DIABETES RESEARCH WELFARE ASSOCIATION

RESEARCH dRWA EDUCATION

Quarterly

NEWSLETTER

JUNE-2021

Patrons	:	Dr Bijay Patni Dr Gautam Banerjee Dr P C Hazra
President	:	Dr Mary D'Cruz
Vice President	:	Dr Tamonash Bhattacharya
Secretary	:	Dr M H Sanwarwalla
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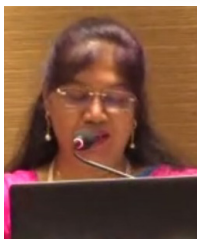
From The President's Desk



The year that has gone by, 2020 has challenged us in many ways. The medical fraternity bore the brunt of the Covid-19 assault, working in stressful conditions, away from home and family and we lost many medical personnel across the world. To those brave and indefatigable souls, we pay our tearful homage. To those who succumbed to the disease- patients and dear ones, we remember with gratitude their presence in our lives.

We are grateful to be alive and well, and will continue to observe all sanitization to prevent the spread of the disease.

Inspite of all the hurdles DRWA, Diabetes Research And Welfare Association, held monthly webinars, conducted a national Diabetes Activist Certified course, had a diabetes update program and held the World Diabetes Day camp in November 2020 at Raghabpur.

As we begin a new financial year, we look forward with hope to a reduced mortality due to disease and increased vaccination of the entire population.

We welcome all our new members and invite them to participate in all our activities.

Dr. Mary D'Cruz

From the secretary desk

Dear friends 2020 was indeed a special year in many ways The initial phase of lockdown was a period of retrospection, fear of the unknown, uncertainty of what the future holds. Our team was actually preparing for the annual conference to be held in May but then that's now history.

However inspite of all the setbacks we managed to communicate with our members virtually and through our Whatsapp group and also kept our academic activities

Alive and on 27th June organised our first national webinar 'Digital Health: Way Ahead which was chaired by Prof. Supten Sarbadhikary. The success of which spured us on to hold our next national webinar on GDM on19th July where we had a galaxy of reputed national speakers and was viewed by a large virtual audience.

On 7th August we organised another national webinar on Lifestyle Modification of Cardiometabolic disease and in the same month on 25th there was a webinar on Diabetes and Skin a Practical Approach.

On 24th September we had our own virtual CME with members presenting their own cases and Dr Patni discussed his Covid data.

On 16th October we had another national webinar on Obesity where Dr Sarita Bajaj was at her best. This was followed by our Bijoya\Diwali meet where besides exchanging greeting we made sure "KuchMeetha Ho Jaye"

On 16th November we organised a Diabetes Camp to observe World Diabetes Day at St Xaviers College Raghabpur maintaining all Covid Protocols and about 80 patients participated. This was only possible due to the efforts of our beloved President Dr Mary D Cruz.

December 17th was the day when we had the first physical meet CME post Covid at Peerless Inn.

This was followed by our prestigious, well appreciated and well attended virtual Diabetes Activist Certificate Program, five modules over 5 weeks and this culminated in our Diabetes Update on 20th and 21st February 2021at the exotic location of Vedic Village which was a Hybrid program. All credit to Dr Bijay Patni for envisaging and executing the Diabetes Activist Certificate Program. You have made the association proud.

On 19th of March this year we again met physically at peerless Inn. Our newly enrolled members were welcomed into the DRWA family, as only 14 members were present there was no quorum for a GB meeting to select the Executive Committee for 2021. However members were selected to form the other committees and sub committees for 2021-22

Committee

Scientific Committee

Dr Bijay Patni
Dr M H Sanwarwalla
Dr Mary D'Cruz
Dr Tamonash Bhattacharya
Dr Anirban Sinha
Dr Sarbajit Ray
Dr Arjun Baidya
Dr M H Rahaman
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Dr Amit Kumar Dey

Scientific Committee (National)

Dr Anuj Maheshwari
Dr Narsing Verma
Dr N K Singh
Dr Dinesh Agarwal
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Sub Committee for Newsletter

Dr.Amit Dey
Dr M H Sanwarwalla
Dr Mary D'Cruz
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Dr Agnik Pal

Mid session Committee

Dr Gautam Banerjee
Dr P C Hazra
Dr Partha Biswas
Dr Saumitra Bhattacharyya
Dr Supriyo Mukherjee
Dr Malay Kanti Das
Dr Bijay Patni
Dr M H Sanwarwalla
Dr Mary D'Cruz

Monthly CME Committee

Dr Meenakshi Biswas
Dr Partha Biswas
Dr Saumaitra Bhattacharyya
Dr M H Sanwarwalla
Dr Mary D'Cruz
Dr Bijay Patni
Dr Ajitesh Ghosal

World Diabetes Day Committee

Dr Mary D'Cruz
Dr M H Sanwarwalla
Dr Partho Biswas
Dr Suman Guha
Dr Malay Kanti Das
Dr Tapas Bhattacharya

Before I sign off I want to say a big thank you to our "Man Friday" Dr Partha Biswas who remains behind the scene but is instrumental in the success of all our ventures.

Looking forward to a eventful year and working in tandem with all our esteemed members Long live DRWA

Dr M H Sanwarwalla

Our website is : www.drwa.in
Our interface page is on clirnet.E-mail: b_patni@ hotmail.com
Ph Number: 9748823702 / 9831216006 / 9831095308

From the Editors' Desk

Dear everyone,

It gives me immense pleasure to present the first newsletter of DRWA. The ongoing covid pandemic over the past one and a half year had been challenging to all of us in many ways. However, these testing times has taught us to set our priorities right, both as a clinician and as an individual. While our minds are clouded with various scientific data in search of that "elixir" drug in the treatment of Covid 19, we must also keep some academic apetite for the non communicable chronic diseases like diabetes that will continue to co exist as "metabolic epidemic" amidst the "covid 19 pandemic"

Presenting, a collection of articles covering topics on diabetes and covid 19 in this edition of the newsletter.

Enjoy your reading.

Dr. Amit Kumar Dey

What should be in your ideal Breakfast?

Dr. Agnik Pal,
MBBS, MD, IDECC, Dip Diabetology (RCP, UK)
Asst. Professor, College of Medicine & JNM Hospital, Kalyani Consultant Physician and Diabetologist

Diabetes is a chronic complex metabolic disorder with a major impact on the lives and well-being of individuals, families, and societies worldwide. It is among the top 10 causes of death in adults, and was estimated to have 463 million adults aged 20-79 years currently living with diabetes, representing 9.3% of the world's population in this age group. The total number is predicted to rise to 578 million (10.2%) by 2030 and to 700 million (10.9%) by 2045 (International Diabetes Federation, 2019). Obesity is a frequent concomitant of type 2 diabetes and in many longitudinal studies has been shown to be a powerful predictor of its development. Obesity has increased rapidly in many populations in recent years because of an interaction between genetic and environmental factors. These include: metabolic characteristics, physical inactivity, habitual energy intake in relation to expenditure and macronutrient lastly composition of the diet. This increase in obesity has been accompanied by an increasing prevalence of type 2 diabetes. Since obesity is such a strong predictor of diabetes incidence, it appears that the rapid increases in the prevalence of type 2 diabetes seen in many populations in recent decades are almost certainly related to increasing obesity. Data from the Nurses' Health Study suggest that the lowest risk of diabetes occurs in individuals who have a body mass index (BMI), with increasing prevalence seen as obesity levels increase. Diet is one of the most important aspects not only for the patients with diabetes or obesity but also for the non-diabetic persons, as all of us are perpetually at high risk of developing diabetes.

Of the diet, breakfast is one of most important part of whole day's consumption. It's more helpful to say that no meal should be categorised as more important than another and daily food intake should be considered as a whole. Breakfast literally means 'breaking the fast', as one has had no food or 'fasted' since the day before. Breakfast helps top up the energy stores one has used up each night whilst his body repairs and renews itself. It also gives energy for morning activities, whether at work, school, home or out. Skipping meals, whether it is breakfast, lunch or dinner, is not advised at all. Establishing a regular eating pattern has been shown to improve glycaemic control and reduce likelihood of weight gain. However, it is estimated that up to one third of us still regularly miss breakfast. Many of us put this down to time pressures in the morning, but with a little planning, one can find a breakfast choice to suit his lifestyle.

