INDIAN MEDICAL ASSOCIATION

Doctors for Doctors and Community at Large

SAY NO TO MIXOPATHY
SAVE HEALTHCARE

CGP NEWS

ASSAM STATE FACULTY

SPECIAL ISSUE ON DIABETES

HAPPY NEW YEAR

and Greetings from IMA CGP ASSAM STATE FACULTY to all the Honourable members of IMA and IMA COLLEGE of GENERAL PRACTITIONERS

Heath first, Healthy profession for Healthy Nation
Take Care
Stay Safe, Happy & Healthy

Official publication of IMA College of General Practitioners

Volume – 6 Issue – 3 July to Sept. & Oct. to Dec.2020

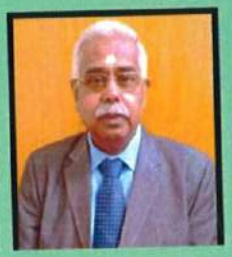
INDIAN MEDICAL ASSOCIATION

HQs, New Delhi

COLLEGE OF GENERAL PRACTITIONERS

IMA HQs, Chennai

DR. RANJAN SHARMA
National President, IMA HQs



DR. R. V. ASOKAN
Hon.Secretary General, IMA HQs

DR. RAMESH DATTA
Hon.Finance Secretary, IMA HQs

DR. HIRANMAY ADHIKARY
Dean, IMA CGP HQs

DR. L. YOSODHA
Hon. Secretary, IMA CGP HQs

IMA COLLEGE OF GENERAL PRECTITIONERS

ASSAM STATE FACULTY, H.Qs - GUWAHATI

DR. SATYAJIT BORA
President, IMA, ASB

DR. HIRANMOY ADHIKARI
Past President, IMA ASB

DR. HEMENDRA KR. BORAH
Director, IMA CGP ASF

DR. SAMSUL HAQUE
Past Director, IMA CGP ASF

DR. D.C. BORBORA
Sr. V.P., IMA, ASB

DR. B.D. BHARALI
V.P., IMA, ASB

DR. HAMANGA BAISHYA
Hony.State Secy, IMA, ASB

DR. RAJUMONI SHARMA
Finance Secy, IMA, ASB

DR. JAGADISH BASUMATARY
Editor, Hony. State Secy
IMA, CGP ASF

DR. MIHIR KR. SEN
Past Secy, IMA,CGP ASF

DR. RUPAM DAS
Jt. Secy, IMA, CGP ASF

- The views expressed in the articles are the authors of each article and not of CGP News
- For all correspondence, write to Hony. Secretary, IMA CGP, ASF Dr. Jagadish Basumatary

Editorial

Greetings to you all from the editorial board. We are glad that the 3rd issue of our journal will reach each one of you after a fortnight from now. It also gives me special sense of pleasure to note that the release of this issue will coincide with a CME which will be conducted by IMA CGP ASB tentebely on last part of the April' 2020 in Tezpur. Indeed a commendable achievement on the part of IMA CGP ASB which has not even completed a year after its formation. Let me reiterate with a deep sense of appreciation that all these have been possible only due to the wisdom and dedication of Dr. Hemendra Kumar Borah, Director, IMA CGP ASB.

For kind attention of esteemed members :

The following editorial was written when Covid 19 infection started to emerge as a major health threat in our country and GOI Health Authority had to issue notification imposing restrictions in all forms of public gathering like social, political, religious, sports, academics etc etc. As a consequence of this organisers of many academic events had no option left but to either postpone or cancel their programmes. We, the IMA ASB followed suit too. We had to postpone our IMA ASB and IMA ASB CGP CME where the 3rd issue of this journal was supposed to be released. We waited and waited but to no avail. Things turned from bad to worse. The spread of CORONA engulfed the whole country alarmingly. In Assam also the scenario is grim. The total number of infected people has crossed over one lakh seventy thousand and about thirty five thounds among these are active cases and they are undergoing treatment. The death toll is nearing seven hundred mark already. Unfortunately, we have lost several of our dear doctor colleagues also to Corona. The whole health department in our state has swung into action since March to effectively tackle the crisis. Among the thousands of health care providers our doctors are leading from the front

day and ceaselessly. Many of them have caught the virus during duty and were quarantined. They, however, returned to resume their duties without fear and hesitation as soon as they were declared negative. Commitment and Dedication of highest order indeed !! Importantly, genuine issues of our concerns also kept emerging time to time during this battle. Violence against on duty corona warriors were also reported now and then. Health authority would suddenly issue notification on duty hours and quarantine period for doctors which, if implemented, would have subjected our doctors to severe degree of physical and mental stress. Issues of health insurance for on duty corona doctors was another issue to ponder upon. It's heartening that IMA voiced constantly and strongly in these matters and talked across the table with Government for the welfare of its members who were fighting the corona crisis. We have reposed our faith in IMA. Our battle will continue and we know not how long? But life must also go on. Show must go on too. With this philosophy IMA ASB is again getting ready to resume its activities. We are hopeful of meeting each other soon and it is there where we will be glad to bring this journal for your happy reading. Finally, I, on behalf of the Editorial Team, take this opportunity to extend our welcome and good wishes and would pray to God to grant you and your family safety and good health all the time.)

Few words about CME.

There is an unceasing requirement of advances in clinical practices and patient care, thereby changing the concepts and approaches towards diagnosis, prevention and treatment. It is therefore a primary responsibility of physicians to keep abreast of the ever increasing advances in the medical field, requiring constant updating of medical knowledge to provide highest quality of care to their patients. CME is an educational activity that contribute to maintaining, improving and updating a physician's knowledge, expertise and professional performance.

The Medical Council of India (MCI) is the national governing body responsible for the establishment and maintainance of high standard of medical education as well as recognition of medical qualifications. The MCI and the 26 State Medical Councils(SMC) are the major CME Regulator in India. They also acredits to the Health Care Professionals (HPC).

In 2002, with the approval from Government of India, the MCI issued a gazette notification(Code of Medical Ethics Regulation, Amended upto 8 October 2016), related to professional conduct, etiquette and ethics for registered medical practitioners.

Regulation 1.2.3 states :

" A physician should participate in professional meetings as part of CME programmes for at least 30 hours every five years, organised by reputed professional academic bodies or any other authorised organisation. The compliance of this requirement shall be informed regularly to MCI or SMC as the case may be."

As a part of it's relentless effort to provide update informations to the doctors working in far flung areas, IMA CGP ASB has this time taken the initiative to hold a 4 hour long IMA ASB CREDIT POINT CME on 22nd March'2020 in Tezpur. It's indeed a laudable step. We take this opportunity to reaffirm our commitments and we take a pledge that IMA CGP will try to do evrything possible for keeping our colleagues abreast of the latest updates on medical science.

Finally, as the Spring is approaching fast, so is our much awaited Rongali Bihu. We wish you all a very happy and healthy Rongali Bihu and New Year in advance. May love, brotherhood and peace prevail upon all of us.

Long ALive, IMA CGP

Dr. Jagadish Basumatary
Hony. Secretary, IMA CGP
Tezpur

Guest Editorial

It is my great pleasure to write the editorial as guest editor of third issue of IMA 'CGP News' of Assam State Faculty. This issue of 'IMA CGP News' ASF, mostly emphasizes on one of the most concerning issue in the medical fraternity i.e. Diabetes Melitus one of the horrific most common non communicable disease in the world- irrespective of developed, developing and under developed countries.

Diabetes mellitus is one of the most prevalent non communicable chronic disease with multi organ morbidity which handicapping the day today activities of the affected person and with significant mortality. How we could explain the significantly increasing trend of the Disease like Diabetis Melitus globally? Is it due because of food, lifestyle, of course mechanization of life has got some role in initiating the problem. Hereditary has got some role to play on it.

As it going on increasing trend simultaneously reachers is also going on to combat this horrific disease. The hallmark of diabetes is dysregulation of glucose homeostasis. As Diabetes is not curable, concept of treatment is to control the sugar in the blood near normal level. So it needs a very good teaching to the patient by the treating doctors. The patient has to understand what is diabetes mellitus and had to deal with it. So it is the responsibility of the physician to make them understand.

Many of the patients with diabetes they do not have preliminary knowledge about the disease which is a dangerous scenario to treat the patient comfortably. Treating doctors should give a try to give a brief idea about diabetes (what it is, how to control) and later on both doctor and patient will be more comfortable in controlling the diabetes. There are major challenges for the medical fraternity to educate the people about diabetes and I think it is one part of our service. But it is crucial that we remain optimistic. This is no time to get discouraged or to think that task is impossible.

Since the beginning of diabetes so much development has made in the field of its understanding and its management. More particularly during last two decades tremendous development has been made in understanding and treatment of diabetes mellitus. Some innovative advances in medical technology are going to be integral part of person living with diabetes. These innovations improve the quality of life by decreasing morbidity and mortality significantly intelligent closed loop Insulin Pump (artificial pancreas), Non invasive glucose sensor are concepts are already established in the field of diabetes management. Artificial Intelligence (AI) is also coming up rapidly for management of diabetes in place of human intelligence.

Although now communicable Disease(NCD) is one of the major challenge in day to day practice, yet some other devastating challenges are coming up at a gap chattering the human life all over the world killing thousand,lakhs of people within a short span of time. If we go through pandemic history of diseases, the influenza pandemic of 1918-19 which is also widely known as Spanish Flu, infected atleast one third of the world's population in just eighteen months. Estimates on the exact number of fatalities vary from 20 million to 100 million deaths. At that time there were no lab test to detect the virus a vaccine to immunise the population and the health department relied on intervention such as quarantime, isolation and limits on public gatherings. In 1960 a bout of Asian Flu virus causes a havoc in some countries. During recent years different virus pandemics like Swine Flu, Ibola, Nipa, Jika, causes a concern to the world population.

Now a serious concern globally is Corona virus,COVID -19 . Till date about three crores of population infected with COVID-19, 10 lacs died of it world wide within 9 months. It is a big number since single infected case in 30th Dec,2019.

India is second on the list of infected tally after United States of America. In India during the month September,2020 average daily 70,000 – 75,000people are infected on COVID-19 and in some days it goes upto 97,000. The total number of infected cases in India today on29th September,2020 is 61 lacs and death is

96,000. In the initial period during first three month of the year COVID -19 cases in Assam was very negligible, but gradually swinging up during last three months with daily average of positive cases more than 2000 per day. And it is an alarming situation for a small State like Assam.

To stop the Corona virus at present we donot have a definitive treatment or vaccine. Although different trials for Corona vaccine is going on world wide yet no good result is coming up . Another one year may have to wait and to get freely available Corona vaccine. In the meantime as the Corona cases increases the scarcity of hospital beds and facilities of treatment getting a trouble some issue. One of the mainstay of treatment of Corona is Oxygen and initial requirement at the starting of disease was 750 tons and now it goes upto 28000 tons daily. The Indian Gas manufacture Organisation claimed that it will be difficult to manage the requirement.

