

Quarterly NEWSLETTER SEPT-2021



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From The President's Desk



Dear Members of DRWA,

We are delighted to bring the latest issue of the DRWA newsletter for your perusal.

We request you to continue your inputs on a regular basis and enrich us with your experience. The editor Dr. Satpathy has worked hard, in a very short time to collate all articles for your viewing. We trust you will enjoy reading them.

We are proud to announce the achievements of our members, members' spouses and children, so please update us regarding your children's performance in the future.

With all good wishes, **Dr. Mary DCruz**



From the secretary desk



Hope all members are keeping good health in this prevailing Pandemic situation. Our organization has continued with all the academic activities either online, hybrid or physical, our last monthly CME was a physical meet following all Covid protocols of course.

We have planned our Mid Term CME on 25th September 2021 at Sundarbans with a Symposium on 'Defeat Diabetes' it will be a hybrid program with the participation of a good number of National faculties.

DRWA is an academic association continuously trying to raise the academic standards. So dear members we need your active participation in all our academic activities throughout the year, only your support and encouragement will help us reach the pinnacle.

Wishing you all remain in the pink of health and happy reading.



From the Editors' Desk

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Dr. Amit Kumar Dey

Message from Editor of this issue

I thank DRWA for giving me this opportunity to be the editor of this issue .

I am overjoyed by the content of the scientific materials sent by our colleagues and congratulate all for contributing towards our newsletter. I hope everytime our academic involvement will grow and we could bring out newsletter of substance.

Thanking all once again

Dr T. Satpathi MBBS, M Sc, Dip Diabetology, PG in Diabetology (Johns Hopkins Univ, USA), PG Dip in Cardiology RCP, UK), Diabetes Foot Edu Prog (ICP, India) CONSULTANT DIABETOLOGIST



How to choose Carbohydrates in our foods?

Dr Agnik Pal, MBBS, MD, IDECC, Dip Diabetology (RCP, UK), Dip Cardiology (RCP, UK) Asst. Professor, College of Medicine & JNM Hospital, Kalyani Consultant Physician and Diabetologist Email: agnik_pal@yahoo.co.in

Dietary risk factors are one of the most common causes of various non-communicable diseases. The role of dietary carbohydrate in diabetes is the subject of much debate – especially with respect to ideal amounts and types of carbohydrate. This relates to both type and quantity of carbohydrate consumed, with low carbohydrate diets increasing in popularity. However, it is important to take a complete dietery approach and not just in terms of single nutrients. Recommendations from the UK suggest 50% of total energy should come from carbohydrate for the general population; however, evidence does not support an optimal carbohydrate intake for people living with diabetes. Carbohydrate quality is important in terms of glycaemic index and fibre, and may have other health benefits; however, the quantity of carbohydrate is a more important predictor of glycaemic response. Besides, people with Type 1 diabetes can improve the accuracy of insulin dosing with carbohydrate counting and technology may also have a role to play in this, with the introduction of bolus advisor meters.

Carbohydrates can be defined as sugars, starches and fibre – often divided as simple (sugars) and complex (starches and fibre). Cereal and cereal based products make the largest contribution to daily carbohydrate intake, principally from white bread, pasta and rice. The greatest contributors to free sugar intake are sugars, preserves and confectionery and non-alcoholic drinks – each group making up about 25% of our free sugar intake. The World Health Organization (WHO) definition for free sugar includes all sugars that are added during food manufacturing and preparation as well as sugars that are naturally present in honey, syrups, fruit juices and fruit concentrates, The WHO issued dietary guidelines which recommend limiting free sugar intake to less than 10% of daily energy intake. The term "sugars," as applied to human diets, is a collective term for several different chemical species. Thus, "table sugar" is essentially pure sucrose, whereas fruit juice, honey and syrups contain mixtures of sucrose, glucose and fructose, and often oligo-saccharides of different size.

Each time we eat or drink something sugary or starchy, the blood glucose level in the body rises. Carbohydrates are digested at different rates and this has an effect on blood glucose levels. The measure of how quickly carbohydrate-containing foods result in a rise in blood glucose is known as glycaemic index (GI); it is a ranking from 1-100. Some of carbohydrates are quickly digested and cause quick and sharp rises in our blood glucose levels - they are called high GI foods and drinks. Low GI foods and drinks are more slowly digested and make our blood glucose rise more slowly. These are sometimes called 'slow release' carbohydrates. The rate of hydrolysis of food in the gastrointestinal tract and the rate of gastric emptying determine the absorption rate which, in turn, determines the extent and duration of the glucose rise after a meal. Circulating insulin levels are determined directly by pancreatic β -cell stimulation by absorbed products of digestion (ie glucose and amino acids) and indirectly by their action on incretins (eg. gut inhibitory peptide) released from gut cells. Neural and endocrine stimuli also play a role. The system is therefore responsive to the amount of carbohydrate and its rate of absorption.

On the other hand, the glycaemic load (GL) of food is a number that estimates how much the food will raise a person's blood glucose level and is measured by multiplying the glycemic index of the food in question by the carbohydrate content of the actual serving. **Glycemic load (GL) = Gl of a food x amount carbohy-**



drate in an average serving (g).

The link between high GI and high GL diets and diabetes may relate to glucose peaks and increased insulin demand. High GI foods lead to rapid rises in blood glucose and insulin levels. Hyperinsulinemia, in turn, may down regulate insulin receptors and therefore reduce insulin efficiency, resulting in insulin resistance. This condition may act in a vicious circle by increasing blood glucose concentrations and insulin secretion. Insulin resistance is a risk factor for Type 2 diabetes. Also, poor glucose control has been shown to result in a greater incidence of long term macrovascular and microvascular complications in both patients with Type 1 and 2 diabetes. Each 1% reduction in mean hemoglobin-A1c (HbA1c) is associated with approximately 21% reduction in risk for severe end points related to diabetes (eg mortality, myocardial infarction, heart failure, stroke, amputation, retinopathy etc)

High GI foods are characterized by fast-release carbohydrate and higher blood glucose levels, resulting in greater insulin demand. Foods with a high GI are not necessarily bad foods. For example, potato crisps have a medium GI but a baked potato has a high GI. Despite this, a baked potato is better for our health than potato crisps, which are higher in fat and salt. And all lower GI foods are not necessarily healthy like chocolate and ice cream have a low to medium GI rating. If we focus only on the GI of foods, we could end up eating a diet that's high in fat and calories, making us more prone to weight gain and heart disease.

So, the key is to use GI in the context of balanced eating. Here are some everyday examples of lower GI carbohydrate choices:

- Multigrain, granary, rye, seeded bread, sourdough bread
- New potatoes in their skins, sweet potato, yam, cold boiled potatoes
- · All pasta cooked until al dente, instant noodles
- Basmati rice, long grain or brown rice
- Bulgur wheat, barley, couscous, quinoa
- · Porridge, muesli, some low-sugar oats and bran-based cereals

Many low GI foods are a good source of fibre or whole grains. A healthy way to use the GI principles is to incorporate a range of lower GI carbohydrates that are also rich in fibre and low in saturated fat. For weight management, we also need to think about reducing our overall calorie intake by incorporating lower calorie foods into our meals and snacks.

British Dietetic Association suggests 10 tips to choose carbohydrates:

- Carbohydrates come in different forms and some are healthier than others.
- 'Slow release' or low GI carbs have been shown to help stabilise blood glucose levels.
- GI can be helpful in diabetes because lower GI meals and snacks can help to keep blood glucose levels steady (which helps to reduce the risk of long-term complications of diabetes).
- The amount of carbohydrate you eat is more important than GI in diabetes.