Apart from providing energy (calories) to kick-start one's day, a healthy breakfast provides essential nutrients that the body needs, such as fibre, vitamins and key minerals like calcium and iron. Research has shown that people who eat breakfast have more balanced diets than those who skip it, are less likely to be overweight, lose weight more successfully

if overweight, and have reduced risk of certain diseases such as cardiovascular disease and diabetes. Missing breakfast may increase feelings of hunger later on in the day, resulting in snacking on less healthy foods without necessarily consuming essential nutrients. Eating breakfast may also help to improve mental performance, concentration and mood.

Now, the question is what makes a healthy breakfast? Breakfast should provide about 20-25% of your daily nutritional requirements, and it's not just about having any breakfast - it's about having a healthy breakfast. The Association of UK Dietitians provides a comprehensive understanding of what should be included in a breakfast.

Starchy foods such as bread, cereals, rice, potatoes, and pasta provide energy, vitamins, some iron and fibre. Cereals are a really good choice: as well as being quick and easy to prepare, they often are fortified with vitamins,

iron and calcium to contribute to daily nutritional requirements. However some of these products have added sugar and salt. Porridge, bread, rolls, muffins, scones, malt loaf, fruit bread, currant buns and bagels all provide good sources of energy, mainly as starchy carbohydrate with low fat. Wholegrain varieties should be included whenever possible to ensure a good fibre intake, and one should try to avoid cereals coated in sugar. Evidence suggests that porridge oats for breakfast may have a positive effect on total cholesterol concentration when compared to skipping breakfast.

Fruit and vegetables are good sources of vitamins and fibre. One should try chopped fresh fruit, like a banana, or some dried, stewed or canned (in juice rather than syrup) fruit, or add half a grapefruit or fruit salad to usual breakfast. A small glass (150ml) of pure fruit juice also counts as one serving of your 5-a-day. For something different, one can try a fresh fruit smoothie – blending some fruit of choice with low fat yogurt or milk. Frozen berries, fruit in season or ripe fruit are all ideal for making smoothies. Alternatively, vegetables can be tried at breakfast time; mushrooms, baked beans or tomatoes on toast also can make a tasty change.

Milk and dairy foods provide protein, calcium and vitamins. Calcium is essential to keep bones strong and healthy, and a serving of milk on cereal can give up to one third of daily calcium needs. Low fat milks like skimmed, semi-skimmed or 1% should be used. If one does not like milk on cereal, a glass of milk on its own or in a smoothie, or a pot of low fat yogurt can be tried instead. Natural yoghurt is delicious topped with fruit and a sprinkle of muesli. If one uses milk and other products not made from cows' milk such as soya, oat, coconut, almond or rice, he should make sure they are unsweetened and fortified with calcium.

Meat, fish, eggs, beans and other non-dairy sources of protein give protein, iron and vitamins. These foods are not essential at breakfast, but they can add variety. One should try not to have meat at breakfast every day, and choose cooking methods such as grilling or poaching instead of frying in fat. Poached, boiled or scrambled eggs, baked beans, grilled kippers or smoked haddock are healthier options than bacon and sausages, which are higher in saturated fat.

Foods and drinks high in fat and sugar give energy but are generally low in vitamins, minerals and other nutrients. One should limit these foods and choose low fat sunflower, olive or vegetable oil based spreads where possible and spread thinly. Low sugar, wholegrain breakfast cereals instead of sugar-coated, refined varieties should be chosen. One must try to avoid fizzy drinks, biscuits and crisps at breakfast and use fruit to add natural sweetness instead of sugar on cereal.

One should always remember to include a drink -water, milk, pure fruit juice, tea and coffee - all supply vital fluids. Low fat milks should be used and ask for 'skinny' coffee should be asked when out and about. Being well hydrated also helps to concentrate better.

Lastly, one must try to eat within two hours of getting up. Healthy whole grain toast or cereal, porridge, low fat yoghurts, pure fruit juices, fresh fruit salads and smoothies with low fat milk should be kept in breakfast. If one is in a hurry, he should have foods to hand that one can grab, such as a banana, yoghurt with muesli, instant porridge or toast. Thus, eating a healthy breakfast every day will give one the best possible start, as well as enhancing the overall nutritional quality of one's daily food intake.

Email: curedoc@rediffmail.com

LP(a)- A Clinical Perspective

Dr. Bijay Patni
Founder, Diabetes Research Welfare Association

Introduction:

- Lipoprotein(a), Lp(a), is a modified atherogenic low-density lipoprotein particle that contains apolipoprotein(a). Its levels are highly heritable and variable in the population.
- There is evidence that Lp(a) is an independent cardiovascular disease (CVD) risk factor. Lipoprotein(a) (Lp(a)) is now established as a causal risk factor for cardiovascular disease (CVD)
- Epidemiology:

  African descent have levels twice as high as Caucasians, Hispanics and certain Asian populations, South Asians have intermediate levels

Present status:

There is little consensus between the different national guidelines on how to use this information on Lp(a) to more accurately estimate and modify cardiovascular risk:

- The 2019 European Guideline on CVD prevention in clinical practice suggests measuring Lp(a) at least once in each adult person's lifetime.
- The 2018 American College of Cardiology/American Heart Association Guideline on blood cholesterol defined Lp(a) as a 'risk-enhancing factor', especially at higher levels of Lp(a)

  Measurement of Lp(a): Measurement of Lp(a) is currently not standardized or harmonized. Available assays report Lp(a) in either mg/dL or nmol/L and may exhibit Lp(a) isoform-dependent bias.

  Evidence is incomplete regarding the utility of using different risk cut points of Lp(a) based on age, gender, ethnicity, or the presence of comorbid conditions.Lipoprotein(a) levels need only be measured once, unless a secondary cause is suspected or specific treatment is instituted in order to lower levels. Fasting is not required for Lp(a) measurement, and despite being genetically determined, levels may be influenced in the presence of inflammation.

  Serum Lipoprotein(a) levels should be measured in those with:

a. A personal or family history of premature atherosclerotic cardiovascular disease (<60 years of age)
b. **First degree relatives with raised serum Lp(a) levels (>200 nmol/l)**
c. Familial hypercholesterolemia (FH), or other genetic dyslipidemias
d. Calcific aortic valve stenosis
e. A borderline increased (but <15%) 10-year risk of a cardiovascular event

- The LPA gene is fully expressed by 1-2 y of age and the concentration of Lp(a) reaches adult levels by 5 y of age. Because Lp(a) is genetically transmitted, youth whose parents have an elevated Lp(a) level are reasonable candidates for screening; conversely, reverse cascade screening is recommended when a child is found to have an elevated level of Lp(a).
- Even if the absence of approved Lp(a)-lowering medications in youth found to have an elevated level of Lp(a), it is important to emphasize early and lifelong adoption of a heart-healthy lifestyle by the child and family members, especially with respect to smoking avoidance or cessation, given the thrombotic risk attributable to Lp(a).

- **Measurement of Lp (a):** in youth with a history of ischemic stroke may be reasonable.
  Importance of reducing LP(a):The importance of reducing Lp(a) associated cardiovascular risk requires a reappraisal for four main reasons:

1) genetic studies have demonstrated an unequivocal strong link between genes associated with increased Lp(a) and cardiovascular risk as well as the protective effect of LPA null alleles or other strongly Lp(a)-decreasing alleles and CVD risk
2) New insights into Lp(a) assay methodology suggest that inaccurate quantitation of Lp(a) has led historically to its underestimation as a cardiovascular risk factor
3) Lp(a) contributes to aortic valve calcification.
4) The emergence of effective therapies for reducing Lp(a) level. Possible mechanism of increased CVD: Lp (a) promotes atherosclerosis by two principal mechanisms:
   a) As an LDL-like particle, Lp(a) can infiltrate into the arterial intima and bind components of the extracellular matrix, enhancing macrophage infiltration and smooth muscle proliferation.
   Within the atherosclerotic plaque of coronary lesions and carotid endarterectomy specimens, a substantial proportion of circulating oxidised phospholipids reside on Lp(a). These oxidised phospholipids are implicated in driving monocyte trafficking into the arterial wall and enhanc ing pro- inflammatory cytokine release
   b) Secondly, through its similarity to plasminogen, Lp(a) is envisaged to have a prothrombotic effect by inhibiting fibrinolysis. Apo(a) may also promote platelet aggregation by mediating the binding of Lp(a) to plasminogen receptors on the platelet surface and granule release via the thrombin receptor. Some biochemical studies have also demonstrated anti-fibrinolytic effects of Lp(a) .
   c) In the past decade there has been consolidation of the evidence that elevated serum Lp(a) is a risk factor for coronary heart disease (CHD), ischaemic stroke, peripheral artery disease, as well as calcific aortic valve stenosis.
   d) Mendelian randomisation studies and genome wide association studies support Lp(a) as an indepen dent cardiovascular risk factor

Treatment possibilities:

- Lifestyle therapy, including diet and physical exercise,has no significant effect on Lp(a) levels.
- Statin therapy does not decrease Lp(a) levels.
- Patients with a history of ASCVD who are taking statinsand have an Lp(a) 50 mg/dL are at increased risk for ASCVD events, independent of other risk factors.
- Niacin lowers Lp(a), has no demonstrated ASCVD risk reduction benefit in patients taking statins, and may cause harm.
- Lomitapide, which is indicated to lower LDL-C in patients with homozygous FH, also lowers Lp(a) but is not recommended for ASCVD risk reduction.
- PCSK9 inhibitors lower Lp(a), but the contribution of Lp(a) reduction to their ASCVD risk reduction benefit remains undetermined.