Now our mainstay to stop the Corona pandemic will be relied on quarantine, isolation ,limits on public gathering plus use of musk, Social distancing and hand sanitisation . Government has time to time published the SOPs for Corona prevention, but general public donot adhere to it. It needs public awareness. Awareness is to be such that general public as a whole understand the issues and take it from their heart and souls. Till now we failed to make people understand the real meaning of use of musk and social distancing. The Government machinaries, NGO and all responsible citizens should take an honest effort to mittigate the life threatening issue of corona virus so that we can save world.

WHO Chief Tedros Adhanom Ghebreyesus warned that it is not the corona virus COVID -19 last pandemic and world must be better prepared for next pandemic and countries must invest in public health to takle the next pandemic in a better way. Another arthropod borne virus (Arboviruses) Cat Que virus spreading in China may spread to India and other countries as well. It will be also a alarming situation which cause febrile illness,meningitis and pandemic encephalitis. Due care to be taken that it will not cause another pandemic.

Since last year Dr. Hemendra Kumar Borah, Director IMA, CGP, ASF have taken very encouraging effort of introducing IMA CGP News of Assam State Faculty and not only giving an extra flavor to IMA ASB but a beautiful academic feast quarterly with different taste. Dr. H. K. Borah who is very sincere good organiser I hope that he will leave his legacy to the next one so that it will flourish and spread beautiful fragrance to everyone of us.

We are very much delighted to see the enthuasim of the writers to this edition of the IMA CGP News and offer our sincere thanks and gratitude to them.

Long Live IMA

Dr. Laksheswar Bhuyan

INDIAN MEDICAL ASSOCIATION

CGP NEWS

ASSAM STATE FACULTY

Printed at : **M/s Extensile**
Anwar Complex, Tezpur
Dist.-Sonitpur (Assam)
Cont.-9435067530

Contents

Dr. Ravi Wankhedka
National President,IMA,
Treasurer World Medical Association and
President SAARC Medical Association.

WHEN DOCTORS SOLDIER ON, SOCIETY NEEDS TO PLAY ITS PART TOO

When doctors soldier on, society needs to play its part too" "Ravi Wankhedkar " "Caught between the ethical and moral responsibility of our commitment towards patients on one side and various assaults on the other, doctors and HCGs (health care givers) find themselves in a bind." "It's eight months into the Covid 19 pandemic, with over 57 lakh cases and 91,000 deaths, and the peak of this crisis is nowhere in sight. Doctors are exhausted, frustrated and the burnout threshold is near." "And, we are scared. More than 10,000 HCGs have been infected and over 500 have lost their lives. The rate of infection and death rate amongst HCGs is 4 times that of the general population. " "But the assaults continue and in different forms. There is the physical violence, despite stringent laws. There are also legal assaults in the form of various directives undermining and encroaching on the autonomy of doctors. There are also financial assaults by way of huge losses in running hospitals, capping of charges without a scientific basis, with no capping of inputs and without any subsidies." "Meanwhile, as the disease spreads in the community, every patient or relative could be a potential source of infection. And that's the difference between soldiers in war and us. Soldiers' families are safe while our families are getting infected and dying because of us." "The frustration is building up in the doctor community because of the carelessness of people, intent on breaking safety norms. Besides, the over bureaucratisation of healthcare is further leading to improper policies." "People need to understand that the virus is constant, it is our behaviour that needs to change! So, to tire of the lockdown and head out without a mask for instance or be in a crowded place is irresponsible behaviour endangering other people and adding to the pressure on the medical community." "This is a pandemic, health experts need to formulate policies and the bureaucracy implement it. But the opposite is happening! The bureaucracy seems to behave like a modern day Autocracy, giving diktats, often contradictory. Fixing the Virus is priority, the rest (including problems with the economy) can then be fixed and not the other way round. In the delicate balance between Life and livelihood, life is the priority. "Today doctors feel like they are in the driver's seat of an old car(our dilapidated

and chronically neglected health system), with no steering-wheel in our hand (backseat driving is by the bureaucracy), a hostile jeering crowd (media-bashing and assaults). And yet, they are expected to win a Formula 1 race!" "The tipping point for all of us is near. Discovery of a safe and effective vaccine is still at least a year away. It's going to be a very long haul for us. Even after vaccine is available, there will be logistical nightmares to get everyone vaccinated. And that too will be rolled out through doctors. " "For society's wellbeing, the medical community should not be driven to the point of no return. Govt, opinion makers and the society we live in need to understand that HCGs are also human." "We don't need 'thalis' (applause), nor "gaalis" (abuse), don't light candles for us. Help keep us safe too, so that no one has to light candles on our graves. " "Putting doctors on the high pedestal of warriors or soldiers, helps the fallacies in the system to get brushed under the carpet where we are supposed to make the supreme sacrifice even without having the necessary tools for safety. " "Funds are spent on many unnecessary projects, when it should have gone into building a good health care delivery system and protective gear for the HCGs. Even doctors, especially the young ones entering the system need encouragement and a safe conducive working environment. The medical community should get hazard pay bonus, incentives, subsidies for health care establishments, insurance cover, recognition of services etc from the Government. "These are unprecedented times for all of us, as they are testing times too. It's a new disease where everyone is learning. Everybody is under stress, most of all the medical community. So the Government and society have a role to play in not pushing doctors and healthcare workers to the brink." "(The writer is former National President, the Indian Medical Association; Treasurer World Medical Association and President SAARC Medical Association. Views are personal)

.....

One most Important

1. *Maskingis the prevention*
2. *RTPCR CT is the test for Diagnosis*
3. *Zinc is the Vitamin*
4. *Dat 5 is the day in covid phase for mortality prevention*
5. *Day 90 is the day after which the word covid ends*
6. *Home isolation is the modality of treatment*
7. *12 years is the age when the mortality starto*
8. *CRP is the Lab test for seriousness*
9. *Loss of smell is the symptoms equal to RTPCR test*
10. *15 minutes is the time to get the infection*

Sources –Medical Voice for Policy Change

Prof. Dr. J. A. Jayalal MS, FRCS (Glasgow),MBA,PhD
Professor of Surgery KGMCH
National senior vice president IMA(2019)
IMA CGP National Hon,Secretary(2008-10)
President Elect- 2020-2021

Diabesity: The Worldwide Twin Epidemics of Obesity and Diabetes

Introduction

Type 2 diabetes mellitus (T2DM) is a major public health problem that is significantly linked to another worldwide epidemic—**obesity**. Although there are many risk factors for developing T2DM, such as age, stress, certain medications, and genetics or family history, having overweight or obesity remains the single best predictor of T2DM.[1] Both T2DM and obesity are metabolic disorders characterized by defects of insulin, insulin resistance and insulin deficiency.[2] Since weight gain and body mass index (BMI) have strong relationships to diabetes and insulin resistance, it is no surprise that nearly 90 percent of people living with T2DM also have overweight or obesity.[1]

In this article, we provide statistics and predictions for both diseases, present management options for specific patient populations, describe multiple theories on why T2DM improves or, in some cases, resolves in individuals with obesity following metabolic surgery, and outline the latest consensus guidelines for T2DM treatment based on the available evidence.

The Evolution of Diabesity

The terminology surrounding obesity, T2DM, and bariatric surgery has evolved through the years. Although surgical procedures are used for treatment of both obesity and diabetes, it is important to make a distinction between **bariatric surgery** and **metabolic surgery**. Bariatric surgery is defined as a procedure performed for weight control, while metabolic surgery is defined as gastrointestinal surgery performed with the goal of treating diabetes and metabolic syndrome.[3]

The concept of combining obesity and diabetes as a single entity began emerging in 1973, when Sims et al[4] reported the discovery

that endocrine and metabolic changes are associated with weight gain. Over the decades, as metabolic syndrome and metabolic surgery began rising to the forefront, the words "diabetes" and "obesity" were eventually unified to create the new, more descriptive term—**diabesity**.

In 2017, The American Association of Clinical Endocrinologists (AACE) and American College of Endocrinology (ACE) adopted a new term—adiposity-based chronic disease (ABCD)—that explicitly identifies a chronic disease, alludes to a precise pathophysiologic basis, and avoids the stigma and confusion related to the differential use and multiple meanings of the term obesity.[5] The term ABCD was later incorporated into the IFSO mission and vision statements.

MORBIDITY AND MORTALITY.

The risk of developing multiple co morbidities, including T2DM, significantly increases with obesity. Other risks include cardiovascular disease, hypertension, stroke, gastro esophageal reflux disease (GERD), obstructive sleep apnea (OSA), nonalcoholic steatohepatitis (NASH), and cancer.[6] The risk of developing diabetes is almost 20 times higher in individuals who have BMIs of 35kg/m2 or higher than in those with BMIs less than 25kg/m2 (relative risk [RR]: 14–20 for women, 19–33 for men; confidence interval [CI]: 95%).[7] One study found a 20- to 30-percent increase in the risk of death among a cohort of patients with obesity, who were 50 to 71 years old at enrollment in 1995 to 1996, compared to those with normal weight.[8]

A recent study concluded that obesity and diabetes were the leading causes of preventable death in the United States, knocking tobacco use from the top spot.

1973, when Sims et al[4] reported the discovery **LIFESTYLE MODIFICATIONS.**

The management of ABCD should incorporate not only the symptoms, but also the severity and genetic characteristics of the disease. Lifestyle modifications, self or professionally directed, should provide the foundation for more invasive options and might ensure longevity for weight maintenance and cardiovascular health. Lifestyle modifications include change in diet rather than just caloric restriction, elimination or replacement of weight-promoting medications, restoration of healthy sleep cycles, and increased physical activity.[9]

In the Look AHEAD trial, individuals who lost at least 10 percent of their body weight in the first year of the study had a 21-percent lower risk of the primary outcome (adjusted hazard ratio [HR]: 0.79; 95% CI: 0·64–0·98; p=0·034) and a 24-percent reduced risk of the secondary outcome (adjusted HR 0.76; 95% CI: 0·63–0·91; p=0·003) compared with individuals with stable weight or weight gain.[10]

In people with insulin resistance, research indicates that lifestyle modifications could reduce the risk of developing diabetes up to 50 percent. The initial goal for patients with overweight or obesity and T2DM is to lose 5 to 10 percent of their weight within the first six months of treatment. Due to the intense need for follow-up and difficulty adhering to a new lifestyle, sustained long-term results are very limited.[3]

If lifestyle modifications fail to achieve satisfactory weight loss or weight maintenance, or if the patient has diabetes, treatment should include medications tailored to the treatment of diabetes and weight reduction.

