IJBPAS, January, Special Issue 2, 2022, 11(1): 497-511
ISSN: 2277–4998

International Journal of Biology, Pharmacy and Allied Sciences (IJBPAS)
'A Bridge Between Laboratory and Reader'

www.ijbpas.com

ANALYZING PRAMEHA (DIABETES MELLITUS) NIDANA (ETIOLOGY) AND SADHYA ASADHYATA (PROGNOSIS)

SACHIN DEVA[1], SUNIL R KHANDARE[2*]

1: Associate Professor & HOD, Ph.D Scholar, PG & PhD Department of Roga Nidana Evum Vikriti Vigyan, Parul Institute of Ayurved, Parul University, Limda, Vadodara, Gujarat - 391760, India

2: Professor, PG & PhD Department of Roga Nidana Evum Vikriti Vigyan, Parul Institute of Ayurved, Parul University, Limda, Vadodara, Gujarat - 391760, India

***Corresponding Author: Dr. Sunil R Khandare: E Mail: sunil.khandare260000@paruluniversity.ac.in**

Received 16th June 2021; Revised 11th July 2021; Accepted 25th Aug. 2021; Available online 25th Jan. 2022

https://doi.org/10.31032/ijbpas/2022/11.1.2048

ABSTRACT

Diabetes mellitus is a chronic disease characterized by derangements in carbohydrate, fat and protein metabolism. It is a clinical condition characterized by increased blood glucose level (hyperglycemia) due to insufficient or inefficient insulin. It is a most common metabolic disorder in India. Diabetic nephropathy is currently one of the leading causes of morbidity and mortality in diabetic population, accounting for greatest proportion of end stage renal disease worldwide. In Ayurveda Prameha is usually correlated with Diabetes Mellitus with important symptoms like Prabhoota Mutrata i.e Increased frequency and quantity of urine and Avila Mutrata which means turbid urine. Prameha is considered as Chirakaaleena Vyadhi (chronic disease) which is Anushangi (recurring) in nature. It is due to vitiation of Tridoshas (Vata-Pitta-Kapha) especially Kapha Dosha which can be considered as important components of protoplasm which governs all the activities of body and also symbolizes the physico-biological properties of compounds made through a different combination of Panchamahabhootas i.e Akasha (space), Vayu (electrons), Teja (energy), Jala (proton) and Prithvi (neutron). Sadhya Asadhyata of any disease plays a pivotal role in treatment aspect. Since Prameha is a Anushangi Vyadhi understanding its prognosis is of prime importance.

Keywords: Prameha, Nidana, Sadhya Asadhyata, Diabetes mellitus

NIDANA VIVECHANA

The word Nidana has been used to mean different aspects of a disease by Madhava; the first one is Vyadhibodhaka Hetu which means, those factors which helps a physician know clearly about the disease and second one is Vyadhijanaka Hetu which means all those factors which are responsible in producing the disease. Ayurveda classifies Nidana broadly into two categories viz. Bahya and Abhyantara [1]. The later are the Doshas and Dushyas whereas, the former are the influence of environmental factors like Ahara, Vihara, Achara, Vichara, Kala and so on. The Bahya Hetus can be again divided into Samanya Hetus and Vishesha Hetus. This conventional way of going into the details of Nidana is extremely useful in making a comprehensive study for a good analysis.

Bahya Hetus:

Samanya Nidana

The key word in Samanya Nidana of Prameha are the Hetus which cause Kapha Vriddhi (Kapha Kriccha Sarvam) [2]. It becomes contextual here to take note of the fact that Kapha is the main Dosha involved [3] in Prameha and hence all those Hetus that cause Kapha Vriddhi automatically become the Hetus for Prameha.

Kapha is made up of Prithvi and Ap Mahabhootas and is in fact a personification of Tamasika Guna. Hence any environmental influence on the body and mind in the form of Ahara, Vihara, Ritu contributing to the Tamasika factor results in a morbid predominance of Prithvi and Ap Mahabhootas in all the forms of Kapha.

Ahara [4]: Any Ahara which is Madhura and Lavana Rasa Pradhana, Guru, Manda, Sheeta, Snigdha, Shlakshna, Sandra, Sthira, Picchila Guna Pradhana, Madhura vipaka and Sheeta Veerya, including unboiled, unroasted, unfried food articles and Anoopa Mamsa, if taken in excess quantity increases Kapha which attains 'Aparipakwa Avastha' and mainly effects the Medas and Kleda leading to Prameha (Sthoola) due to Avarana.