- LDL apheresis lowers Lp(a) and is sometimes used for those with elevated Lp(a) and recurrent ASCVD events.

- Gene silencing by antisence and use of various ligands to increase the effectiveness is another modality to manage LP (a).

- The role of antiplatelet therapy in primary prevention in patients with elevated Lipoprotein(a) need to be considered.

Conclusion:

- More than 45 years after the initial identification of Lp(a) by Kåre Berg, and over 20 years after the genetic sequence of apo(a) was reported by McLean et al., Lp(a) remains an enigmatic lipoprotein.

- Although a risk factor role of Lp(a) for cardiovascular disease has been controversial, studies during the past decade have provided robust support for a role of Lp(a) in promoting CVD.

- Despite its recognition as a risk factor for CVD, the atherogenic mechanism for Lp(a) is poorly understood. The substantial heterogeneity in apo(a) gene size and considerable variability in the contribution of apo(a) size to plasma Lp(a) levels post significant challenges

- This underscores the importance of isoform-specific or allele-specific apo(a) levels, that is, the amount of Lp(a) associated with each apo(a) size allele.

- Further, Lp(a) particles with smaller, compared with larger, apo(a) isoforms may be a stronger risk factor for CVD. Although plasma Lp(a) levels are to a major extent regulated by genetic factors, some metabolic and environmental factors, such as diet and exercise, together with proinflammatory conditions have been shown to impact Lp(a).

- These alterations in Lp(a) levels may in turn enhance its proatherogenic properties and lead to increased risk for CVD

Future focus points:

- a) Development of commercial truly isoform insensitive assays

- b) Randomised, controlled interventional studies that selectively lower Lipoprotein(a) in primary and secondary prevention of ASCVD

- c) Future population-based and metabolic studies are warranted to understand how Lp(a) participates in the development of atherosclerosis, and how apo(a) molecular properties may modulate the Lp(a) risk factor role.

- d) Lipoprotein(a) reference ranges in different ethnic groups

Enigma of Pancreatogenic Diabetes (Type 3c)

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

INTRODUCTION

Pancreatogenic diabetes occurs as a result of benign and malignant diseases of the exocrine pancreas. It is classified as type 3c diabetes mellitus (T3cDM) according to the current classification of diabetes mellitus by the American Diabetes Association. Prevalence of T3cDM is 5% - 10% among all diabetes mellitus cases in Western populations. Its prevalence is around 25% - 30% in South East Asian Countries and India where tropical or fibrocalcific pancreatitis is endemic. T3cDM is distinct from Type 1 and Type 2 DM as it arises due to chronic inflammation and has unique clinical and laboratory parameters and is associated with high incidence of pancreatic carcinoma.

PATHOPHYSIOLOGY

Chronic pancreatitis (CP) is a fibroinflammatory disorder. Inflammatory environment early in the disease results in beta cell dysfunction leading to development of diabetes. In advanced disease as a result of fibrosis and islet cell destruction there is insulin and glucagon deficiency responsible for poorly controlled diabetes. In addition, pancreatic polypeptide deficiency early in the course of disease is responsible for hepatic insulin resistance and development of diabetes. As a result of nutrient maldigestion in chronic pancreatitis, there is impaired incretin effect (Impaired GLP-1 secretion from intestines) leading to diminished insulin release and development of diabetes.Presence of variant in 5` UTR of Bach2 gene along with primary susceptibility to CP can aggravate the disease towards a more advanced/severe form and is associated with development of diabetes. Reduction in abundance of Faecalibacteriumprausnitzii is associated with increase in endotoxin levels and increase in blood sugars and reduction in insulin levels in CP patients with DM. It is known that lipopolysaccharide (LPS) could induce inflammation in beta cells via Toll like receptors (TLRs) and nuclear factor of kappa-B (NFk-B) thereby resulting in beta cell dysfunction.

CLINICAL FEATURES AND DIAGNOSIS

The altered glucose metabolism of T3cDM ranges from mild impairment to a severe form, characterized by frequent episodes of iatrogenic hypoglycaemia, referred to as “brittle diabetes” which is difficult to treat. Hyperglycemia is due to unsuppressed hepatic glucose production (HGP), whereas enhanced peripheral insulin sensitivity and deficiency of pancreatic glucagon secretion results in hypoglycemia. It is not always easy to diagnose and classify a patient with type 3c diabetes mellitus correctly. It has been suggested that 40% of T3cDM are misdiagnosed as type 2 diabetes. In distinguishing between the different diabetes types, the presence of islet cell antibodies is consistent with T1DM. An absent pancreatic polypeptide response can distinguish between T3cDM from T2DM, which is characterized by elevated pancreatic polypeptide levels. All patients with chronic pancreatitis should be screened for the development diabetes mellitus with fasting blood sugar and HbA1c annually. If any impairment in either one, requires further evaluation with 75g oral glucose tolerance test.

PROPOSED DIAGNOSTIC CRITERIA FOR TYPE 3C DIABETES MELLITUS

- **Major criteria (must be present)**
  - Presence of exocrine pancreatic insufficiency (monoclonal fecal elastase-1 test or direct function tests)
  - Pathological pancreatic imaging (endoscopic ultrasound, MRI, CT)
  - Absence of type 1 diabetes mellitus associated autoimmune markers
- **Minor criteria**
  - Absent pancreatic polypeptide se cretion
  - Impaired incretin secretion (e.g., GLP-1)
  - No excessive insulin resistance (e.g., HOMA-IR)
  - Impaired beta cell function (e.g., HOMA-B, C-Peptide/glucose-ratio)
  - Low serum levels of lipid soluble vitamins (A, D, E and K).

MANAGEMENT

As pancreatogenic diabetes is associated with slight increase in risk of malignancy and treatments which employ insulin and insulin secretagogues appear to increase the risk further by enhanced activation of signalling pathways. In all patients with T3cDM, initial treatment should begin with a concentrated effort to correct lifestyle factors which contribute to hyperglycaemia and the risk of malignancy. In T3cDM, metformin is the initial drug of choice for oral therapy due in part to its insulin-lowering effects on glucose metabolism, and also due to its specific anti-neoplastic actions on cellular mediators of replication and protein synthesis. When insulin administration is required as primary therapy to adequately control hyperglycaemia, adjunct therapy with additional oral agents (specially metformin) to reduce the required insulin dose is beneficial. Thiazolidinediones increase both hepatic and peripheral insulin sensitivity but carry an increased risk of bone fracture, so their use might not be well suited for patients who are already at increased risk for this complication. Alpha glucosidase inhibitors can be added with beneficial effects but in patients with severe exocrine deficiency may result in bloating. GLP-1 and its analog are growth-promoting agents of pancreatic tissue due to their expression of the transcription factor PDX-1. Injectable GLP-1 analogues and oral DPP-4 inhibitors are typically avoided in chronic pancreatitis because of their potential role in increasing risk for acute pancreatitis and pancreatic ductal adenocarcinoma. SGLT2 inhibitors help in reduction of insulin requirement but should be used with caution as there is increased risk of euglycemic diabetic ketoacidosis and should be avoided in patients with recurrent pancreatitis. Pancreatic enzyme replacement therapy in addition to improving exocrine insufficiency, may also help in controlling hyperglycaemia by regulating incretin secretion.

