Diabetes Deceptivus

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The issue of diabetes was summed up by Boyd: "The more we know about diabetes, the less we seem to understand it." From times prechristian to the neomillennial, Boyd seems concise and relevant. An *International Textbook of Diabetes* Mellitus (Wiley, 1997) credits "two Indian physicians" Charak and Susruta as pioneers of the classification of diabetes into the currently accepted two types - juvenile, and maturity-onset, about both of which all pathies know so little that it would be intellectually honest to coin a new term *Diabetes deceptivus* - misleading, beguiling, delusive, illusory, tricky, questionable. Let us know that we know not that we know naught about diabetes deceptivus.

Diabetes connotes a siphon, from *dia* = through, and bainein = to go or fall. Aretaeus (30 - 90 A.D.), a Greek physician, gave the name diabetes for excessive urination - "something like passing of water by a siphon." Thomas Willis, the English physician after whom the intracranial arterial circle has been named, discovered the sweet taste of urine, to call it diabetes mellitus, and to distinguish it from diabetes insipidus, both mellitus and insipidus meriting the appellation deceptivus. Unhelpably, what now follows is on diabetes mellitus et deceptivus, to be abbreviated as DMD.

A few years ago, diabetologists from the world over in California, attempted a definition of DMD. After deliberations over 4 days, they gave it up as a bad job. DMD is a systemic syndrome, affecting protein/fat/carbohydrate as also arterial metabolism. The glucose-card is overplayed so that agents which are merely glucostatic are christened as anti-diabetics to the neglect of protein/fat/arterial aspects. Lewis Thomas, the most eminent medical philosopher of recent times, sums up historically: "Although the discovery of insulin fifty years ago made possible the survival of most diabetics who would otherwise have died in diabetic coma, the blood-vessel disorder which is a major aspect of the disease is unaffected by insulin and remains a mystery." This 1977 generalization is valid as of today.

While on the subject of insulin, some drama behind it is in order. Mering and Minowski, in the 1880s, produced canine DMD by removing the pancreas. Around the same time, Paul Langerhans described the pancreatic islets named after him. Frederick Banting, a fledgling Canadian surgeon, asked a junior intern Charles Best to team up with him to isolate in 1921, an extract that saved the life of Thompson, a juvenile diabetic and Banting rightly named the active agent as *insulin* - a gift of the pancreatic insula or islands. The fastest Nobel came to Banting, in 1923, but Best got neglected. It is to the credit of Banting's magnanimity that he gave exactly half of his Nobel-money to Best.

Another Frederick (Sanger) got his Nobel in 1958 for establishing the 51-amino-acid chain that comprises insulin.

From the celestial, it is time to move to the clinical. The kill-joy modern medicine has botched up our meals by denying sugar in the name of *DMD*, and butter in the name of Coronary. How hollow is this? Martin Fischer of Harvard found that "Many a diabetic has stayed alive by stealing the bread denied him by his doctor." No wonder, Louis MacNeice poetically bemoaned "The excess sugar of a diabetic culture/ Rotting the nerve of life and literature." Humanity's predicaments are that the brain only wants glucose

and the heart only burns the high octane fat, two vitals heavy-handedly banned by the medicos. Many an adult mind is starved of the vital glucose and many a fracture-neck-femur has resulted therefrom, because of the sugar-n-sweet that the mind was denied. Alex Comfort, the pioneer biologist-n-sexologist, has described *Anxiety-making* as the "curious preoccupation of the medical professionals." To add insult to all injury, the medicos have coined a new term DIABESITY, (on which conferences have started.) making you guilty sugar-wise and fat-wise.

Let us end with the twin immunological ironies that plague DMD. The insulin-dependent juvenile variety, forming a small percentage of the total, is ascribed to the loss of Beta-cells because of an auto-immune assault on the islets. Immunity, it seems, is no friend, but a potent foe, for there are n-number of maladies that are a gift of one's own immune system gone berserk. The maturity-onset DMD is ascribed to an excess of insulin, a sort of hyperinsulinemia, to which the body's insulin-receptors have grown resistant, possibly because of anti-insulin antibodies. *Et tu, Immunity!* Little wonder that the Nobel-laureate Burnet lamented the lack of definition of the much-abused term immunity. And Glemser exposed experts' hollowness when he generalized that the science of immunology is so advanced that one immunologist cannot comprehend what another is talking about. Let us know we know naught.