Vihara: The following Viharas have been implicated as Kaphamedokara.

Divaswapna: Causes inertia in the body and the accumulation of Prithvi and Ap Mahabhoota, leading to aggravation of Kapha.

Avyayama: A man is generally supposed to balance between the nutrient intake and energy spending to maintain equilibrium. When this balance is not maintained, it results in accumulation of Medas and Kapha. Hence adequate amount of Vyayama is necessary to avoid Prameha.

Asya Atisukha: Excessive Asya Sukha and Swapna Sukha causes Snigdhata leading to Kapha Vriddhi.

Alasya: Is an attribute of a human being, which is mostly controlled by Manas. Alasya is nothing but that state of lethargy of mind where a man is unable to carry out or undertake any enthusiastic task not because he is incapacitated due to ill health but only because he is unwilling to do it. This results in inactivity causing excessive nourishment.

Chinta Tyaga: An attribute of the mind that is antagonistic to Kapha and Medas. When a person becomes free from Chinta, he starts sitting idol without doing any work which helps in accumulating excess Kapha and Medas.

Samshodhana Varjana : Samshodhana therapy is essential in any individual for cleansing the body of Doshas. Being a form of Langhana, Samshodhana causes Medas and Kapha Kshaya. If this is not resorted to, it causes accumulation of Kapha and Medas.

Mruja varjana : Mruja is Shuddhi. One who avoids therapies like Udvartana, Rutu Prakara Samshodhana etc leads to Kapha Dusti causing Prameha.

It can be explained with some examples like:

Table 1

Nidana	Mahabhoota	Properties	Dosha/Dushya
Gow & Mahisha Ksheera	Prithvi & Ap	Madhura Rasa & Abhishyandi in nature	Kapha/Meda
Ikshu Rasa	Prithvi & Ap	Madhura rasa & Sheeta virya	Kapha/Meda
Trapusha (cuccumis sativas)	Prithvi	Madhura rasa,Guru Guna etc.	Kapha/Meda
Asya & swapna sukha	-	-	Kapha & Tamoguna/Meda & Mamsa
Not performing any samshodhana	-	-	Tridosha vriddhi

Vishesha Nidana

Though the Kapha is the Arambhaka Dosha in the Samprapti of Prameha, Pitta and Vata play an important role in complicating the disease. For e.g. if an affected person starts indulging in Pittakara Ahara Viharas then 6 types of Pittaja Mehas manifest. Similarly when Kapha and Pitta are in Ksheenavastha, the Vayu causes 4 types of Vataja Prameha.

Kaphaja Prameha Nidana: Are the same as explained in the Samanya Nidana

Pittaja Prameha Nidana [5]

Ahara Sambandhi **-** Ushna Guna Ahara Atisevana, Amla Rasa Ahara Atisevana , Lavana Rasa Ahara Atisevana, Katu Rasa Ahara Atisevana, Ahara Sevana when there is already Ajeerna, Vishama Ahara Sevana.

Vihara Sambandhi - Ati Stapa Sevana, Ati Santapa, Shrama, Krodha.

Pitta is predominantly a Tejo Mahabhoota Pradhana Dosha and all those environmental influences that cause Tejoguna Vriddhi aggravate Pitta. These

are subdivided into Ahara and Vihara Sambandhi for convenience. Ushna, Amla, Lavana and Katu Rasas are potent Pittavardhaka, hence cause aggravation of Pitta. Ahara Sevana when there is already Ajeerna and Vishama Ahara Sevana leads to aggravation of Tridoshas, starting with Pitta first, because this is directly related to Agni.

Atapa Sevana in excess proportions has a direct bearing on the Pitta that gets aggravated first. Shrama or strain (physical or mental) and Krodha (anger) also leads to Pitta Prakopa causing Pittaja Prameha.

Vataja Prameha Nidana

The causes for aggravation of Vata can be mainly grouped into two categories i.e Dhatu Kshaya [7] and Margavarana [6].