SGLT2 Inhibitors: Emerging Landscapes

Dr N.K. Singh
MD, FICP, Diabetologist physician, Dhanbad
Editor: www.cmeindia.in

Some of the untold stories related to beyond glycemic lowering of SGLT2 Inhibitors need serious considerations This journey of SGLT2 inhibitors is going through an unimaginable curve showing tremendous benefits to meeting the multitudes of unmet needs of diabetic patients.

New Exciting Indications

1.Prevention of diabetes:

First evidence emerges from DAPA-HF trial which revealed among the participants who did not have T2DM at the start of the trial, treatment with dapagliflozin reduced the risk of developing diabetes by a whopping 32% compared to placebo.

2.Prevention of non-diabetic HF

All guidelines have been changed recently as CV events prevention, renoprotection and reduction in hospitalization for heart failure are top most priority in diabetes. In 2019, when DAPA-HF results came, it amazed the scientific community. DAPA-HF trial showed 26% relative risk (RR) reduction (HR 0.74;95% CI,0.65-0.85) in primary end point– composite of CV death, or hospitalization or urgent emergency visit for HF, 30% RR reduction (HR 0.70; 95% CI,0.59-0.83) in worsening HF and 18% RR reduction (HR 0.82; 95% CI,0.69-0.98) in CV death with all-cause death reduction of 17%. Interestingly, DAPA-HF had 55% non-diabetics and when results were compared in pre-specified subgroup of diabetics and non-diabetics, the benefits were observed in equal proportion in both groups without greater incidence of adverse outcome, suggesting the drug's efficacy is independent of glycemic control.

3.SGLT2 inhibitors are yet to show proven benefits in HFpEF

Few important trials are on the way- EMPEROR-Preserved and EMPERIAL-Preserved with empagliflozin, DELIVER and DETERMINE-Preserved with dapagliflozin, and CHIEF-EF with canagliflozin. A pair of trials involving sotagliflozin-a unique diabetes drug suggest it offers benefits for patients who have heart failure with preserved ejection fraction (HFpEF), marking the first-time investigators have shown this in a prospective trial. (Results from SOLOIST-WHF and SCORED presented this evening at the American Heart Association Scientific Sessions 2020)

4.Evidence in hypertension:

SGLT-2i have been shown to be effective in modest reduction in systolic and diastolic blood pressure. This reduction takes account for the 5–6 mm Hg decrease in systolic and the 1–2 mm Hg decrease in diastolic blood pressure (BP). The BP reduction seems independent from the improvement in glycemic control.

5.Emerging as Nephrologist's favorite to treat diabetic kidney disease (DKD):

Emerging evidence derived from trials and meta-analyses point to cardio-protective and Reno protective effects of SGLT-2i that are maintained in patients with full blown DKD with baseline eGFR up to 30mL/min/1.73m2 Scientific societies have moved to recommend the preferential use of SGLT-2i in patients with DKD.

6. **Primary prevention of cardio-renal complications in T2DM:**
While initial trials clearly pointed out reduced CV morbidity and mortality events with use of SGLT-2i among T2DM patients with high established atherosclerotic cardiovascular diseases (ASCVD), the drug's potential CV benefits among diabetics even without cardio-renal diseases are being actively explored.

7. **Lowering gout risk by SGLT2 Inhibitors:**
A meta-analysis of 62 studies, comprising 34941 patients shows that any of the SGLT-2i significantly decreased serum uric acid levels compared to control. A more recent real-world experience from 295307 diabetic patients showed patients prescribed with SGLT-2i had 39% lower incidence of gout than those prescribed GLP1 agonist (4.9 events versus 7.8 events per 1000 person-years). The resulting urate lowering effect of SGLT-2i secondary to renal glucosuric actions might be a potential mechanism through which these drugs provide CV and renal benefits

8. **Abdominal Obesity:**
SGLT-2i, in combination with other drugs (such as GLP-1 agonists) have been shown to reduce body weight, visceral adiposity, ectopic fat deposition and improves adipose tissue function and weight-related quality of life

9. **Use in NASH/NAFLD:**
SGLT-2i are showing promising results in non-alcoholic steatohepatitis (NASH) and non-Alcoholic fatty liver disease (NAFLD), a common accompaniment among diabetic and obese. Various studies have already shown that SGLT-2i improve serum level of liver enzymes, decrease liver fat, fibrosis, necroinflammation

10. **SGLT-2i in T1DM:**
SGLT-2i has been a lucrative option to treat T1DM due to its insulin-independent action. Some recent trials like DEPICT-1,2. EASE-2,3, and inTandem-1,2,3 were done to evaluate its effectiveness in T1DM. Although the glucose-lowering effect was modest, the risk for diabetic keto-acidosis (DKA) was substantial . The increased risk of DKA is attributable to the failure to promptly recognize early metabolic derangements that occur at much lower than usual glucose levels (euglycemic DKA) among SGLT-2i users.

Some other possible uses:

11. DIVE study with empagliflozin among healthy adults with artificially induced syndrome of in appropriate ADH secretion (SIADH) gives insights that SGLT-2i may be used in diseases associated with hyponatremia due to their effect on free water clearance

12. SGLT-2i points beneficial effect in weight loss and improved anthropometric parameters in poly cystic ovarian syndrome comparable to metformin

13. SGLT-2i significantly reduced the risk of new-onset obstructive sleep apnea (OSA) probably as a secondary gain of weight loss

14. Study done with dapagliflozin showed that it reduced atrial fibrillation/flutter (AF) risk by 19% in T2DM regardless of previous history of AF, ASCVD or HF

15. Paper presented at ADA virtual meeting 2020 showed that empagliflozin significantly reduced the requirement of insulin by 59% (new-initiation of insulin, or >20% increase in insulin requirement). Thus, it appears SGLT-2i are emerging as insulin dose modifier.

16. Although researchers have cautioned against continuing SGLT-2i among diabetic COVID-19 patients, trial is underway for its possible use against SARS-CoV2.

Further Readings:

1. Mohan V, Mithal A, Joshi SR, Aravind SR, Chowdhury S. Remogliflozin Etabonate in the Treatment of Type 2 Diabetes: Design, Development, and Place in Therapy. Drug Des DevelTher. 2020;14:2487-501.
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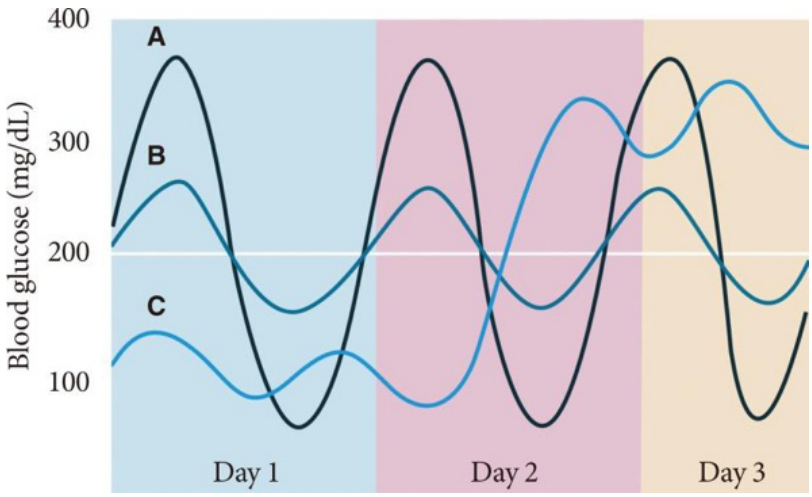
Addressing GV and Improving Time in range-an unmet need in Diabetes management

Dr. Supratik Bhattacharyya

Glucose variability (GV) has been identified as a potential risk factor for diabetic complications. Considerable evidence supports the negative role of GV in the development of diabetic macrovascular and microvascular complications

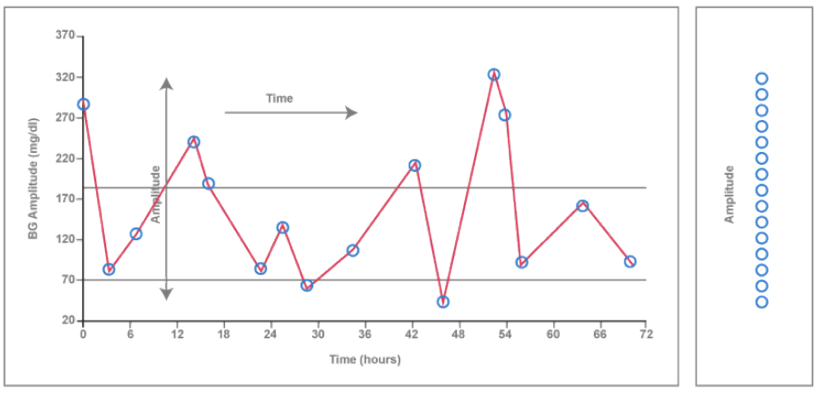
Meta-analysis of the published data from 20 studies of 95,783 individuals found a progressive relationship between the GV and cardiovascular risk

GV is likely to be incompletely expressed by HbA1c, particularly in patients with good metabolic control



Glycemic variability in three hypothetical patients who have the same mean blood glucose concentration. Patient B has relatively small variations during the day and on different days; this patient should have little difficulty in lowering daily mean blood glucose concentrations without inducing hypoglycemia. In comparison, patient A has marked blood glucose variations on the same day and patient C has marked blood glucose variations on different days. (Graph adapted from Suh S, Kim JH. Diabetes Metab J. 2015;39(4):273-282.)

