ISSN 2456-3110
Vol 4 · Issue 1
Jan-Feb 2019

Journal of Ayurveda and Integrated Medical Sciences

www.jaims.in

JAIMS

Charaka Publications

Indexed

Journal of
Ayurveda and Integrated Medical Sciences
ISSN 2456-3110
ISSN: 2456-3110 REVIEW ARTICLE Jan-Feb 2019

Conceptual study of malnutrition related diabetes mellitus

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ABSTRACT

The Diabetes mellitus (DM) is a metabolic disease characterized by absolute or relative insulin deficiency. Type 1 diabetes is commonly known as juvenile diabetes, because it typically strikes during childhood and sometimes adolescents, and young age group. Now in the whole world nearly about 24% of population is suffering from diseases. The first widely accepted classification of diabetes mellitus was published by WHO in 1980 named them as IDDM (type-1) and NIDDM (type-2) and Malnutrition related diabetes mellitus (MRDM) was introduced in 1985. In India Malnutrition has high prevalence rate Malnutrition during intrauterine and early childhood period may impair growth and development. This review provides an overview of Juvenile Diabetes in children with MRDM. The juvenile diabetes and MRDM is a palliative disease. It cannot be completely cured, but can be controlled by medication, food, Ayurvedic *Chikitsa* and Lifestyle Changes.

Key words: *MRDM, Diabetes Mellitus, Malnutrition, Prameha.*

INTRODUCTION

Malnutrition related diabetes mellitus (MRDM) is a rare type of diabetes associated with long term malnutrition. This type of diabetes is characterized by insulinopenia, insulin resistance, hyperglycemia and failure of the beta-cells in the pancreas. It is also known as tropical diabetes or tropical pancreatic diabetes mellitus. These patients are thin, young, severely hyperglycemic, but in contrast to IDDM do not have ketonuria and requires high doses of insulin for control. Implication of malnutrition as a possible factor in the genesis and atypical features of this form of diabetes was first envisaged by Kar and Tripathy (1963) from Cuttack, Orissa, India.[1],[2]

Pancreatic calculi were first described by R. de graaf (1664), and diabetes associated with pancreatic calculi was first reported by Thomas Cawley (1788).[3] The pathology of chronic pancreatitis was first documented by Riedle in 1896. Asamann (1912) recognized pancreatic calculi radiologically. Elman et.al. (1929) related changes in serum amylase with pancreatitis, and introduced this as a diagnostic test in inflammatory disorders of pancreas. The association of malnutrition with diabetes was possibly first elucidated by Zuidema (1959).[4] From Indonesia who found pancreatic calcification and diabetes in patients majority of whom suffered from clinically evident protein malnutrition. From Kerala in (1962) reported a large series of patients with chronic pancreatitis, calculi and diabetes.[5],[6] This initiated a country wide awareness of calcific pancreatitis and diabetes. In India, one third of neonates are born low-birth weight and 63% of children less than five years are malnourished.[7] Thrifty phenotype hypothesis proposes that malnutrition during intrauterine and

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Submission Date: 13/01/2019 Accepted Date: 19/02/2019

Access this article online

Quick Response Code

Website: www.jaims.in

DOI: 10.21760/jaims.4.1.18

ISSN: 2456-3110 REVIEW ARTICLE Jan-Feb 2019

early childhood period may impair development and maturation of various organ systems, thereby predisposing the individual to a variety of illnesses like diabetes mellitus, coronary artery diseases and hypertension in later part of life. Malnutrition in early part of life was proposed to be etiological agent for Malnutrition Related Diabetes Mellitus.[9-11]

However, it has also been argued that malnutrition, in person, does not cause diabetes mellitus but modifies its clinical picture. Hence a new term, Malnutrition Modified Diabetes Mellitus (MMDM) or Malnutrition Related Diabetes Mellitus (MMDM) has also been proposed. Ayurveda describes *Prameha* or polyurea. It is basically *Kapha* disease affecting all the *Dhatus* in the course of disease. *Pitta* and *Vata* play supporting role to create a complex nature of this disease.

Sushruta describes *Prameha* of two types.

- *Sahaja* (genetic) caused by defect in sperm and /or ovam of parents. The parents are generally thin, and emaciated.
- *Apatthya Nimitta* - caused by defects in diet, exercise, rest. The patient is usually thick, stout and overweight.

Vagbhata describes it as a defect of *Meda-Kleda-Kapha* metabolism. Abnormal cough creates flaccidity, fluidity in *Meda. Mansa* and *Kapha* related *Dhatus* and their related structures like heart, kidney, skin, nerves and blood vessels.

