YOGIC MANAGEMENT OF

DIABETES MELLITUS

Morarji Desai National Institute of Yoga
Ministry of AYUSH, Government of India
68, Ashok Road, Near Gole Dak Khana,
New Delhi - 110 001

DIABETES MELLITUS

Diabetes mellitus is one of the most common endocrine diseases in all populations and all age groups. It is also a chronic metabolic disorder in which the body cannot metabolize carbohydrates, fats and proteins because of a lack of or ineffective use of the hormone insulin. The basic defect is in utilization of sugar by the body.

Glucose is the main source of energy for all the bodily functions. After digestion, it passes into the blood stream where cells use it for energy and growth. For its utilization, a hormone, Insulin must be present in proper quantity, which is produced by the pancreas. The defect in pancreas to produce proper amount of Insulin leads to decreased uptake of glucose from the blood and hence increased level of blood sugar.

Diabetes Mellitus (DM) is not a single diseases entity, but rather a group of metabolic disorders sharing the common underline feature of hyperglycemia. Hyperglycemia in diabetes results from defect in insulin secretion, insulin action or most commonly, both. The chronic hyperglycemia and attendant metabolic dysregulation may be associated with secondary damage in multiple organ systems, especially the kidneys, eyes, nerves and blood vessels.

Diagnosis

Blood glucose values are normally maintained in a very narrow range, usually 70-120mg/dl. The diagnosis of diabetes is established by noting elevation of blood glucose by any one of three criteria:

1. A random glucose>200mg/dl, with classical signs and symptoms (discussed below)
2. A fasting glucose > 126 mg/dl on more than one occasion.
3. An abnormal oral glucose tolerance test (OGTT), in which the glucose > 200 mg/dl. 2 hours after a standard carbohydrate load.

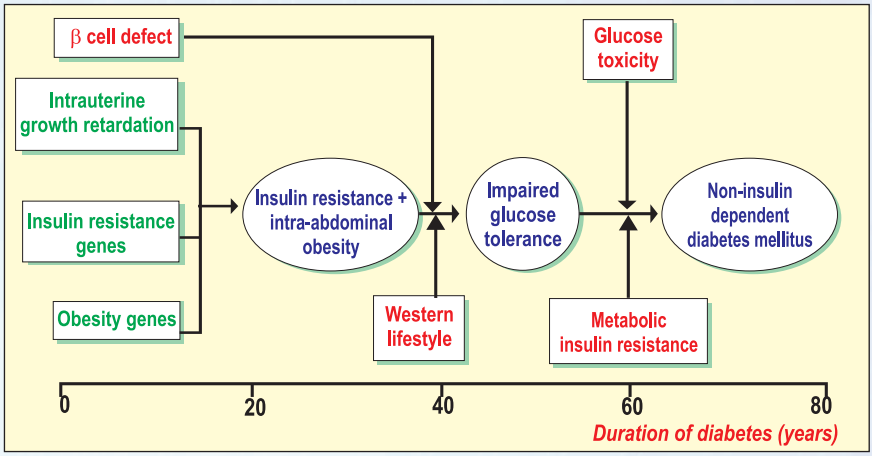
Classification

A vast majority of cases of diabetes fall into one of two broad classes.

Type 1 Diabetes is characterized by an absolute deficiency of insulin due to pancreatic beta cell destruction. It accounts for approximately 10% of all cases.

Type 2 Diabetes is caused by a combination of peripheral resistance to insulin action an inadequate secretory response by the pancreatic beta cells (relative insulin deficiency). Approximately 80% to 90% of patients have type 2 Diabetes.

Another reason for Diabetes mellitus is that the cells cannot use insulin properly, which is known as Insulin resistance.



CLINICAL SYMPTOMS

- **Polyuria :** Excessive urination
- **Polyphagia :** Increased appetite
- **Polydypsia :** Excessive thirst
- Loss of body weight
- Dehydration due to excessive urinary output
- Decreased resistance

Problems, which Diabetics encounter more often are:

- Heart Attacks, Strokes
- Gangrene of foot
- Repeated attacks of infections/boils
- Rapid Deterioration of Vision & Blindness
- Sexual Problems
- Kidney diseases
- Blurring of vision or double vision (Diplopia)

LONG TERM COMPLICATIONS WITH DIABETES MELLITUS

- **Retinopathy :** The blood vessels and lens of the eyes are swollen and dilated due to increase in blood glucose level, resulting in distorted vision. It has damaging effect on the optic nerve also.
- **Nephropathy :** Impaired kidney function
- **Neuropathy :** When blood sugar goes very high, nerves can produce pain specially in legs.