Glucose fluctuations are a process in time that has 2 dimensions - amplitude and time



Main types of metric for assessment of GV

Time in range (TIR) is identified as a key metric of glycemic control

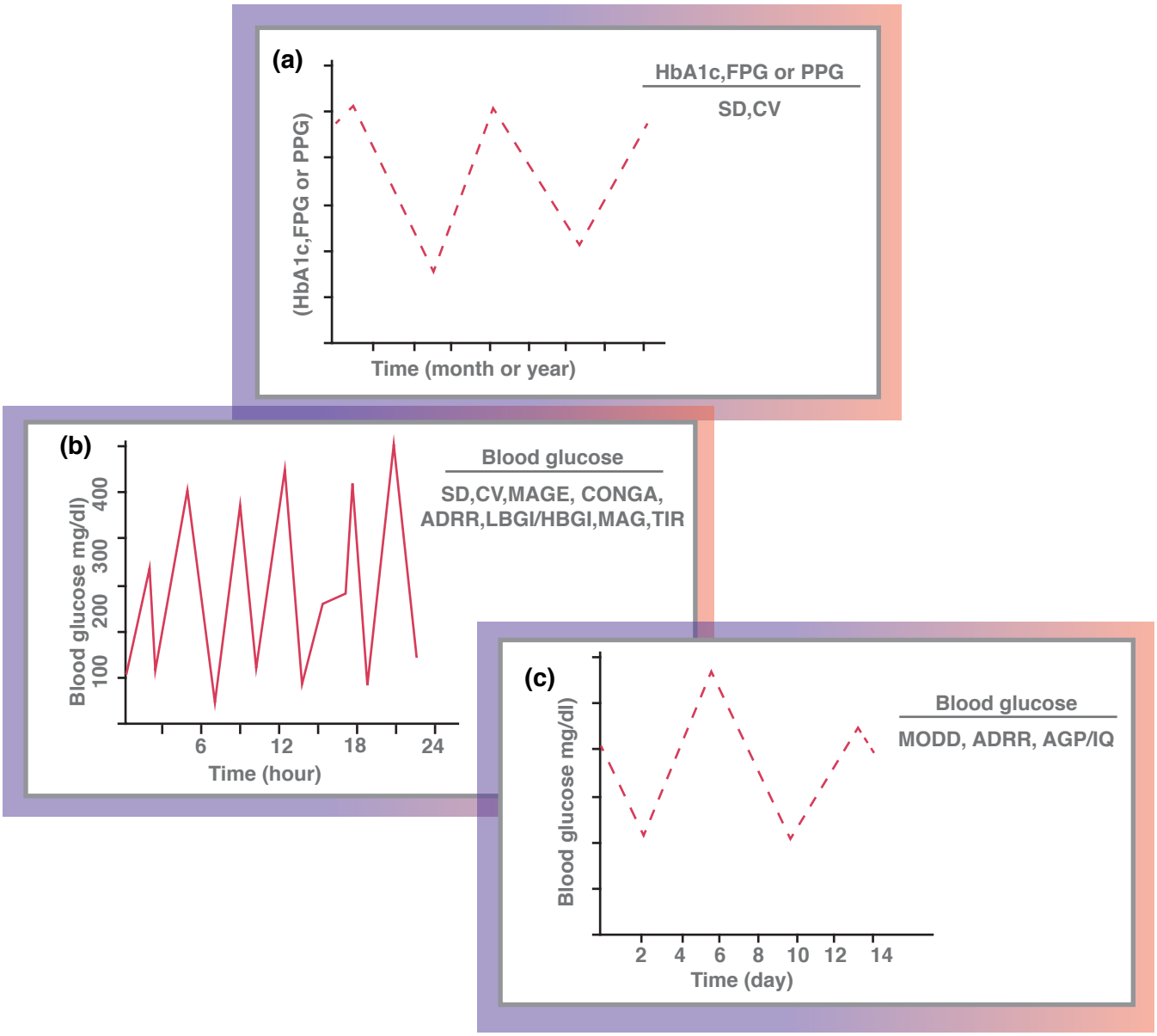
TIR and GV are mathematically and conceptually linked, they are not interchangeable.

Types of metric	Computation or description
Long-term GV	
Visit-to-visit measurements of HbA1c, FPG or PPG	Measures of SD or CV of HbA1c, FPG and PPG between sequential visits
Short-term GV	
ADRR	Sum of the daily peak risks for hypoglycemia and hyperglycemia
AGP/IQR	Distribution of glucose data at a given timepoint and resulted as interquartile ranges
CV	Magnitude of variability relative to mean blood glucose
CONGA	Difference between a current blood glucose reading and a reading taken hours earlier
LBGI/HBGI	Preceded by a log transform to render symmetric the skewed distribution of glucose values
MAGE	Mean differences from peaks to nadirs
MAG	Absolute differences between sequential readings divided by the time between the first and last blood glucose measurement
MODD	Absolute differences between two glucose values measured at the same time with a 24 h interval
SD	Variation around the mean blood glucose
TIME IN RANGE (TIR)	**Percentage of time per day within target glucose range (3.9–10.0 mmol/L)**

Time-in-Range (TIR) is established as a treatment target in diabetes to complement the time-tested A1c

Two principal types of glucose variability based on the length of time-interval:

Long-term GV based on visit-to-visit changes of HbA1c, FPG or PPG.
Short-term GV represented by within-day and between-day GV.



a: *Long-term GV based on visit-to-visit changes of HbA1c, FPG or PPG, b, c: Short-term GV represented by within-day and between-day GV FPG, fasting plasma glucose; PPG, postprandial glucose; SD, standard deviation; CV, coefficient of variation; MAGE, mean amplitude of glycemic excursions; CONGA, continuous overlapping net glycemic action; MAG, mean absolute glucose; MODD, mean of daily differences; AGP, average glucose profile; IQR, interquartile ranges; LBGI, low blood glucose index; HBGI, high blood glucose index; ADRR, average daily risk range; TIR, time in rang*

ADA Time in Range Guidelines

Diabetes Type	Glucose Range	Recommendations (% of readings; time per day)
Type 1/type 2 diabetes	<54 mg/dl (<3.0 mmol/L)	<1% (<15 min)
	<70 mg/dl (<3.9 mmol/L)	<4% (<1 h)
	70 - 180 mg/dl (3.9 - 10.0 mmol/L)	**>70% (>16 h, 48 min)** **>60% for individuals <25 yrs old (>14 h, 24 min)**
	>180 mg/dl (>10.0 mmol/L)	<25% (<6 h)
	>250 mg/dl (>13.9 mmol/L)	<5% (<1 h, 12 min)
Older/high risk type 1/type 2 diabetes	<70 mg/dl (<3.9 mmol/L)	<1% (<15 min)
	70 -180 mg/dl (3.9 - 10.0 mmol/L)	**>50% (>12 h)**
	>250 mg/dl (>13.9 mmol/L)	<10% (<2 h, 24 min)
Pregnancy with type 1 diabetes	<54 mg/dl (<3.0 mmol/L)	<1% (<15 min)
	<63 mg/dl (<3.5 mmol/L)	<4% (<1 h)
	63 -140 mg/dl (3.5 - 7.8 mmol/L)	**>70% (>16 h, 48 min)**
	>140 mg/dl (>7.8 mmol/L)	<25% (<6 h)
Pregnancy with gestational or type 2 diabetes	**63 -140 mg/dl (3.5 - 7.8 mmol/L)**	**>90% (>21 h, 36 min)**

Concluding remarks:

The measurement of GV using SMBG or CGM data has the potential to complement A1C data, and to provide a more comprehensive assessment of glycemic control in order to better inform treatment decisions.