- If you have diabetes and want to know more about how to use GI to help regulate your blood glucose, ask your GP to refer you to a dietitian.
- Lower GI foods can only help you to manage your weight if they are eaten as part of a calorie-controlled diet combined with regular physical activity.
- Lower GI foods like whole grains, fruit, beans, lentils, and vegetables are generally lower in calories too.
- GI is about the physical make-up of a food and it is important to consider the mix of foods you eat, not just the GI value of the carbohydrates.
- Some lower GI foods (such as chocolate cake) may be high in fat or calories and so they are not a healthy choice.
- In general, high fibre lower GI foods such as beans, peas, lentils, porridge, muesli, fruit and vegetables are good choices and can help you keep to an overall healthy eating plan.

So, to conclude we should consider the overall balance of our meals by looking at labels and choosing foods that are lower in saturated fat, salt and sugar, and keeping an eye on our portion sizes. There is good scientific evidence to suggest that basing diet on low GI foods may help to control blood glucose levels for people with type 2 diabetes. Choosing low GI foods as part of a balanced diet can help to minimise the glycaemic variability. In the long term this can help reduce the risk of late complications of diabetes.



A case of T2DM with other comorbidities

Dr. Arindam Sur MBBS,MD,CCEBDM Consultant Physician & Diabetologist

Case History:

S.M. is a retired 61-year-old obese man with a 5-year history of type 2 diabetes. Although he was diagnosed in 2018, he had symptoms indicating hyperglycemia for 2 years before diagnosis. He had fasting blood glucose records ranging between 110–125 mg/dl, which were described to him as "borderline diabetes." He also remembered past episodes of nocturia. At the time of initial diagnosis, he was advised to lose weight, but he did not pay much heed to the advice.

Referred by his family physician to a diabetes specialty clinic, S.M. presents with recent weight gain, suboptimal diabetes control, and foot pain. He has been trying to lose weight and increase his exercise for the past 6 months without success. He had been started on glimiperide 1 mg every morning, but had stopped taking it because of dizziness, often accompanied by sweating and a feeling of restlessness, particularly in the late afternoon.

He also takes Atorvastatin 10 mg daily, for hypercholesterolemia (elevated LDL cholesterol, low HDL cholesterol, and elevated triglycerides). He has tolerated this medication and adheres to the daily schedule. He does not test his blood glucose levels at home and expresses doubt that this procedure would help him improve his diabetes control.

S.M. lives with his wife, 58 years of age and has two children. Although both his mother and father had Type 2 diabetes, S.M. has limited knowledge regarding diabetes self-care management and states that he does not understand why he has diabetes since he never eats sugar. In the past, his wife has encouraged him to treat his diabetes with herbal remedies and weight-loss supplements. During the past year, S.M. has gained 8 kg. Since retiring, he has been more physically active, doing morning walk 4 times a week and gardening, but was unable to lose more than 2–3 Kgs. He never consulted a dietitian and or has not been instructed in self-monitoring of blood glucose (SMBG).

S.M.'s diet history reveals excessive carbohydrate intake in the form of rice. He is an occasional drinker. He stopped smoking more than 10 years ago.

The medical documents that S.M. brings to this appointment indicate that his hemoglobin A1c (A1C) has never been <8%. His blood pressure has been measured at 150/70, 148/92, and 166/88 mmHg on separate occasions during the past year. S.M. has never had a foot exam as part of his primary care exams, nor has he been instructed in preventive foot care. However, his medical records also indicate that he has had no surgeries or hospitalizations and in general, he has been healthy for many years.

Physical Exam

A physical examination reveals the following:

- Weight: 96 Kg; height: 5"7";
- Fasting capillary glucose: 166 mg/dl,Blood pressure: 154/96 mmHg; Pulse: 88 bpm;
- Eyes: corrective lenses, pupils equal and reactive to light and accommodation, Fundi-clear, no retinopathy
- Thyroid: not palpable
- Lungs: clear
- Heart: Rate and rhythm regular, no murmurs or gallops
- Neurological assessment: diminished vibratory sense to the forefoot, absent ankle reflexes, monofilament felt only above the ankle



Lab Results:

Results of laboratory tests are as follows:

- Glucose (fasting): 168 mg/dl
- Creatinine: 1.2 mg/dl
- Blood urea nitrogen: 20 mg/dl Sodium: 141 meq/l
- Potassium: 4.3 meq/l
- Lipid profile:
- Total cholesterol: 242 mg/dl HDL cholesterol: 38 mg/dl LDL cholesterol : 144 mg/dl, Triglycerides: 207 mg/dl
- AST: 54 IU/I ,ALT: 69 IU/I ,Alkaline phosphotase: 156 IU/I
- HbA1C: 8.1%
- Urine ACR: 45 mg /gm of Creatinine

Diagnosis:

Based on S.M.'s medical history, records, physical exam, and lab results, he is assessed as follows:

- Uncontrolled Type 2 diabetes
- Obesity
- Hyperlipidemia (controlled with atorvastatin)
- Peripheral neuropathy (distal and symmetrical by exam)
- Hypertension (by previous chart data and exam)
- urine microalbuminuria
- Discussion

S.M. presented with uncontrolled Type 2 diabetes and a complex set of comorbidities, all of which needed treatment. The first task was to select the most pressing health care issues and prioritize his medical care to address them. Although S.M. stated that his need to lose weight was his chief reason for seeking diabetes specialty care, his elevated glucose levels and his hypertension also needed to be addressed at the initial visit.

He recognized that his glucose control was affected by large portions of rice and agreed to start improving dietary control by reducing his portion size by one-third during the week before his dietary consultation. Weight loss would also be an important first step in reducing his blood pressure.

Although his physical activity had increased since his retirement, it was fairly sporadic and weather-dependent. After further discussion, he realized that a week or more would often pass without any significant form of exercise and that most of his exercise was seasonal. Whatever weight he had lost during the summer was regained in the winter, when he was again quite sedentary. The patient was adviced basics of foot care by podiatrist

A first-line medication for this patient had to be targeted to improving glucose control without contributing to weight gain. Thiazolidinediones effectively address insulin resistance but have been associated with weight gain. A sulfonylurea can reduce postprandial elevations caused by increased carbohydrate intake, but they are also associated with some weight gain. When glimepiride was previously prescribed, the patient exhibited signs and symptoms of hypoglycemia (unconfirmed by SMBG). α -glucosidase inhibitors can help with postprandial hyper-glycemia rise by blunting the effect of the entry of carbohydrate-related glucose into the system. However, acarbose requires slow titration, has multiple gastrointestinal (GI) side effects, and reduces A1C by only 0.5–0.9%. Acarbose may be considered as a second-line therapy for S.M. but would not fully address his elevated A1C results. Metformin which reduces hepatic glucose production and improves insulin resistance, is not associated with hypoglycemia and can lower A1C results by 1%. Although GI side effects can occur, they are usually self-limiting and can be further reduced by slow titration to dose efficacy. After reviewing these options and discussing the need for improved glycemic control, he was prescribed metformin, 500 mg twice a day subject to up-titration.

He was started with ACE inhibitor enalapril 5 mg daily. The use of an ACE inhibitor was indicated both by the level of hypertension and by the presence of microalbuminuria. The dose of Atorvastatin increased to 20 mg once daily.



Diabetic Foot: A well-known but less well-attended complication of diabetes mellitus

Dr. Arjun Baidya

Diabetes mellitus is a chronic metabolic disorder which exerts its effects over a period and manifests with multi-organ involvement leading to complications. There is clear evidence that onset and progression of chronic complications is associated with duration and control of diabetes [1-3]. In India, diabetic foot ulcers (DFUs) affect 15% of diabetics during their lifetime. Among all diabetics in India, 25% develop DFUs, of which 50% become infected, requiring hospitalization while 20% need amputation. DFUs contribute to approximately 80% of all non-traumatic foot amputations in India, annually. Patients with a history of DFU have 40% higher 10-year deaths rate, than those without [4].