MEDICATION:

Prospective studies comparing metformin with placebo have shown that patients taking metformin were able to lose weight for at least 10 years of treatment. This result was statistically significant compared with the placebo group. Unfortunately, some common diabetic medications are associated with weight gain, especially insulin. On the other hand, the glucagon-like peptide 1 receptor (GLP-1R) agonists are associated with modest weight loss.[11]

Medications primarily targeting weight loss, such as phentermine, topirmate, lorcaserin, and combination drugs (e.g., naltrexone HCl and bupropion HCl, phentermine and topiramate

extended-release can be effective for treating T2DM in some patients.

Liraglutide ,a GLP-1R agonist, was approved by the United States Food and Drug Administration (FDA) for weight loss in the absence of diabetes, but is generally cost-prohibitive for most patients at this time.

SURGERY:

Bariatric/metabolic surgical procedures, such as Roux-en-Y gastric bypass (RYGB), have been shown to be effective not only with absolute weight loss, but also with weight maintenance. Compared with medical management alone, these surgical procedures have direct effects on glucose metabolism and reduce the micro- and macrovascular complications associated with diabetes.In addition, bariatric surgery has been shown to significantly improve survival in patients with obesity compared with those who didn't undergo surgery.[12]

The efficacy of bariatric surgery for weight loss and T2DM remission in descending order is as follows:

- biliopancreatic diversion with or without duodenal switch (BPD+/-DS),
- RYGB, sleeve gastrectomy (SG),
- laparoscopic adjustable gastric banding (LAGB).[13]

COMBINATION THERAPY:

Complete resolution of diabetes may not always be possible. There are many factors that can influence the chances of remission, such as the surgical procedure, duration of diabetes, preoperative glycemic control, and number of medications taken preoperatively. For this reason it is not uncommon that a combination of surgical and medical therapy might be used.[15]

Since metabolic changes occur immediately after bariatric surgery independently of weight loss, it is important to titrate down a patient's diabetes medications postoperatively because there is a higher propensity to hypoglycemia during this period. For patients who were on insulin preoperatively, the short-acting insulin should be decreased by at least 50 percent, and long-acting insulin can be stopped if the dose was less than 30 units or decrease by more than 50 percent if it was higher 30 units.

Metformin can be continued as a stand-alone therapy in the postoperative period for patients with HbA1c less than nine percent. If HbA1c is higher than nine percent, a second oral agent can be administered. Long-acting

discouraged in general due to the risk of hypoglycemia or decreasing the bone density, respectively. Long-term management with sodium-glucose cotransporter-2 (SGL2) inhibitors and e glucagon-like peptide-1 (GLP-1) analogues have been favored as well as an adjusted insulin regimen.[16]

These are all general recommendations; however, individualized treatment should be tailored based on a patient's response and glycemic levels.

METABOLIC SURGERY: MECHANISMS OF ACTION: PROPOSED THEORIES:

There are multiple theories on why diabetes improves or, in some case, resolves, in individuals with obesity following metabolic surgery. These theories are not necessarily related to weight loss since many of the changes in glucose control are seen before there is significant weight loss. The mechanisms of action are still not completely understood, and no a single theory is considered an absolute truth. The reality is that bariatric surgery causes a complex interaction of many known and certainly some unknown metabolic factors.

In order to understand some of the mechanistic processes of the effect of bariatric surgery on T2DM, it is important to define incretins. Incritins are intestinal hormones that promote postprandial secretion of insulin. Two major peptides have been identified in the mechanistic processes of bariatric surgery—gastric inhibitory peptide (GIP), which are secreted by the K cells in the duodenum, and GLP-1, which is secreted by the L cells in the ileum. GLP-1 stimulates insulin secretion, proliferation of pancreatic beta cells, and increases gluconeogenesis in the liver and skeletal muscle. GIP stimulates beta cells and inhibits their apoptosis. GIP also stimulates lipoprotein lipase activity.

METABOLIC THEORIES.

There are two main theories relating to the metabolic effects of various bariatric surgical procedures. Other incretins and naturally occurring substances and microorganisms likely influence these two major theories: the foregut theory and hindgut theory.

The hypothesis of the foregut theory is that there are "anti" incretins in the proximal small bowel that lead to abnormal glycemic control, theoretically to prevent hypoglycemia. With surgeries like the RYGB or BPD, this region is bypassed and the secretion from those substances avoided.

The hypothesis of the hindgut theory is that the rapid delivery of nutrients to the ilium stimulates secretion of GLP-1 and thus helps to better regulate blood sugar and improve diabetic control.

GHRELIN

Ghrelin is an incretin that is also known as the "hunger hormone" and is secreted mainly at the gastric fundus. It is believed to not only increase hunger, but to also inhibit insulin secretion. Most cells producing ghrelin are removed in the SG procedure, and the fundus is bypassed in RYGB.

BILE ACIDS

Bile acids play a role in the digestion and absorption of fat and liposoluble vitamins and are involved in the enterohepatic circulation (EHC), which is the movement of bile acid molecules from the liver to the small intestine and back to the liver. Bile acids interact with intranuclear receptors in the liver, inhibiting gluconeogenesis and promoting release of GLP-1. Some studies have shown that levels of bile acids increase after SG.

GUT FLORA

Human gut flora is dominated by two major groups of bacteria: *Bacteroides* and *Firmicutes*. Research has shown that there is a larger presence of *Firmicutes* in patients with obesity. Studies in mice models have demonstrated that insulin sensitivity improved after transferring intestinal flora from lean mice donors to insulin-resistant mice. Certain microbiota also induce the breakdown of substances like fatty acids, which could lead to an increase of GLP-1 and peptide YY (PYY). PYY stimulates the vagus nerve to produce satiety.

Main recommendations from the DSS-II Consensus include the following:

There is sufficient clinical evidence that supports the inclusion of metabolic surgery in the management of diabesity and treatment algorithms that include surgery should be considered

- RYGB, SG, LAGB, and BPD +/-DS each have their own risk/benefit ratio. Any other procedure is still considered investigational. Recommendations on patient selection for metabolic surgery include the following:

-Class III obesity (BMI e"40kg/m2)

-Class II obesity(BMI 35.0–39.9kg/m2) with inadequate glycemic control

-Class I obesity (BMI 30.0–34.9kg/m2) with inadequate glycemic control despite optimal medical treatment by either oral or injectable medication.

- RYGB appears to have a more favorable risk/benefit profile in most patients with T2DM.
- SG is an effective procedure for weight loss and control of T2DM, at least in the short and medium term. Long-term studies are required.

- LAGB is effective in improving glycemic control to the degree that it causes weight loss; however, this procedure has no metabolic effect when used alone and is associated with a greater risk of revisions and complications.

- BPD +/- DS is the most effective procedure in terms of glycemic control and weight loss; however, it's associated with a greater risk of nutritional deficiencies, which make it less favorable than other metabolic procedures and should be considered only in patients with super obesity (BMIe"50kg/m2).

- After surgery, a multidisciplinary team with expertise in diabetes should manage the patients.[18]

BARRIERS

It has been estimated that about 80 percent of people with diabetes live in low- or middle-income communities.[8] Access to metabolic surgery in these populations might be limited, with cost being the primary barrier. Despite the proven track record of bariatric/ metabolic surgery as it pertains to safety, efficacy and durability; a low number of patients are referred to bariatric/metabolic surgeons. This could be due to misconceptions about the risk of surgery versus the natural progression of the disease itself, inadequate knowledge about diabetes remission with metabolic surgery, and simple dismissal of the well-documented outcomes with metabolic/bariatric surgery.

The ADA and the IDF helped develop and have ratified the DSS-II consensus statements and guidelines, and several other organizations have formally endorsed the DSS-II consensus statements and guidelines. This is significant when one considers that just a few years ago, metabolic surgery was not in the treatment algorithms of the major diabetes treatment organizations. It was not until 2009 that the ADA included metabolic surgery in their annual guidelines, but even then, the recommendations were based on weight, not T2DM management.

They also included lower weight-based parameters in their recommendations, in order to address the Asian populations who have a higher risk of diabetes for the same basal metabolic index.

Participants at the Policy Lab, held during the 3rd World Congress on Interventional Therapies For Type 2 Diabetes, identified four building blocks to facilitate change in practice and policy, with the ultimate goal of increasing the use of metabolic surgery for the patients who can benefit. The building blocks are:

1) Communicate scale of diabetes challenge;
2) Properly articulate the role for bariatric/ metabolic surgery;
3) Identify cost effectiveness/savings;
4) Explore resources/processes to support surgery access.

CONCLUSION

The belief that diabesity is avoidable through self-control and caloric restriction needs to be dismantled. This can be achieved through constant education. With continued lobbying and policy change, access to care and referrals for metabolic/bariatric surgery by other physician groups will improve, ultimately helping clinicians achieve optimal results among the diabesity population.

References

1. World Health Organization. Obesity and Overweight Fact Sheet. http://www.who.int/dietphysicalactivity/media/en/gsfs_obesity.pdf Accessed August 30, 2018.

2. Verma S, Hussain ME. Obesity and diabetes: An update. Diabetes Metab Syndr. 2017;11(1):73–79. Epub 2016 Jun 17.

3. Gadde KM, Martin CK, et al. Obesity: pathophysiology and management. J Am Coll Cardiol. 2018;71(1):69–84.

4. Sims EA, Danforth E Jr, Horton ES, Bray GA, Glennon JA, Salans LB. Endocrine and metabolic effects of experimental obesity in man. Recent Prog Horm Res. 1973;29:457–496.

5. Mechanick JI, Hurley D, Garvey WT. Adiposity-based chronic disease as a new diagnostic term: The American Association of Clinical Endocrinologists and American College of Endocrinology position statement. Endocr Pract. 2017;23(3):372–378.

6. Field AE, Coakley EH, Must A, et al. Impact of overweight on the risk of developing common chronic diseases during a 10-year period. Arch Intern Med. 2001;161:1581–1586.

7. Adams KF, Schatzkin A, Harris TB, et al. Overweight, obesity, and mortality in a large prospective cohort of persons 50 to 71 years old, N Engl J Med. 2006;355:763–778.

8. Cleveland Clinic. Obesity is top cause of preventable life-years lost, study shows. ScienceDaily. April 22, 2017. www.sciencedaily.com/releases/2017/04/170422101614.htm. Accessed August 30, 2018.

9. Wadden TA, Webb VL, Moran CH, Bailer BA. Lifestyle modification for obesity: new developments in diet, physical activity, and behavior therapy. Circulation. 2012;125(9):1157–1170.

10. Look AHEAD Research Group. Association of the magnitude of weight loss and changes in physical fitness with long-term cardiovascular disease

outcomes in overweight or obese people with type 2 diabetes: a post-hoc analysis of the Look AHEAD randomised clinical trial. Lancet Diabetes Endocrinol. 2016;4(11):913–921.