The Margavarana is a result of accumulation of Kapha or Pitta Doshas in the Vatavaha Srotas. Due to the respective Nidana Sevana, this leads to Vataja Prameha. In Prameha, Dhatu Kshaya is an invariable consequence of Aparipakvata of Dhatus. This leads to aggravation of Vata causing Vataja Pramehas. Following factors are also responsible for aggravation of Vata

Ahara Sambandhi

Katurasa Ahara Atisevana, Kashaya Rasa Atisevana, Tikta Rasa Ahara Atisevana, Laghu and Rooksha Guna Ahara Atisevana.

Vihara Sambandhi - Vyavaya atiyoga, Vyayama Atiyoga, Vamana Atiyoga, Virechana Atiyoga, Asthapana Atiyoga, Vega Sandharana, Anashana, Abhighata, Atapa Sevana, Udwega, Shoka, Shonita Ati seka, Ratri Jagarana,Vishama Shareera Nyasa.

All these factors are basically designed to provoke the Rajasika Guna in the body. Vata that is predominantly Rajasika gets aggravated leading to Vataja Pramehas.

Abhyantara Hetus

The Abhyantara Hetus are the Doshas which are responsible for causing the disease. The description of mode of action of these Doshas will be explained in Samprapti Vivechana.

Study of Nidana in Sthoola and Krusha Pramehis in relation to Beeja Dosha

The occurrence of Prameha in Sthoola and Krusha has been identified by the authors. Sushruta has categorically stated the existence of two varieties of Pramehis [8] and Charaka has prescribed different lines of treatment for the Sthoola and Krusha Pramehis [9]. He has also asserted that whenever a patient presents with Madhupama Mootra, a wise physician should always consider the possibilities of Madhumeha due to Vata as a result of Ksheenata of Doshas and Madhumeha due to Kapha as a result of Santarpana.

It is very clear from Charaka's explanation that Prameha in Krusha occurs in the event of a relative Vata Vriddhi in comparison to the other Doshas which would have attained a state of Ksheenavastha in an already affected person. This person may have been Sthoola in the beginning but would have become Krusha due to Dhatu Kshaya.

On the other hand, Krusha Pramehis are usually Rogis with a Beeja Dosha. Hence, when such a Rogi consumes the Nidanas for Vata Vriddhi, he develops Madhumeha sooner than a Sthoola Rogi who always indulges in Tarpana Ahara. Sthoulyata is also a condition due to Beeja Dosha here. Though the patient consumes normal food, Upachaya of only Medo Dhatu occurs, not the other Dhatus [10]. Though both the Rogis are favorably disposed for developing Madhumeha, the Beeja Doshaja Sthoulya Rogi is more under risk of developing the disease than his counterpart.

Study of aetiology according to modern medicine [11]

The exact cause of the disease DM has remained elusive, but many factors have been found to be closely linked or associated with the disease.

Genetics

The mechanism of inheritance of DM either insulin dependent on non-insulin dependent is unclear. The genetic predisposition is probably permissive and not casual.

Genetic susceptibility in IDDM - This probably involves more than one gene. Candidate loci have been proposed on chromosomes 2, 6, 11, & 15 though primary genetic site in humans is believed to be located in the major histocompatibility locus on the short arm of the 6^{th} chromosome. While definite associations exist between class I alleles & type I DM, the D locus is considered of primary importance (A, B, C & D are the four loci of HLA human leucocyte antigen) encoded by the MHC (Major Histocompatibility Complex) found to be closely associated with IDDM]

Genetic Susceptibility in NIDDM-Modes of inheritance of NIDDM in variant called maturity onset DM of the young have been more or less conclusive than the other forms. It is highly likely that ordinary NIDDM is polygenic. Genetic influence is powerful. Since the concordance rate for DM in monozygotic twins with type 2 disease may be as high as 80%, risk to offspring and siblings of patients with NIDDM are higher than in type I DM. Nearly $^{4}/_{10}^{ths}$ of siblings and $^{1}/_{3}^{rd}$ of offsprings eventually develop abnormal glucose tolerance or frank DM.

Autoimmunity

The basic pathology of DM spins around one factor i.e. insulin and its source of production viz. islet β cells. Complete or partial destruction of islet β cells or peripheral resistance of insulin is causal of DM, but an autoimmune process destroying the islet β cells is nevertheless Insulin dependent. The autoimmune destruction of the β cells may be best explained by the existence of a β cell specific protein that for unknown reasons acquires auto-antigenic properties and eventually becomes target for autoimmune reaction. Gradual β cell loss becomes clinically manifest only when β cell mass is reduced to a critical part after which metabolic compensation is not possible.

