interconnections to understand the use of complimentary and alternative medical therapies\textsuperscript{12}. A survey done under which the therapies used for diabetes included solitary prayer/spiritual practices (28\%), herbal remedies (7\%), commercial diets (6\%) and folk remedies (3\%). Excluding solitary prayer, only 20\% of respondents used complimentary and alternative medicine to treat diabetes\textsuperscript{13}. The diabetic education program also presents an excellent opportunity for all health care professionals, who are interested in diabetes care to lead the way in promoting the control of blood glucose, lipids, and blood pressure to other provider groups and their diabetic patients\textsuperscript{14}. The growing utilization of complementary and alternative medicine therapies represents one of the characteristic phenomena facing scientific medicine. Studies of the patient’s opinions and attitudes toward CAM therapies are scarce. Among doctors, it is widely considered that the use of CAM therapies is only linked to a particular social or cultural background. A cross-sectional study designed to evaluate the spontaneous use of CAM therapies. Almost 62\% (353) of participants make use of CAM therapies, a higher percentage than that reported in the U.S. (8\%) and Canada (37.3\%). Mexican patients who use CAM therapies prefer herbal remedies (94.2\%), while the remaining 5.8\% use other treatments. In Mexico the use of plants has a long historical tradition, while in the U.S. only 20\% of diabetic subjects use herbal medicine\textsuperscript{15}.

Type II diabetes is accompanied by many severe complications, such as blindness, renal failure, lower-limb amputations, cardiovascular disease and stroke. The study was conducted to determine the prevalence of chronic complications and associated factors in type 2 diabetes of 500 diabetic patients having age \( \geq 25 \) years. Of the 500 diabetic patients examined retinopathy was seen in 43\%, neuropathy in 39.6\% and foot ulcers in 4\%. Nephropathy was found in 20.2\%, and was significantly associated with hypertension. The prevalence of micro-vascular complications was higher in the group of patients with HbA1c \( \geq 8\% \) and was significantly related to duration of diabetes, hypertension and obesity. Hypertension was manifest in 64.6\% patients, 61\% had raised Body Mass Index and Waist Hip Ratio was more than normal in 88\% subjects. Macrovascular complications were encountered in 102 diabetic patients, with angina in 85 (17\%), heart attack in 25 (5\%) and stroke in 13 (2.6\%). The prevalence of diabetic
micro-vascular complications was higher in people with poor glycaemic control, longer duration of diabetes and associated hypertension and obesity\textsuperscript{16}. Time-dependent effects were consistently elevated for low education and male blue-collar occupation, but non-significant after full adjustment. Socioeconomic disadvantage, especially with low educational attainment, is a significant predictor of incident Type 2 diabetes, although associations were largely eliminated after covariate adjustment. Obesity and overweight appear to mediate these associations\textsuperscript{17}. The prevalence of diabetes in Canada is increasing. One of the surveys carried out in Canada illustrates that the proportion of the disease rose from 3.4\% in 1994/95 to an estimated 4.5\% in 2000/01. The rate for men increased to 4.8\% from 3.4\%, while the rate for women rose to 4.2\% from 3.3\%\textsuperscript{18}.

In the concern with clinical trials, it is suggested that one should rely only on randomized, clinical trials with clinically meaningful endpoints to determine which agent to be use for the treatment of type 2 diabetes. One could consider HbA\textsubscript{1c} levels a surrogate endpoint, since it is obviously not a direct measurement of diabetes-related complications. The oral glucose tolerance test not be routinely used to identify people with either diabetes or impaired glucose tolerance (IGT) has fueled considerable controversy regarding the importance of such testing in either a clinical or epidemiological context\textsuperscript{19}.

In one of the study the hypothesis was tested that HbA\textsubscript{1c} levels might be more sensitive than Fasting glucose concentrations in diagnosing diabetes in the 150 subjects. As expected, the proportion with elevated Fasting glucose and HbA\textsubscript{1c} values was higher. However, the hypothesis was still not supported, because 74\% had Fasting glucose concentrations 126 mg/dl and only 59\% had elevated HbA\textsubscript{1c} levels\textsuperscript{20}. It is come to know that there are insufficient data to determine accurately the relative contribution of the Fasting and post-prandial to HbA\textsubscript{1c}. It appears that FPG is somewhat better than post-prandial in predicting HbA\textsubscript{1c}, especially in type 2 diabetes. Absolute Fasting glucose is not a reliable tool for management of type 2 diabetes. Published data don’t support the conclusion that Fasting glucose is somewhat better than PPG in predicting HbA\textsubscript{1c}, especially in type 2 diabetes\textsuperscript{21}. In practice, fluctuations occur all the time and one effective way is to monitor the HbA1c, which gives the average blood glucose level of the preceding 2-3 months\textsuperscript{22}. It is come to know that lipoprotein concentrations were
directly correlated with LDL cholesterol and negatively correlated with triglyceride levels in diabetic patients. The HbA1c level was negatively associated with the polyunsaturated fat-to-saturated fat ratio (P:S ratio) of the diet and positively associated with the total level of fat intake adjusted for age and total energy intake. It provides further support to efforts promoting modifications in the intake of dietary fat. It is reported that fruit and green leafy vegetable consumption and vitamin C intake are negatively associated with HbA1c.

The study was conducted on some plants contains appreciable amount of elements like K, Ca, Cr, Mn, Cu and Zn, which are responsible for potentiating insulin action. In another study the exploration was made on the Anti-diabetic activity of medicinal plants and its relationship with their antioxidant property.
The idiom Ayurveda means the science of life. After thousands of years of the ancient Ayurvedic history, was transmitted from oral to the textual form written in Sanskrit, which has further grown into a medicinal science. The human and the plant evolution consist of the same basic matter (Panchamahabhoota). The material and nonmaterial properties dictate the medicinal and healing properties of the plants and other healing processes. It was meant essentially to promote health, rather than fight against disease. We can find so many of the herbs explained in the sutra form for so many diseases. Prameha is one of them and it was explained in a very broad manner.

There is an estimated 143 million people worldwide suffering from diabetes, i.e. almost five times more than what was the estimate before ten years. This number may probably be double by the year 2030. In spite of this much high rates of prevalence and increasing prevalence all over the world, scientists are still struggling to search an effective and harmless remedy. At present, approximately 80% of the population of third world countries (64% of total world population) is almost dependent on traditional therapies for their health care, a fact that has been substantiated by the WHO recommendation to include traditional medicines in the primary health-care.

Man always struggled with present and attempted for the better future and these can be achieved with a better perspective when the past and present experiences are revived and organized at proper time. Most of the traditional therapies are plant based and a large number of these plants are studied to test the claimed activity. In Bhavaprakash Nighantu there are more than thirty single plants have been described for the Pramehaghna activity. It is widely accepted that all the Ayurvedic medicine acts holistically and their action is described in a very broad manner. As Diabetes is a complex disorder having the chain of symptoms and affect many of the systems and organs in the body, it is considered that it requires a complex drug having complex chemical composition. Extensive clinical observation, intuition and insight, and interpretation based on the Tri-Dosha philosophy will further become the guidelines of research for complex diseases like Diabetes.
DEFINITION AND MEANING OF DIABETES

Diabetes mellitus is a clinical syndrome characterized by hyperglycemia due to absolute or relative deficiency of insulin. The condition may be accompanied by other biochemical disturbances and the presence of progressive tissue damage with macro-vascular and micro-vascular complications. It is a chronic metabolic disorder with multifactorial etiology.

The term “Diabetes” means the condition in which a large amount of urine is passed. The term “Mellitus” (Sweet) dates back to the time when the urine was tested and found to be sweet in taste.

DIABETES AND RELATION WITH PRAMEHA / MADHUMEHA

Diabetes mellitus strikingly resembles with Madhumeha, which is one of the twenty type of Prameha as described in various Ayurvedic texts. The patient who has turbid urine in excessive quantity along with increased frequency of urination is known as Prameha. In whomsoever the prodromal features of Prameha are seen and even if there is increase in urine output, it is consider the patient is suffering from Prameha. In Ayurvedic literature Madhumeha is described along with Prameha. It appears that Prameha is a group of urinary syndromes including Diabetes mellitus and Diabetes Insipidus. On the basis of their clinical manifestation 20 types of Prameha can be correlated with various clinical conditions. If Prameha is not well treated properly and continue for longer duration, in due course of time it will convert into Madhumeha (Diabetes mellitus). Considering the seriousness of the disease and its prognosis is also known as Mahagad or Maharoga. It is obvious that the description of Medhumeha is available in ancient treatise of Ayurveda even before modern scientists knew it.