11. Diabetes Prevention Program Research Group. Long-term safety, tolerability, and weight loss associated with metformin in the Diabetes Prevention Program Outcomes Study. Diabetes Care. 2012;35:731–737.

12. Tremmel M, Gerdtham UG, Nilsson PM, Saha S. Economic burden of obesity: a systematic literature review. Int J Environ Res Public Health. 2017;14(4):pii: E435.

13. American Diabetes Association. Economic Costs of Diabetes in the U.S. in 2017. Diabetes Care. 2018;41(5):917–928.

14. M acDaniels JS, Schwartz TL. Effectiveness, tolerability and practical application of the newer generation anti-obesity medications. Drugs Context. 2016;5:212291.

15. Adams TD, Arterburn DE, Nathan DM, Eckel RH. Clinical outcomes of metabolic surgery: microvascular and macrovascular complications. Diabetes Care. 2016;39(6):912–923.

16. Adams T, Gress RE, Smith SC, et al. Long-term mortality after gastric bypass surgery. N Engl J Med. 2007; 357:753–761.

17. Sjöström L. Review of the key results from the Swedish Obese Subjects (SOS) trial—a prospective controlled intervention study of bariatric surgery. J Intern Med. 2013;273(3):219–234. Epub 2013 Feb 8.

18. Koliaki C, Liatis S, le Roux CW, Kokkinos A. The role of bariatric surgery to treat diabetes: current challenges and perspectives. BMC Endocr Disord. 2017;17(1):50.

xxxxxxxxx

COVID

C- Change is the constant = Accept it
O – Optimize own Health = Invest in it
V- Value your Education = Respect it
I – Innovate and Integrate = Inculcate it
D – Doctors for Doctors = Support it

Dr. K.K. Aggarwal
President CMAAO, HCFI and
Past National President IMA

What to do if you are asked to do Self-Home Quarantine

Stay home unless you must see a doctor. No work, school, college, cinema halls or shopping. If you must come out of your room, wear a surgical mask. And don't share towels.

Self-quarantine and self-isolation are different. The first measure is for the large numbers of healthy people who may fall sick following possible exposure. The second is for people who are ill with the coronavirus — they are a danger to their family and visitors, and must be watched carefully in case they deteriorate.

At the moment, with testing still not available all over it is best to self quarantine.

Governments have the power not just to advise quarantine but to order it.

Stay Home: if returned from any affected country; have symptoms of **fever and a dry cough,** spent time in other countries or on cruise ships; or ill without any known source of infection.

Home quarantine of 2 weeks may be unpleasant especially if you have young children or elderly relatives to care for, or live in cramped quarters with a lot of roommates.

Separation If you are potentially infectious, it is important that you separate yourself from your partner, your housemates, your children, your elderly aunt. Shouldn't even pet your dog, although pets are not known to transmit the coronavirus.

A room must be designated for your exclusive use. A bathroom should be, too, if possible. Every surface you cough on or touch could become contaminated with the virus.

Have no visitors, and keep three to six feet away from others. Don't take the bus or metro, or even a taxi.

Masks If you must be around other people — in your home, or in a car, because you're on your way to see a doctor (only after you have called) — you should wear a surgical mask, and everyone else should, too. If you can't, you can create a makeshift one from a scarf or other garment.

Hygiene If you cough or sneeze, you should cover your mouth and nose with a tissue, and discard the used tissue in a lined trash can. Then you must immediately wash your hands with soap and water for at least 20 seconds.

You can use sanitizer, if you can find it, but soap and water is preferred.

Even if you haven't coughed or sneezed, you should wash your hands frequently, and avoid touching your eyes, nose and mouth, if you haven't just washed them.

Disinfect Don't share dishes, drinking glasses, cups, eating utensils, towels or bedding with anyone (including your pets). Wash these items after you use them.

Countertops, tabletops, doorknobs, bathroom fixtures, toilets, phones, keyboards, tablets and bedside tables are considered "high-touch surfaces" — wipe them often with a household cleanser or 0.1% bleach.

Frequently wipe down surfaces that may be contaminated by bodily fluids, including blood and stool.

Monitoring Keep an eye on your health and call a doctor if you develop symptoms or if they worsen. Make sure to tell the medical staff that you are at risk of coronavirus.

Household members Housemates can go to work or school, stock up on groceries, pick up prescriptions, take care of the quarantined and keep the place clean.

They'll be wiping down doorknobs and countertops, doing loads of laundry and washing their hands — a lot.

Family members and other occupants should monitor the patient's symptoms and call a doctor if they see a turn for the worse.

When around a symptomatic patient, household members must wear a face mask, as well as gloves if they have contact with his or her bodily fluids. These should be thrown away immediately, never reused.

Elderly members of the household and those with chronic medical conditions risk severe complications, even death, if they become infected. Pregnant women may also be at particular risk, although the data aren't clear.

In China, 70 to 80 percent of transmission occurred within family clusters. Local governments there were forced to set up isolation wards with thousands of beds in gymnasiums and stadiums to care for people who lived alone or were at risk of infecting their families.

Family members should monitor their own health, and call a doctor if they develop a cough, fever or shortness of breath.

Need of the day

Govt, work insurance and mediclaim must pay for self-quarantine. Not everyone can work remotely. A two-week absence from work can take an enormous financial toll. [Source Newyork times]

Dr. Soumich Ranjan Sahoo
MBBS, DNB, Felloship in Otology

Dr. Pranab Kumar Sarma, MS (ENT),
Senior ENT Consultant
FCGP, P.Hony Secy, IMA,ASB,
Governing Council Member
IMA CGP H.Q.

DIABETES MELLITUS AND ENT DISEASES

INTRODUCTION

Among the most common chronic disorders of modern time, diabetes mellitus (DM) remains unique because of its multisystem ramifi cations.[1,2]Type 2 DM is a complex disease with both metabolic and vascular components that affects about 285 million worldwide in 2010 and will rise to 438 million at the end of 2030 with age group of 20-70 years.[3] Type 2 DM is associated with a number of microvascular complications affecting most commonly the eyes and kidneys, and histopathological studies have shown damages to the nerves and vessels of the inner ear of individuals with diabetes. [4,,5]Necrotizing (malignant) external otitis, an infection involving the temporal and adjacent bones, is a relatively rare but dangerous complication of external otitis.[6] Cutaneous infections, which may be bacterial, fungal or viral, are common among patients with diabetes,[7] and can affect the skin and soft-tissue structure of the ear and the nose as well. To the best of our knowledge, the prevalence of otorhinolaryngological disorders in patients with diabetes has not been properly documented .So we are doing a review of literature and trying out to find out the various ENT diseases prevalent in diabetics.

KEYWORDS
Diabetes Mellitus,Mucormycosis, sensorineural, otitis externa

LITERATURE REVIEW AND RESULTS

In the subsequent sections we are going to review various ent diseases seen in case of diabetic patients

RHINOLOGY

DM patients may be prone to Gram negative bacterial sinus infections,

Mucormycosis is an aggressive, frequently fatal invasive fungal infection that can develop in immunocompromised patients.[8,9] It is usually documented in patients with uncontrolled diabetes. Mucor is often recognized as a triad of symptoms, such as uncontrolled diabetes mellitus, periorbital infection and meningoenchephalitis [10]Early diagnosis is of prime importance in treating patients with mucormycosis. Correcting or controlling predisposing problems is also essential for improving the treatment outcome. In diabetic ketoacidotic patients, hyperglycemia and acidemia should be corrected[11]

OTOLOGY

Both hypo- and hyperglycemia have been associated with inner ear dysfunction and hearing may fl uctuate with blood-glucose levels.[12] The relationship between DM, sensorineural hearing loss and vestibular dysfunction has been known for sometimes and histopathological changes in the temporal bone have been clearly documented. [13]But, according to one study, there was a weak association between diabetes and hearing loss.[14] Taylor and Irwin noted that patients with diabetes as a whole were deafer particularly in the lower frequencies than the controls, gradually approaching each other in the middle range (1-4kHz) and were similar at 8 kHz.[15] Friedman and Schulman studied 20 diabetic patients with peripheral neuropathy.[16] They concluded that 55% of their subjects had symmetrical hearing loss of the sensorineural type, involving at least one frequency, although none gave a history of hearing loss and ear diseases. The hearing loss was unrelated to age, and the impairment was similar at low and high frequencies, with a maximum defi ciency between 750 and 2000Hz.19[16] A small group of patients with idiopathic sudden hearing loss was investigated to study the possibility of a relationship with diabetes but no correlation was found in the audiological pattern. However, patients with diabetes failed to recover as well in high-frequency loss compared to non diabetic patients.[17]

Invasive otitis externa affects predominantly those over fi fty years of age and most patients have evidence of micro vascular disease such as DM.[18] Poorly controlled diabetics have a greater susceptibility to bacterial and fungal infections of the skin, such as furunculosis and carbuncles.[19,20] In the preinsulin era, the prevalence of common pyodermas such as furunculosis, carbunculosis, and erysipelas was

much higher in patients with diabetes than for their non-diabetic counterparts.[20] The duration of diabetes is an important factor that determines the appearance of microvascular complications of diabetes. It seems that the longer duration of DM predisposes more to the development of deafness in many studies, however, mild degree of hearing impairment was detected in many children with diabetic duration of less than four years.[21] Such observation was unusual and might be explained by poor glycemic control. Elamin et al. have proven the relationship of hearing loss in children and adolescent with type 1 DM at middle and high frequencies, which was not present in their controls.[21].

ORAL CAVITY

SALIVARY

It is known that diabetes mellitus is associated with chronic complications such as neuropathy, micro-vascular abnormalities and endothelial dysfunction that lead to deterioration of microcirculation and this may play a role in reduction of the salivary flow rate and composition.[22,23]Sialosis is defined as asymptomatic, non-inflammatory, non-neoplastic, bilateral chronic diffuse swelling mainly affecting the parotid glands. Sialosis has been found to be more prevalent in patients with diabetes mellitus. [24]

FUNGAL INFECTIONS

The incidence of fungal infections in patients with diabetes mellitus has been recognised for many years. [25] Candidal infection is reported to be more prevalent in patients with diabetes especially in those patients who smoke, wear dentures, have poor glycaemic control and use steroids and broad spectrum antibiotics.[26] In addition, salivary dysfunction in patients with diabetes can also contribute to higher carriage of fungi in this group of patients. It is clear from these studies that both local and systemic predisposing factors might increase candidal carriage rate and hence increase the risk of oral candidal infection in patients with diabetes.[27,28,29]

BACTERIAL INFECTIONS

Patients with diabetes are more susceptible to developing oral bacterial infections.[30] They are well known to have an impaired defense mechanism hence considered to be immuno-compromised. Diabetics with diabetic complications and poor metabolic control are more prone to spreading and recurrent bacterial infection.