Over time, neurological complicationsespecially diabetic neuropathy creeps up mainly due to nerve damage and manifests with tingling and pain, and further leads to numb feeling in feet. In other side, blood flow in feet lowers due to sustained hyperglycemia which make it hard for a sore or an infection to heal which might lead to gangrene. Amputation of toe, foot, or part of leg is a common phenomenon when gangrene and foot ulcers that do not get better with treatment. Any deformity occurring in a foot with other risk factors increases the ulcer risk. Clawing of the toes is common, leading to increased metatarsal head pressures that, in neuropathic patients, may result in breakdown due to repetitive moderate stress to an insensate area. Other examples include Charcot deformities and hallux valgus. Even due to nerve damage from diabetes causes Charcot's foot which can cause feet to have an odd shape, such as a "rocker bottom." An important prelude to proper management of the diabetic foot is the correct diagnosis of its two main syndromes; the neuropathic foot, in which neuropathy predominates but the major arterial supply to the foot is intact, and the neuro ischaemic foot, where both neuropathy and ischemia resulting from a reduced arterial supply, contribute to the clinical presentation. There are numerous functional abnormalities which may be important for structural abnormalities which includes increased blood flow, widespread vascular dilation, increased vascular permeability, impaired vascular activity, and limitation of hyperemia.

Major advances in last decade have led to improved outcomes in foot ulcer management and reduced numbers of amputations [5]. Increased interest in the diabetic foot has resulted in systematic reviews [6-8], guidelines [9] and consensus development [10,11]. These reports have stressed the importance of early recognition of the 'at risk' foot, the prompt institution of preventive measures and the provision of rapid and intensive treatment of foot infection in multidisciplinary foot clinics. Such measures can reduce the number of amputations in diabetic patients. Successful management of the diabetic foot needs the expertise of a multidisciplinary team which should include diabetes specialists, podiatrist, diabetic nurse, orthotist, radiologist, and surgeon working closely together, within the focus of a diabetic foot clinic.

Infection is rarely a sole factor but often complicates neuropathy and ischemia and is responsible for considerable tissue necrosis in the diabetic foot. Effective neutrophil microbial action depends on the generation of several oxygen-derived free radicals. These toxic species, which include the superoxide, are formed during the respiratory burst activated after chemotaxis and phagocytosis. In diabetes, especially if poorly controlled, deficiencies in neutrophil chemotaxis, phagocytosis, superoxide production, respiratory burst activity and intercellular killing have all been described. Several other conditions are known to be associated with an increased risk of foot ulceration. Visual impairment because of retinopathy is an established risk factor for foot lesions. Perhaps the most high-risk group for ulceration is the dialysis population.



It can be safely presumed that patients at all stages of nephropathy have increased risk of ulceration. Dialysis treatment is an independent risk factor for foot ulceration.

It is important to assess the neurological, vascular, dermatological, and musculoskeletal status of people with diabetes at least annually. The American Diabetes Association (ADA) developed a Comprehensive Foot Examination and Risk Assessment that can be performed rapidly with minimal equipment [12,13].

After assessment of the foot, outlines suggested indications, priorities, and timelines for referral based on ADA guidelines [13]. The table below shows ADA patient risk categories (i.e., very low, low, moderate, and high risk) and follow-up recommendations.

Priority	Indications	Timeline	Suggested Follow- up
URGENT (active pathology)	 ! Open wound or ulcerative area, with or without signs of infection ! New neuropathic pain or pain at rest ! Signs of active Charcot deformity (red, hot, swollen midfoot or ankle) ! V ascular compromise (sudden absent D P/PT pulses or gangrene) 	Immediate referral/consultatio n	A s determined by specialist
HIGH (ADA risk category 3: the diabetic foot in remission)	 Presence of diabetes with a previous history of ulcer or lower- extremity amputation Chronic venous insufficiency (skin color change or temperature difference) 	Immediate or "next available" outpatient referral	Every 1–2 months



MODERATE (ADA risk category 2)	-! PAD ± LOPS -! DP/PT pulses diminished -! Presence of swelling or edema	Referral within 1–3 weeks (if not already receiving regular care)	Every 2–3 months
LOW (ADA risk category 1)	 LOPS ± longstanding, nonchanging deformity Patient requires prescriptive or accommodative footwear 	Referral within 1 month	Every 4–6 months
VERY LOW (ADA risk category 0)	No LOPS or PAD Patient seeks education on topics such as routine foot care, athletic training, appropriate footwear, or injury prevention	Referral within 1–3 months	At least annually for all people with diabetes

Patients who present with tissue loss are assigned to a higher risk category. In suchcases, the overall degree of limb threat should be assessed.

The three key factors associated with limb loss include degree of tissue loss (wound severity), severity of ischemia, and severity of foot infection. The acronym WIfI can be used as shorthand for these factors, which can assist the health care team in describing patients' overall limb threat status [14,15].

If the foot is normal and not at risk, the patients does not have the risk factors that render him vulnerable to foot ulcers (neuropathy, ischemia, deformity, callus, and oedema). If the foot is at high risk, the patients has developed one or more of the risk factors for ulceration of the foot. Patients when develops foot ulcer, ulceration in most of the cases is on the plantar surface in the neuropathic foot and on the margin in the neuro-ischemic foot. In few complicated cases, the ulcer gets infected with the presence of cellulitis, which can complicate both the neuropathic and the neuro-ischemic foot. In the neuropathic foot, infection is usually the cause of necrosis. In the neuro-ischemic foot, infection is still the most common reason, although severe ischemia can lead to necrosis directly.



At each stage, it is necessary to take control to prevent progression. Management will be considered under the stages such as wound control, microbiological control, mechanical control, vascular control, metabolic control, and education. Metabolic control is important at every stage. Tight control of blood glucose, blood pressure and blood cholesterol and triglycerides should be achieved to preserve neurological and cardiovascular function. Advice should be given to stop smoking. In higher stages, metabolic decompensation may occur in the presence of infection and intensive management of the diabetic state is often required. As education, advice on basic foot care including nail cutting techniques, the treatment of minor injuries and the purchase of shoes should be given. Patients who have lost protective pain sensation need advice on how to protect their feet from mechanical, thermal, and chemical trauma. Educational programs involving behavioral contracts and organizational intervention for health care providers have shown a significant reduction in foot ulceration at one year follow up [16].

The diabetic foot is often an inching painless surprise that holds in its dark portals a soon rising flood of complications. It is a quiet dread of disability, long stretches of hospitalization, mounting impossible expenses, with the ever-danglingend-result of an amputated limb.

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A perspective on MSK and DM

Dr. Bijay Patni Founder, Diabetes Research Welfare Association

Diabetes mellitus (DM) is a multisystem disease that affects millions of people worldwide. The vascular and cardiac effects of DM have been well-studied and understood but controversy remains about the prevalence of musculoskeletal (MSK) conditions in patients with DM.

MSK disorders often accompany chronic diseases such as diabetes mellitus (DM), which is a multisystem disease that poses a major public health problem.

The most common MSK disorders were OA, shoulder capsulitis and CTS (carpel tunnel syndrome). The 2 factors associated with significantly increased prevalence of MSK conditions were age older than 50 years and dyslipidemia. High prevalence of upper extremity MSK conditions in patients with diabetes, including flexor tenosynovitis (5%-36%) and diffuse idiopathic skeletal hyperostosis (13%-40%). Diabetic adults also had a higher prevalence of chronic low back pain (LBP) compared with nondiabetic adults.