Diabetic Nephropathy: Pathophysiology, Diagnosis& treatment

Dr Arindam Sur
MBBS,MD,CCEBDM Consultant physician

Diabetes Mellitus (DM) refers to a group of common metabolic disorders that share the phenotype of hyperglycemia .The two broad categories of Diabetes are designated as Type1 and Type2.All forms of diabetes, both inherited and acquired, are characterized by a relative or absolute lack of insulin, and the development of diabetes-specific micro-vascular pathology in the retina, renal glomerulus, and peripheral nerve as well as macrovascular changes in various organ systems.

Diabetic Nephropathy is the leading cause of End Stage Renal Disease and affects approximately 40% of Type 2 Diabetic patients and most of the patients entering dialysis are diabetic. It is clinically characterized by increasing rates of urinary albumin excretion starting from normoalbuminuria, which progress to micro-albuminuria then macroalbuminuria and eventually to end stage renal disease .Screening for diabetic nephropathy is currently done by monitoring patients for the development of microalbuminuria and as adjunct for the estimation of GFR, the determination of serum creatinine (sCr), and creatinine clearance (CCr). The appearance of pathological levels of urinary albumin excretion (UAE) represents the most common clinical sign of early renal

involvement in patients affected by diabetes mellitus. Moreover, impaired renal function may be present even in patients with normal urinary albumin excretion (UAE) which further is affected by several extra renal factors.In addition, reduced kidney function is associated with increased incidence of cardiovascular morbidity and mortality.

In diabetic nephropathy glomerular hyperperfusion and renal hypertrophy occurs at the early stages of DM and are associated with an increase in GFR During first 5 yrs of DM, thickening of the glomerular basement membrane, glomerular hypertrophy occurs as the GFR returns to normal. After 5-10 years 40% individuals begin to excrete small volumes of albumin in urine. Although the appearance of microalbuminuria (30-299 mg/day

albumin in urine) is an important risk factor for progression to macroalbuminuria (>300 mg/day albumin in urine), only 50% individuals progress to macroalbuminuria over the next 10 years. Once macroalbuminuria is present, there is a steady decline in GFR and 50% individuals reach ESRD in 7-10 years.

With advance in diabetes and consequent hyperglycemia induced damage to the podocytes and basement membrane increases the permeability to albumin.Co-exsistent glomerulosclerosis and basement membrane thickening causes decreased filtration function of kidney. Consequently this leads to rise in the serum level of creatinine as it is exclusively filtered by the glomerulus with catabolism in tubules without tubular secretion.

Treatment consists of glycemic control, BP control (ACE inhibitors & Angiotensin receptor blockers) & lipid profile control. Weight reduction & dietary modifications (restricted protein intake& sodium restriction) are done to delay or minimize development of nephropathy.

Bunch of Thoughts

Dr. Tamonash Bhattacharya

Since the early phase of the outbreak of CoVid 19 infection an unexpected flow of empiricism has been observed. It's beyond any critical commentary that science finds its way through extensive experience of experiments. But, here's a doubt in the linear projection we see. For the last few decades,we find people (who are decision-makers or policymakers) endorsing only the EBM (Evidence based medicine). In CoVid situation it's gone away, might be as some situational compulsions. The people started to discover hitherto unacclaimed anti-viral properties of a variety of drugs, and introduced as guidelines. But soon or later, those got discarded. Azithromycin, later HCQ, and Famotidine (iv), so many drugs came into play, were prescribed and got used a lot, before they were sent away. Some were claimed to have anti-viral action, some were said to be master of immunity.

And the already acknowledged anti-viral drugs like Favipiravir or Remdesevir are not accepted as the first line drugs in the management of CoVid 19.

Infective or immune pathology, which one needs to be addressed primarily,is yet to be ascertained. Hyper-viscosity due to a state of hypercoagulability is one of the pathological cascades, for which anticoagulant drugs are accepted. Similarly, for immune-pathology including cytokine storms, age-old friend of mankind, the steroids are still the prime mover in saving lives, the other being Oxygen.

This time CoVid days will go worse, so far this morning shows.
But, what's the real time comparison between the mortality rates in the consecutive two years?
This epidemic couldn't give us any definitive medicine, say anti-viral, but we could bring a series of vaccines, unlike the recent previous pandemics of Avian influenza or Swine flu, where some specific medications were successful.

I'm not in a position to believe or disbelieve any conspiracy theory, and astronomically away from the scientific development of vaccines.

Still, a crack in my mind compels me to ask our friends and masters, could this untamed wave of infection be a collateral natural phenomenon of bringing out vaccines within so shorter span?

The virus CoVid 19 with its higher transmission potential showed comparatively little mortality among the common people, excepting the high risk groups including the HCW and people with co-morbidities. What is or will be in this present wave of transmission?

Which would be more helpful to prevent (second) transmission, infection induced herd immunity or the vaccine immunity?

Protective behaviour is judiciously asked to be stringent, but for how long in future?

How much sustainable immunity will be provided by the vaccines vis-a-vis a subclinical (or symptomatic) infection?

FAQs on single-source double-mutant triple-helix Corona variant in India

Dr. Prashant Advani

Q. Why curfew from 8pm to 7am?
A. Due to rising temperatures with the onset of summer, Corona likes to step out when it gets cool. Hence the curfew.

Q. Is it safe to attend Kumbh Mela?
A. Yes. Corona is afraid of religion and does not attack the lakhs who gather to pray to God.

Q. Is it safe to attend political rallies?
A. Absolutely. Corona is terrified of politicians, especially the ones who occupy the highest posts.

Q. Why are only 20 people or less allowed at cremations?
A. Cremations generate lot of heat, which weakens the virus. It tries to escape by jumping from one person to the other. Research has shown that a critical mass of 21 or more people ensures survival of the virus. Hence the restriction of 20.

Q. Why are only 50 people or less allowed at weddings?
A. Indian weddings generate lot of sound & light, which scares the virus (as proved by the PM last year). The virus requires a critical mass of 51 or more people to survive.

Q. Why does Corona not attack crowded buses?
A. The virus has mutated and acquired human qualities. It hates road traffic and avoids buses due to the longer commute. Hence crowded buses are safe.

Q. Are local trains dangerous?
A. Yes. Corona prefers trains due to the quick commute. Also, the virus enjoys the breeze when standing on the footboard.

Q. Does online shopping prevent Corona?
A. Yes. Corona is not able to jump on to fast moving motorcycles and hence does not reach your home.

Q. Why does Corona not attack slum dwellers?
A. Corona cannot stand the competition from malaria & TB and hence focuses on higher income flats.

Q. Does lockdown help?
A. Indeed it does. Corona hangs around outside the door, waiting for people to step out to pick the newspaper, get the milk, go outside etc. Hence lockdown protects you by ensuring you stay inside your home. Note that Corona never enters your home, it only stays outside.

Q. Can I see Covid?
A. Covid is like Maya. It is everywhere & it is nowhere.

Stay home. Stay safe. Stay mad.

CME INDIA COVID-19 Management Protocol- April 2021

Summary

1. Older age, male sex, and comorbidities increase the risk for severe disease.
2. For people hospitalized with Covid-19, 15-30% will go on to develop covid-19 associated acute respiratory distress syndrome (CARDS).
3. When used appropriately, **high $ow nasal cannula (HFNC)** may allow CARDS patients to avoid intubation and does not increase the risk for disease transmission.
4. Steroid/**Dexamethasone treatment** improves mortality for the treatment of severe and critical covid-19.
5. Remdesivirmay have modest bene"tin time to recovery in patients with severe disease but shows no statistically signi! cantbene!t in mortality or other clinical outcomes.
6. Active symptomatic support remains the key treatment for **mildly to moderately ill patients,** such as maintaining hydration, nutrition, and controlling fever and cough.
7. Hospitalized for mild to moderate Covid-19 (not hypoxemic) Supportive care:
   No clear bene"t for Remdesivir or Convalescent plasma.
   Steroids have no demonstrated bene!t and may cause harm.
8. Hospitalized for severe covid-19, but not critical (hypoxemic needing low #ow supplemental oxygen): Corticosteroids (dexamethasone 6 mg/day × 10 days or until discharge or an equivalent dose of hydrocortisone or methylprednisolone).