ORAL MUCOSAL DISEASE

Both lichen planus and recurrent apthous stomatitis have been reported to occur in

atients with diabetes. [31,32] Oral lichen planus OLP) is a skin disorder that produces lesions n the mouth. OLP is reported to occur more requently in patients with type1 diabetes ompared to type 2 diabetes.[31]The reason or this is that type 1 diabetes is considered an utoimmune disease, and OLP has been eported to have an underlying autoimmune nechanism. Patients with diabetes are subjected o a prolonged state of chronic immune suppression especially in type 1 diabetes. In ddition, acute hyperglycaemia causes alteration n the immune responsiveness in diabetes nellitus.

NEUROSENSORY ORAL DISORDER

Oral dysesthesia or burning mouth syndrome (BMS) is a painful condition affecting the oral cavity (palate, tongue, throat and gingivae).[33,34]Other abnormal oral sensations may co-exist with the burning mouth sensation such as tingling, numbness, dryness or sore mouth at the same time. The exact cause of BMS is unknown, but it has been attributed to several conditions such as dry mouth, menopause, candidal infection, diabetes mellitus, cancer therapy, psychological problems and acid reflux. BMS is classified into two types: primary idiopathic, and secondary as a result of a systemic process; secondary BMS has been reported to occur with diabetes mellitus. It could adversely affect the ability to maintain good oral hygiene in patients with diabetes. Diabetic neuropathy could be the underlying cause of BMS in patients with diabetes. The nerve damage in diabetic neuropathy has been reported to show an increase in the Langerhans cells that are associated with immune disturbance.[35,36]Therefore, it is crucial to screen patients who have symptoms of BMS for diabetes mellitus.

CONCLUSION

Diabetes mellitus is a disorder which has significant correlation with many ENT diseases affecting ear ,nose and the oral cavity.Proper glycemic control can effectively reduce the burden of the ENT diseases associated with diabetes mellitus.

REFERENCES

1. G azzaz, Zohair & Makhdom, M.N. & Dhafar, & Maimini, O. & Farooq, Mian & Rasheed, A.. (2011). Patterns of otorhinolaryngological disorders in subjects with diabetes. International Medical Journal Malaysia. 10. 13-16.

2. Amos AF, McCarty DJ, Zimmet P. The rising global burden of diabetes and its complications: estimates and projections to the year2010. Diabet Med 1997;14: S1-85.

3. International Diabetes Federation (Belgium).In: Diabetes facts and fi gures (online). Available from: http//www.idf.org/diabetesfacts-and-fi gures. Accessed Oct 28, 2010.

4. Dalton DS, Cruickhanks KJ, Klein R, KleinBEK, Wiley TL. Association of NID DM and hearing loss. Diabetes Care 1994;17:1158-63.

5. Jordao AMD. Consideration sur un cas dudiabete. Union Medicale Paris 1857; 11:446. [English translation]

6. Handzel O, Halperin D. Necrotizing (Malignant) External Otitis. Am Fam Physician 2003; 68: 309-312.

7. Schwartz B, Schuchat A, Oxtoby MJ. Invasivegroup B streptococcal disease in adults. Apopulation based study in metropolitanAtlanta. JAMA 1991; 266:1112-14.

8. Vijayabala GS, Annigeri RG, Sudarshan R. Mucormycosis in a diabetic ketoacidosis patient. *Asian Pac J Trop Biomed.* 2013;3(10):830–833. doi:10.1016/S2221-1691(13)60164-1

9. Fogarty C, Regennitter F, Viozzi CF. Invasive fungal infection of the maxilla following dental extractions in a patient with chronic obstructive pulmonary disease. J Can Dent Assoc. 2006;72(2):149–152. [PubMed] [Google Scholar]

10. Breiman A, Sadawsky D, Friedman J. Mucormycosis discussion and report of a case involving the maxillary sinus. Oral Surg Oral Med Oral Pathol. 1981; 52:375–378. [PubMed] [Google Scholar]

11. Mathebula SD. Mucormycosis. S Afr Optom. 2008;67(1):36–41. [Google Scholar]

12. Rudd MJ, Harrie ML, Lynch CA, Moffat DA.Hearing loss fl uctuating with blood sugar levels in meniere's desease. J Laryngol Otol1993; 107:620-622.

13. Moffat DA, Booth JB, Morrison AW. Metabolicinvestigations in meniere's disease. J Laryngol Otol 1981; 95:905-913.

14. Al-Nozha MM, Al-Maatouq MA, Al-Mazrou YY,et al. Diabetes mellitus in Saudi Arabia. Saudi Med J 2004; 25(11):1603-10.

15. Taylor IG, Irwin J. Some audiological aspectsof diabetes mellitus. J Laryngol Otol 1978;92: 99-113.

16. Friedman SA, Schulman RH. Hearing anddiabetic nephropathy. Arch Inter Med 1975;135:573-576.

17. Wilson WR, Laird N, Soeldner JS, Moo-youngG, Kaveshi DA, Macmeel JW. The

relationshipof idiopathic sudden hearing loss to diabetesmellitus. Laryngoscope 1982; 92:155-160.

18. Necrotising otitis externa. Lancet 1982;319:207. doi:10.1016/S0140-6736 (82)90769-3.

19. Sreedevi C, Car N, Pavlic-Renar I. Dermatologic lesions in diabetes mellitus. Diabetol Croat2002; 3:147-159.

20. Huntley A. Diabetes Mellitus Review. Dermatology (Online) 1995:1. Available at: http/ /dermatology.cdlib.org/DOJvol1num2/ diabetes/dmreview.html. Accessed October 30, 2010.

21. Elamin A, Fadlallah M, Tuvemo T. Hearing lossin children with type 1 diabetes. Indianpediatrics 2005; 42:15-21.

22. Conner S, Iranfour B, Mills J. Alteration in parotid salivary flow in diabetes mellitus. Oral Surg Oral Med Oral Pathol. 1970;30:55–9. [PubMed] [Google Scholar]

23. Chomkhakhai U, Thanakun S, Khovidhunkit S-P, Khovidhunkit W, Thaweboon S. Oral health in Thai patients with metabolic syndrome. Diabetes Metab Syndr. 2009;3:192–7. [Google Scholar]

24. Scully C, Bagán JV, Eveson JW, Barnard N, Turner FM. Sialosis: 35 cases of persistent parotid swelling from two countries. Br J Oral Maxillofac Surg. 2008;46:468–72. [PubMed] [Google Scholar]

25. Lamey PJ, Darwaza A, Fisher BM, Samaranayake LP, MacFarlane TW, Frier BM. Secretor status, candidal carriage and candidal infection in patients with diabetes mellitus. J Oral Pathol. 1988;17:354–7. [PubMed] [Google Scholar]

26. Willis AM, Coulter WA, Fulton CR, Hayes RJ, Bell PM, Lamey PJ. Oral candidal carriage and infection in insulin treated diabetic patients. Diabet Med. 1999;16:675–9. [PubMed] [Google Scholar]

27. Hill LV, Tan MH, Pereira LH, Embil JA. Association of oral candidiasis with diabetic control. J Clin Pathol. 1989;42:502–5. [PMC free article] [PubMed] [Google Scholar]

28. Khosravi AR, Yarahmadi S, Baiat M, Shokri H, Pourkabireh M. Factors affecting the prevalence of yeasts in the oral cavity of patients with diabetes mellitus. J Mycol Med. 2008;18:83–8. [Google Scholar]

29. Soysa NS, Samaranayake LP, Ellepola NB. Diabetes mellitus as a contributory factor in oral candidosis. Diabet Med. 2006;23:455–9. [PubMed] [Google Scholar]

30. Al-Maskari AY, Al-Maskari MY, Al-Sudairy S. Oral Manifestations and Complications of Diabetes Mellitus: A review. *Sultan Qaboos Univ Med J.* 2011;11(2):179–186.

31. Amerikanou CP, Markopoulos AK, Belazi M, Karamitsos D, Papanayotou P. Prevalence of oral lichen planus in diabetes mellitus according to the type of diabetes. Oral Dis. 1998;4:37–40. [PubMed] [Google Scholar]

32. Torrente-Castells E, Figueiredo R, Berini-Aytés L, Gay-Escoda C. Clinical features of oral lichen planus - A retrospective study of 65 cases. Med Oral Patol Oral Cir Bucal. 2010;15:685–90. [PubMed] [Google Scholar]

33. ADA Division of Communications Burning mouth syndrome. J Am Dent Assoc. 2005;136:1191. [PubMed] [Google Scholar]

34. Scala A, Checchi L, Montevecchi M, Marini I, Giamberardino MA. Update on burning mouth syndrome: Overview and patient management. Crit Rev Oral Biol Med. 2003;14:275–91. [PubMed] [Google Scholar]

35. Moore PA, Guggenheimer J, Orchard T. Burning mouth syndrome and peripheral neuropathy in patients with type 1 diabetes mellitus. J Diabetes Complications. 2007;21:397–402. [PubMed] [Google Scholar]

36. Tavakoli M, Boulton AJ, Efron N, Malik RA. Increased Langerhans cell density and corneal nerve damage in diabetic patients: Role of immune mechanisms in human diabetic neuropathy. Cont Lens Anterior Eye. 2010. [Epub 2010 Sep 16] [PMC free article] [PubMed]

Dr. Atul Chandra Saikia MBBS, MD, FICP
Retd. Joint Director of Health Services
Past President, API Assam
Tezpur, Assam

MANAGEMENT OF HYPERTENSION IN DIABETES MELLITUS

Introduction :-

The incidence of Diabetes mellitus is increasing gradually in our country. According to recent data, 62.4 million people have diabetes in India (1) and the number may increase to 101.2 million by the year 2030 (2). According to World Health Organization, " India would very soon become the Diabetes Capital of the world". On the other hand Hypertension (HT) is another major health problem in India and the incidence of HT is gradually increasing due to lack of control and awareness. According to recent data more than 207 million Indians (Men 112 million and Women 95 million) have hypertension (3). The prevalence of incidence is more in urban (24-30%) than in rural (12-14%) people in India (4).

Hypertension (HT) is more common in both Type I and Type II than that of common people in the society. On the other hand, both Type I and Type diabetic patients are more prone to develop HT during their course of diseases. In Type II diabetes, the incidence of HT is about40-60% (5) and in Type I diabetes it is about 14.71% (6). Diabetes mellitus is a metabolic disease with high incidence of mortality and morbidity due to macro vascular and micro vascular complications. On the other hand HT is the main cause of coronary artery disease, heart failure, stroke and kidney diseases. When HT develops in diabetes, the morbidity and mortality increases by more than seven fold (7). According to HDS-II (8) study, HT patients had a greater incidence of death from diabetes related events and develops major morbidity like myocardial infarction, angina, strokes and amputation of limbs (9).