Heredity

The mechanism of inheritance of IDDM is unclear. At various times, transmission has been postulated to be autosomal dominant, autosomal recessive and mixed. While IDDM occurs with increased frequency in some families, familial aggregation is uncommon. So, deduction of mechanism of inheritance is difficult. Analysis of pedigrees shows a low prevalence of direct vertical transmission. The chance of a child developing type I DM when another first-degree relative has the disease is only 5-10%. HLA identity in a sibling increases the risk. The presence of NIDDM in a parent increases the risk for IDDM in the offspring. It is not known whether the intermixing of IDDM and NIDDM in the same family represents operation of a single genetic trait or whether two common genetic predispositions coexist in a family by chance. While the low rate of transmission of IDDM makes it difficult to discern the mechanisms of inheritance through study of families, they are reassuring to diabetics who wish to have children. The risk of type I DM is up to five times higher when the father has the disease than when the mother is a diabetic.

Environmental factors

Viral Infections-As noted earlier, the fact that a significant proportion of monozygotic twins remain discordant for IDDM suggests that non-genetic factors are required for the development of DM. An environmental factor in many cases is believed to be a viral infection of a β cell. A viral etiology was originally suggested by seasonal variations in the onset of the disease and by what appeared to be more than a chance relationship between the appearance of DM and preceding episodes of mumps, hepatitis, infections, mononucleosis and coxakie virus infections. The isolation of coxakievirus B4 from pancreas of a previously healthy boy who died after an episode of ketoacidosis and induction of DM in animals inoculated with isolated virus also suggested a viral

etiology. Further support for viral theory comes from the observation that about 1/5th of individuals with congenital rubella develop DM. Despite its attractiveness, the viral theory should be treated with considerable caution. Serological studies seeking evidence of recent viral infection in patients with new onset IDDM are inconclusive at best.

Bovine albumin: [14] - It has been suggested that exposure to cow's milk or milk products early in life predisposes to autoimmune DM. In an initial study, diabetic subjects were found to have attributes to bovine albumin. Exposure to cows milk is presumed to induce an immune response to 17-amino acid fragment in some infants and cross reactivity of the antibody. This hypothesis has not received wide support.

i. Obesity: Type 2 DM is almost non-existent in individuals with a body mass index below 22 Kg/m2 and increased risk of DM with obesity has a strong familial tendency. When one or both parents are diabetic, 100% offspring's will develop DM, if they become sufficiently obese. If neither parent has DM, fewer than 20% of obese offspring develop DM.

ii. Life Style [12] :Epidemiological studies of type 2 DM provide evidence that over eating, especially when combined with obesity and under activity is associated with development of type 2 DM.

iii. Malnutrition [12]: It is proposed that malnutrition in utero and in infancy may damage β cell development at a critical period predisposed to type 2 DM at a later stage. Impaired β cell function and peripheral insulin resistance have been major factors involved in NIDDM. Pregnancy also induces NIDDM in genetically susceptible individuals.

Causes of secondary DM [11]

I. Secondary to Pancreatic disease:Congenital Pancreatic Aplasia, Acute and Chronic Pancreatitis, Pancreatic Carcinoma, Cystic fibrosis & Haemochromotosis.

II. Secondary to Endocrine Disorders: Hormones with an insulin antagonistic affect such as, Growth hormone, glucocorticoids, catecholamines, thyromine and glucagon cause impaired glucose tolerance or even overt DM when produced in excess as a result of tumor or hyperplasia of the respective gland of origin. Endocrine syndromes frequently associated

with carbohydrate abnormalities of variable intensity are Acromegaly, Cushing's syndrome, Phaeochromocytoma, Glucogonoma, Hyperthyroidism, Cons syndrome and Carcinoid syndrome.

III. Chemically induced DM: Two groups of drugs causing permanent or transient hyperglycaemia can be distinguished. 1) Substances with cytotoxic effect on β cells and 2) Substances inhibiting insulin secretion without β cell destruction. The first group includes Alloxan, Glyoxal Streptozotocin, Oxine dithiazone and recently Asparaginase, Pentamidine Esthionate, N-3-Phyridyl methyl N- p- mitro ethyl urea (PNU). The second group includes Diazoxide, Diphenyldydation Cyproheptadine and Manaoheptolose. Majority of these drugs have been used only experimentally in laboratory animals.