REVIEW LITERATURE PRAMEHA / MADHUMEHA
HISTORICAL REVIEW

A Critical review of the history, from the primitive stage, to the new millennium assists, one to understand the future in a better way. History helps to reveal the widen facts and ideas of the concerned subject. Here an attempt has been made to compile the references on Diabetes as a part of Prameha found in Ayurvedic literature.
VEDIC PERIOD (5000 B.C. to 1500 B.C.):

In Vedic literature there is a reference of Prameha in Kaushik Sutra of Atharva Veda. Two terms have been used there: Aasrava and Prameha. Vedic commentators Sayana and Kesava have interpreted Aasrava as Mutratisara i.e. excessive urination. Whintey (1962) interpreted Aasrava as flux while Griffith as morbid flow. The word Prameha is used in Kautilya Arthashastra (321-296 B.C.) in the context of inducing Prameha. Leman has translated the meaning of Asrava as Diabetes Mellitus; Sayanacharyya has highlighted the Vata nature of this ailment.

SAMHITA PERIOD (2000 B.C. to 800 A.D.)

Charaka Samhita: In this ancient treatise of Ayurveda, Charaka has given a detailed description of the etiology, pathogenesis, symptomatology and complications in Nidanasthana, where as detailed explanations of treatment in Chikitsasthana. The aetio-pathogenesis of Madhumeha along with its complications is narrated in Sutrasthana. This is the unique contribution of this treatise.

Susruta Samhita: Acharya Susruta has given elaborate explanations of Prameha Nidan Panchaka in Nidanasthana. Especially he has described Pramehanivritti Lakshanas, i.e. how to detect that the patient is relieved from the disease. He has described the treatment in three different chapters under the heading of Prameha-Chikitsit, Prameha Pidaka Chikitsit and Madhumeha Chikitsit. He used 'Ksaudrameha' synonym to Madhumeha in Nidana 6th chapter. He typically mentioned the decoctions for each type of Prameha and mentioned the symptoms related to Sahaja and Apathyanimittaja Prameha.

Ashtang Hridaya: Description of Prameha Nidana is found in Nidanasthana and its treatment can be seen in Chikitsasthana. Acharya Vagbhata has described that Madhumeha occurs as a result of Vata Avarana or Dhatukshaya. He has described 'Dhatri Nisha' for the Chikitsa of Prameha. Vagbhata categorized the disease under the heading 'Mutraatipravrttija' and mentioned two types of Madhumeha i.e. Dhatukshayat and Avarita and added Sweda among the Dushyasangraha.

Bhela Samhita: In Nidanasthana, description of two types of Prameha is given i.e. Swakritija Prameha and Prakritija Prameha.
Harita Samhita: Acharya Harita has narrated Prameha as Papajanya Roga. He has enumerated types of Prameha with different nomenclature like Puya Prameha, Takra Prameha, Rasa Prameha, Ghrita Prameha etc.\(^3\)

Kashyapa Samhita: Acharya Kashyapa mentioned the symptoms of Pramehi child in Vedanadhyana\(^4\) and noted the disease as Chirakari.

MEDIEVAL PERIOD (800 A.D. to 1900 A.D.):

In this period, mainly commentaries were written, but most of their contents were only the collection of thoughts from previous authors.

Madhavakara: He collectively repeated the description of Charaka, Susruta and Vagbhata\(^5\).

Gayadasa: Gayadasa explained the Avilamutrata because of the presence of Dushya in it\(^6\).

Chakrapanidatta: Chakrapanidatta described the treatment of Prameha in his documentation Chakradatta.

Sharangdhara: Sharangdhara has described 20 types of Prameha in Poorvakhanda 7\(^{th}\) chapter 59-62.

Dalhana: In his commentary on Susruta Samhita, ‘Nibandha Samgraha’, opined that females do not suffer from Prameha.

Bhavamishra: He described Prameha and Madhumeha along with some new herbo-mineral preparations\(^7\).

Yogaratnakara: Prameha Chikista has been described vividly in Yogratnakara.\(^8\)

MODERN PERIOD:

Clinical description of polyuric states resembling Diabetes mellitus has been described in the Egyptian Papyrus (15\(^{th}\) Century B.C.). The period between 16\(^{th}\) and 18\(^{th}\) centuries was the “diagnostic period” when diabetes was recognized as a separate disease entity, while the 19\(^{th}\) century has been regarded as the “experimental period” when the role of pancreas was elucidated and biochemical abnormalities of diabetes were characterized. The following are the important landmarks in the modern history of Diabetes\(^9\).

1552 B.C. - Earliest known record of diabetes mentioned on 3\(^{rd}\) Dynasty Egyptian papyrus by physician Hesy-Ra, mentions polyuria as a symptom.
1st Cent. A.D. - Diabetes described by Areatus as the melting down of flesh & limbs into urine.

164 A.D - Greek Physician Galen of Pergamum mistakenly diagnoses diabetes as an ailment of the kidneys.

Up to 11th Cent. - The Latin word for honey, mellitus is added to the term diabetes.

16th Cent. - Paracelsus identifies diabetes as a serious general disorder.

Early 19th Cent. - First chemical tests developed to indicate and measure the presence of sugar in urine.

Late 1850s - French Physician, Priorry, advises diabetes patients to eat extra large quantities of sugar as treatment.

1870s - French Physician, Bouchardat, notices the disappearance of glycosuria in his diabetic patients during the rationing of food in Paris while under Siege by Germany during Franco-prussian war, formulates idea of individualized diets for diabetic patients.

19th Cent. - French researcher Claude Bernard studies working of pancreas and glycogen metabolism of the liver. I.V. Pavlov, discovers the links between the nervous system and gastric secretion, making an important contribution to science's knowledge of the physiology of the digestive system.

Late 19th Cent - Italian diabetes specialist, Catoni, isolate his patient under lock and key in order to get them to follow their diets.

1869 - Paul Langerhans, a German medical student announces in a dissertation that the pancreas contains two systems of cells one set secretes the normal pancreatic juice, the function of the other was unknown several years later, these cells are identified as Islets of Langerhans.

1889 - Oskar Minkowski and Joseph Von Mering at the university of Strasbourg, France, who initially removed the pancreas from a dog to determine the effect of an absent pancreas on digestion.

1908 - German scientist, Gerog Zuelzer develops the first injectable pancreatic extract to suppress glycosuria, however, there are extreme side effects to the treatment.

1921 - Insulin is discovered. A depancreatized dog is successfully treated with insulin.

Mid 1940s - Alfred Free and Hellen Murray invented the home diabetes test.

1940s - Link was made between diabetes and long-term complications.
1944 - Standard Insulin syringe is developed.
1955 - Oral drugs are introduced to help to lower blood glucose levels.
1959 - Two major types of diabetes are recognized: Type-1 (Insulin dependent) diabetes and Type-2 (Non-Insulin dependent) diabetes.
1960 - The purity of insulin is improved. Home testing for sugar levels in urine increases level of control for people with diabetes.
1966 - First pancreas transplantation performed at the University of Manitoba.
1970 - Blood glucose meters and Insulin pumps are developed. Laser therapy is used to help to slow or prevent blindness in some people with diabetes.
1983 - First biosynthetic human insulin is introduced.
1986 - Insulin pen delivery system is introduced.
1993 - Diabetes control and complication Trial (DCCT) results demonstrate that intensive therapy delays the onset and progression of long-term complications in individuals with type 1 DM.
1998 - The United Kingdom prospective Diabetes Study (UKPDS) results identify the importance of glucose control and blood pressure control in the delay and/or prevention of complications in type 2 diabetes.

Etymology of Prameha:

The word 'Prameha' consists of two sub-words. i.e. 'Pra' and 'Meha'. The word Meha is derived from the root "Mih Sechane" by adding 'Lue' Pratyaya to it "Mehati Sinchati Mutraretsani" which means to excrete. Rigveda mentioned this word first is Mehanadthanam Karanallium. Shayanacharya, the commentator of Rigveda has interpreted the word Mehana as Medhرا, which indicates to Shishna (Penis). In Sanskrit literature, 'Mih' is used to denote to make water, to wet, to emit semen. So this root 'Mih' is added to prefix 'Pra' the word becomes 'Prameha'.

प्रमेह = प्र + मिह = मेहति मूत्रवति इति अर्थ ।
= प्रकर्षण मेहति रोगे स प्रमेहः ।
= प्रकर्षण प्रौढ़े वार्तवार वा मेहति यो रोगे स प्रमेहः ।

Following the explanation it can be interpreted that the disease Prameha is the result of excessive diminution or excretion of something (Ati-pravritti). Acharya
Vagbhatta describes Prameha as frequent and copious urine with turbidity i.e. Prabhutavil Mutrata.