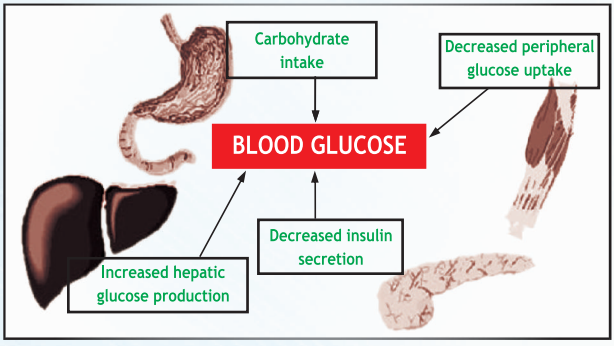
EFFECTIVE DIABETES MANAGEMENT

The chief tools in the treatment of diabetes are diet, exercise and suitable medication to keep the symptoms in control.

Insulin is the principal hormone that regulates uptake of glucose from the blood into most cells (primarily muscle and fat cells, but not central nervous system cells). Therefore deficiency of insulin or the insensitivity of its receptors plays a central role in all forms of diabetes mellitus.

Most of the carbohydrates in food are converted within a few hours to the monosacch-aride glucose, the principal carbohydrate found in blood and used by the body as fuel. The most significant exceptions are fructose, most disaccharides (except sucrose and in some people lactose), and all more complex polysaccharides, with the outstanding exception of starch. Insulin is released into the blood by beta cells (ß-cells), found in the Islets of Langerhans in the pancreas, in response to rising levels of blood glucose, typically after eating. Insulin is used by about two-thirds of the body's cells to absorb glucose from the blood use as fuel, for conversion to other needed molecules, or for storage.

Insulin is also the principal control signal for conversion of glucose to glycogen for internal storage in liver and muscle cells. Lowered glucose levels result both in the reduced release of insulin from the beta cells and in the reverse conversion of glycogen to glucose when glucose levels fall. This is mainly controlled by the hormone glucagon which acts in an opposite manner to insulin. Glucose thus recovered by the liver re-enters the bloodstream; muscle cells lack the necessary export mechanism.



Higher insulin levels increase some anabolic ("building up") processes such as cell growth and duplication, protein synthesis and fat storage. Insulin (or its lack) is the principal signal in converting many of the bidirectional processes of metabolism from a catabolic to an anabolic direction and vice versa. In particular, a low insulin level is the trigger for entering or leaving ketosis (the fat burning metabolic phase).

If the amount of insulin available is insufficient, if cells respond poorly to the effects of insulin (insulin insensitivity or resistance) or if the insulin itself is defective, then glucose will not be absorbed properly by those body cells that require it nor will it be stored appropriately in the liver and muscles. The net effect is persistent high levels of blood glucose, poor protein synthesis, and other metabolic derangements such as acidosis.

PATHOGENESIS OF TYPE 1 DIABETES MELLITUS

This form of diabetes results from a severe lack of insulin caused by an immunologically medicated destruction of beta cells.

Type-1 diabetes is an autoimmune disease in which islet of langerhans destruction is caused primarily by T-lymphocytes reacting against the poorly defined beta-cell antigens.

PATHOGENESIS OF TYPE-2 DIABETES MELLITUS

Environmental factors, such as a sedentary lifestyle and dietary habits, evidently play a role in occurrence of diabetes. Nevertheless, genetic factors are even more important than in type 1 diabetes.

The two metabolic defects that characterize type 2 diabetes are :

1. A decreased ability of peripheral tissues to respond to insulin (insulin resistant) and
2. Beta cell dysfunction that is manifested as inadequate insulin secretion in the face of insulin resistance is the primary event, and is followed by increasing degrees of beta- cell dysfunction.

Insulin Resistance: Insulin resistance leads to decreased uptake of glucose in muscle and adipose tissue and an inability of the hormone to suppress hepatic gluco-neogenesis. Functional studies in individuals with insulin resistance have demonstrated numerous quantitative and qualitative abnormalities of the insulin signaling pathway.