Rare causes of DM

Numerous rare genetic syndromes (Lipoatrophic DM, Leperchaunism, Acanthosis Nigricans types A & B, Ataxia Telangiectasia, Stiff man syndrome, Bardet Biedl syndrome) may be associated with either glucose tolerance or overt DM. Special islet lesions have not been described in any of these syndromes, there is an absolute or relative insulin deficiency in such diseases. The high coincidence of cirrhosis and DM is long established (Naunyn's DM). This may be due to to reduced insulin degradation by cirrhotic liver with subsequent hyperinsulinism and insulin resistance.

i. Pituitary DM [13]: The growth hormone of the pituitary appears to have diabetogenic power (Houssay). Administration of hormone leads to atrophic changes in the β cells associated with an early reversible phase of DM, followed by an irreversible phase with complete destruction of β cells. DM may be associated with Acromegaly. Hypophysectomy will arrest the course of DM in the experimental animal.

ii. Adrenal DM [13]: There is convincing evidence that Adrenal cortical hormones may affect both the experimental and human disease. Adrenalectomy will arrest or modify the progress of experimental DM. Adrenal hyperplasia or tumors may be

associated with DM. The administration of hydrocortisone causes DM by inhibiting insulin action.

iii. Gestational DM [12]: During normal pregnancy, insulin sensitivity is reduced through the action of placental hormones and this affects glucose tolerance. The term gestational DM refers to hyperglycaemia occurring for the first time during pregnancy. Repeated pregnancy increases the risk of developing permanent DM especially in obese women.

The detailed study of cause according to Ayurvedic classics and modern medicine unveils the following facts.

Table 2

Nidana of Prameha	Etiology of DM
Beeja, Beejabhaga & Beejabhaga Avayava Upatapa leading to Prameha Arambhaka Dosha Dushti in Sahaja Madhumeha	Genetic susceptibility in the 6th Chromosome leading to IDDM
Kulaja Vikara- Pitr Pitamahadi Karma	Familial inheritance more in IDDM
Kaphamedokara Ahara Vihara Sevana, Avyayama and Chinta Tyaga	Over eating and under activity
Vikara Vighata Abhava and Sahaja Asatmya	Auto immunity
Ksheera, Dadhi as Kaphakara Ahara	Bovine albumin
Sthoulya Upadrava	Obesity leading to NIDDM
Anashana	Malnutrition in infancy predisposes to IDDM
Shoka Udwega in Vataja Prameha	Stress leading to IDDM

SADHYA-ASADHYA VIVECHANA

The physician who desires to be successful in treating a disease, should posses the knowledge of Sadhya Asadhyata of that particular disease, hence in our classics a separate sections of chapters have been dedicated to decide the Sadhya Asadhyata of disease.

In classics while discussing about the Sadhya Asadhyata, it has been told that Prameha is Anushangi [15] and Mahatyaya [16] which means Punarbhavi and one which causes the Kshaya of the Sarabhuta Hatu like Majja etc. respectively. As described earlier, the Prameha disease attains Sthairya and Asadhya status because of its Prakriti and Vikriti Bhootatvat. Here the word Prakriti and Vikriti Bhootatvat refers to Prameha Arambhaka Dosha i.e Prakrita Kapha which attains Vikrutatvat, by its Guna when the Premaha gets chronic and if Kapha gets provoked further there will be involvement of Raktadi Dhatu which are not similar in qualities to Kapha are considered as Vikriti and ultimately leading to a condition of incurability [17]. This Prakriti Vikriti Bhootatvat of the Prameha can be described in different stages as follows.

Sadhyata of Kaphaja prameha [17]

Kaphaja Prameha is said to be Sadhya based on following reasons

i. Samakriyatvat
ii. Atishaya medo na dushtatvat
iii. Meda Dhatu and Kapha Dosha Sthana.