**Etymology of Madhumeha:**

The word Madhumeha consists of two words i.e. Madhu and Meha. The word Madhu is derived from the root ‘Manyante Visheshena Janati Jana Yasmin’. The root “Manjane” is applied by ‘Dha’ Adesha and it shows the similarity of urine in taste, color and appearance etc. In Sanskrit literature the word Madhu is used in various contexts like Pushparasa, Makarandah, Makshikam, Madhyam, Ksiram, Jalam, Madhurarasa etc.

\[
\text{मधुमेह} = \text{मधु} + \text{मेह}
\]

\[
= \text{मधु इव मेहति} \quad \text{रुमात कारणात मधु इव मेहति मधु सदृशं मेहति} \\
\text{अस्मात कारणात मधुमेह सज्जा} \\
\text{अर्जुदत्ता, वा. नि} 10
\]

In this way the etymology clearly indicates that the disease in which the excretion is having quality concordant like Madhu (honey) i.e. its color, taste, smell and consistency is called Madhumeha.

**Definition of Madhumeha:**

The word ‘Meha’ is mainly related to the excretions through urine. In this way Madhumeha is defined as the clinical entity in which patient voids the urine having concordance with Madhu i.e. having Kashaya and Madhura taste, Ruksha (dry) texture, color like honey and body acquires sweetness called Madhumeha.

However, Sushruta has used the term Ksaudrameha in place of Madhumeha. Kshaudra is nothing but variety of Madhu (honey), which is Kapila (tawny) in color. It undoubtedly resembles with Madhumeha. Further, he asserts that when all the Pramehas are ill-treated or neglected, they get converted into Madhumeha. He also especially emphasizes that the disease Prameha along with Pidaka and Upadrava should be termed as Madhumeha.

**Classification of Prameha:**

To properly understand of the disease and to formulate an effective treatment protocol, one should have a well-designed knowledge of a disease. It is found that the
classification is mentioned in various manners in Ayurvedic text, in which the most important is of Dosha predominance.

1. According to Dosha – (Clinico-pathological Classification):

In Ayurvedic Classics the Samhitakara has contributed twenty types of Prameha, out of which 10 are of Kaphaja, 6 of Pittaja and 4 belong to Vataja type.

**Table No. 3.1- Doshaja Classification of Prameha**

<table>
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<tr>
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<th>Susruta</th>
<th>Vagbhata</th>
<th>Madhava</th>
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<td>Kalameha</td>
<td>+</td>
<td>Amlameha</td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>Nilmeha</td>
<td>+</td>
<td>+</td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>Lohitameha</td>
<td>+</td>
<td>Shonitameha</td>
<td>Raktameha</td>
<td>Raktameha</td>
</tr>
<tr>
<td>Manjishtameha</td>
<td>+</td>
<td>+</td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>Haridrameha</td>
<td>+</td>
<td>+</td>
<td></td>
<td>+</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Vataja Meha</th>
<th>Charaka</th>
<th>Susruta</th>
<th>Vagbhata</th>
<th>Madhava</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vasameha</td>
<td>+</td>
<td>+</td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>Majjameha</td>
<td>+</td>
<td>Sarpimeha</td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>Hastimeha</td>
<td>+</td>
<td>+</td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>Madhumeha</td>
<td>+</td>
<td>Kshaudrameha</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>
The basic ideology behind such classification is the relation between Dosha, Dushya and their specific combination and in each subtype where specific pattern of urine is voided. According to Charaka, all these types are because of the specific qualities and combinations of Dosha with each other. But, the nomenclature is mainly based upon the predominance of one quality. Chakrapani also explained that the nomenclature is because of the close resembles of urine with particular quality (Guna) e.g. Shitameha, Shulkameha etc.

2. According to Sadhyasadhyatva (Prognostic Classification):

Prognosis is a predictable part of Chikitsa and a wise physician is always concerned with it. The success of treatment depends on an unbiased prognosis.

<table>
<thead>
<tr>
<th>Sadhya</th>
<th>Yapya</th>
<th>Asadhya</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kaphaja</td>
<td>Pittaja</td>
<td>Vataja</td>
</tr>
<tr>
<td>Sthula (Obese)</td>
<td>Usually not much obese</td>
<td>Krisha (Asthene)</td>
</tr>
<tr>
<td>Apathyanimittaja (Acquired)</td>
<td>Acquired</td>
<td>Sahaja (hereditary)</td>
</tr>
<tr>
<td>Early Stage</td>
<td>Acute Stage</td>
<td>Advanced Stage</td>
</tr>
<tr>
<td>Without complication</td>
<td>With Complication</td>
<td>With Complication</td>
</tr>
</tbody>
</table>

Classification of Madhumeha:

Clinico-pathological status of a disease has an invariable relation with physical constitution of the body in Madhumeha. This has to be taken into consideration when treatment is formulated. According to this, in Ayurveda, Madhumeha is of two types.

Sthula and Krisha:

The root cause of disease has enough importance for the prognosis and treatment of the disease. The occurrence of Madhumeha from this point of view is of two types. Sahaja (Hereditary) and Apathyanimittaja (Acquired) 

Beejadosha is one of the major causes behind Sahaja Prameha and considered to be a result of genetic origin; while describing its prognosis, Acharya Charaka has mentioned that the Prameha or Madhumeha occurs due to Beeja Dosha, which is incurable.

Apathyanimittaja type itself suggests its etiology, which occurs due to unplanned and vague dietary habits. The Samprapti-ghataka (contributing factors for disease origin)
has to be understood to know about the prognosis and treatment of the disease. As per the Samprapti, Apthyanimittaja Madhumeha is of two types.

According to Samprapti

**Avaranjanya and Dhatukshayajanya:**

In Avaranjanya Madhumeha, Kaphavardhaka Nidanasevana leads to Vata Avarana that makes Ojas Karshana at Basti level. The whole process in turn shows the symptoms of Mutrotpatti (urination) in the form of Madhura, Kashaya, and Ruksha Mutra, which is said to be a Madhumeha condition. In Dhatukshayajanya Madhumeha, Vataprakopa occurs due to Vatavardhaka Nidan and Madhuratwa of Oja is displaced by Kashaya Rasa, which is brought to the Basti leading to Madhuvat Mutratyaga ultimately leading to Madhumeha.

**According to Etiological factors:**

**Santarpanjanya and Apatarpanjanya:**

Santarpanjanya Madhumeha is due to intake of high nutritious diet, having excessive Kaphavardhaka properties. The excess intake of such substances will primarily lead to the vitiation of Kapha, Pitta, Meda and Mamasa, which in turn cause Madhumeha due to Avarana of Vata\(^5\). In Apatarpanajanya Prameha the main causative factors are consumption of the substances, which deplete the Dhatu and aggravate Vata. They act through vitiation of Vata, which again in turn leads to the manifestation of Madhumeha.

In a nutshell, Sahaja and Apathyanimittja are types of Madhumeha. In it the Krisha, Dhatukshayajanya and Apatarpanjanya can be correlated with under Sahaja Madhumeha. The Sthula, Avaranjanya and Santarpanjanya are considered to be under Apathyanimittaja Madhumeha.

**Nidana:**

An Ayurvedic classic elaborately describes the general etiological factors of Prameha and classifies into Sahaja and Apathyanimittja\(^5\).\(^7\)

**Sahaja:**

Due to certain defects in Stri and Pumbeeja (Ovum and Sperm), which are said to be Matru-pitru Beejadoshakrita, result in Sahaja Prameha. Acharya Charaka has mentioned that excessive indulgence of Madhura Rasa by parents is the chief cause of
changes and damages in the Beeja (Sperm and Ovum). Over indulgence of Madhura Rasa by mother during pregnancy is also likely to induce Prameha.

Charaka, while describing the prognosis of the Madhumeha, clearly highlights that the Kulaja Vikara is the main causative factor for the defect in the Beeja\(^5^\). Chakrapani opines father that mother or grand parents can cause it. It means the disease is inherited from generation to generation\(^5^\). Charaka narrated that Sahaja type of diseases can occur due to defect in Beeja, Beejabhaga or Beejabhagavayava\(^6^\). Charaka narrated that indulgence of Madhura Rasa by mother at the time of pregnancy causes Madhumeha and Sthaulya\(^6^\).

**Apathyanimittaja:**

**Etiological Factors of Prameha**

Asyasukham (Without control eating the substances as per wish), Swapnasukham (Excessive sleep, day and night without any specific time), Dadhini (Excessive indulgence in various preparation of curd), Gramya, Audaka, Anupa Mamsa i.e. meat of domestic, aquatic, wet land animals. Payamsi i.e. excessive use of milk and its preparation, Navannapanam (new grains and drinks), Guda Vaikrutam i.e. various preparation of sugar and jaggery and other substances, which increase Kapha, may cause prameha\(^5^\). Susruta adds Snigdha (unctuous), Medhya (fatty) and Drava (liquid) type of food are also among causative factors\(^5^\). According to Vagbhatta, the diet and activities, which increase Meda, Mutra and Kapha, are supposed to cause prameha\(^6^\). Charaka narrated etiological factors according to Dosha predominance in Nidana Sthana and common etiological factors in Chikitsasthana.