Despite the lack of association between duration of DM, HbA1c, and chronic MSK conditions, patients with either type 2 DM or unclassified DM were 1.5 times more likely to report chronic widespread MSK conditions

Research has shown that OMT can be of benefit in the previously mentioned MSK disorders such as carpal tunnel and adhesive capsulitis. A 2018 systematic review13 on knee OA found that OMT with and without exercise resulted in reduced pain, increased function, increased physical performance, and a short-term benefit in range of motion with studies showing an effect ranging from 3 weeks to 1 year.

Reduced pain due to OMT has also been reported to reduce analgesic use for MSK disorders in patients with DM. Use of NSAIDs has been linked to increased risk of cardiovascular events and chronic kidney disease in patients with DM, which increases mortality in such patient population. Therefore, a comprehensive screening and appropriate treatment of MSK conditions in patients with DM is critical to optimize their possible outcomes. Osteopathic manipulative treatment (OMT) and lifestyle modifications are beneficial for DM management and prevention.

Conclusion:

A link between DM and certain MSK conditions has been identified in the literature. Studies have also shown that OMT can be effective in improving symptoms or reducing pain in these comorbid MSK conditions. Although promising research has been completed, further investigations are needed to establish the exact link between DM and MSK conditions because the role of HbA1c as an indicator for MSK abnormalities remains unclear. An understanding of the potential bidirectional relationship between DM and MSK disorders and the role of OMT may help guide the osteopathic physician in the appropriate application of OMT and exercise therapy for patients with DM. Increasing mobility and decreasing pain with OMT and exercise therapy can help patients with DM, and hopefully mitigate cardiovascular, neural, renal, visual, and MSK complications.



Covid vaccination: as I see-

Dr. Bijay Patni Founder, Diabetes Research Welfare Association

My inputs...

- 1. If people have received and still at higher risk should be given a booster dose 6 months of post second dose.
- 2. If people get covid post first dose the second shot can be delayed by 6 months.
- 3. If people get serious covid before any vaccination, natural antibodies is good enough to protect for 6 months or more so vaccination can be delayed for at least 6 months but if symptoms were most asymp tomatic then a three month period is good for first vaccination.
- 4. All vulnerable group or at risk group should be double vaccination within 6-8 weeks of 1st dose.
- 5. All adults (Non vulnerable) should be first vaccinated with first dose at the earliest.
- 6. Children below 18 yrs should be vaccinated if they are at risk due to physical condition.
- 7. All staff of schools should be vaccinated twice before they are allowed for physical schooling
- 8. Vaccination should be completed first in tier 1, Tier 2 and tier 3 cities at rapid pace.
- 9. Vaccination at far flung places is only for photo session. They should be the last group to be vaccinated.
- 10. All those who come to cities for work should be vaccinated at their workplace rather than at the villages from where they come
- 11. Village vaccination is must but less priority. All adults should be vaccinated with 1st dose initially.



Gender differences in Cardiovascular Disease

Dr. Lily Rodrigues

This article is to highlight the differences of CVD in women as compared to men, particularly womenliviwith Diabetes .It is high time that we understand the gender differences and therefore become smarter in managing both.

As per the AHA update of 2004: 38.2 million women are suffering from CVD and much larger population in women is at risk. Heart disease is no1 and stroke are no 3 are killers in women.1 in 30 die of breast cancer but 1 in 2.5 die of cardiovascular disease and stroke every year.66,000 more women die of Cardiovascular disease as compared to men which is 54% and in men it is about 46%.

Women develop disease 10-20 years later than men but if younger women do develop heart disease they suffer more malignant clinical course. Diabetes and HTN has more roles to play in women, and clinical manifestations and treatment responses differ in both genders.

- Type 2 diabetes mellitus (T2DM) is a common co-morbidity in Heart failure (HF) patients and is associated with worse prognosis.
- The effect of T2DM on cardiovascular complications seems to have a sex- specific interaction. Female sex
 confers special protection against cardiovascular disease, but this protection is lost in the presence of
 T2DM.
- Diabetes is also associated with a significant increase in HF incidence in men and women, although diabetic women have a greater relative risk of developing heart disease/HF. The reasons are:
- 1. Women receive less intense glycemic control and treatment of T2DM and HF compared with men.
- 2. We may also speculate that T2DM and HF are diagnosed at later stages in women.
- 3. Women frequently have confounding factors such as obesity and lower natriuretic peptides, which may delay correct diagnosis and treatment
- 4. Estrogen affects collagen synthesis and degradation, inhibits the renin-angiotensin system and the loss of its protective mechanisms may render the heart of postmenopausal women more vulnerable.
- 5. Other gender differences in underlying mechanisms identified include calcium handling, the NO system, and natriuretic peptides



Women have smaller coronary arteries

After correcting for body surface area, womens' arteries are smaller

This can seriously affect symptoms from anything that reduces diameter

- Stenosis
- Endothelial dysfunction



Estrogen and heart disease:

Natural estrogen is protective for the heart prior to menopause; therefore South Asian women tend to get heart attacks 10 to 15 years later than men. However after menopause, with declining estrogen levels woman's risk for heart disease not only approaches but surpasses that of a man with similar risk factors once a women is greater than 75 years of age.

Post-menopausal women also have a poor lipid profile with higher LDL (bad) cholesterol, more harmful dense and smaller LDL particles, lower HDL (good) cholesterol, and higher triglycerides. South Asians also have low levels of a very important type of good cholesterol called HDL2b, with nearly two thirds of South Asian women deficient.

Long-term hormone replacement therapy often used to be prescribed to relieve post-menopausal symptoms and was believed to reduce the risk for heart disease. The largest randomized, scientific study to date, however, found a small but significant increase in heart disease in postmenopausal women using combined (both estrogen and progestin) hormone therapy. If you are considering estrogen supplementation for post-menopausal symptoms such as hot flashes, speak to your doctor about the risks versus benefits of estrogen supplementation

Heart disease risk factors in women:

Similar to South Asian men, women have both standard and non-standard risk factors for heart disease. Standard risk factors include high LDL cholesterol, low HDL cholesterol, diabetes, family history of heart disease, hypertension (BP), lack of physical activity, and tobacco smoking. Smoking and diabetes in particular has a stronger impact on heart disease risk in women than in men.

Metabolic syndrome (high lipids, abdominal obesity, diabetes, high blood pressure) in particular is a stronger predictor for heart disease in South Asian women than their male counterparts. A South Asian woman with an abdominal circumference over 32 inches is considered obese, while this number is 36 inches for other ethnic groups.

Women who have had recurrent miscarriages (including those with elevated homocysteine levels) are at particularly high risk for heart disease. In addition, non-standard risk factors such as elevated lipoprotein(a) and C-reactive protein (CRP) are important in women



Based on recent USA statistics the prevalence of HF among older women (> 75 years) is 10.9% and in men of the same age group is 9.8%.

- Myocardial infarction (MI) as an etiologic factor for HF which is detrimental for both genders. However in women within six years of acute MI, 46% of women are disabled because of HF, as compared to a 22% rate of disability in men.
- The prevalence and incidence of HF are increasing in Western countries, particularly in individuals older than 80 years of age.
- The life time risk for developing HF according to data from Framingham Heart Study (FHS), independent of gender, is one in five for those less than 40 years old.
- In the same cohort, at the age of 40, the lifetime risk of HF without myocardial infarction is 1 in 9 in men and 1 in 6 for women. Over the past 20 years in this mainly white cohort, incidence of HF was rising faster in women than in men (9% vs. 6%).
- Women in the Studies of Left Ventricular Dysfunction (SOLVD) registry had a higher one year mortality rate (22%) than men (17%).
- Difference has been attributed to hypertension being the most common etiology for HF in women, whereas myocardial infarction is the most common factor causing HF in men.
- Women with HF tend to be older than men with HF and may have higher level of co-morbidities at base line.
- Hypertension and MI together account for about three quarters of the population-attributable risk of HF.
- According to FHS, the hazard for developing HF in hypertensive compared with normotensive subjects was about 2-fold in men and 3-fold in women.
- There was a significant association between HTN and HF in both men and women.
- The risk doubled with blood pressure (BP) \geq 160/90 mm Hg compared to BP < 140/90.
- HTN had the highest population attributable risk (PAR) of all risk factors; 39% for males and 59% for females.
- The PAR for MI was 34% for men, 13% for females and for DM was 6% for men and 12% for females.