Samakriyatvat

Here the word Samakriyatvat refers to the treatment of similar Doshas and Dushyas which are involved in the manifestation of disease. In Kaphaja Prameha the involved Pradhana Dosha is Kapha and the Dushyas are Medas, Mamsa and Shreera Kleda which are said to be having similar qualities of Kapha, this gives the clearcut idea about Thulya Dushyatva of Kaphaja Prameha. It is said to be Sadhya because the Katu, Tikta, Kashaya Rasas and Tikshna, Ushna Gunas, which are antagonistic to Kapha, are the same for Medas, Mamsa, Kleda, Lasika, Rasa etc Dhatus. Thus due to Samakriya Kaphaja Prameha is Sadhya.

Atishaya medo na dushtatvat

Kaphaja Mehas are characterised by less involvement of Dhatus. Hence the term "Atishayena medo na dushtatvat". Here the predominant symptoms of only Kapha are seen, therefore Prameha is Sadhya in this stage. Moreover other Dhatus are not much involved in this stage and Upadravas are not manifested.

Meda Dhatu and Kapha Dosha Sthana

Even the Sthana of Meda Dhatu and Kapha Dosha plays an important role in severity of the disease, as Sthana of Kapha Dosha is Uras and that of meda dhatu is Vapavahana, because of more distance between these two Sthana Vriddhi of Kapha Dosha doesn't influence the Meda Dhatu immediately.

Sadhyata of Pittaja Prameha [18]

In Pittaja Prameha if Meda Dhatu has not attained Pradusta then it is considered to be Sadhya to treat.

Yapyata of Pittaja Prameha [19]

The six Pittaja Pramehas are considered to be Yapya as per the classics because of the following reasons.

i. Vishamakriyatvat
ii. Atrapi atishayena medo na dushtavat
iii. Samsrushta dosha meda sthanatvat

Vishamakriyatvat

In Pittaja Prameha involved Dosha is Pitta and Dushyas are same as that of Kaphaja Prameha i.e Meda, Mamsa and Shareeraja Kleda. These Dushyas have opposite Gunas of Pitta which makes the Chikitsa difficult i.e if Pitta is treated with Sheeta and Madhuradi Dravyas, they are Viruddha to Pitta but are Medo-Mamsa and Kledakara and if Medadi Dhatus are treated with Tikshna Aushadhi Dravyas, they cause Pitta Vruddhi. The Dosha Pitta and Dhatu Medas, Mamsa and Kleda have Viruddhagunas, which overpower the

purpose of Chikitsa. Therefore the six Pittaja Pramehas are Yapya.

Atrapi atishayena medo na dushtatvat

Pittaja Pramehas are also characterised by relatively less involvement of Dhatus as suggested by the term "Atrapi" which means that the involvement of Medas and other Dhatus is too severe for it to be Sadhya, yet not severe enough to be Asadhya. Therefore this stage of the disease is Yapya.

Samsrushta dosha meda sthanatvat

Means "Sannikrushtam doshasya pittasya medascha sthanam yasmat" i.e closeness in the Stahnas of Medas and Pitta. Hence Pittaja Pramehas have been called Durjaya. The Sthana of Pitta is Amashaya and that of Medas is Vapavahana. There is proximity or Pratyasannata of these Sthanas in the Kostha as described by the term Ekadesha. Hence there is mutually contradictory environment in Ekadesha, the result is Vishama Kriya of the Chikitsa and therefore Pittaja Pramehas are Yapya.

Asadhyata of Vataja Prameha [16]

The four Vataja Pramehas are considered as Asadhya due to following reasons

i. Mahatyayatvat
ii. Viruddhopakramatvat

Mahatyayatvat

The term Mahatyaya has following interpretations

a. Mahata gambhira dhatunam atyaya nasho yena sa
b. Ashukaritva [20]
c. Bahuvyapattikartrukatva [20]
d. Majja prabhrutisarabhoota dhatukshaya [16]
e. Majjadi gambhiradhatu apakarshakatvena [20]
f. Uttarottara saratara dhatu sravakatvat [21]
g. Sheegram vinasha karitva [21]

The above mentioned interpretation signifies the severity of the Vataja Mehas, where Mahat and Gambhira Dhatus undergo destruction very fast. The word Ashukari indicates its rapidity in spreading and in destruction of the body. The word Bahuvyapattikartrukatva denote its ability to cause many complications. The words Majja prabhrutisarabhoota dhatukshaya, majjadi gambhiradhatu apakarshakatvena and Uttarottara saratara dhatu sravkatvat indicates the Kshaya of Gambhira Dhatus in the form of Mootra and the word Sheegram vinasha karitva signifies the ability of the disease to destroy the Shareera very fast.