Initially, the patient feels heavy, lethargic, sleepy, hungry, and thirsty. As gradually *Pitta* and *Vata* enter in the pathogenesis, the disease becomes complicated and *Tridoshaja*. It becomes *Mahagada* and *Asadhya.*

OBJECTIVES

To study the disease malnutrition related diabetes mellitus (MRDM) with sub types and treatment.

MATERIALS AND METHODS

Type of Study - Literature Review from modern text book, research journals and web references and Ayurvedic Classics etc.

Classification MRDM

The WHO study group report has set it apart from IDMM and NIDDM and Malnutrition related diabetes mellitus (MRDM) was introduced in 1985 designated it as MRDM in view of the striking features of malnutrition seen in these patients.The 2 subtypes of MRDM i.e. (a) fibrocalcific or fibrocalculaous pancreatic (FCPD) : Chracterized by a socioeconomic setting of poverty and malnutrition, onset is in young only below age of 30 years, clinically evidence of malnutrition, insulin-requirement for control, ketosis resistance and radiologically demonstrate pancreatic calcification and evidence of exocrine pancreatic dysfunction. (b) Protein-deficient pancreatic diabetes (PDPD) or protein deficient diabetes mellitus (PDDM): Has the same characteristics but differs from FCPD in absence of clinical and radiological evidence of pancreatic dysfunction and relative resistance to insulin (previously referred to as J-type).

Pathogenesis of MRDM

The pathogenesis of FCPD (Fibrocalcific or fibrocalculous pancreatic diabetes): That a genetic susceptibility play a role. It was demonstrated by restriction fragment length polymorphism (RFLP) studies showing that FCPD patients shared susceptibility genes in common with both NIDDM.[12]

Genetic susceptibility of Diabetes Mellitus or Pancreatitis

Undernutrition (calories, proteins, fats). Deficiency of sulphur containing amino acids, deficiency of micronutrients (Vit A, Vit E, selenium, zinc), decreased free radical and peroxide removal, known and unknown environmental agents, like Cyanoglucosides, Organic Nitrides, Cytochrome P450 Mediated decreases free radical and peroxidal removal leads to pancreatic injury by damaging beta cell leads to diabetes, and if there is exocrine injury and acinar, ductal damage of pancreas which may alter composition of exocrine secretion leads to steatorrhea and also by dilatation of duct. Calcium salt are precipitated which may form calculi and unknown mechanism in pancreatic injury by ductal damage may cause abdominal pain. This is genetic susceptibility of

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diabetes mellitus or pancreatitis. These are diagnostic triad of FCPD.[13]

The pathogenesis of PDPD (Protein-deficient pancreatic diabetes)

Central to the genesis of this type of DM is undernutrition, and possible, dietary habits.[14] Malnutrition per se with or without deficiency of other micronutrients, initiates a functional impairment of the pancreatic B cell. The persistence of subnormal B cell function provides a pathophysiological rationale for the well known clinical observation of ketosis resistance under basal condition. The possible nature of this subtype has been much debated and current theories are,[14]

- Early stage of FCPD
- Early Stage of IDDM
- Atypical form of IDDM modified by undernutrition
- Poorly treated NIDDM -Distinct Entity

Clinical Features

MRDM is associated with distinct clinical features. Most of the patients are aged between 10 and 30 years, with male preponderance. Most patients belong to poor socio-economic status. Typically, they are lean even before onset of symptoms and appear poorly nourished. Onset in insidious but may be relatively rapid. The syndrome commonly presents as diabetes, with polyphagia, polydipsia, polyuria and gross emaciation, fatigue, asthenia, weakness and cramps often lead to prostration in course of time.[15]

The report by Shaper[17] from Uganda reported that the common findings in the patients were softening and redness of the hair, parotid enlargement, steatorrhoea and radiologically demonstrable pancreatic calcification. Sensory neuropathy with numbness, sensation of pins and needles, episodic sweating and burning paraesthesia is frequently found. The development of secondary sexual characters is often lacking.[16]

There is a peculiar cyanotic hue of the lips. Varying grades of undernutrition and vitamin deficiencies may be observed. Nutritional status with glossitis, cheilitis; angular stomatitis, dry protuberant belly is characteristic.[17]

In FCPD (Fibrocalcific or fibrocalculous pancreatic diabetes)

Most patients seen in the hospital diabetes clinic present with symptoms usual for young patients with diabetes. The classical triad is of abdominal pain, pancretic calculi and diabetes but the occurrence of this is variable. Mohan's criteria for diagnosis of FCPD have been accepted widely as the most appropriate i.e. Occurrence in a tropical country Diabetes by WHO study group criteria. Evidence of chronic pancreatic disease : pancreatic calculi on X-ray or any three of the following abbominal pancreatic morphology with ducatal dilatation detected by sonography, CtsScan or ERCP, abdominal exocrine pancreatic function test chronic recurrent abdominal pain since childhood steatorrhoea absence of other cause of chronic pancreatitis i.e. alcoholism hepatobilliary disorders or hyperparathyroidism etc.