American heart association guidance for the prevention of CVD in women

- Limit the intake of SALT
- Use 500ml of oil / person / month
- Include Heart Friendly foods like almonds, walnuts garlic, oats, fish, onion, soybean in your diet.
- Drink plenty of fluids like buttermilk, lemon water, coconut water and plain water.
- Include plenty of fruits and vegetables
- Aerobic At least 150 minutes / week
- Muscle Strengthening Alternate days
- Yoga, Suryanamaskar Everyday 20 min
- Pranayama, Meditation, Breathing exercise on a regular basis



Walking is the best medicine

Dr. M.H.Sanwarwalla Secretary. DRWA

All national and international guidelines recommend as first line management of diabetes whichever type, diet exercise and life style modifications. It is taken for granted and as physicians we are more concerned with the glucose levels, comorbidities, HbA1c, time in range etc. Patients too do not give as much emphasis to lifestyle modifications, they expect the physician to do the needful, prescribe a magic remedy.

If you look at the RSSDI defeat diabetes effort the emphasis is on diet, exercise lifestyle modification, early detection, screening of population there by preventing or delaying onset of full-fledged diabetes.

We organize walk for Diabetes every World Diabetes Day but what is walking all about?

Walking is the most ancient but still the best modern exercise. 'It is one mode of human locomotion by which a man proceeds on his own two feet, upright, erect as a man should be, not squatting on his rear haunches like a frog ' said Edward Abbey There is a lot to know about walking, but first why should one walk? What evidences are there to prove that it is one of the best forms of exercise?

Diabetes prevention program showed that walking 150 mins per week and losing 7% of your body weight could reduce your risk of developing diabetes by 58%. Walking is an excellent aerobic exercise and strengthens your heart. Nurses Health study showed 72,488 nurses who walked 3 hours or more per week reduced their risk of coronary arterial disease by 35%. Studies have shown that regular walking can reduce depression and improve cognitive brain function, which means it is good for your brain.

It is also good for you bones and reduces risk of breast and colon cancer. Walking improves overall fitness, appearance, and so on and so forth the list will continue and if you continue reading when will you go out there and walk suffice to say that walking is an excellent exercise.

There are different types of walking but I will write about the normal brisk walk. Tips on walking technique include:

Legwork – in an attempt to walk fast don not over stride, biomechanically it is inefficient and will lead to early fatigue concentrate on a powerful push off while front foot is placed close to the body.

Footwork- walk heel to toe to increase speed, contact ground with heel and push off with your toes.

Hip – Twist your waist by rotating forward and backward.

Body – Keep it upright.

Arms – keep elbow at 90degree, hands relaxed and swing them close to the body without crossing the midline, increasing arm swing will automatically increase speed.

Head Neck and Shoulders – Keep them relaxed, head upright and eyes looking forward.

It is also important to know your foot type namely flat foot, high arch foot or neutral. This is particularly



important with regards to the type of walking shoes one must purchase.

Some tips for purchasing appropriate shoes:

- Shoes should feel comfortable when you first wear them, don't buy shoes if seam, stitching can be felt as these will lead to callous/blister formation.
- Allow one-half inch between end of longest toe and shoe end.
- Always wear both shoes before purchasing buy the larger size if one foot is larger than the other.
- Shop at the end of the day when your foot is at its largest as feet swell during the day.
- Breathable shoes are more comfortable than leather shoes.
- Buy from a reputable store.

Replace shoes which hurt or are worn out at the earliest.

If you are a first timer start with 5 minutes forward and 5 mins return, gradually increase by 3 minutes per week till you are doing about 30 minutes. As your fitness improves you can do power walking, interval training etc. A good average speed of walking is 3-4 miles per hour. treadmill and outdoor walking yield the same benefits. How much to walk? American College of Sports Medicine recommends 20-40 mins of continuous activity 3-5 times in a week at 60-90% of Maximal Heart Rate.

The question frequently asked is which is better walking or jogging? Risk of injury is low for walking, about everyone can do it, burns calories, maintains health and fitness, jogging yield the same benefits but there is more impact on your knee, hip and other joints. Choice is yours.

"Walking is mans best medicine," said Hippocrates so what are you waiting for get started and advise your patients too.



Finding Peace & Joy

Dr. Mary DCruz President DRWA

Root of health is in the Brain The trunk of health is in Emotion The branches and leaves are the Body The flower of health blooms when all parts work together.

Kurdish Folk Wisdom.

Anything that costs your peace is too expensive. We all yearn for peace of mind at the end of the day and all through the day. We need to slow down the mad momentum of the mind with its raging storms and emotions. We need to stop all unnecessary, superfluous and shallow activities.

Most of us are busy attending to patients, attending webinars, carrying out mundane activities through the day. A constant 'busyness' keeps us under constant stress and the resultant chronic stress gives rise to many of the NCDs- Non-Communicable Diseases- like obesity, diabetes, hypertension and CVD.

Try NIKSEN

The Dutch people practice Niksen which means 'doing nothing'. It means to be idle or do something without any utility. It is switching off, doing nothing and spending time with yourself. It is an effective remedy for stress and anxiety. Meditation is different from Niksen, because we are required to be present in the moment and observe our thoughts usually with eyes closed.

Do few things but do them thoroughly- Eckhart.

Build healthy habits gradually and consistently

We all live by habits- get up, brush, bathe, pray, breakfast. It is a routine we follow everyday so why is a new habit difficult to follow?

- Probably lack of consistency.

1. Stack your habits

To form a new habit tie it to an existing one like a morning routine of tea or coffee, newspaper reading can be linked to a 5 minutes meditation or squats while brushing or stand on one feet for balance. End of the day yoga and meditation is helpful.

2. Do it everyday

Researchers found that the amount of time it took for a task to become automatic- a habit- ranged from 18-254 days. Median is 66 days. Habits take long to form but form faster if we do more often. Start small- few jumps, yoga rather than thrice a week gym.

3. Start small

Big behavior changes are challenging. Start small- sit ups, jumping jacks, sleeping in Track suit to get up and go early.

4. Reward yourself

Reinforce an exercise habit by rewarding yourself- not with food but with music, exercise, running or picnics.



Pancreatic Exocrine Insufficiency In Diabetes Mellitus

Dr. Mohsin Aslam, MD. Asian Institute of Gastroenterology, Hyderabad

Introduction

Pancreatic exocrine insufficiency (PEI) is frequently associated with diabetes, with high prevalence in either insulin-dependent or insulin-independent patients. Exocrine pancreatic failure has often been perceived as a complication of diabetes. In contrast, recent clinical observations lead to the notion that nonendocrine pancreatic disease is a critical factor for development rather than a sequel to diabetes.

PEI is present in about 50% of patients with Type 1 diabetes mellitus (T1DM) and 30–50% of Type 2 diabetes mellitus (T2DM).

Clinical Consequences of Exocrine Pancreatic Insufficiency in Patients with Diabetes

Chronic pancreatitis: without major clinical symptoms is present in a high percentage of patients with diabetes. Chronic pancreatitis and exocrine dysfunction have been associated with impairments of the incretin system.Glucose-dependent insulinotropic polypeptide (GIP) and Glucagon-like peptide 1 (GLP-1) secretion is reduced in patients with steatorrhea due to pancreatitis and can be normalized by enzyme replacement therapy.