Viruddha upakramatvat

The Chikitsa of Vataja Prameha involves Viruddhopakrama which means there is a mutual contradiction in the treatment modalities as use of Snigdha etc are Pathya

for Vata but apathy for Medas. Hence the disease is Asadhya.

Other conditions determining Asadhyata of Prameha

a. Prameha associated with Upadrava is Asadhya [22].
b. Prameha with all its Poorvaroopas is Asadhya [23]
c. Jatah Prameha is Asadhya. [23]
d. Pramehi with Balamamsa Kshaya can be left untreated [24].
e. All Pramehas if left untreated will land into Madhumeha which is Asadhya [25]
f. Prameha with Arista Lakshanas is Asadhya.
g. A patient who hates hygenic activities like Snana, Chankramana and one who has Manda Utsaha, who is Atisthoola, Snigdha and Mahashana dies of Prameha.
h. If Prameha associated with Pidakas then it is Asadhya [26].
i. If Prameha associated with Atiprasruta (atishayam dhatu mootra sravayuktam) then it is Asadhya [26]
j. If Prameha is Ghada (Kaala Prakarshat) then it is Asadhya [26]

Prognosis in DM [27]

The prognosis of the diabetes usually refers to the likely outcome of diabetes. The prognosis of the diabetes may include the duration of disease, chances of complications, probable outcomes, prospects for recovery, recovery period of diabetes, survival rates, death rates and other possible outcomes in the overall prognosis of disease. Naturally, such forecast issues are by their nature unpredictable.

A summary of the study which was undertaken to show the prognosis of diabetes mellitus in a geographically defined population is as follows which gives the clear cut picture of the life expectancy of diabetic patients.

SUMMARY

The centralised registration and care of all diabetics in a geographically defined population has provided an epidemiological basis for a longitudinal investigation of the prognosis of this disease. Records of all newly diagnosed diabetics who had been registered in the Erfurt district (population 1.25million) in 1966 were studied in relation to the time period 1966-1976.Of the known 2560 diabetics (910men, 1650 women; 93.7% more than 40yrs of age), 1054 had died during the 10year follow-up period. Cardiovascular causes accounted for the majority of deaths (63%). In almost all age classes proportionally more men than women had died at follow-up. There was significant difference in the 60-69year group(men 61.6%,women 46.2%). In

comparison with the general population, excess mortality ranged from 2.1 to 1.0,decreasing with age at onset without significant differences between men and women. Excess mortality was present in most age classes and was evident within the first year after diagnosis. Current life-table analysis confirmed the shortened life expectancy of the diabetics. The lower life expectancy of the non-insulin dependent diabetics may not be due to hyperglycemia alone but probably involves a variety of atherogenic risk factors.

REFERENCES

[1] Agnivesha, Charaka, Dridhabala, Chakrapanidatta, Vaidya Trikamji, Charaka Samhita, Reprint-2009, Varanasi (INDIA), Chaukhambha Orientalia, Nidanasthana, Chapter-4, Verse-3, Page -211.

[2] Sushruta, Dalhana, Gayadasa, Vaidya Trikamji, Nibandhasangraha, Susruta Samhita, 2003 Edition, Varanasi (INDIA), Chaukhambha Surbharati Prakashan, Uttaratantra, Chapter-33, Verse-4, Page-144.

[3] Vagbhata, Translation by Prof.K.R.Srikanthamurthy, Edition-9th, Chaukhambha Orientalia, Varanasi (INDIA), Sutrasthana,Chapter-13,Verse-2-4, Page-279.

[4] Agnivesha, Charaka, Dridhabala, Chakrapanidatta, Vaidya Trikamji, Charaka Samhita, Reprint-2009, Varanasi (INDIA), Chaukhambha Orientalia, Nidanasthana, Chapter-4, Verse-6, Page-212.

[5] Agnivesha, Charaka, Dridhabala, Chakrapanidatta, Vaidya Trikamji, Charaka Samhita, Reprint-2009,Varanasi (INDIA), Chaukhambha Orientalia, Nidanasthana, Chapter-23, Verse-5-7,Page-122

[6] The Global burden of diabetes and its complications: An emerging pandemic, Review article, Van Dieren S, Beulens JW, Van der Schouw YT, Grobbee DE, Neal B, pmid 20489418 (Pubmed Indexed)

[7] Clinical Biochemistry & Haematology with clinical concepts, Naveenchandra N.H, Published by SDM College of Ayurveda, Chapter-9, Page-188.