1. **Kaphaja Prameha Nidana\(^5^\):**

The etiological factors responsible for the manifestation of Kaphaja Prameha are like frequent and excessive intake of fresh corns like Hayanak, Yavaka, Chinaka, Uddalaka, Naishadha, Itkata, Mukundaka, Mahavrihi, Pramodaka and Sugandhaka. It may also happen because of the excessive intake of pulses like fresh Harenu and Masha with ghee and of the meat of domestic, marshy and aquatic animals. Frequent intake of vegetables, Tila, Palala, Pishtanna, Payasa (a type of milk preparation), Krishara, Vilepi and preparations of sugarcane also cause the same. Also an intake of milk, fresh wine, immature curd and curd which are mostly liquid, Sheeta and immature in nature are
responsible factors along with avoidance of unction and physical exercise and indulgence in sleep, bed rest and sedentary habits. Restoring to even such regimens produce more of Kapha, fat and urine.

2. Pittaja Prameha Nidana

The etiological factors help in the manifestation of Prameha due to Pitta are intake of Ushna, Amla, Lavana, Kshara and Katura Dravyas. Intake of food before the digestion of the previous meal, exposure to excessively hot sun, heat of the fire, physical exertion and anger and intake of mutually contradictory food articles are the other responsible factors.

Vataja Prameha Nidana

The etiological factors help in the manifestation of Prameha due to Vata are excessive intake of Dravyas having predominantly Kashaya, Katu, Tikta Rasa, Ruksha, Laghu and Sheeta Veerya. Excessive indulgence in sex and physical exercise, Excessive administration of Vamana, Virechana, Asthapana and Shirovirechana also cause Vatajanya Prameha. Resorting to suppression of the manifested urges, fasting, assault, exposure to sun, anxiety, grief. Excessive bloodletting, keeping awake at night and irregular posture of the body are the main manifesting causes for Prameha of Vata origin.

Specific Etiology of Madhumeha:

Charaka narrated specific etiological factors for Vata Prameha, which led to depletion of Dhatus. He described that the person indulging in food substances having Guru, Snigdha qualities and excessive indulgence of Amla and Lavana Rasa substances and Navannapana, excessive sleep, sitting in a same place for longer duration, avoiding exercise and thinking process and also not performing the Shodhana process in a proper time also becomes victim in it. These etiological factors are mainly vitiates Kapha and Pitta with Meda and Mamsa and obstructs the normal path of Vata (Avarana) lead to vitiation of Vata which in turn leads to Madhumeha. Charaka in Nidanasthana emphasized the etiological factors as follows:

(1) **Nidanvishesha:** These are the typical Nidanas that cause excessive quantity of deranged Shleshma, Meda and Mutra

(2) **Doshavishesha:** The Nida / etiological factors cause typical characteristic of Kapha i.e. more liquidity with excess quantity.
(3) **Dushyavishesha**: Etiological factors mainly affect the normal physiological qualities of Dhatus and transfer them into abnormal texture.

Acharya Susruta has narrated that untreated Prameha in its initial stage gets converted into Madhumeha and becomes incurable. According to Acharya Vagbhata, the urine of Madhumeha person will be simulating with that of Madhu. He has also mentioned two types of Vata vitiation; one is due to Dhatukshaya and second is of Margavarana.

**Poorvarupa**

Premonitory symptoms are very helpful to diagnose the disease at its early stage, which in turn help for good prognosis. Poorvarupa are a valuable signs and symptoms to predict the nature of disease and a way to check the full-blown symptoms by proper medicaments. As a matter of fact, premonitory symptoms are produced at the stage of Sthana Samshraya and it is one kind of warning to the person to stop the ingestion of causes of Prameha. As Madhumeha is classified under the Vata type of Prameha, Poorvarupa of Prameha can be taken as Poorvarupa of Madhumeha.

**Table No. 3.2- Poorvarupa of Prameha**

<table>
<thead>
<tr>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td>Kesheshu Jatilibhava</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Asya Madhurya</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Karapadadaha</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Karapada Suptata</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Mukha Talu Kantha Shosha</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Pipasa</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Alasya</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Kayemalam</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Kaya Chhidreshu Upadeha</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Paridaha Angeshu</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Suptata Angeshu</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Shatpada Pipilika Mutrabhisaranam</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Mutre cha Mutradosham</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>
Visra Sharira Gandha | + | + | + | - | -
Sarvakala Nidra | + | - | - | + | -
Sarvakala Tendra | + | + | - | + | -
Snigdha Gatrat | - | + | - | + | -
Pichhila and Guru Gatrat | - | + | - | - | -
Madhura Mutrata | - | + | - | - | -
Shukla Mutrata | - | + | - | + | -
Sada | - | + | - | + | -
Shwas | - | + | - | + | -
Keshanakhativriddi | + | + | + | - | -
Sheeta Priyata | + | - | + | + | -
Sweda | + | - | + | + | -
Dehe chikkanata | - | - | - | - | +

**Rupa**

According to Susruta, the person should be diagnosed as Pramehi when complete or a partial prodromal symptom of Prameha accompanied by polyuria gets manifested. Gayadasa opines on this assertion that in this ailment all prodromal symptoms gets converted into Rupa because of the specific nature of the disease i.e. Vyadhiprabhava.

**Table No. 3.3- Rupa of Prameha**

<table>
<thead>
<tr>
<th>Signs</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Jatilibhava Keseshu</td>
<td>1. Asyamadhurya</td>
</tr>
<tr>
<td>2. Satapipilikasu Sharirmutrabhi Saranam.</td>
<td>2. Karadaha</td>
</tr>
<tr>
<td>5. Ghanangata</td>
<td>5. Padasuptata</td>
</tr>
<tr>
<td>7. Angasaithilya</td>
<td>7. Pipasa</td>
</tr>
<tr>
<td>8. Snigdhagatrata</td>
<td>8. Alasya</td>
</tr>
</tbody>
</table>
Conversion of Sadhya Roga into Krichrasadhya or Asadhya is considered as commonly occurring in the untreated condition. Acharya Susruta explains that, if all the Prameha are not treated initially, they will gradually get converted to stage of Madhumeha. According to Samprapti, Vagbhata divides Madhumeha into two types. Madhumeha is included in Vataja type and if Vataprakopa occurs due to Sarvadhatukshaya, it is called Dhatukshayajanya Madhumeha; and if it manifests as result of Vatavarana, it is called Avaranjanya Madhumeha. According to Vagbhata two types of pathogenesis get precipitate because of the two types of etiological factors, which are Dhatukshayaja and Avaranjanya.\(^2\)

**Dhatukshayaja:**

It happens due to depletion of the vital Dhatus and its pathogenesis. Charaka explained the pathogenesis in Nidana Sthana that due to specific etiological factors Vata gets provoked. It draws out the vital Dhatus and carries them towards Basti resulting into Madhumeha.\(^3\) The provocation of Vata is because of its own etiological factors that cause diminution of Dhatus; so called Sampraptivisishta Anilatmaka Madhumeha. In this pathogenesis, genetic predisposition makes patient prone to Madhumeha.\(^4\) The Kshaya of vital Dhatus like Vasa, Majja, Lasika and Oja leads to Vataprakopa. This vitiated Vata further makes Ksharana of these Dhatus through Mutravaha Srotasa resulting in Vasameha, Majjameha, Hastimeha and Madhumeha respectively. The Kapha and Pitta depletion provokes Vata provocation and it leads to depletion of Dhatus like Vasa.\(^5\)

**Sahaja Prameha:**

Susruta mentioned that Sahaja Prameha precipitates because of defect in beeja and Charaka explains the same thing that Sahaja Madhumeha is a Kulaja Vikara, because of the defect in Beeja (Sperm / ovum).\(^6\) In such cases the patient is prone to Dhatukshaya because of above-mentioned factors.
SAMPRAPTI GHATAKAS: 78

Kapha:

This Dosha has the status of dominant-Dosha in the Meha Samprapti; the first vitiated Dosha is Kapha itself. Acharya Charaka while describing the causative factors stated ‘Kaphakrut cha Sarvam’ as it indicates the importance of this Doshadushti in Meha. Especially Sharirshaithilya is the result of Bahudrava Kapha and other resulted manifestations are like Atinidra, Tandra, Alasya etc. Kapha has also normal function like Sthiratva. Chakrapani commented upon the word Sthiratva, which means Ashaithilya.