Beta Cell Dysfunction: Beta cell dysfunction in type 2 diabetes reflects the inability of these cells to adapt themselves to the long-term demand of peripheral insulin resistance and increased insulin secretion.

Several mechanisms contribute to beta cell destruction

- T-lymphocytes react against beta cell antigens and cause cell damage.
- Locally produced cytokines damage beta cells.
- Auto-antibodies against islet cells and insulin are also detected in the blood of 70% to 80% of patients.

Obesity and Insulin Resistance: The link between obesity and diabetes is mediated via effects on insulin resistance. Insulin resistance is present even in simple obesity unaccompanied by hyperglycemia, indicating a fundamental abnormality of insulin signaling in states of fat excess. The risk for diabetes increase as the body mass index (a measure of body fat content) increase. It is not only the

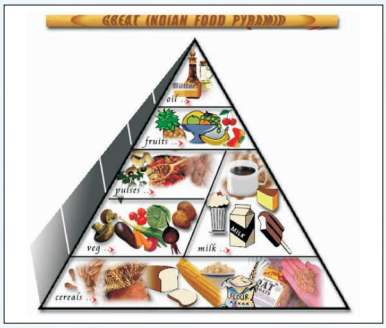
absolute amount but also the distribution of body fat that has an effect on insulin sensitivity: Central obesity (abdominal fat) is more likely to be linked with insulin resistance than are peripheral (gluteal/ subcutaneous).

MANAGEMENT OF DIABETES MELLITUS

I. DIETARY MANAGEMENT

It varies according to the severity of the disease, activity and metabolic needs. There are certain general principles for all diabetic diets:

- The caloric intake should allow the patient to loose or to gain weight as required.
- The protein recommended in the diet is normal
- Carbohydrates should be sufficient to prevent Ketosis.
- High fibre in diet helps in controlling diabetes by preventing excessive rise in blood glucose helps in decreasing blood cholesterol and triglycerides and also helps in reducing weight.



Foods to be avoided

- Root Vegetables: Potato, Yam etc.
- Fried foods and sweets
- Soft drinks and Alcoholic drinks
- Fruits like Mango, Banana, Chiku, Custard Apple, Grapes & Dates
- Oily, fried, frozen and fast foods should be avoided.

II. YOGIC MANAGEMENT

The Yogic practices are found to be useful in the management of Diabetes mellitus through various research studies. The aim of the Yogic treatment in the management of diabetes is of two fold:

a) To stimulate the pancreatic cells to produce adequate amount of Insulin

b) To reduce the Insulin resistance.

The practices prescribed for the Diabetic patients are as under:

- **Kriyas:** Kunjal, Vastradhouti, Kapalabhati, Agnisar and Nauli.
- **Asanas:** Tadasana, Katichakrasana, Pavanamuktasana, Sarvangasana, Matsyasana, Halasana, Ushtrasana, Gomukhasana, Ardhamatsyendra sana, Mandukasana, Paschimottanasana, Bhujangasana, Shalabhasana, Dhanurasana, Shavasana
- **Pranayama:** Nadishuddi, Suryabhedana, Bhastrika and Bhramari
- **Bandhas:** Uddiayana Bandha
- **Meditation:** Breath awareness, Om chanting and Om meditation.

Disclaimer : This literature is for general awareness about disease management through Yoga. It should not be considered as treatment prescription.

YOGIC PRACTICES FOR THE MANAGEMENT OF DIABETES MELLITUS


Kapalabhati Kriya


Kunjal

Sūryanamaskār


Nauli


Tadasana


Katichakrasana


Pavanmuktasana


Sarvangasana


Matsyasana


Bhujangasana

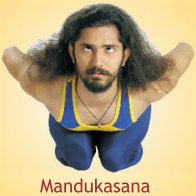

Shalabhasana


Dhanurasana


Paschimottanasana


Ardha Matsyendrasana


Ushtrasana


Mandukasana


Gomukhasana


Shavasana


Nadishodhana Pranayama


Bhramari Pranayama


Dhyana

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For further details please contact:

Director

Morarji Desai National Institute of Yoga

Ministry of AYUSH, Government of India

68, Ashok Road, Near Gole Dak Khana, New Delhi-110 001
Ph : 011-23730417-18, 23351099, Telefax : 011-23711657
Email : mdniy@yahoo.co.in Website : yogamdniy.nic.in

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