[8] Joslin's, Diabetes mellitus, Edited by C. Ronal Kahn, Gordon C.Weir, George.L. King, Alan M. Jacobson, Alan C. Moses, Robert J. Smith, Indian Edition, 14th edition, Lippincott Williams & Wilkins publications,2006,Pp.1208.

[9] Wikipedia

[10] Etiopathogenesis of Prameha Upadrava with special reference to skin manifestations in Diabetes Mellitus by Dr. Sachin. Deva, Dessertation submitted to RGUHS, Bangalore in partial fulfilment of the requirement for the degree of doctor of medicine in Roga Nidana 2012, Pages 192, Pg.No-11

[11] Etiopathogenesis of Prameha Upadrava with special reference to skin manifestations in Diabetes Mellitus by Dr. Sachin. Deva, Dessertation submitted to RGUHS, Bangalore in partial fulfilment of the requirement for the degree of doctor of medicine in Roga Nidana 2012, Pages 192, Pg.No-12.

[12] Madhavakara, Madhava Nidana, Shri Vijaya Rakshita & Shrikantha dutta, Brahmananda tripati, Vol-1, Madhukosha, Choukamba surabhi prakashana, Varanasi, 2nd edition, 1998, Pp-660, page no 18.

[13] Agnivesha, Charaka samhita, Acharya Yadavji Trikamji, Choukambha publication 2001 Varanasi. Pp 738, page no 212.

[14] Agnivesha, Charaka samhita, Acharya Yadavji Trikamji, Choukambha publication 2001 Varanasi. Pp 738, page no 212.

[15] Agni Vesha, Charaka Samhita, Acharya Yadavji Trikamji, Choukambha publication 2001 Varanasi.Pp.738,Page no-133

[16] Agni Vesha, Charaka Samhita, Acharya Yadavji Trikamji, Choukambha publication 2001 Varanasi. Pp.738, Page no-215

[17] Agni Vesha, Charaka Samhita, Acharya Yadavji Trikamji, Choukambha publication 2001 Varanasi.Pp.738, Page no-213

[18] Agni Vesha, Charaka Samhita, Acharya Yadavji Trikamji, Choukambha publication 2001 Varanasi.Pp.738, Page no-449

[19] Agni Vesha, Charaka Samhita, Acharya Yadavji Trikamji, Choukambha publication 2001 Varanasi.Pp.738, Page no-214

[20] Vagbhatacharya, Astanga hridaya, Dr. Anna Mereshwara Kunte, Choukambha publication 2000, Varanasi. Pp-956, page no-502.

[21] Maharshi Sushrutha, Sushrutha Samhita, 7th ed, Varanasi U.P, Sri Dalhana Acharya Sri Gayadas Acharya, Vaidya Yadavji Trikamji Acharya, Chaukambha Sanskrit Sansthan, 2002, Pp-824, page no-290

[22] Madhavakara, Madhava Nidana, Shri Vijaya Rakshita & Shrikanta dutta, Brahmananda tripati, vol-2, Madhokosha, Choukambha Surbharati Prakashana, Varanasi, 2nd edition, 1998, Pp.622, Page.no-20

[23] Agni Vesha, Charaka Samhita, Acharya Yadavji Trikamji, Choukambha publication 2001 Varanasi.Pp.738,Page no-449

[24] Agni Vesha, Charaka Samhita, Acharya Yadavji Trikamji, Choukambha publication 2001 Varanasi.Pp.738, Page no-368

[25] Maharshi Sushrutha,Sushrutha Samhita,7th ed, Varanasi U.P, Sri Dalhana Acharya Sri Gayadas Acharya, Vaidya Yadavji Trikamji Acharya, Chaukambha Sanskrit Sansthan, 2002, Pp-824, page no-294

[26] Maharshi Sushrutha, Sushrutha Samhita,7th ed, Varanasi U.P, Sri Dalhana Acharya Sri Gayadas Acharya, Vaidya Yadavji Trikamji Acharya, Chaukambha Sanskrit Sansthan, 2002, Pp-824, page no-144

[27] Springer Link-Journal Article.mht