In India also 50% of diabetic individuals are hypertensive . The Screening India's Twin Epidemic (SITE) cross sectional study conducted in 10 Indian states (10) reported that 20.6% of patients are suffering from both diabetes and hypertension. So the burden of hypertension in diabetes is increasing gradually in India.

Pathophysiology –

Diabetes itself is a risk factor for development of hypertension (11). Similarly the

hypertensive patients have the tendency of developing diabetes in due course. Central obesity, insulin resistance and hyperinsulinemia, dyslipidemia and ventricular hypertrophy are very common in Type II diabetes and all these lead to the development of hypertension.

* Central obesity, insulin resistance and hyperinsulinemia affect the nitric oxide path way, smooth muscle cells, sympathetic activity and sodium fluid level. All these lead to the development of hypertension.

* Hyperglycemias sometimes directly suppresses the release of nitric oxide leading to the development on hypertension (12). It increases vascular wall stiffness impairing endothelial-dependent vasodilatation.

* In type I diabetes, hypertension at an early stage with the onset of nephropathy. In type II diabetes, age related hypertension and obesity are very common but the incidence of hypertension is about 90% or more if associated with impaired renal function and diabetes.

* Some other pathological conditions like hyperthyroidism, hypothyroidism, Cushing's syndrome, acromegaly, pheochromocytoma in diabetic patients, the incidence of hypertension is very common.

Pathological changes due to hypertension in Diabetes :-

Most of the time, the hypertension in diabetic patients are of Resistant type of hypertension. In some cases patients develop isolated systolic hypertension due to autonomic neuropathy and subsequently develop atherosclerotic changes.

Left ventricular hypertrophy is very common in this group and sometimes it may be the independent risk factor for sudden death. Stroke results from thrombosis, thromboembolism or haemorrhages are very common in this group.

The development of CVD and strokes in this group are 5-7 times higher than that of normotensive and non-diabetic group (13). Arrhythmia occurring during hypoglycaemia may be the main cause of CVD death.

In case of pregnancy with diabetes, the chances development of Eclampsia and foetal abnormality is very high if associated with hypertension.

Management of Hypertension in Diabetes :-

a. General guidelines :-

* Blood pressure should be monitored from the first visit at the consultation room and should be recorded by the physician himself with the certified BP instruments.

* Home monitoring of Blood pressure should be encouraged to each and every patient.

* Targeted goal should be as per guidelines – according to American Diabetes Association, recommended Blood pressure should be less than or equal to 130/80 mm of Hg for hypertensive diabetic patients. JNC 7 also recommended the targeted BP in this group as less than or equal to 130/80 mm of Hg (14). Persistent blood pressure more than 140/90 in diabetes need early treatment with any agents.

b. Life style modification :- If blood pressure is more than or equal to 130/80 in two successive occasion than we can advise life style modification without starting any medication. Life style modifications are -

* Simple physical activities like walking 30-45 minutes per day 5 days in a week, jogging, cycling and swimming improve insulin resistance, reduces weight and thus improve control of blood pressure.

* Complete cessation of smoking cigarette, tobacco and restriction of alcohol is always advisable.

* Common salt should be restricted to less than 5 gm per day.

* Daily diet of low sodium, low calorie, high potassium and fibre should be recommended.

* Lastly adequate and proper sleep is an important factor in the management of hypertension in diabetes. No lifestyle change would be completed without enough sleep.

c. Drugs treatment :-

*ACE inhibitors :- ACE inhibitors are the first line of drugs in the treatment of HT in diabetes with or without microalbuminuria. According to HOPE study, they had lower risk of cardiovascular morbidity and mortality. Common drugs are Ramipril, Captopril, Enalepril and Lisinopril. In case if intolerability to ACE inhibitors, we can start with ARBs.

* Diuretics :- If hypertension still persists, we can add diuretics along with ACE inhibitors. They lower BP, improve heart failure, reduce risk of stroke and cardiovascular mortility. The commonly used

diuretics are Thiazide diuretics, Loop diuretics and potassium Sparing one.

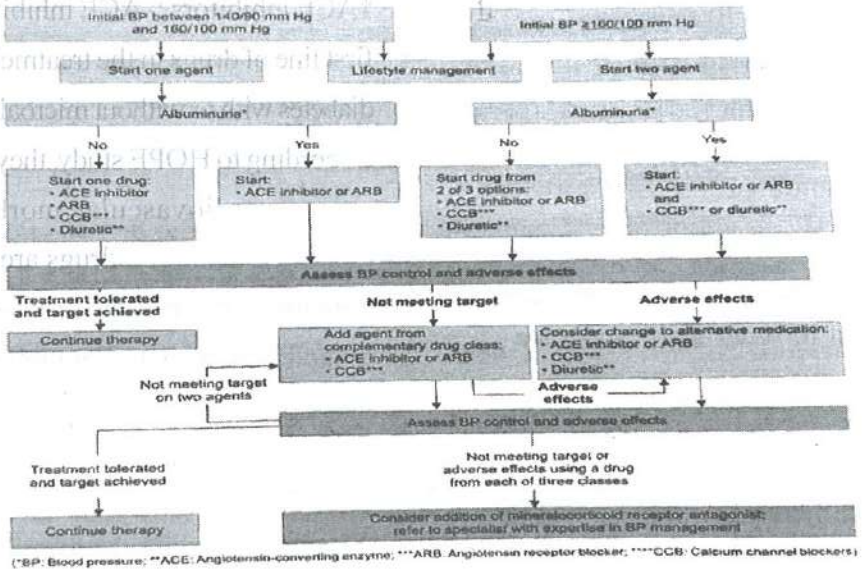
* Angiotensin Receptor Blockers :- Like ACE inhibitors, ARBs may be used as first line of drug in Type II diabetes with hypertension with or without microalbuminuria. Commonly used ARBs are Losartan, Valsartan, Telmisartan and Olmisartan. ARBs are usually recommended when ACE inhibitors are not tolerated by the patients .

*Beta blocker :- Beta blockers can be used along with other agents to prevent heart failure and coronary artery disease. To avoid the masking hypoglycaemic effect, it is better to use cardio selective beta blockers. Metoprolol and Bisoprolol are the best drug to use in diabetes with hypertension.

* Calcium channel blockers are used as a second line of drugs in the management of hypertension in diabetes. Commonly used are Nifedipine, Amlodipine and Cilnidipine.

Other agents like Alfa blockers, Loop diuretics, Centrally acting drugs are of less important to control blood pressure i diabetes.

The flowchart of the American Diabetes Association 2018 recomendation are given herewith for the treatment of hypertension in diabetic patients for ready references.



(*BP: Blood pressure; **ACE: Angiotensin-converting enzyme; ***ARB: Angiotensin receptor blocker; ****CCB: Calcium channel blockers)

Conclusion :- Diabetes affects almost every system of the body particularly Eyes, Kidneys, Heart , Nerves and Feet with micro vascular and macro vascular complications. On the other hand, hypertension is also associated with high incidence of stroke related death and coronary heart diseases. In most cases both the illnesses are frequently co-existing morbid illness. So proper and adequate blood pressure control as per guide line is most essential with single or combination therapy along with strict glycaemic control to prevent the CVD and other complications of Hypertension and Diabetes.

References :-

1. Anjana RM, Pradeepa R, Deepa M et al. On behalf of the ICMR-INDIAB Collaborative Study Group. Prevalence of Diabetes and Prediabetes in urban and rural India : 2011:54:3022-7

2. Unwin N, Whiting D et al. IDF Diabetes Atlas, 5th Edition, International Diabetes federation; 2011 pp11:74.

3. Gupta R, Gaur K, Venkata C et al. Emerging trends in Hypertension epidemiology in India. J Hum Hypertension, 2018:33;575-87.

4. Sawase GB, Kumthekar SG, SalphaleSN, et al. A study of prevalence of hypertension and socio- demographic factors in urban slum, maharastra. Int. J. community Med Public Health 2019;2

5. Sowers JR, Epstein M, Frohlich ED. Diabetes, hypertension and Cardiovascular Disease: an update: Hypertension 2001:37:1053-9.

6. Norgaard K et al. Diabetology 1990.

7. KDOQI. KDOQIclinical practice guidelines and clinival practice recommendations for diabetes and chronic kidney disease. Am J Kidney Dis, 2007; 47:S12-154.

8. Hypertension in Diabetes Study(HDS) I Prevalence of hypertension in Newly presenting Type II diabetic patientsand the association with risk factors for cardiovascular and diabetic complications. J Hypertension1993:11;:309-17.

9. Hypertension in Diabetes Study (HDS)II. Increased risk of Cardiovascular complicationsin Hypertensive Type II Diabetic patients. J Hypert . 1993;11:309-17.

10. Joshi SR, Saboo B, VadivaleM et al. prevalence of diagnosed and undiagnosed Diabetes and Hypertension in India- result from the screening India's twin epidemic (SITE) Study Diabetes Technol Ther. 2012;14:8-15.

11. National High Blood Pressure Education Programmee working group report on hypertension in diabetes. Hypertension;1994;23:145-58.

12. Major SG. Blood Pressure in Diabetes Mellitus: a statistical study. Arch Intern Med. 1989;44:797-812.

13. KK Pareek, G Mathur, GD Ramchandani. Hypertension and Diabetes. API medicine UPDATE 2020;225-2

14. Chobanian AV, Bakris GL, BlackHR et al. The Seventh Report of the Joint National Committee on the Prevention, Detection, Evaluation and treatment of High Blood Pressure; The JNC 7 report. JAMA. 2003;289:2560-72.

Dr. Laksheswar Bhuyan
MS (Surgery)
Tezpur

SURGERY AND DIABETES MELLITUS

Diabetes mellitus is one of the more common co-morbidity associated with surgical patient which needs to be well assessed and well controlled before surgery as far as practicable. Particularly in elective surgical procedure hyperglycemic control is must to avoid peroperative and postoperative complications due to high blood glucose. Good hyperglycemic control before surgery creates a comfortable situation to the patient as well as to the treating team of doctors. The situations in an emergency surgical patient may be of slightly different as complete hyperglycemic control may not be possible before putting the knife. Strategic management of hyperglycemia may be needed as the situation arises.

Diabetes is associated with increased requirement for surgical procedures and increased postoperative morbidity and mortality. The stress response to surgery and the resultant hyperglycemia, osmotic dieresis and hypoinsulinemia can lead to peroperative keto acidosis or hyper osmolar syndrome. Hyperglycemia impairs leucocyte function and wound healing. The management goal is to optimize metabolic control through close monitoring, adequate fluid and caloric repletion and judicious use of insulin.