Steatorrhea: Patients with diabetes and PEI as measured by FEC develop overt steatorrhea in 60% of the cases. Even in patients considered to have normal exocrine function by FEC, steatorrhea can sometimes be present. In patients with diabetes and pancreatic exocrine insufficiency, meteorism and stool texture correlate with steatorrhea, whereas abdominal pain and stool frequency do not.

Malabsorption: Many patients with PEI manifest some degree of fat malabsorption, regardless of the presence of symptoms. Since clinically overt steatorrhea is usually not observed until over 90% of exocrine pancreatic functions have vanished, exocrine pancreatic insufficiency and maldigestion might remain undetected. However, the relevant maldigestion, which is present in the majority of patients with PEI, may cause qualitative malnutrition. This is especially important concerning the absorption of fat-soluble vitamins (A, D, E and K).

Vitamin D deficiency is seen in > 90% of patients with PEI. High rates of exocrine pancreas dysfunction have been reported in patients with osteoporosis and vitamin D deficiency. Measuring serum-25-hydroxyvitamin D levels and supplementing deficient patients might be beneficial.

Diagnosis of Pancreatic Exocrine Insufficiency in Diabetes Mellitus

It is often difficult to detect PEI in patients with diabetes in routine clinical practice. Majority of these patients are usually asymptomatic in the early stage of PEI. The classical symptoms of steatorrhea and weight loss only tend to occur in patients with very severe PEI.

There should be a high index of suspicion for PEI in diabetics, especially in patients presenting with loose bowel movements, abdominal discomfort and flatulence.Patients with diabetes often describe symptoms of fatigue and difficulty controlling blood glucose levels.



PEI should be suspected in patients with long standing type 1 and type 2 DM. Other factors that have also been shown to increase the incidence of PEI in diabetic patients include

- 1. Poor glycemic control
- 2. Insulin dependence
- 3. Elderly age
- 4. Presence of microangiopathy and autonomic neuropathy

A number of other causes such as gastroparesis, celiac disease, and side effects of blood glucose lower-

ing medications should be excluded before considering PEI.

Diagnostic Tests for Pancreatic Exocrine Insufficiency

Since the last two decades indirect pancreatic function tests are available. Especially the measurement of fecal elastase concentrations (FECs) that can easily be performed in any setting and demonstrate PEI in diabetic patients. FEC of < 100 ug/g is diagnostic of severe PEI.

Direct Function Tests

- 1. **72 hours fecal fat estimation:** is a gold standard test but has several disadvantages like 72 hours stool collection, patient has to be on 100 g of standard fat diet and should not be on pancreatic enzyme replacement therapy (PERT) and it is not specific for pancreas.
- 2. **Cholecystokinin/secretin stimulation:** is the gold standard for scientific evaluation of PEI, but it is invasive and complicated.
- 3. **Endoscopic pancreatic function test:** is useful in diagnosing early chronic pancreatitis, cause of malabsorptive diarrhoea but has disadvantages like it is time consuming, invasive, unable to quantify and requires further validation.
- Secretin MRCP test. is useful in evaluation of acute & chronic pancreatitis and pancreatic neoplasms. Disadvantages are subjective nature of reports and there are no large trials supporting it. Indirect Function Tests
- 1. *Fecal chymotrypsin activity:* is good for compliance control as this test needs single stool sample but has several disadvantages like low sensitivity, not for mild PEI, watery stools decrease enzyme activity and PERT must be discontinued.
- 2. *Fecal Elastase 1 concentration:* has the advantage of single stool sample and patient can be continued on PERT, but it has poor sensitivity in mild PEI, watery stools and small bowel disease.
- 3. C-mixed triglyceride breath test: is useful even in mild PEI cases, detects fat maldigestion with sensitivity of > 90%, but further validation needed and is not available in India. Management of Pancreatic Exocrine Insufficiency in Diabetes Mellitus The mainstay of treatment of PEI in diabetes is PERT. Since there are scant data or guidelines on PERT specifically in patients with diabetes, treatment should be based on literature pertaining to treatment of PERT in chronic pancreatitis. Several pancreatic enzyme preparations have been tested and used extensively for treating PEI associated with pancreatic diseases. Recent recommendations support the use of PERT in patients with diabetes and PEI.



For optimal digestive action, a pancreatic enzyme preparation should survive the gastric acidic milieu, get released into the duodenum along with chyme, and contain the correct dose of lipase, which is the most crucial component of the preparation.PERT is best given with meals rather than before or after meals

Impaired GLP-1 secretion, in patients with diabetes and PEI can by normalized by pancreatic enzyme supplementation and help in better control of diabetes. Besides helping to control symptoms of steatorrhea, PERT also seems capable of preventing qualitative malnutrition and metabolic complications. Routine nutritional assessment of patients with PEI is important PERT helps to improve weight and Quality of Life.

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Remembering Centenary year of Insulin

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Insulin's centenary year appears to be 2020 if you appreciate the birth of an idea.

Everything begins with an idea." - Earl Nightingale

Is Oct 31, 1920, is an appropriate date for tribute, if so let us reverberate it now, November 14 is the World Diabetes Day and this month is so precious-Robert A Hegele, Grant M Maltman

"At 2:00 h on Oct 31, 1920, Frederick G Banting, a surgeon practising in London, ON, Canada, conceived an idea to isolate the internal secretion of the pancreas."



Consideration to commemorate insulin's centenary

May 17, 1921,

• Frederick G Banting and Charles H Best (under the supervision of John J R Macleod) commenced preclinical studies at the University of Toronto, Toronto, ON, Canada.

Aug 3, 1921

• Banting and Best's crude extracts from the pancreas of a dog first showed activity in reducing hyperglycaemia in a pancreatectomised dog.

Jan 23, 1922

• Leonard Thompson, a 14-year-old patient with type 1 diabetes at Toronto General Hospital, Toronto received the first dose **The Discovery of Insulin**

We recount the events before and after the morning of Oct 31, 1920, which transformed the field of endocrinology.





Frederick Grant Banting

- Born on Nov 14, 1891, in Alliston, ON, Canada, a village 70 km north of Toronto,
- In 1912, he was transferred to the Faculty of Medicine and chose surgery as his specialty.

• His classmate was Norman Bethune, who later served in Mao Zedong's army and helped to reform medical care in China.

- Banting completed his accelerated medical degree in December, 1916, and reported for duty as medical officer in the Canadian Army Medical Corps. Arriving in England in spring 1917
- Returned to Canada in early 1919
- Banting worked at the Toronto Military Orthopaedic Hospital, Toronto
- Started his own practice but business was slow from the outset, he cared for an average of **two patients per month**.

Why 1920, October 30 is memorable? EUREKA MOMENTS

- · An idea can change the life
- Oct 30, 1920, Banting dropped by the medical school library to review journals in preparation for his lecture on the pancreas
- He noted an article of interest in his copy of the November issue of Surgery, Gynaecology and Obstetrics. When he saw an article by Moses Barron of the University of Minnesota, Minneapolis and Saint Paul, MN, USA.
- Barron had published four case reports; a stone blocked the pancreatic duct, leading to atrophy of the exocrine pancreas, but preserving the islets of Langerhans.
- The patient did not develop diabetes
- Banting read it with great interest. After reading this article---Banting seemingly had a so-called eureka moment.
- Banting later wrote "It was one of those nights when I was disturbed and could not sleep. I thought about the
 lecture and about the article...Finally about two in the morning after the lecture and article had been chasing
 each other through my mind for some time, the idea occurred to me. I got up and wrote down the idea and
 spent most of the night thinking about it."

The original entry of "Oct 30/20": "Diabetus by Banting



- Ligate pancreatic duct of dog.
- Keep dogs alive till acini degenerate leaving Islets.
- Try to isolate internal secretion of these to relieve glycosurea."