In PDDM (Protein-deficient pancreatic diabetes (PDPD) or protein deficient diabetes mellitus)

As opposed to FCPD, patients of PDMM present as young subjects with emaciation, asthenia and extreme weakness. In most cases, dietary history could be ascertained from parents and other accompanying persons and the diet was found to be utterly deficient. Height and body weight indicated retardation of growth. Marks of micronutrient deficiency were evident in many cases.

The clinical features of PDDM

Severe diabetes, fasting blood glucose more than 200 mg/dl, Onset of diabetes before the age of 30 years, Leanness, Body mass index<18 kg/m2. Absence of ketosis on withdrawal of insulin, Poor socio economic status, history of childhood malnutrition, Insulin requirement more than 60U/day or more than 1.5 to 2 U/Kg/day Of rural origin Absence of radiographic or sonographic findings of pancreatic calculi ductal dilatation and fibrosis; laboratory evidence of exocrine pancreatic dysfunction.[18]

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Table 1: Shows the broad differences between PDDM and FCPD

Comparison	PDDM	FCPD
Age of Onset	10-30 years	10-40 year
Rual	All	78%
Socioeconomic Status	All	60%
BMI<16 kg/m^2	92%	60%
Ketonuria	Nil	16%
C-Peptide (2 hr postprandial)	0.1	1.0pmol/1
Fecal fat (on 100g fat diet/day	6.2g/day	29g/day

Diagnostic Criteria

For the diagnosis of MRDM, a defined set of criteria was suggested by Bajaj et.al. i.e.[19]

SN	Clinical profile	Point score
1.	Age onset of 10 - 30 yrs	1
2.	leneneness with BMI <19	2
3.	H/O Malnutrition in child hood	1
4.	Childhood Stigmata of past or present malnutrition or deficiency State	2
5.	Moderate to severe Hyperglycemia Lack of protineness to Ketosis in absence of stress	3
6.	Insulin required to achieve metabolic controlled but no dependence on insulin for prevention	2
7.	Pancreatic calcification	3

An aggravated score more Than >10 is suggestive of MRDM.

Complications

MRDS patients are relatively resistant to ketotic coma and may be designated ambulant ketotics. Skin, respiratory and urinary infections are common. Pulmonary tuberculosis, vascular complications, autonomic neuropathy involving the bladder and premature loss of teeth with periodontal disease are common. Hypertension may occur with advanced renal disease. Infertility, oligomenorrhea and amenorrhoea are also common.[20-23]

Acute complications

Pyogenic and fungle infection, scabies and pulmonary tuberculosis, hypoglycemia, preodontities leading to premature loss of teeth.

Chronic complications

Autonomic neuropathy, Cataract Retinopathy and nephropathy.

Investigation

1. Fasting plasma glucose level and mean insulin requirement
2. Lipid Profile
3. Stool examination to detect undigested fat.
4. Plain x-ray of abdomen to detect pancreatic calcification
5. Pancreatic ultrasonography to measure dimensions of pancreatic head, body, tail and duct.

MANAGEMENT

Modern management

Most important is the glycaemic control and many patients require large doses of insulin, (Insulin needs) which is generally beyond their means. The dose of insulin required may go upto 150-200 units even in the absence of *co*mplications, indicating insulin resistance. Patients with FCPD require insulin to control hyperglycemia, and remainder respond to

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sulfonylureas. If pain is severe and intractable often lead to surgical intervention and various procedures have been attempted like;

1. Pancreatic Lithotomy
2. Pancreaticojejunostomy,
3. Sphincterotomy,
4. Choledocho-jejunostomy have all been treatead with variable results.

Steatorrhoea may be relieved by pancreatic extract and low fat diet. Incontradiction, patients with PDPD are totally unresponsive to the sulphonylurea and need insulin for the treatment of hyperglycemia.[24-25]

Ayurvedic Management

1. Dietic Modification
2. Lifestyle Modification
3. Ayurvedic Medicine.

Dietic Modification

A specially designed 'Diabetes Diet' is prescribed to the patient.