The stress of surgery itself results in metabolic disturbation that alters the glucose homeostasis and persistent hyperglycemia may cause endothelial dysfunction, postoperative sepsis, impaired wound healing and cerebral ischemia. The stress response itself may precipitate diabetic crises (Diabetic Ketoacidosis), hyperglycemic hyperosmolar syndrome during surgery or post operatively with negative prognostic value. Gastrointestinal instability provoked by anesthesia, medications and stress related vagal over activity can lead to nausea, vomiting and dehydration. Thereby increasing the risk for ishemic events and acute renal failure. The person with uncontrolled diabetes who are posted for elective surgery should preferably be scheduled after acceptable glycemic control has been achieved after treating as indoor patient for 1-2 days in hospital.

The actual treatment recommendations for a given patient should be individualized based on diabetic classification, usual diabetic regimen, state of glycemic control, nature and extent of surgical procedure and available expertise. Some general rules to be adhere while treating a patient for surgery with diabetes mellitus. The best rule to take up for surgery as soon as condition is optimum without further delay.

****

Dr. Jagadish Basumatary MD
Department of Anaesthesiology & Critical Care Tezpur Medical College, Tezpur, Assam

PRE ANAESTHETIC ASSESSMENT OF DIABETIC PATIENTS POSTED FOR SURGERY

The prevalence of diabetes mellitus in both adults abd children has been steadily rising through out the world for past 20 to 30 years. Inevitably, diabetic patients presenting for incidental surgery or surgery related to their disease, will place an increasing burden on anaesthetic services.

Better glycaemic control in diabetic patients undergoing major surgery has been shown to improve peri operative morbidity and mortality.

PRE OPERATIVE ASSESSMENT IS THE KEY TO SUCCESS OF SURGERY AND ANAESTHESIA IN A DIABETIC PATIENT

Diabetes is a metabolic disorder resulting from an (absolute or relative) deficiency or resistance to insulin. 50% of diabetes patients present for surgery in their life time.

Classification

Type I
Absolute insulin deficiency secondary to immune mediated or idiopathetic.

Type II Adult onset secondary to resistance/ relative deficiency

Type III Specific types of diabetes mellitus secondary to genetic defects

Type IV Gestational

The stress of surgery/ Anaesthesia results in metabolic disturbance that alter glucose homeostasis, persistent hyperglycaemia resulting in:

- Depressed immunity
- Impaired wound healing
- Endothelial dysfunction like IHD, CVA
- Diabetes crises

PRE OPERATIVE ASSESSMENT

The main Objectives are:

- To establish the indication for the surgery and extent of the diabetes
- To determine the presence and chronicity of the diabetes.
- To find out detailed medical follow up and control. SYSTEMIC REVIEW

1. Autonomic Neuropathy

- Presence of postural hypotension
- Gastroparasis
- Gustatory sweating
- Nocturnal diarrhoea

Heart rate variability with deep breath:

- Normal > 15 bpm
- Neuropathy is likely if < 10 bpm

RESPIRATORY SYSTEM

Diabetics are more prone to respiratory infections and may also have abnormal spirometry.

CARDIOVASCULAR SYSTEM

Diabetics are more prone to develop

- Ischaemic heart dieses (MI)
- Hypertension
- Peripheral vascular disease
- CVA
- Cardiomyopathy

GASTRO INTESTINAL SYSTEM

Gastroparasis is a common complication characterised by delay in gastric emptying without any gastric outlet obstruction. Increased gastric contents increase the risk of aspiration.

AIR WAY

Glycosylation of Collagen in the cervical and temporo-mandibular joint can cause difficult intubation.

KIDNEY

Diabetes is one of the common causes of End Stage Renal Failure. IMMUNE SYSTEM

Diabetes is prone to all types of infection. Indeed an infection might actually worsen diabetic control.

INVESTIGATION

1. RBS, FBS, 2Hrs PG

WHO Guidelines	Glucose concentration (mmol/ l)	
	Plasma Venous	Whole Blood Venous
Diabetes Mellitus		
Fasting	e" 7.0	e" 6.1
2 Hrs. Post load/ Random	e" 11.1	e" 10.0

1. Glycated Hb (Hb A1c) < 7% Good control, > 9% Poor control

2. Urine analysis. Ketones (poor control), Protein (possible renal complications) and bacteriology.

3. LFT, RFT, Lipid profile

4. ECG

5. CXR

GENERAL PRINCIPLES FOR PRE OPERATIVE PREPARATION

- 1- Preoperative management of DM patient is MULTIDISIPLINARY
- 2- Diabetes should be well controlled prior to elective surgery
- 3- Avoid hypoglycaemia (under 4mmol/l)
- 4- Avoid severe hyperglycaemia (over 14mmol/l)
- 5- Type 1 diabetics need insulin to prevent ketogenesis and metabolic derangement
- 6- Aim for a blood glucose between 6 and 10 mmol/l
- 7- Accurate and close glucose monitoring MUST BE ENSURED
- 8- Diabetic patients should be placed first on the operating list
- 9- Patients must be given clear written instructions concerning the management of their diabetes both pre- and post-operatively

PERIOPERATIVE MANAGEMENT:

Should be individualized:

Type of DM

- Pre operative Treatment
- Metabolic status
- Presence of complication: Cardiac, Renal, Autonomic Surgery:
- Type: Emergency or Elective Minor or major procedure
- Type of Anaesthesia GA or Regional PRE OPERATIVE OPTIMIZATION
- Ensure good hydration
- Correct electrolyte abnormality
- Stop long acting OHG (eg. Chlorpropamide) 48- 72 hrs before surgery
- Stop long acting insulin a day before surgery
- Convert to soluble insulin
- Check blood glucose early in the morning of surgery
- Give Premedicaion
- Fast patient overnight
- Commence glucose/ potassium/ insulin(GKI) infusion
- Grouping & Cross matching blood accordingly
- Obtain intra- operative antibiotics
- Obtain informed consent
- Catheterize patient going for major surgery SPECIFIC PRE OPERATIVE TREATMENT

Patient on Dietary Control

Elective	Emergency
• Treat as non diabetic	* Check BG before surgery
• Check FBG before surgery	* 1-2 hourly BG till return to oral intake
• Intra- operative BG 2 hourly	* Insulin may be required depending on BG level
• Return to usual diet as	* Return to usual diet before discharge soon as possible

ORAL HYPOGLYCAEMIC CONTROLLED

Elective	Emergency
• Admit 2 days before surgery	* Commence insulin infusion on admission
• Stabilize on soluble insulin	* Continue as elective
• Omit insulin on day of operation	
• Insulin infusion, to continue till oral Intake resumes	
• 1-2 hourly BG	
• Change to usual OHG before discharge	
• If minor surgery monitor BG	

PATIENT ON INSULIN

Elective	Emergency
• Admit 48 hrs before surgery	* Commence insulin infusion on admission
• Change long acting/ intermediate acting	* Continue as elective to soluble insulin
• Omit morning dose	
• Insulin infusion, to continue till oral intake resumes	

- 1-2 hourly Blood glucose
- Change to usual Insulin before Discharge

Diabetic Crisis

- Patient with DKA or HHS usually have gross volume deficite, electrolyte derangement and acid base imbalance.
- Active resuscitation is must be done b4 surgery
- GKI or sliding scale should be commenced immediately
- 1 hourly Blood Glucose Insulin Infusion Regimens

1- No Glucose No Insulin Regime

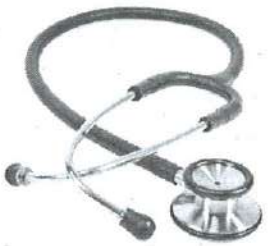
2- Glucose 5g/h and Insulin 1 IU/h via infusion pumps

3-Alberti regime:

- 500 ml of 10% dextrose+10 iu of soluble Insulin + 10mmol of KCI to run at 125ml/hr
- 500 ml of 5% dextrose+5 IU of soluble Insulin + 5mmol of KcI

4- Sliding scale

Plasma glucose (mmol/L)	* Insulin infusion rate (iu/hr)
<4.0	* No insulin
4.1-7.0	* 1
7.1- 9.0	* 1.5
9.1- 11.0	* 2
11.1- 17.0	* 3
17.1- 28.0	*4
>28.0	* 6



Courtesy
Dr. Nareswar Dutta
Sr. Consultant (Opthalmology)
Past State President
IMA, ASB

Dr. Nilutpal Borah, M.S.
Guwahati Eye Institute,
Aksi Advance Eye Center
Guwahati
in vitrio Ratinal Surgeon

Diabetic Retinopathy and Management-A Review

Diabetic retinopathy is a highly specific vascular complication of both type I and type II diabetes mellitus. All over the world among working-aged individuals, diabetic retinopathy is a leading cause of blindness. About 30% of people with diabetes mellitus have diabetic retinopathy and highestincidence is expected to be in India, China and USA. The prevalence of vision-threatening diabetic retinopathy is approximately 5%.

India is considered as the Diabetes capital of the world where the number of people with diabetes is expected to rise to 69.9 million by 2025 unless urgent preventive measures are taken.

Indian people carry certain unique clinical, biochemical abnormalities including increased insulin resistance, greater abdominal adiposity, lower BMI, lower adiponectin and higher highly sensitive C reactive protein levels.This 'Asian Indian phenotype' makes Asian Indians more prone to diabetes and premature coronary artery diseases. Also changes in dietary patterns, less physical activity are primary cause of increased incidence.In Indians, prevalence of micro vascular complications of diabetes like retinopathy and nephropathy are comparatively lower but the prevalence of premature coronary disease is much higher compared to other ethnic groups.

Mechanism of onset and progression of diabetic retinopathy still fully not understood. High glucose-driven metabolic dysfunction and the induction of chronic, low-grade inflammatory signalling in the retina play an important role as low level systemic and local inflammation have been detected in patients with diabetic retinopathy.There is thickening of vascular basement membranes, up-regulation of membrane components and/or reduced breakdown by proteolytic enzymes and loss of endothelial pericytes. Capillary microaneurysms are normally the first clinically recognisable feature of diabetic retinopathy. Other major features of diabetic retinopathy are capillary occlusion, retinal ischaemia, intraretinal and extraretinal(vitreous) haemorrhage

Types of diabetic retinopathy are non-proliferetive diabetic retinopathy(NPDR) and proliferative diabetic retinopathy(PDR). NPDR is the milder form of the disease and is usually symptomless. Diabetic macular edema may coexist with both non-proliferative and proliferative diabetic retinopathy.