• The spelling errors are ironic but are characteristic of Banting's personal writings. *Banting to pitch his idea to Macleod*

- Miller and his colleagues at Western University persuaded Banting to pitch his idea to Macleod.worked at the University of Toronto.
- On Nov 8, 1920.2,7–9 Macleod heard the idea. Initially, after hearing Banting's idea regarding pancreatic duct ligation, Macleod was sceptical.
- 400 previous attempts to treat patients with diabetes by use of pancreatic extracts, proved a futile approach.
- The concept of a pancreatic internal secretion was at least 30 years old.
- In 1890, Josef Freiherr von Mering and Oskar Minkowski (Strasbourg, Germany) reported a dog that survived a total pancreatectomy. Surprisingly, the dog developed polyuria, caused by glycosuria from hyperglycaemia,

implicating the pancreas in diabetes.

- Between 1905 and 1920 the accepted wisdom was that impurities were inextricably linked to the pancreas.
- Macleod showed that islets were the source of insulin, although in quantities that were insufficient to be commercially viable.
- Despite reservations, Macleod eventually agreed to allow Banting, a battlefield surgeon with minimal research experience, to enter as a volunteer in his laboratory
- Banting again met with Macleod on May 17, 1921, to begin the project. They were joined by an undergraduate student assistant, Charles Herbert Best, who either won or lost a coin toss with another student to work with Banting over the summer.
- Macleod also outlined a preparation method for the pancreatic extracts, assisted on the first operation, and oversaw the experiments. A month later, Macleod departed for a vacation in Scotland but kept in touch with the young researchers over the summer.

On Aug 3, 1921 History met a turning Point

- It is interesting to know that the Lab condition was pathetic, where Insulin was discovered.
- Enduring sweltering heat
- Poor conditions
- Repeated setbacks—
- An animal mortality rate of 70%
- Banting and Best began an experiment that eventually showed that their extract, which was administered four times over four days, reduced glucose and improved the status of a dog with diabetes. They called this preparation isletin, which was later renamed insulin.
- Banting wrote to Macleod of their findings on Aug 9, 1921.
- Over this time, Best provided both technical and psychological support for the volatile Banting.
- The results were sufficiently promising
- Macleod then tried to take the credit, it well known fact.
- Macleod recounted that he had gone out of his way to consistently acknowledge Banting. Macleod honoured Banting's request to invite James Bertram Collip into the project to help purify the crude



extract.

Collip not to be forgotten

- On Jan 23, 1922, Collip's isolate substantially improved the status of Leonard Thompson, a 14-year-old patient with type 1 diabetes
- Banting and Best's preparation had caused only sterile abscesses in the same patient.
- The use of insulin in patients was soon published with clinician co-authors.
- The substantial effects of insulin profoundly affected the general public and international diabetologists, such as Elliott Joslinwho later wrote-
- "By Christmas of 1922 I had witnessed so many near resurrections that I realized I was seeing enacted before my very eyes Ezekiel's vision of the valley of dry bones"
- The first US patent application for insulin was filed in the names of Collip and Best on June 3, 1922.39
- In the autumn of 1922, George Walden, an Eli Lilly scientist, used isoelectric precipitation to drastically increase both the purity and yield of insulin.

The Final Twist

- Furthermore, in November, 1922 Danish Nobel Prize winner August Krogh visited to Macleod's laboratory
- After meeting the principal investigators personally, Krogh wrote nomination letters to the Nobel Prize commit tee.
- Krogh received approval from the University of Toronto to produce insulin in Denmark at the Nordisk Insulin labatorium, which he founded and later became Novo Nordisk.
- The climax event occurred on Oct 25, 1923, when the Karolinska Institute awarded the Nobel Prize in Physiolo gy or Medicine to Banting and Macleod for the discovery of insulin

Considering all four scientists for Nobel Prize?

- · Banting with Best + Macleod with Collip. All four individuals made essential contributions
- Other diabetes researchers contested the decision to award the prize to the Toronto researchers but were unconvincing.
- History now judges that, if only two of the scientists could be recognised, Krogh's initial case for Banting and Macleod was appropriate
- Still, there is heat in scientific world and an argument could be made that all four should have been co-recipi ents.
- Ultimately, the prize mainly acknowledged the importance of insulin's discovery.
- Banting is the youngest recipient of the Nobel Prize in Physiology or Medicine.
- Collip grew into a person of great importance in endocrinology, having isolated parathyroid hormone, thyroid stimulating hormone, and follicle stimulating hormone, among other hormones.

Banting's original idea was physiologically flawed.

- Unique Fact: There was no need to ligate the pancreatic duct to preserve the β cells or insulin. Although trypsinogen, the precursor of trypsin, is resident in the acinar cells, it does not directly possess digestive capacity until it becomes activated in the intestinal lumen
- Neither Banting nor Macleod realised this fact at first.
- Banting and Best eventually found that whole fresh pancreas, non-duct ligated, could serve as the source of insulin.

Further Reading

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Myths about Diabetes

Dr. Smitha Bhat *MD, MRCP, FRCP*

Many times, when treating diabetes, we are faced with an inexplicable resistance from our patients regarding the treatment modalities we suggest. This might be due to change resistance, fear of increased expense or distrust, but often it is due to myths and the beliefs that have permeated through the population regarding various aspects of diabetes treatment. It behooves a good physician to be aware of these beliefs, as prior information means half the battle is won.

- 1. "Thin people don't get diabetes" while it is true that the majority of cases of Type 2 diabetes are overweight and obese, thin people can get DM too, and must be informed that their low BMI does not give them com plete protection against the disease.
- 2. An extremely widespread and difficult to eradicate belief is that "rice is evil." Unfortunately, this leads to patients overeating other forms of carbohydrates like wheat; sometimes even rice containing foods like dosas, idlis are considered superior to rice.
- 3. "In the absence of symptoms, diabetes is not severe." Many patients refuse to escalate their OAD prescrip tion, stating that they don't have fatigue, and their wounds are healing, even when the blood sugar is more than 200 mg%, with the contention that they feel healthy, so that there is no need to modify treatment.
- 4. People believe that "insulin damages the body" apart from the cost and the inconvenience, many patients, even those who are educated, believe that insulin can damage the body, especially the kidneys. This belief that insulin is more detrimental than oral antidiabetic drugs is widespread. This needs to be dealt with gently; crucially, the future use of insulin should not be used as a threat to encourage patients to follow diet and prescription.
- 5. A study in Karachi shows that some patients believe that "diabetes is contagious." This might lead to ostra cization among family and friends, adding to depression in the patients.
- 6. Disturbingly, even in the present era, almost a quarter of patients surveyed opined that "diabetes is a conse quence of past sins."
- 7. People believe strongly that "diabetes is only a disease of old age."
- 8. "Soaking feet in water cures diabetes." was an opinion held by many.
- 9. Almost all patients believed that "diabetes was solely due to excess consumption of sugar" and conversely could be controlled by eating bitter foodstuffs (like bitter gourd)
- 10. "Insulin and OADs must be withheld when the patient is sick." Almost all diabetologists have had the experience of patients presenting in diabetic ketoacidosis due to this pernicious belief.
- 11. Women with diabetes cannot get married or become pregnant. This is a dangerous and hurtful belief, especially to the many young women who are diagnosed with diabetes or prediabetes.



These myths are widespread, hidden and negatively impact patient care. They spread by word of mouth and once heard, tend to take root tenaciously. Many patients tend to follow up with native medicine practitioners and present to us with diabetic emergencies. Others may present late in the disease, with microvascular andmacrovascular damage already having set in.

The fact that these incorrect beliefs are so widespread emphasizes the truth that education is as crucial as medication, diet and exercise in the management of diabetes and other chronic diseases.