Dietary changes mainly include low calorie sugar, high protein, low fat and high fiber Diet.

Lifestyle modification

Simple changes in daily routine are suggested. They include,

1. Avoiid water, fruits, beverages or sweets immediately after food.
2. A walk for four to five minutes after each meal.
3. Consume warm water during meals.
4. A healthy breakfast is necessary. Avoid late and heavy dinners.
5. Consume two almonds soaked overnight in water and two walnut seeds each morning.
6. Eat an apple or an mango each morning.

Since exercise is equally important, we generally prescribe following exercises out of which a patient is advised to follow at least one routinely. A set of two *Suryanamaskara* (Vedic method of salute to Sun. It includes different postures)

Ayurvedic Medicine

1. *Chandraprabha Vati* - 250mg tablet
2. *Panchatikta Ghrut Guggulu.*
3. A blend of following herbs – half tablespoon 0.5 mg twice a day, at morning with empty stomach with half a cup of warm water and one tablespoon at bedtime at least one hour after dinner. Each 1 gm powder has approximately following quantity of *herbs.*

- *Patola (*Fruit of *Trichosanthes dioica* Roxb*)* - 30 mg.
- *Indrayava(*Seeds of *Wrighita tinctoria)* - 30 mg.
- *Kutki (Picrorhiza kurroa)* - 30 mg.
- *Vasa (Adathoda vasica)* - 30 mg.
- *Nimblood pressureatra (*Leaves of *Azardichta indica)* - 30 mg.
- *Methika (Fenugreek seed - Trigunnela foenumn-graccum* linn*)* - 30 mg.
- *Gudamaar (*Leaves of *Gymnema sylvestre)* - 30 mg.
- *Chitraka (*Roots of *Plumbago zeylanica Linn)* - 10 mg.
- *Tejapatra (*Leaves of *Cinnamomum tamala* Nees and Eberm*)* - 10 mg.
- *Anantmool (*Root of *Hemidesmus indicus* R.Br.*)* - 30 mg.
- *Bilvapatra (*Leaves of *Aegle marmelos)* - 30 mg.
- *Usheera (*Root of *Vetiveria zizanioides)* - 30 mg.
- *Bhoomyamlaki (*Leaves of *Phyllanthus niruli)* - 30 mg.
- *Jeeraka (*Seeds of *Cumminum cyminum)* - 30 mg.
- *Chirayata (Swerita chirataBuch* – Ham – *Ex C.B.Clarke)* - 30 mg.
- *Jambu (*Seeds of *Eugenia jambolana)* - 40 mg.

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- *Lodhra (Symplocos racemosa* Roxb*)* - 30 mg.
- *Khadeera (*Bark of *Acacia catechuWild)* - 30 mg.
- *Amalaki (*Fruit of *Emblica officinals Gaertn)* - 30 mg.
- *Bibhitaka (*Fruit of *Terminalia Bellerica Roxb)* - 30 mg.
- *Haritaki (*Fruit of *Terminalia chebula)* - 30 mg.
- *Ashwagandha (Withania somnifera)* - 40 mg.
- *Nimba twak (*Bark of *Azadirachta indica)* - 30 mg.
- *Karela (Powder of Momordica Charantia* Linn*)* - 40 mg.
- *Shweta Chandana (Sandalwood – Santalum album)* - 30 mg.
- *Haridra (Curcuma longa* Linn*)* - 30 mg.

CONCLUSION

This will help for proper understanding of pathogenesis and management of Malnutrition Related Diabetes Mellitus and provide valuable key for the effective management through Ayurveda. Despite discussion at several different international levels, the controversies on the term MMDM remain open. Whether it could be placed along with the classes PDDM and FCPD is still a point of controversy. It was assumed that this situation persisted due to lack of opportunity for diabetologists from all the regions to have a first hand exposure to such clinical disease. There has been no suggestion from any quarter as to what could be the alternative. The tentative classification proposed by WHO consultation group has not provided a place for MMDM, although they have discussed the current views on the topic.

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How to cite this article: Dr. Pallavi Bhirud, Dr. Borase Jayaprakash Balasaheb. Conceptual study of malnutrition related diabetes mellitus. J Ayurveda Integr Med Sci 2019;1:89-95.
http://dx.doi.org/10.21760/jaims.4.1.18

Source of Support: Nil, **Conflict of Interest:** None declared.

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