   May consider Remdesivir. May benefit from use of tocilizumab.
9. Hospitalized for covid-19 and critically ill (needing HFNC, NIV, IMV, or ECMO) Supportive care: Corticosteroids (Dexamethasone 6 mg/day × 10 days or until discharge or an equivalent dose of hydrocortisone or methylprednisolone).

   May consider Remdesivir. May benefit from use of tocilizumab.
10. Timely and accurate diagnostic SARS-CoV-2 testing is a crucial step in managing the patient.
11. **Avoid Fancy anti-virals and HCQS.** At present Ivermectin is also not scienti!cally supported.
12. Low molecular weight heparin, Aspirin, NOACs, (novel oral anticoagulants: dabigatran, rivaroxaban, apixaban, and edoxaban) to be used as it was earlier.
13. **Colchicine might help.**
14. Tocilizumab: More sepsis-related complications, can be used in selected cases with cytokine storm.
15. Anticoagulants may need to be given for 3 weeks or more (2-4 weeks).
16. People are developing Myocardial infarctions, Stroke, etc. few weeks after Corona infection-the Thromboembolic phenomenon.
17. **Oxygen is the superhero.**
18. To be sensible, clinicians must recognize that highly selective, fully e"ective treatments are uncommon in acute care. Focus on High-Quality Evidence Some clinical research is biased.
19. Even the best research methods, such as randomized trials, can be unreliable.This has been ampli!ed by the rapid pace of research undertaken during the COVID-19.
20. It follows that treatment guidelines, national mandates, and bedside care adapt to new data only when the evidence is rigorous, reproducible, and su$ciently strong.

National Webinar During Covid ERA 2020

Diabetes Research and welfare association

DRWA organises

"Digital Health: Way Ahead"

Saturday 27th June 2020 17:00-18:00 hours IST

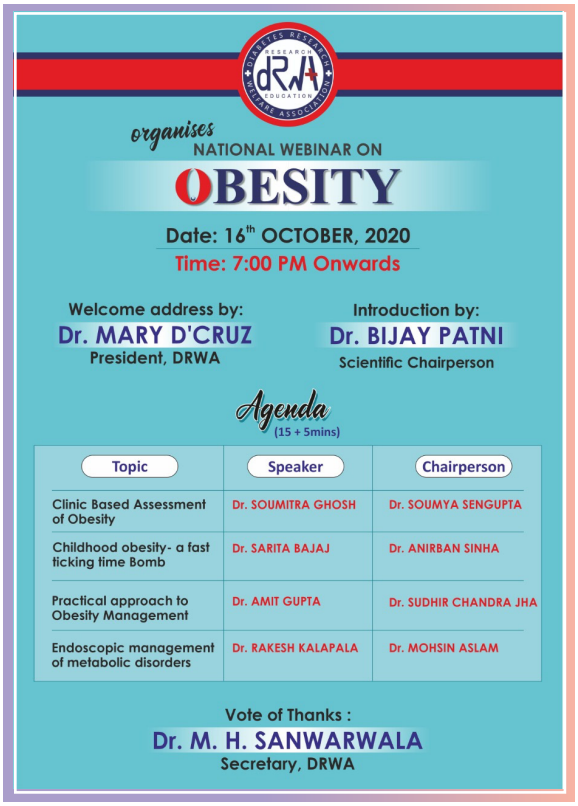
Virtual Webinar

Tentative Agenda

Coordinator: Dr Bijay Patni

Chair: Prof. Supten Sarbadhikari: Introduction to the Panelists and sharing perspective on "***Digital Health Technology to Prevent and Treat Diabetes Mellitus***" (17:00 – 17:10 hrs.)

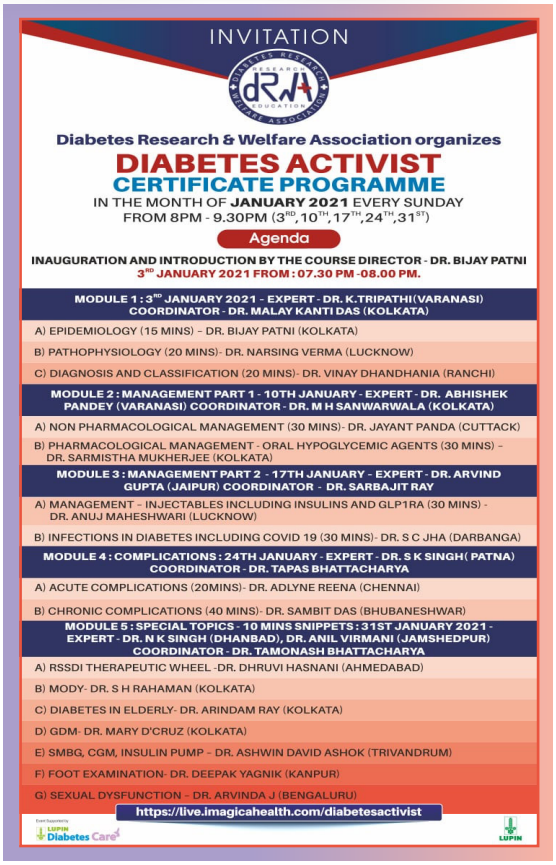
Speaker	Topic	Time
Syed Kadam Murshed	Barriers and Challenges of Adopting Digital Health in Hospitals	17:10 – 17:20 hrs.
Dr. Satadal Saha	Digital Health in Rural Context	17:20 – 17:30 hrs.
Surupa Chakraborti	Developing Workforce to leverage better Outcomes through Digital health	17:30 – 17:40 hrs.
Audience + Panelists	Q&A	17:40 – 18:00 hrs.



World Diabetes Day Camp 14th November-2020

DRWA facilitated Covid 19 Vaccination for Pharmaceutical friends and their family on 7th,8th and 10th June.

DRWA Academic Programmes-2021



INVITATION

Diabetes Research & Welfare Association organizes

DIABETES ACTIVIST

CERTIFICATE PROGRAMME

IN THE MONTH OF **JANUARY 2021** EVERY SUNDAY
FROM 8PM - 9.30PM (3RD,10TH,17TH,24TH,31ST)

Agenda

INAUGURATION AND INTRODUCTION BY THE COURSE DIRECTOR - DR. BIJAY PATNI
3RD JANUARY 2021 FROM : 07.30 PM -08.00 PM.

MODULE 1 : 3RD JANUARY 2021 - EXPERT - DR. K.TRIPATHI(VARANASI) COORDINATOR - DR. MALAY KANTI DAS (KOLKATA)
A) EPIDEMIOLOGY (15 MINS) – DR. BIJAY PATNI (KOLKATA)
B) PATHOPHYSIOLOGY (20 MINS)- DR. NARSING VERMA (LUCKNOW)
C) DIAGNOSIS AND CLASSIFICATION (20 MINS)- DR. VINAY DHANDHANIA (RANCHI)
MODULE 2 : MANAGEMENT PART 1 - 10TH JANUARY - EXPERT - DR. ABHISHEK PANDEY (VARANASI) COORDINATOR - DR. M H SANWARWALA (KOLKATA)
A) NON PHARMACOLOGICAL MANAGEMENT (30 MINS)- DR. JAYANT PANDA (CUTTACK)
B) PHARMACOLOGICAL MANAGEMENT - ORAL HYPOGLYCEMIC AGENTS (30 MINS) – DR. SARMISTHA MUKHERJEE (KOLKATA)
MODULE 3 : MANAGEMENT PART 2 - 17TH JANUARY - EXPERT - DR. ARVIND GUPTA (JAIPUR) COORDINATOR - DR. SARBAJIT RAY
A) MANAGEMENT – INJECTABLES INCLUDING INSULINS AND GLP1RA (30 MINS) - DR. ANUJ MAHESHWARI (LUCKNOW)
B) INFECTIONS IN DIABETES INCLUDING COVID 19 (30 MINS)- DR. S C JHA (DARBANGA)
MODULE 4 : COMPLICATIONS : 24TH JANUARY - EXPERT - DR. S K SINGH(PATNA) COORDINATOR - DR. TAPAS BHATTACHARYA
A) ACUTE COMPLICATIONS (20MINS)- DR. ADLYNE REENA (CHENNAI)
B) CHRONIC COMPLICATIONS (40 MINS)- DR. SAMBIT DAS (BHUBANESHWAR)
MODULE 5 : SPECIAL TOPICS - 10 MINS SNIPPETS : 31ST JANUARY 2021 - EXPERT - DR. N K SINGH (DHANBAD), DR. ANIL VIRMANI (JAMSHEDPUR) COORDINATOR - DR. TAMONASH BHATTACHARYA
A) RSSDI THERAPEUTIC WHEEL -DR. DHRUVI HASNANI (AHMEDABAD)
B) MODY- DR. S H RAHAMAN (KOLKATA)
C) DIABETES IN ELDERLY- DR. ARINDAM RAY (KOLKATA)
D) GDM- DR. MARY D'CRUZ (KOLKATA)
E) SMBG, CGM, INSULIN PUMP – DR. ASHWIN DAVID ASHOK (TRIVANDRUM)
F) FOOT EXAMINATION- DR. DEEPAK YAGNIK (KANPUR)
G) SEXUAL DYSFUNCTION – DR. ARVINDA J (BENGALURU)