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Future of 'Telemedicine'

Dr. Supratik Bhattacharya, Director SKN Diabetes & Endocrine Centre

COVID-19 has presented one of the most formidable challenges leading to a paradigm shift about how medical practitioners perform consultations and communication with their patients.

Health systems increasingly supporting the use of telemedicine

According to the National Institutes of Health (NIH), telemedicine or telehealth is defined as using technology to provide and help healthcare at a distance. Telemedicine can greatly improve access to quality, affordable care for patients while maintaining physical distancing for the safety of both patients and healthcare providers.

Longstanding technological challenges before the COVID-19 pandemic

Professional scepticism, ethical, financial, administrative and legal barriers had limited the uptake and use of remote consultations. It was being considered for a limited proportion of patient consultations only. In addition to this, it had made less progress than either the technology or the regulations allowed. For example, remote consultations often used telephone links rather than video or other platforms to enable the simultaneous sharing of test results, diagnostic images or other files.

The COVID-19 pandemic has been a triggering factor in making progress in the implementation of telemedicine and overcoming these longstanding challenges.

Telemedicine, a boon for people with comorbid conditions

People living with comorbid conditions such as diabetes, hypertension, heart diseases, cancer, chronic lung diseases etc., are a vulnerable population who need to be protected from avoidable outpatient clinic visits, particularly in this pandemic.

A meta-analysis in 2019 focusing on the clinical effectiveness of telemedicine in diabetes showed significant HbA1c reductions in the group that was supported by telehealthcare. Telemedicine for diabetes patients has positively impacted glucose control, therapy adherence.

Video consultations to be the perfect solution

Video consultations in healthcare present an approximation of face-to-face interaction and are a "visual upgrade" of widely used telephone consultations. Evidence for video consultations effectiveness is scarce, but points towards efficacy, safety, and high satisfaction in patients and healthcare providers have been remarkable.

How healthcare professionals have perceived video consultations

They considered video consultations to provide numerous benefits, minimal differences according to their professional category or workplace. They also thought that it offered efficient screening for consultations, avoiding crowds and waiting lists, allowing quick resolutions of common and minor diseases, and decreasing workloads and costs in healthcare centres.



Limitations of Teleconsultations

There are however certain limitations with video consultations e.g. internet connectivity issues at either ends, not getting to examine or record vitals in person, patient's desire to meet his/her physician in person rather than virtually

Video consultations in the covid-19 pandemic and beyond

During the COVID-19 pandemic, the implementation of video consultations yielded information on telemedicine's future to provide care not only to chronic patients but also to those with acute diseases. Video consultations have shown to be useful in many ways especially for those who want convenience, elderly or select group of individuals who would prefer less wait time at clinic and those who live far off and may not be able to assess the physician of his/her choice because of the distance. Hence, telemedicine should not be sidetracked when the pandemic is mitigated. Although telemedicine may not be able to replace face to face consultations completely in near future, it would however be an useful adjunct to in person consultations.

To effectively implement video consultations as a modality of health within the healthcare system, it is important to analyse how healthcare professionals and patients are perceiving it.



Post- CoVid 19 Syndromes

Dr. Tamonash Bhattacharyya

Post-CoVid 19 Syndrome has become important, no less than the viral infection itself. Here we like to touch only two of them. There are research works going around the world. And almost every other day we are getting more and more information, often contradictory to the previous results. This is always acceptable in science. We would like to remember a few things even after discharge of a (cured) CoVid 19 patient. It's not only about serial assessment of some blood parameters like d-Dimer or CRP, and decision of continuing anti-coagulant (Heparin or NOAC) or steroid. We have to follow up the patients for glycemic control and evolving other infections including Tuberculosis.

If the postulates of direct affection of the pancreatic beta cells by the Corona virus is the key phenomenon in a good number of hitherto euglycemic patients, the dysglycemia may lead to frank Diabetes Mellitus. Whether the rate of beta cell apoptosis gets aggravated by the CoVid 19 infection, and if there is retarded programmed cell death once the cured patient becomes free from infection—to be assessed thoroughly. Continuation of insulin till adequate and sustained glycemic control and/or introduction of SGLT2i +/- DPP4i after absolute control of infection seem to be beneficial. Steroids being the most important life-saving player in CoVid 19 and at the same time being the most common offender here to cause iatrogenic dysglycemia, need proper address. Some advocate continuation with the existing oral drugs (OAD) in patients with pre-existing T2 DM with Corona, insulinisation under supervision and frequent monitoring (SMBG/CBG in domestic or IPD care + venous sampling) seem ideal.

Another ailment particularly one infection must not be forgotten, in all CoVid patients.

Covid 19 is a disease with immunological disordered phenomenon. To address the immunological complexities steroids are used and the success is significant in reducing mortalities in most studies. And this is perhaps the only group of pharmacological agents which has proven its credential. But, just like deterioration of the glycemic status (in euglycemic and diabetic patients with Covid) whether unsupervised or prolonged untapered dose of oral/parenteral corticosteroids can be responsible for new onset Tuberculosis, needs evaluation. We are getting newer PTB (Pulmonary Tuberculosis) and flaring of previous Koch's in post-CoVid 19 cases coming with persisting cough and/or haemoptysis.

Another proposed game-changer in hyperinflammatory condition of CoVid 19 pneumonia is anti-IL6 receptor antibody Tocilizumab which is found to be effective to address the notorious Cytokine Storm in the infection usually appearing in the later part of disease. Even in the NEJM original article (January 7 2021) it was concluded to reduce the progression to the composite outcome of mechanical ventilation or death in CoVid pneumonia not receiving mechanical ventilation, though it did not improve survival.

But Tocilizumab being a humanized monoclonal antibody primarily indicated as an immunosuppressive in several auto-immune diseases has some basic contraindications including Active Tuberculosis, Inactive Tuberculosis /LTBI, opportunistic fungal infections including Pneumocystis jirovecii, untreated or advanced malignancy.

But, probably owing to logistic issues in most cases exclusion of TB in CoVid 19 patients is not possible. And this is also another contributory factor to develop Tuberculosis in post-Covid situation. So it would be the task of the treating Physician of a CoVid 19 patient, beyond the cure of primary infection, not only addressing the sequalae of coagulopathy myocarditis or pulmonary fibrosis, but also to take care of glycemic control and exclusion of appearing Koch's.



DRWA Academic Programmes July to September 2021

National Webinar

Date: Sunday 18 July 2021 | Time: 18:00 - 20:00 hrs Scientific Chairperson: Dr Bijay Patni Moderator: Dr M H Sanwarwalla Experts: Dr Anil Virmani, Dr S.C.Jha



Registration URL: <u>https://attendee.gotowebinar.com/register/8960279111833185807</u> Webinar ID: 229-427-883

TIME	ТОРІС	SPEAKER
18:00 - 18:20	History of Insulin-celebrating 100 years Chairperson: Dr Mary D'Cruz	Dr Munna Sherpa
18:20 - 18:40	ADA 2021-Highlights on Insulin: Chairperson: Dr Lily Rodrigue	Dr Daljit Singh Sethi
18:40 - 19:00	ADA 2021-Highlights on GLP-1RAs: Chairperson: Dr Supratik Bhattacharya	Dr Salaam Ranabir
19:00 - 19:20	Basal insulins –Flatter than ever before Chairperson: Dr Arjun Baidya	Dr Basab Ghosh
19:20 - 19:40	The Game Changer in Diabetes Management: Oral Semaglutide Chairperson: Dr Bijay Patni	Dr Rupam Das
19:40 - 20:00	COVID-19 and Diabetes - Choosing what matters for Total Control Chairperson: Dr N K Singh	Dr Parvati Nandy



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DRWA mid-session Snaps