Pitta:

Role of Pittadosha in Avaranajanya Madhumeha is due to Pittavardhaka diet. In Dhatukshayajanya Madhumeha, Vata is found in Kupitta state, which highlights the symptoms of Pittakshaya like Mandagni, Prabhahani etc; where as the symptoms like Kshudhadhikya, Atisweda etc. are of Pittavridhi and are found in Avaranajanya Meha. Rakta, Sweda, Lasika and Rasa are the seats of Pitta Dosha. So, when Pitta gets provoked, it undoubtedly causes the vitiation of above Dushyas. That's why the symptoms manifested are Swedavruddhi, Visra Shariragandha, Paridaha, Pipasa and Agnivaisyama indirectly.

Vata:

Vata Dosha is having special status as it is controlling all the major life activities along with the movements of Kapha and Pitta because of its properties like Gati and Yogavahi. In Madhumeha, the provocation of Vata occurs in two ways i.e. by Margavarodha and Dhatukshaya, which drag the vital Dhatus like Vasa, Majja, Lasika and Oja to Basti and results in Madhumeha. Thus, due to severe depletion of Dhatu, the symptoms that manifests are Karshya, Daurbalya, Angasuptata and Parisaransheela nature. Charaka narrated that Dhatugati is the function of normal Vata on this Chakrapani opined that the Gati is related with the transfer of Rasadi towards Poshya. Thus, these functions of Vata in digestion get hampered causing imbalance in Dhatu formation and their transformation in the body.

Dushya:

Nidana, Dosha and Dushyas are the three factors responsible for the manifestation of every disease. This happens only at the time of Anukulatwa of all these factors to
establish a disease. The 10 Dushyas described in Madhumeha are Rasa, Rakta, Mamsa, Meda, Majja, Shukra, Kleda, Vasa, Lasika and Oja. Out of these, Meda is common Dushya in all Prameha Samprapti. While considering the Poorvarupas at the Sthanasamshraya stage, Keshanakhativriddhi is also mentioned, which is considered as Malabhavas of Asthi Dhatu. Thus almost all the Dhatus are involved in this disease, which leads to either Asadhyaatwa or Krichrasadhyaatwa.

All Acharya narrated Dushya Sangraha and their involvement in the Samprapti, but Charaka specially enumerated a group and named it as a Dushvyvishesha.\(^9\) Again he mentioned them in Chikitsa Sthana. In Charaka, Kleda has been referred as Ambu. Susruta also narrated the Dushyas, but he typically mentioned them along with the Doshik type.\(^8\) but he commonly included Meda in each type. Only Vaghbata mentioned Sweda as a Dushya along with above dusyas\(^3\)

**Rasa:** This is one of the Dhatus mainly vitiates in Madhumeha because of its close resemblance with Kapha qualitatively. It is one of the seats of Kaphadosha and Pittadosha. So, if Kapha gets vitiate Rasa is also gets vitiated as mentioned by Vaghbata\(^8\) i.e. Rasoapi Shleshmavat.

**Rakta:** Rakta is not having much involvement in the precipitation of the Madhumeha but it mainly gets vitiated in Pittaja Prameha. In later stage Rakta also get vitiated prominently causing complications like Pidaka, Vidradhi, and Alasi.\(^5\)

**Mamsa:** This is one of the main Dushya described by Charaka, especially in Kaphaja Prameha and Avaranjanya Madhumeha. Mamsa and Kapha are having same qualities, which give strength to the body. When Mamsa gets vitiated, it loses its normal consistency and develops Shaithilya, which provides accumulation of morbid matter in between.

**Meda:** Meda vitiation is common and dominant Dushya in the Samprapti of Madhumeha. Kapha and Meda are having close resemblance in regard to functions as well as qualitative parameters. Both are getting vitiated more or less by same etiological factors. The normal function of Meda is to produce unctuousness in the body along with Dardhatva i.e. compactness. In Madhumeha, vitiation of Meda results by two way; one is of qualitative i.e. Abaddha which causes derangement in the structure of Meda, producing Shaithilya in the body, where as the other is of quantitative which is explained
as Bahu. In the Samprapti Nirmana, Meda is found to be in excess quantity, which is Aparipakva in nature.\textsuperscript{86} It obstructs the path of Vayu in combination with Kapha, which unnecessarily provokes Vata and results in Atiagni. So the patient eats more causing excessive deposition of Aparipakva Meda. This, in turn, causes severe depletion of the other Dhatus and produces various sign and symptoms\textsuperscript{87}.

\textbf{Majja:} Majja is not much vitiated in Madhumeha but its vitiation caused by Vata, results in Kshaya and produces the symptoms like, Alpashukrata, Parvabhedha, Asthistododa andAsthishunyata\textsuperscript{88}. Majja Dhatu is not vitiated to the maximum extent but Vata causes its Kshaya i.e. depletion.

\textbf{Shukra:} Shukra also get vitiated in the pathogenesis and produces symptoms like Daurbalya and Kruchchha-Vyavayata. Vata causes depletion of Shukradhatu and thereby causes Shukrameha. It also plays important role in the precipitation of Sahaja Prameha and occurs as a result of Beeja Dosha. Vyana and Apana are the causative factors for Shukra Dosha and Prameha.

\textbf{Oja:} Oja as Dushya mainly involved in Vataja Prameha i.e. Ojomeha (Madhumeha), provokes Vata due to its own etiological factors or due to Avarana\textsuperscript{89} and carries Oja towards Basti and excrete outside through urine. Ojakshaya manifests the symptoms like Gurugatrata, Nidra, Tandra\textsuperscript{90} and Daurbalya\textsuperscript{91}.

\textbf{Kleda:} Kleda means wetness, moisture, dampness, etc. and its physiology is mainly related with Mutra and Sweda along with Meda. Proper quantity of Kleda is important to maintain the unctuousness in between the tissues. As per this opinion Mutra and Sweda maintain the balance of Kleda. Especially Sweda holds it in the body and Mutra excrete it outside the body, as per the body condition and requirement. If this Kleda is vitiated, it directly affects the physiology of Mutra and Sweda and disrupts the assemblage of bodily elements causing Shaithilya. Thus, the symptoms manifest due to Kleda vitiation is Prabhumurtata, Swedavriddhi, Shaithilya,ourandhya and Avilamurtata. Kleda itself is an important Dushya in Prameha. It makes other Dushyas susceptible for the progression of the Samprapti. Kleda establishes the Abaddhatwa in the Dhatus, which creates Shareera Shaithilya. Kleda promotes analogy between Dosha and Dushya. The increased Kleda has the similarity with Bahudrava Shleshma and Bahu Abaddha Meda.
**Vasa:** Vasa is the unctuousness present in the Mamsa Dhatu and it is consider as the Upadhatu of Mamsa. In Prameha Mamsa is one of the main Dushya, so in term Vasa too get vitiated.

**Lasika:** There is vitiated condition of Vasa and Lasika described in Prameha. The Dushti of Lasika is described in Hastimeha. Vitiated Vata vitiates Lasika, which results ultimately in Lasikameha.

**Sweda:** Vagbhata has separately mentioned Sweda as Dushya. Sweda is mainly related to Meda and Kleda. Thus the Swedovaha Srotodushti occurs due to vitiation of Kleda and Meda. Sweda gets disturbed resulting in manifestations like Swedavruddhi, Daurgandhya, Picchilagatrata, Snigdhagatrata etc. Susruta has mentioned that in Madhumeha Sweda becomes Sweet in nature.\(^2\)

**Srotas:**

Madhumeha is mainly of systemic consideration where there is involvement of each and every constituent of the body. The reference is mainly found to be of Mutravaha Srotodushti, but inherently the symptomatology highlights the involvement of Medovaha, Mamsavaha, Swedavaha and Udakavaha Srotasa, too. Prabhuta-avila Mutrata is a result of Mutravaha Srotodushti where as Poorvarupa of Prameha like, Kaye Malam, Snigdha Gatruta, Pichhila Gatruta is manifestations of Medovaha Srotodushti. Udakavaha Srotodushti produces symptoms like Pipasa, Mukha-talu-kantha Shosha. Sharavika, Kachhapika etc. Pidakas gets manifested when Mamsavaha Srotodushti occurs.

In the Dosha-dushya Sammurchhana two types of Srotodushti can be presented like Atipravrutti and Vimargagaman.

**Agni and Ama:**

No direct reference related to the Agni can be found in Madhumeha but both Agnimandya and Tikshnagni situation are present in the pathogenesis. Ama is considered to be the main thing behind this pathogenesis, which is Aparipakva in nature. Aparipakva condition of Dhatu is related with Ama mainly according to Susruta.\(^3\)

**Upadrava**

The term Upadrava is applied to a disease, which has taken place in the Samprapti Ghatakas of a born disease and can be cured only if the original disease is treated.
successfully. Acharya Charaka enumerated the general complications whereas Acharya Susruta and Vagbhata described it in terms of the Dosha predominance.