https://live.imagicahealth.com/diabetesactivist

Event Supported by LUPIN Diabetes Care — LUPIN

January 2021 Certificate Programme

Diabetes Research & Welfare Association
Presents

DIABETES UPDATE 2021

Organising Chairperson
Dr. Mary D'cruz

Organising Secretary
Dr. M. H. Sanwarwalla

Scientific Chairperson
Dr. Bijay Patni

Patrons
Dr K. Tripathi (Varanasi)
Dr. Lily Rodriques (Hyderabad)
Dr. Narsing Verma (Lucknow)
Dr. Anuj Maheshwari(Lucknow)

Scientific Subcommittee
Dr. Anirban Sinha (Kolkata)
Dr. Sarbajit Ray
Dr. Tamonash Bhattacharya
Dr. T. Satpathi

Logisitic Team
Dr. Partha Biswas
Dr. Malay Kanti Das
Dr. Tapas Bhattacharya
Dr. Ajitesh Ghoshal
Dr. S S Poddar
Dr Agnik Pal

SCIENTIFIC AGENDA
PROGRAMME 20TH February:

04:00 – 05:00 PM	Oral paper presentations (5 mins each) Judges : Dr. K. K. Tripathi, Dr Ashutosh Misra, Dr Sarbajit Ray
05:00 – 05:15 PM	Prediabetes – Disease or Transitional State – Dr Anil Virmani Chairperson : Dr Pallavi Mishra , Dr K. Tripathi
05:20 – 05:35 PM	RSSDI/ESI – Standard Of Care2020/2021 Salient Features –Dr Anuj Maheshwari (lucknow) Chairperson: Dr Harish Darla, Dr Smitha Bhat
05:45 – 06:10 PM	"Choosing a GLP-1 RA designed with patients in mind" Dr Sujeet Jha :Chairperson: Dr Lily Rodriquez, Dr Anil Virmani ,
06:15 – 06:40 PM	T2DM management: Track for young , Trail for elderly Dr H N Chakraborty: Chairperson : Dr N K Singh, Dr S C Jha
06:45 – 07:10 PM	Insulin in Management Of Diabetes: The Essential French Black Dress Since 100 Years Dr Amartya Shankar Chowdhury Chairperson: Dr Anirban Sinha, Dr M H Sanwarwala
07:15 – 07:35 PM	International Speaker Symposium: Type 1 Diabetes – Getting the basics right – Dr Parijat De (UK) Chairperson – Dr Sarmistha Mukherjee, Dr Bijay Patni
	Chairpersons for virtual Symposium: **Dr Narsing Verma, Dr Anuj Maheshwari, Dr Bijay Patni**
07:40 – 08:00 PM	Diabetes and Mental Health– Dr. A.K. Das (Puducherry)
08:05 – 08:25 PM	Relationship between TIR and Diabetes Complication Dr. Banshi Saboo (Ahmedabad)
08:30 – 08:50 PM	Reversal Of Diabetes –is it a myth or fact – Dr. Rajeev Chawla(Delhi)
05:00 – 06:00 PM	High Tea
9:00 PM Onwards	Dinner

Digital Partner CITRNET — Google Play — App Store

PROG ON 21/02/2021:	
08:00 – 08:40 AM	Workshop on HBPM – Dr. Bijay Patni (Kolkata) Moderator – Dr Narsing Verma
09:00 – 09:20 AM	Journey of Insulin from Initiation to Intensification for optimal glycemic goal– Dr Arjun Baidya:Chairperson– Dr Ashutosh Misra, Dr Pallavi Mishra
09:25 – 09:40 AM	DM and COVID 19& COVID Vaccines – DR N K Sing Chairperson: Dr Shaibal Guha, Dr Sarbajit Ray
09:45 – 10:00 AM	DiabetesTechnologies Today– Dr Supratik Bhattacharya (Kolkata) Chairperson : Dr Harish Darla , Dr Dinesh Agarwal
10:05 – 10:20 AM	Dark arts or a welcome disrupter – Dr Prashant Advani Chairperson : Dr Supratik Bhattacharya , Dr Tamonash Bhattacharya
10:25 – 10:40 AM	Understanding the link between CVD& DKD– Dr Ashutosh Misra(15min) Chairperson – Dr K K Tripathi, Dr H N Chakraborty.
10:45 – 11:00 AM	The Gut microbiome: connections between nutrient sensing, glucose and disease: Dr Jayant Panda: Chairperson : Dr Lily Rodriquez,
11:05 – 11:25 AM	The brain as a target of complication in Diabetes Dr Narsing Verma Chairperson : Dr Sujoy Ghosh
11.35 – 11.55 AM	A hepato,– centric view of Diabetes therapy– Dr Sujoy Ghosh Chairperson: Dr Narsing Verma
12.00 – 12:30 PM	DRWA Oration Dr Kamlakar Tripathi Chair person : DR Narsing Verma , Dr Supriyo Mukherjee ,Dr M H Sanwarwala DM and COVID 19& COVID Vaccines – DR N K Singh Chairperson: Dr Shaibal Guha, Dr Sarbajit Ray
12:45 – 01:15 PM	Debate : Moderator : Dr Sarmistha Mukherjee GLP1 RA Or SGLT2 inhibitors – First line in T2DM with known ASCVD or CVD risk factors (Indian perspective) – Dr Lily Rodriquez (SGLT2)/ DrDinesh Agarwal(GLP 1 RA)
01:20 – 01:35 PM	Management of T2DM– make new friends but keep the old Dr. Shaibal Guha (Patna) Chairperson: Dr Ajitesh Ghosal, Dr Partha Biswas
01:40 – 02:00 PM	Two Tales One Story– Improved Outcome of Dapaglifozin– Dr Harish Darla Chairperson: Dr S C Jha,Dr Arindam Sur
02:05 – 02:20 PM	The enigma of Type 3c DM– Dr Mohsin Aslam Chairperson– Dr Tapas Bhattacharya, Dr S S Poddar
02:25 – 02.40 PM	Management of Diabetes during and after Pregnancy Dr. Smitha Bhat (Hyderabad) Chairperson– Dr Mary D'Cruz, Dr Gopabandhu Dutta
02:45 – 03:00 PM	DM and Thyroid– Dr Anirban Sinha(kolkata) Chairperson: Dr Mohsin Aslam , Dr Saumitra Bhattacharyya
03:05 – 03:20 PM	DM and Periodontal Diseases– Dr Jayanta Bhattacharya (Kolkata) Chairperson : Dr T. Satpathi, Dr Shreyasi Satpathi
03:25 – 03:40 PM	Common drug interactions in management of metabolic syndrome– Dr Amod Kr Sachan Chairperson – , Dr Malay Kanti Das, Dr Divendu Bhusan
03:45 – 05.20 PM	PMAntihyperglycemic molecules : (10 Mins Each) Metformin– Dr Partha Biswas SU – Dr Malay Kanti Das Glinides – DR. M. H. Sanwarwalla DPPIV Inhibitors: Dr Agnik Pal AGI – Dr Tamonash Bhattacharya HCQ– Dr T.Satpathi Glitazones – Dr. Mary D'Cruz Early combination therapy – Is this the right step – Dr Divendu Bhusan Q&A (10 MINS) Moderator : Dr Amod Kr Sachan , Dr Sarbajit Ray
05:20 – 05:40 PM	Sexual Dysfunction in metabolic disorders– Dr Deepak Jumani(Mumbai) Chairperson : Dr Tamonash Bhattacharya, Dr Agnik Pal
05:45 PM	Valedictory
01:00 – 03:00 PM	Lunch Hours
04:00 – 05:00 PM	High Tea

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Memories of Diabetes Update-2021