The genes have failed to mitigate our sense of helplessness, for DMD is turning out to be multifactorial or polygenic in origin, a way of saying that it is a herd of phenomena wherein an individual obeys the herd by taking upon herself/himself the manifestation of a syndrome called, *faute de mieux*, DMD. Neil, an eminent geneticist, worked on DMD's inheritance for a lifetime to sum up the exercise as "a geneticist's nightmare." DMD is not a disease, but a part of human growing, some prematurely, many naturally. Do not bemoan it unless it bothers you and bother it only to the extent that it bothers you.

Diabetes Mellitus - A Constitutional Dyscrasia

Abstract: In this write-up, we have attempted to explain different facets of this disease and the approach a scientific homoeopath should adopt so as to become rational prescribers of this healing art.

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With changing times, Diabetes Mellitus (DM), one of the most prevalent metabolic disorders has reached alarming proportions. It is rapidly occupying centre stage in medicine due to the speed with which it is spreading its tentacles. It is a collection of diverse morbid symptoms from different systems.

Homoeopathy has a wide scope in its management and can play an effective role not only in preventing DM but also curing it, if the patient opts for treatment early. Even in the stage of complications there are a host of effective

homoeopathic remedies that can palliate the suffering without any untoward effects.

ETIOPATHOGENESIS: DM is a symptom syndrome that is fabricated as a result of a permutation of a host of factors, notable being -

1. PREDISPOSING FACTORS: Which render an individual vulnerable to develop DM:

a) MIASMS - These are the fundamental factors responsible for rendering the individual susceptible for the disease. Initially, Tubercular Miasm - a blend of Psora and Sycosis is responsible for the development of the disease proper. After sometime, as the miasm is sensitized due to various external factors, syphilitic miasm takes hold of the economy.

I) When both parents are diabetic and when Tubercular Miasm is active, the fetus has 80% chances of getting DM after birth. The age and manifestations shall depend upon the environmental influences. Hence Juvenile DM is diagnosed years after the child is born.

II) If only one parent or grandparent has DM, then the fetus is born with DM diathesis. The baby may be fat, flabby, indolent, is easily fatigued, has erratic hunger, unhealthy skin, tendency to obesity, etc. All these are the expressions of Tubercular Miasm which points towards DM in the future.

b) REPEATED VACCINATIONS

c) SANGUINE TEMPERAMENT - Sensitive, brooding, introvert.

2. EXCITING FACTORS- Shocks, mechanical interventions, pregnancy, menopause, severe acute disease, drugs.

3. MAINTAINING FACTORS- Habits, addictions, rich diet, sedentary habits, job dissatisfaction, frequent symptomatic treatment, lifestyle, environment, etc.

INSULIN METABOLISM– When the tubercular miasm selectively affects the beta cells of Islets of Langerhans of pancreas, it leads to disturbance in Insulin metabolism. Insulin is a key player in the control of carbohydrate, lipid, protein and mineral metabolisms. Thus derangements in insulin signaling have widespread effects on many organs. Since Insulin is a protein hormone, its action on target organs is effectuated with the help of peripheral receptors that are embedded in the plasma membrane of target cells. Glucose is liberated from dietary starch or sucrose by hydrolysis in the small intestine. It is then absorbed into the blood. Elevated concentrations of glucose in blood stimulate insulin release. It acts on cells throughout the body to stimulate uptake, utilization and storage of glucose. The effects of insulin are:

1. Insulin facilitates entry of glucose into muscle, adipose and several other tissues except brain, RBC and liver
2. It stimulates liver to store glucose in the form of glycogen after it is absorbed from the small intestine. If glycogen accumulates to high levels further synthesis is strongly suppressed.
3. It decreases the concentration of glucose in blood.
4. In the absence of insulin, glycogen synthesis in the liver ceases and enzymes responsible for breakdown of glycogen become active. Glycogen breakdown is also stimulated by anti-insulin hormone Glucagon.
5. Insulin promotes synthesis of fatty acids in the liver which are exported from the liver as lipoproteins. The lipoproteins dissociate in the blood and provide free fatty acids for use in other tissues. Adipocytes use them to synthesize triglycerides. Insulin inhibits breakdown of fat in adipose tissue.
6. It stimulates the uptake of amino acids and is an anabolic hormone. When insulin levels are low, intracellular protein degradation starts.
7. It also increases the permeability of many cells to potassium and magnesium
8. Insulin activates sodium-potassium ATP pump in many cells. Thus due to dysfunction of a single hormone, we can see its effects on multiple metabolisms of carbohydrate at first (DM), then fat (obesity and dyslipidemia), protein (hypertension) and

minerals (complications of Metabolic syndrome)

SICKNESS EVOLUTION: DM evolves from DM diathesis to disease proper to its end results and finally metastatizes into other metabolic disorders.

A] PRE-DIABETIC STATE OR DIABETIC DIATHESIS

B] DISEASE PROPER

1. EARLY ONSET DM – Frequently presents in children and adolescents. Apart from the symptoms mentioned below, the first symptom may be coma or some other crisis.

2. LATE ONSET DM - Patients manifest any of the following symptoms – polyuria (frequent and profuse urine, nocturia), frequent drying of lips, polydipsia (increased thirst), polyphagia (increased appetite), weight loss inspite of adequate food intake, undue tiredness, tingling or numbness in the extremities, burning feet, generalized pruritis, pruritis vulvae, balanitis, delayed wound healing, impotency, premature cataracts, visual disturbances etc. Caution should be exercised while labelling persons with DM who show sudden rise in the blood glucose levels at time of physical (myocardial infarction, cerebrovascular accidents, acute infections, trauma etc) or mental stress. In any acute event there is always a transient physiological rise in blood sugar levels. Likewise caution should also be taken to diagnose patients as DM who are on drugs, which are known to increase blood glucose levels as steroids, thiazide diuretics, oral contraceptives, beta- blockers, phenytoin sodium etc.

C] END RESULTS- Retinopathy, Nephropathy, Neuropathy, Dermopathy, etc.

D] Finally when the Tubercular Miasm further evolves, the sickness is compelled to channelize in other dysmetabolic states as Hypertension, Hyperlipidemia, etc. Hence these diseases must be understood as part of an evolved sickness.

MANAGEMENT – At the Academy we manage a case of DM as follows:

1. In the first sitting there is a casual conversation with the patient to establish a rapport. If there are any acute symptoms, a short acting drug is selected on symptom similarity. The patient is appraised of homoeopathic working, the understanding of his disease, what is palliative and constitutional homoeopathy, how to give a homoeopathic history, etc. The homoeopath also notes if the patient is on some other drugs or not.

2. After 2-3 sittings, a detailed history-mental state, life space, physical generals, past, family and personal history is taken. The particulars with the ongoing treatment are noted.

3. The patients are then grouped into the following Case Types to decide the management strategies and approaches –

ACUTE CASE – Approach is to give relief of distress, give a sense of well being and start the suspended elimination process. No new symptom should appear.

CHRONIC CASES

1. NATURAL CURABLE CASE- When the patient is in diathesis stage or has many individualistic symptoms, the pathology is reversible, and there are few or no secondary symptoms, a constitutional deep acting anti-Miasmatic similimum on essential totality is administered.

2. INCURABLE CASE- The patient has gross pathological changes or an irreversible pathology or drug disease. A palliative remedy is selected on common symptoms

3. ONE SIDED CASES- When symptoms are few or multiple but incomplete, then drugs are prescribed on the basis of Family/Past history.

4. SETTLED CASES- Such patients have DM since a long time and are settled. They are kept on maintenance doses of symptomatic drugs.

5. MIXED CASES- These patients have multiple types of symptoms (primary, secondary, environmental, habits, etc. They are again managed symptomatically.

6. SILENT CASES– These are accidentally diagnosed on investigations done for some other reasons. They are treated at par with natural cases if a basic constitutional remedy can be selected.