1. General Complications

These are like Trishna, Atisara, Daha, Daurbalya, Arochaka, Avipaka, Putimamsa Pidaka, Alaji, Vidradhi etc.

2. Specific Complications:

a. Kaphaja Meha\textsuperscript{95} - Makshikopasarpanam, Alasya, Mamsopachaya, Pratishyaya, Shaithilya, Arochaka, Avipaka, Kaphapraseka, Chhardi, Nidra, Kasa and Shwasa.

b. Pittaja Meha\textsuperscript{96} - Vrushanayorvadaranam, Bastibheda, Medhra Toda, Hridshula, Amlika, Jwara, Atisara, Arochaka, Vamathu, Paridhumayanam, Daha, Murchha, Pipasa, Nidranasha, Panduroga, Pittavidmutranetratva and Vidbheda.

c. Vataja Meha\textsuperscript{97} - Hridgraha, Laulya, Anidra, Stambha, Kampa, Shula, Baddha Purishatva and Shosha, Kasa, Shwasa.

Complications related to Madhumeha:

Charaka mentioned the ‘Sapta Pidaka’ as a complication arising, due to the negligence of the Madhumeha\textsuperscript{98}, while Susruta mentioned that Madhumeha along with Pidaka is Asadhya. He quoted that these Pidaka caused by the Tridosha, vitiated Meda and Vasa.\textsuperscript{99} Susruta and Vagbhata mentioned 10 Pidaka while Charaka mentioned only seven Pidaka. These are also follows-

Table No. 3.6- Upadrava Related with Prameha

<table>
<thead>
<tr>
<th>Sr. No.</th>
<th>Pidaka</th>
<th>Charaka</th>
<th>Susruta</th>
<th>Vagbhata</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Sharavika</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>2</td>
<td>Kacchapika</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>3</td>
<td>Jalini</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>4</td>
<td>Vinata</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>5</td>
<td>Alaji</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>6</td>
<td>Masurika</td>
<td>-</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>7</td>
<td>Sarshapi</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>8</td>
<td>Putrini</td>
<td>-</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>9</td>
<td>Vidarika</td>
<td>-</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>10</td>
<td>Vidradhika</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>
Sadhyasadhyata:

All Acharyas has give the concept of prognosis in the case of Prameha,
Kaphaja Prameha -Sadhya
Pittaja Prameha -Yapya
Vataja Prameha -Asadhya (Dhatukshayajanya, and if due to Avarana -Krichrasadhya).

Charaka illustrated the prognosis of this disease by considering the presence or absence of Poorvarupas. Kaphaja Meha with Poorvarupa is considered Krichrasadhya while those associated with Pittaja Meha are mentioned as Pratyakhyeya. Chakrapani says that the presence of Poorvarupa is a sign of incurability; this he says as a general rule and can be applied to any disease. Here in the context of Prameha, the presence of all or few Poorvarupas like Visrasharirgandham can be considered as Asadhyatva.

The second concept of prognosis is related with Medodushti. If the Medodushti is to a lesser extent, then the disease can be easily cured. So it is important to consider the gradation of Poorvarupas as well as the extent of Medodushti for having a concept of prognosis in Meha.

Arishta Lakshana:

Charaka mentions the following two features as Arishta Lakshana i.e. the signs of incurability or indication of ensured death\(^{100}\).

- A person whose body attracts the flies even after the bath is sure to die due to Prameha.
- The person who drinks various kinds of oils and ghee or other unctuous preparations with Chandala in his dreams may die of Prameha in future.

CHIKITSA (TREATMENT):

Madhumeha is a systemic disorder, which involves various body constituents, affecting the normal physiology of body organs. Thus, to concentrate on the treatment modalities, it is prime concern to think about each and every factor involved in the pathogenesis, disease severity and associated complications in due regard to provide better management. Chikitsasutra (principles of treatments) and Chikitsa (Proper Management) are the two divisions, where the concepts and methods are different in different conditions, considering the Vyadhi Swabhava and patient.
Charaka, Susruta and Vagbhata consider that the body constitution and strength of
the patient must be assessed, when dealing with the management of Prameha. Charaka
considers two types of patients; one is with stout body structure having strength
(Balawan) and the other one, which is without strength and Krisha in nature. Susruta also
says that Sahaja Meha Rogi is Krisha and Apathyanimitta Rogi is Sthula. After
considering above factors two types of management emphasized as:
1. Samshodhana Chikitsa (Elimination Therapy)
2. Samshaman Chikitsa (Normalizing Therapy)

The other parameters are also important to choose the therapy, which includes
- Nidanaparivarjana
- Treatment as per Dosha
- Treatment as per Dushya
- Treatment as per Mala
- Treatment as per Complications

It is advisable that the patient of Prameha once diagnosed should be treated at the
earliest possible to avoid complications. It is also important to understand that there is a
systemic Agnimandya and constant derangement of Ojus. It is important to maintain the
status of Agnibala and Ojus of the patient, which becomes the main theme of the
treatment.

**Nidanaparivarjanam**-

This is the prime treatment principle narrated by every Acharya before describing
the treatment of every disease. Charaka enumerated that one should avoid the etiological
factors, which are causing the disease Prameha and considered it as the prime treatment.

**Treatment According to Body Constitution**-

In Krisha patient it is necessary to use such a foods, which are increasing the
strength of patient without the vitiation of Dosha. Later, after achieving the proper
strength a mild purification measures in the form of Shodhan Chikitsa can be applicable.
In Sthula patient the application of Apatarpana Chikitsa along with powerful purification
measures are most suitable.
**Treatment According to Dosha Predominance:**

Though the disease is of Tridosha predominant, but individual Dosha consideration for the treatment is important for good prognosis. This includes Samshodhan Chikitsa, where elimination of vitiated Doshas performed with the process of Vamana, Virechana and other allied therapies depends upon the condition and its utility. Samshaman Chikitsa is also necessary for proper management of the diseases. The selection of the therapies and allied medicines must correlate with the vitiation of Doshas and their severity.

**Treatment According to Dushya Predominant**

The main Dushya involved in the pathogenesis are Rasa, Meda, Mamsa and Kleda. They are closely related with each other because of same the qualities and same etiological factors, so the treatment principles are more or less same to alleviate them.

**Treatment According to Maladusti**

The selection of proper therapy also considers the manifestation of Mala involved. The Dushti of Dushyas like Sweda manifests in large extent in Madhumeha, so the treatment like Lepa, Jalavaseka, Udvartana can be applicable. In Dushti of Mutra a common Pramehahara therapies must be applied. In the Dushti of Purisha, mild purgatives and Basti are essential depends upon the requirement.

**Treatment According to Complications:**

If there is complication resulted in the patients then it should be necessary to use multiple treatment modalities for its management. The complicate measures mostly related with Pidaka. In this condition Vranaropana, Udvartana and Parisechana process are applicable. Thus the proper combination of above treatment modalities according to the consideration of each and every factor may prove beneficial for the patient of Madhumeha.

There are various compound formulations and their mode of administration is described in the text. The list is very big but some of them can be like

- **Swarasa**: Amalaki, Haridra, Nimbapatra, Bilwapatra, Guduchi
- **Kwatha**: Vidangadi, Phalatrikadi, Mustadi, Manjishtadi, Pathadi
- **Churna**: Triphaladi, Mustadi, Gokshuradi, Arkadi
- **Gutika**: Chandraprabha, Pramehantak Vati
Gugglu: Gokshuradi Guggul

Modaka: Kastur Modaka

Avaleha: Kushavleha, Bangavleha

Paka: Pugapaka, Ashwagandhadi Paka, Draksha Paka

Asava-Arishta: Lodhrasava, Dantyasava, Madhukasava, Devadarvyadiarishta.

Ghrita: Dhanvantar Ghrita, Trikantakadi Ghrita, Dadimadi Ghrita.


There are also various single herbal drugs had been described as Pramehahara in Bhavaprakasha. some of them like:

List of some single herbal drugs acts as Pramehaghna Drugs

1. Arkapushpi
2. Amalaki
3. Atibala
4. Brahmi
5. Bakuchi
6. Bhurjapatra
7. Devadaru
8. Devadali
9. Dhava
10. Gojihwa
11. Guggulu
12. Guduchi
13. Haridra
14. Haritaki
15. Kakamachi
16. Kampillaka
17. Kutki
18. Katphala
19. Karanjaphala
20. Karanji
21. Katabhi
22. Kadaliphala (Pakwa)
23. Murva
24. Meshashringi (Phala)
25. Moksha
26. Manjishtha
27. Mahanimba
28. Nimba
29. Pippali
30. Pashanabheda
31. Suvarchala
32. Sarjaka
33. Shaivala
34. Tinisha
35. Triphala
36. Trikatu
37. Tindukaphala
38. Varahikanda

Bhavaprakasha Nighantu - Vatadi Varga -67
Bhavaprakasha Nighantu - Amradi Varga-34
Bhavaprakasha Nighantu - Guduchyadi Varga -245
Bhavaprakasha Nighantu - Guduchyadi Varga -254
Bhavaprakasha Nighantu - Vatadi Varga -69
Bhavaprakasha Nighantu - Hareetkyadi Varga -191
Bhavaprakasha Nighantu - Guduchyadi -99
Bhavaprakasha Nighantu - Hareetkyadi Varga -96
Bhavaprakasha Nighantu - Hareetkyadi Varga -57
Bhavaprakasha Nighantu - Hareetkyadi Varga -185
Bhavaprakasha Nighantu - Guduchyadi Varga -286
Bhavaprakasha Nighantu - Vatadi Varga -20
Bhavaprakasha Nighantu - Pushpadi Varga-21
Bhavaprakasha Nighantu - Vatadi Varga -76
Bhavaprakasha Nighantu - Hareetkyadi Varga -43
Bhavaprakasha Nighantu - Hareetkyadi Varga -63
Bhavaprakasha Nighantu - Amradi Varga-65
Bhavaprakasha Nighantu - Guduchyadi Varga -179

PATHYA-APATHYA

Pathya is having a key role in the management of Madhumeha, so before stepping to manage it should be necessary to consider for its implementation. These are classified as follows:

Pathya:

Aahara: Jeerna Shali, Shashtika, Kodrava, Yava, Godhuma, Uddalaka, and Shyamaka in Shuk Dhanya, where as Chanaka, Adhaki, Kulattha, Mudga in Shimbi Dhanya. The leafy vegetables predominant of Tikta-Kashaya Rasa like Patola, Karvellaka, Shigru, etc. under Shaka Varga should be selected. The Phala Varga includes Jambu, Dadima, Shringataka, Amalaki, Kapittha, Tinduka, Kharjura, Kalinga, etc. Vishkira Mamsa like Pratuda, and Jangala Mamsa are also useful in the disease. Taila Varga includes Danti, Ingudi, Sarshapa and Atasi, becomes the drug of choice. In Udaka
Varga, Sarodaka, Kushodaka, Madhudaka and in Kritanna Varga, Apupa, Saktu, Yavodana, Vatya, Yusha should be selected. The other category includes Madhu, Hingu, Saindhava, Maricha, Lashuna, etc are useful for the management of the disease.

Vihara: It is advised to the patient for walks, traveling on elephant, horse and different plays in the form of martial arts are useful and roaming in different places without chappal and umbrella is also beneficial.

Apathya:

Aahara: Jala, Milk, Ghee, Oils, Curd, Sugar, different types of rice preparations, Anupa, Gramya and Audaka Mamsa, Ikshurasa, Pishtanna, Navanna must be avoided.

Vihara: Ek-sthana Asana, Divaswapa, Dhoompana, Sweda, Raktamoksha, Mutravega Dharana.

DIABETES MELLITUS IN MODERN MEDICINE

Diabetes mellitus (DM) comprises a group of common metabolic disorders. Several distinct types of DM exist and are caused by a complex interaction of genetics, environmental factors, and lifestyle choices. Depending on the etiology of the DM, factors contributing to hyperglycemia may include reduced insulin secretion, decreased glucose usage, and increased glucose production. The metabolic dys-regulation associated with DM causes secondary patho-physiologic changes in multiple organ systems that impose a tremendous burden on the individual with diabetes and on the health care system. DM is the leading cause of end-stage renal disease, nontraumatic lower extremity amputations, and adult blindness. With an increasing incidence worldwide, DM will likely continue to be a leading cause of morbidity and mortality for the foreseeable future.

CLASSIFICATION

Recent advances in the understanding of the etiology and pathogenesis of diabetes have led to a revised classification (Figure No 3.1). Although all forms of DM are characterized by hyperglycemia, the pathogenic mechanisms by which hyperglycemia arises differ widely. Some forms of DM are characterized by an absolute insulin deficiency or a genetic defect leading to defective insulin secretion, whereas other forms share insulin resistance as their underlying etiology. Recent changes in classification
reflect an effort to classify DM on the basis of the pathogenic process that leads to hyperglycemia.

**Figure 3.1** Spectrum of glucose homeostasis and diabetes.

<table>
<thead>
<tr>
<th>Types of Diabetes</th>
<th>Normal Glucose Tolerance</th>
<th>Impaired Fasting Glucose or Impaired Glucose Tolerance</th>
<th>Hyperglycemia</th>
<th>Diabetes Mellitus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1</td>
<td></td>
<td></td>
<td></td>
<td>Not Insulin</td>
</tr>
<tr>
<td>Type 2</td>
<td></td>
<td></td>
<td></td>
<td>Insulin Requires</td>
</tr>
<tr>
<td>Other Specific</td>
<td></td>
<td></td>
<td></td>
<td>Control</td>
</tr>
<tr>
<td>Types</td>
<td></td>
<td></td>
<td></td>
<td>Insulin Requires</td>
</tr>
<tr>
<td>Gestational</td>
<td></td>
<td></td>
<td></td>
<td>Survival</td>
</tr>
<tr>
<td>Diabetes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time Years</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FPG (mg/dL)</td>
<td>&lt; 110</td>
<td>110-125</td>
<td>≥ 126</td>
<td></td>
</tr>
<tr>
<td>2hr PG mg/dL</td>
<td>&lt; 140</td>
<td>140-199</td>
<td>≥ 200</td>
<td></td>
</tr>
</tbody>
</table>

Type 2 DM is a heterogeneous group of disorders usually characterized by variable degrees of insulin resistance, impaired insulin secretion, and increased glucose production. Distinct genetic and metabolic defects in insulin action and/or secretion give rise to hyperglycemia in type 2 DM.

**EPIDEMIOLOGY**

The worldwide prevalence of DM has risen dramatically over the past two decades. It is projected that the number of individuals with DM will continue to increase in the near future. The findings, based on national epidemiological data, include individuals with a diagnosis of DM and those with undiagnosed DM (based on identical diagnostic criteria). Likewise, prevalence rates of impaired fasting glucose (IFG) increased from 6.5% to 9.7% over the same period. Although the prevalence of both type 1 and type 2 DM is increasing worldwide, the prevalence of type 2 DM is expected to rise more rapidly in the future because of increasing obesity and reduced activity levels.
There is considerable geographic variation in the incidence of both type 1 and type 2 DM. The prevalence of type 2 DM and its harbinger, impaired glucose tolerance, is highest in certain Pacific islands, intermediate in countries such as India and the United States, and relatively low in Russia and China. This variability is likely due to both genetic and environmental factors.

**DIAGNOSIS**

Revised criteria for diagnosing DM have been issued by consensus panels of experts from the National Diabetes Data Group and the World Health Organization. Symptoms of diabetes plus random blood glucose concentration ≥11.1 mmol/L (200 mg/dL) or Fasting plasma glucose ≥7.0 mmol/L (126 mg/dL) or Two-hour plasma glucose ≥11.1 mmol/L (200 mg/dL) is consider as diabetic one.

Some investigators have advocated the hemoglobin A1c (HbA1c) as a diagnostic test for DM. Though there is a strong correlation between elevations in the plasma glucose and the HbA1c, the relationship between the FPG and the HbA1c in individuals with normal glucose tolerance or mild glucose intolerance is less clear, and the test is not universally standardized or available.

**INSULIN ACTION**

Once insulin is secreted into the portal vein, ~50% is removed and degraded by the liver. Unextracted insulin enters the systemic circulation and binds to its receptor in target sites. The insulin receptor belongs to the tyrosine kinase class of membrane-bound receptors. Insulin binding to the receptor stimulates intrinsic tyrosine kinase activity, leading to receptor autophosphorylation and the recruitment of intracellular signaling molecules, such as insulin receptor substrates (IRS) 1 and 2 (Figure No 3.2).

**Figure 3.2** Insulin signal transduction pathway.
These and other adaptor proteins initiate a complex cascade of phosphorylation and dephosphorylation reactions, ultimately resulting in the widespread metabolic and mitogenic effects of insulin.

Glucose homeostasis reflects a precise balance between hepatic glucose production and peripheral glucose uptake and utilization. Insulin is the most important regulator of this metabolic equilibrium, but the effects of other pathways including neural input, metabolic signals, and hormones (e.g., glucagon) result in integrated control of glucose supply and utilization. The major portion of postprandial glucose is utilized by skeletal muscle. Other tissues, most notably the brain, utilize glucose in an insulin-independent fashion.

**PATHOGENESIS - TYPE 2 DM**

Type 2 DM is a heterogeneous disorder with a complex etiology that develops in response to genetic and environmental influences. Central to the development of type 2 DM are insulin resistance and abnormal insulin secretion. Although controversy remains regarding the primary defect, most studies support the view that insulin resistance precedes insulin secretory defects.

**Pathophysiology** - Type 2 DM is characterized by three pathophysiologic abnormalities:

- Impaired insulin secretion,
- Peripheral insulin resistance, and
- Excessive hepatic glucose production.

Obesity, particularly visceral or central, is very common in type 2 DM. Insulin resistance associated with obesity augments the genetically determined insulin resistance of type 2 DM. In the early stages of the disorder, glucose tolerance remains normal, despite insulin resistance. As insulin resistance and compensatory hyper-insulinemia progress, the pancreatic islets become unable to sustain the hyper-insulinemic state. A further decline in insulin secretion and an increase in hepatic glucose production lead to overt diabetes with fasting hyperglycemia. Ultimately, beta cell failure may ensue.

**Prevention**

Because type 2 DM is preceded by a period of impaired glucose tolerance, a number of life-style modifications and pharmacologic agents have been suggested to prevent or delay its onset. Individuals with a strong family history or those at high risk...
for developing DM should be strongly encouraged to maintain a normal body mass index and to engage in regular physical activity. Beyond this general advice, however, there are no specific interventions proven to prevent type 2 DM.

COMPLICATIONS OF DM

Acute Complications

Diabetic ketoacidosis and non-ketotic hyper-osmolar state are acute complications of diabetes. Diabetic ketoacidosis is seen primarily in individuals with type 1 DM, and non-ketotic hyper-osmolar state is seen in individuals with type 2 DM. Both disorders are associated with absolute or relative insulin deficiency, volume depletion, and altered mental status.

Chronic Complications

The chronic complications of DM affect many organ systems and are responsible for the majority of morbidity and mortality associated with the disease. Chronic complications can be divided into vascular and nonvascular complications.

Microvascular-

- Eye disease- Retinopathy, Macular edema, Cataracts, Glaucoma
- Neuropathy- Autonomic, Sensory and Motor (mono- and poly-neuropathy)
- Nephropathy
- Macrovascular-
  - Coronary artery disease
  - Peripheral vascular disease
  - Cerebrovascular disease
- Other
  - Gastrointestinal (Gastroparesis, Diarrhea)
  - Genitourinary (Uropathy / Sexual dysfunction)
  - Dermatological

EDUCATION OF THE PATIENT, NUTRITION AND EXERCISE

Patient participation is an essential component of comprehensive diabetes care. The patient with type 1 or type 2 DM should receive education about nutrition, exercise, care of diabetes during illness, and medications to lower the plasma glucose. Along with improved compliance, patient education allows individuals with DM to assume greater
responsibility for their care. Patient education should be viewed as a continuing process with regular visits for reinforcement; it should not be a process that is completed after one or two visits to a nurse educator or nutritionist.

**TREATMENT**

**General Aspects**

The goals of therapy for type 2 DM are to improve glycemic control with near normalization of the HbA1c. Attention to the treatment of conditions associated with type 2 DM (obesity, hypertension, dyslipidemia, cardiovascular disease) and detection /management of DM-related complications. DM-specific complications may be present in up to 20 to 50% of individuals with newly diagnosed type 2 DM. Reduction in cardiovascular risk is of paramount importance as this is the leading cause of mortality in these individuals.

Diabetes management should begin with MNT (discussed above). An exercise regimen to increase insulin sensitivity and promote weight loss should also be instituted. After MNT and increased physical activity have been instituted, glycemic control should be reassessed; if the patient's glycemic target is not achieved after 3 to 4 weeks of MNT, pharmacologic therapy is indicated.

**Glucose-Lowering Agents:** Recent advances in the therapy of type 2 DM have generated considerable enthusiasm for oral glucose-lowering agents that target different pathophysiologic processes in type 2 DM. Based on their mechanisms of action, oral glucose-lowering agents are subdivided into agents that increase insulin secretion, reduce glucose production, or increase insulin sensitivity.

**Insulin Secretagogues:** Insulin secretagogues stimulate insulin secretion by interacting with the ATP-sensitive potassium channel on the beta cell. These drugs are most effective in individuals with type 2 DM of relatively recent onset (<5 years), who have endogenous insulin production and tend to be obese.

**Biguanides:** Metformin is representative of this class of agents. It reduces hepatic glucose production through an undefined mechanism and may improve peripheral glucose utilization slightly. Metformin reduces fasting plasma glucose and insulin levels, improves the lipid profile, and promotes modest weight loss. The initial starting dose of 500 mg once or twice a day can be increased to 850 mg tid or 1000 mg bid.
**α-Glucosidase Inhibitors**: α-Glucosidase inhibitors (acarbose and miglitol) reduce postprandial hyperglycemia by delaying glucose absorption; they do not affect glucose utilization or insulin secretion. Postprandial hyperglycemia, secondary to impaired hepatic and peripheral glucose disposal, contributes significantly to the hyperglycemic state in type 2 DM.

**Thiazolidinediones**: Thiazolidinediones represent a new class of agents that reduce insulin resistance. These drugs bind to a nuclear receptor that regulates gene transcription. Thiazolidinediones reduce the fasting plasma glucose by improving peripheral glucose utilization and insulin sensitivity. Circulating insulin levels decrease with use of the thiazolidinediones, indicating a reduction in insulin resistance.

**Insulin Therapy in Type 2 DM**: Modest doses of insulin are quite efficacious in controlling hyperglycemia in newly diagnosed type 2 DM. Insulin should be considered as the initial therapy in type 2 DM, particularly in lean individuals or those with severe weight loss, in individuals with underlying renal or hepatic disease that precludes oral glucose-lowering agents, or in individuals who are hospitalized or acutely ill.

**Choice of initial Glucose-Lowering Agent**: Though insulin is an effective primary therapy for type 2 DM, most patients and physicians currently prefer oral glucose-lowering drugs as the initial pharmacologic approach. The level of hyperglycemia should influence the initial choice of therapy.

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32. Cha. Ni .4

33. Cha. Chi. 6

34. Cha. Sut. 17

35. Su. Ni. 6

36. Su. Chi. 11, 12, 13

37. A.H. Ni. 10

38. A. H. Chi. 12

39. Ha. Tritiya Sthana / 28

40. Ka. Sut. 25/22

41. Ma. Ni. 33

42. Gayadas Commentary, Su. Ni. 6/6

43. Bhavaprakasha Chikitsa. Madhyam Khanda / 38
44. Yogaratnakara, Pramehachikitsa Adhikara
45. Rigveda 10/163.15
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47. Arundatta Commentary, A. H. Ni 10/21
48. Cha. Ni. 4/44, A. H. Ni. 10/18
49. Su.Chi.12/6
50. Cha. Ni.4/9
51. Cha.Chi.6/15, Su.Chi.11/3
52. Su.Chi.11/3
53. Su.Chi.11/3
54. Cha.Chi.6/57
55. Cha. Sut. 17/78-81
56. Su.Chi.11/3
57. Cha. Chi. 6/57
58. Cha. Chi. 6/57
59. Cha. Chi. 6/57
60. Cha. Sa. 4/30
61. Cha. Chi.6/4
62. Su. Ni.6/3
63. A. H. Ni. 10/1-3
64. Cha. Ni.4/5
65. Cha. Ni.4/24
66. Cha. Ni.4/36
67. Cha. Sut.17/78
68. Cha. Ni. 4/5
69. Cha. Ni. 4/36
70. Su. Ni. 6 / 22
71. Cha. Ni. 4/44
72. Su. Chi. 11/3
73. Cha. Ni. 4/36
74. Cha. Chi. 6/6, 11

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75. Cha. Sut. 17/78-80
76. Cha. Chi. 6/57
77. Cha. Ni. 4/36, 37
78. Cha. Ni. 4/3
79. Cha. Sut. 19/51
80. Chakrapanai Commentary, Cha. Su. 19/49
81. Cha. Ni. 4/7
82. Su Ni 6/9
83. A. H. Ni. 10/14
84. A. H. Sut. 11/8
85. Cha. Ni. 4/48
86. Su. Ni. 6/4
87. Cha. Sut. 21/5
88. Su. Sut. 15/13
89. Cha Sut. 17/ 78
90. Su. Sut. 15/24
91. Su. Ni.6/15
92. Su. Chi. 12/4
93. Su. Ni. 6/4
94. Cha. Ni. 4/48
95. Su. Ni. 6/15
96. Su. Ni. 6/15
97. Su. Ni. 6/15
98. Cha. Sut. 17/81
99. Su. Ni. 6/14
100. Cha. I. 5/16-17
101. Cha. Chi.6/16, Su.Chi.12/6, A.H. Chi.12/1