Initially symptomless,vision loss occur in diabetic retinopathy due to diabetic macular edema and vitreous hemorrhage(in proliferative diabetic retinopathy) causing sudden and severe vision loss.Advanced stages of diabetic retinopathy i.e. proliferative diabetic retinopathy is characterized by the development of retinal neovascularization.Neovascularization develop at the junction of perfused and non-perfused retina and grow abnormally out of the retina into the clear vitreous gel. They are very prone to bleeding any jerking motion or even a rise in the blood pressure can lead to a rupture and cause hemorrhage. Other features are fibrovascularepiretinal membranes, and complications including vitreous hemorrhage,tractional retinal detachment and occasionally combined retinal detachment.

Direct and indirect ophthalmoscopy,fundus fluorescein angiography(FFA) and optical coherence tomography(OCT) are few tests which can detect and classify diabetic retinopathy.

Variousmodalities are available for the treatment of vision threatening diabetic retinopathy. These include laser photocoagulation, anti VEGF,steroids and vitrectomy.Management of diabetic retinopathy revolves around laser photocoagulation, intra-vitreal anti-vascular endothelial growth factor(VEGF) agents(e.g. bevacizumab, ranibizumab, aflibercept, brolucizumab) and intravitrealsteroids(depotdexamethasone sodium) and vitrectomy surgery for vitreous haemorrhage, traction and/or combined retinal detachment and premacularvitreoretinal traction.

It is established that duration of diabetes, poor glycaemic control, and hypertension are the primary factors accounting for the risk of developing diabetic retinopathy.

In addition to recognized risk factors, multiple new potential risk factors for diabetic retinopathy have been identified. Several key studies have identified risk factors including blood glucose (DCCT, diabetes control and complications trial; UKPDS(UK prospective diabetes study), Blood pressure(UKPDS), Duration of diabetes (DCCT), Lipid (ACCORD, action to control cardiovascular risk in diabetes trial), Pregnancy (DCCT), Nephropathy (UKPDS, WESDR, Wisconsin epidemiologic study of diabetic retinopathy),

Obesity(WESDR, SiMES, Singapore Malay eye study), Genetics (GOLDR, genetics of Latino diabetic retinopathy study, TUDR, Taiwan-US diabetic retinopathy study) and Nutrition (Japan Diabetes Complications Study Group).

Many studies have reported an association between high BMI and obesity with diabetic retinopathy. But some other studies have reported a possible protective role for higher BMI in the development of diabetic retinopathy.

A study done in Japanese type 2 diabetic patients showed that increased fruit intake was associated with reduced incident diabetic retinopathy among patients with a low-fat energy-restricted diet. It is suggested that the preventive effects of fruits are mediated through glycaemic control as fruits can slow glucose response after ingestion.

Systemic approaches to treatment are aimed at preventing diabetic retinopathy, delaying the onset of retinopathy, reversing retinopathy and delaying the progression of early to advanced stages of retinopathy.

Early identification and appropriate intervention help in preventing the onset of diabetic retinopathy or delaying the progression of diabetic retinopathy thereby preserving good vision. A multifactorial approach, targeting intensive control of hyperglycaemia, strict control of blood pressure, Control of dyslipidaemia, maybe with fenofibrates and blockade of renin angiotensin system have all shown some benefit in the occurrence or progression of diabetic retinopathy.

Patients with diabetic retinopathy need lifelong attention with multiple treatment approaches. Lifestyle habits and an assessment of blood glucose control and overall medical fitness should be addressed. Importance of controlling systemic risk factors to achieve a maximal response to any intervention need to be stressed.

References:

1.Cheung N, Mitchell P, Wong TY. Diabetic retinopathy. Lancet. 2010;376:124–136.

2.Yau JW, Rogers SL, Kawasaki R, Lamoureux EL, et al. Meta-Analysis for Eye Disease (META-EYE) Study Group. META-EYE) Study Group. Global prevalence and major risk factors of diabetic retinopathy. Diabetes Care. 2012;35:556–564.

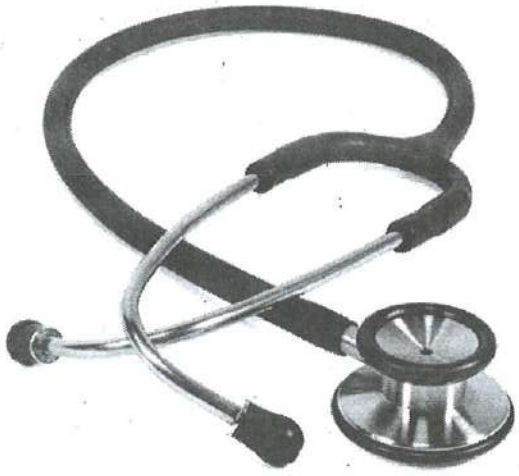
3.Gopal Lingam and Tien Yin Wong: Systemic Medical Management of Diabetic Retinopathy;Middle East Afr J Ophthalmol. 2013 Oct-Dec; 20(4): 301–308.

4.Hovind P, Tarnow L, Rossing K et al. Decreasing incidence of severe diabetic microangiopathy in type 1 diabetes. Diabetes Care. 2003;26:1258–1264.

5. Yau JW et al: Meta-Analysis for Eye Disease. Global prevalence and major risk factors of diabetic retinopathy. Diabetes Care. 2012;35:556–564

6. Park CY et al. Prevalence of and risk factors for diabetic retinopathy in Koreans with type II diabetes: baseline characteristics of Seoul Metropolitan City-Diabetes Prevention Program participants. Br J Ophthalmol. 2012;96:151–155

XXX



DR. S. P. BORDOLOI, MS
ENT SPECIALIST
SONITPUR, TEZPUR

PREVALENCE OF DIABETES IN ENT DISEASES

A SHORT DISCUSSION

Amongst the various non-communicable diseases diabetes appears to hold a major share to figure in global health hazards. India has the highest number of diabetes cases in the world with 72 million reported in 2017. Again, the prevalence of the disease is rapidly increasing in India at a much higher rate than global average and closely linked to various other non-communicable diseases like cardiovascular diseases (CVDs), hypertension, heart attack, stroke, kidney diseases, pulmonary diseases, various arthritis, connective tissue diseases, nerve related diseases etc. In order to epitomize this subject I will restrict this article to a discussion of some ENT (Ear, Nose and Throat) related commonly encountered problems in diabetic people.

In ENT diseases, increased infections are the most frequent problems. An uncontrolled diabetic person is prone to various infections because his defense mechanisms are not geared towards the same level of efficiency as a person without diabetes and hence a large number of complications can occur in a poorly controlled longstanding condition of diabetes.

The most common infections, in general, are the **upper respiratory infections**. These are the infections of the throat, para nasal sinuses and bronchi which are usually viral, but rarely bacterial as well. These infections tend to be recurrent in diabetic persons and also require longer time for the patients to recover. These infections are characterized by running nose or sore throat and fever, the symptoms may be worsened in uncontrolled diabetes.

People with poorly controlled diabetes are at greater risk for **mouth cavity** and **dental problems**. Damage to the gums, erosion or carries of the teeth, delayed healing of gums after teeth extraction, damaged to the teeth at the bases caused by increased acidity of saliva, toothache, spongy bleeding gums, staining of teeth by deposition of lime salts – these are the conditions usually seen in uncontrolled diabetic persons.

Some lesions are usually observed over **tongue** are **aphthous stomatitis** or recurrent canker sores, thrush(fungal infection with white areas), large red tongue sometimes covered with a black fur along the centre, redness of the mucous membrane of the mouth

which looks glazed and feels dry. The throat becomes persistently dry with secretions being thick and the throat looking red and shiny. There may be sweet sickly odour of the breath due to presence of acetones. The patient may complain of a sweet taste in the month.

As per reports of European archives of oto-rhino-laryngology 2010, some gustatory and olfactory dysfunctions like **loss of taste and smell sensation** have been noted only in complicated diabetes mellitus, but its clinical significance is not clear. **Loss of smell sensation** is due to damage of the olfactory nerve carrying smell sensation.

Mucormycoses is an another serious threat in diabetic people and is caused by fungus which affects the sinuses. It usually starts in the nasal sinuses, where it affects the nasal cartilage which gradually turns dark and is destroyed, at time causing holes or perforations in the nasal cartilage. It also spreads to the palatal area where it can deform the bone and creates holes. This infection is diagnosed by a quick biopsy of the nasal cartilage. The treatment is conservative as well as surgery to remove the dead bone and cartilage to eradicate the infection. After the infection is cured, some reconstructive surgery may be needed to restore the bones and cartilage.

Ear infections, mainly **Acute Middle Ear infections** are more frequent in people with diabetes. These occur when a throat or sinus infection spreads into the middle ear through a tube, called Eustachian tube. These infections cause severe pain in the ear with fever especially in children and if not treated promptly, result in a perforated ear drum. This can be followed by a chronic discharging ear, especially in cases of in-adequate treatment.

In case of diabetes with ear diseases the major burden of **Chronic Suppurative Otitis Media (CSOM)** is the non-healing ear discharge which may last for few months to many years or even a lifetime with increasing risk of complications in untreated cases where diabetes becomes a major risk factor to enhance the complications like auditory nerve damage hearing loss, mastoiditis with abscess, dreaded intracranial complications etc.

Malignant Otitis Externa or Necrotising Otitis Externa

Though rare, it is a very severe bacterial infection of outer ear occurring specifically in a diabetic person older than 35 years and is almost always due to psendomonas aeruginosa and Staphyllococcus aureus. The infection starts in the external auditory canal and sometimes spreads to the adjacent soft tissues, cartilage and boes including the bones of the jaw and face specially in uncontrolled diabetes. The term **malignant** is used in such cases to mark its severity, as once acquired it is quite

disabling and life threatuing. With a trivial injury or even without a recognizable injury the external ear get acutely inflamed with very high fever and the disease process rapidly progresses like fire causing difficult to treat specially in uncontrolled diabetes. Over 90 percent of people who develop malignant otitis externa have diabetes.

DIABETES AND HEARING LOSS

Hearing loss has been found to be twice as common in people with diabetes in comparison to those without diabetes. What causes or contributes to hearing loss in people with diabetes is not clear, but it has been determined that hearing loss in diabetes is causes by neuropathy (nerve damage), which is a common complication of both type I and type II diabetes. It is also said that prolong high blood glucose levels may lead to hearing loss by affecting the supply of blood or oxygen to the tiny nerves and blood vessels of the inner ear. Over time, the nerves and blood vessels become damaged, affecting the person's ability to hear causing sensori neural hearing loss due to pathological changes in the cochlea and auditory nerve.

Hearing loss can also be a maternally inherited problem for some people with diabetes. In fact, 1% of all diabetes patients are diagnosed with a sub-type of disease known as – **Maternally Inherited Diabetes and Deafness (MIDD)** and of these patients, 75% experience sensori neural hearing loss. MIDD is a non-insulin-dependent type of diabetes that usually develops before the age of 40 years as a result of mutation in mitochondrial DNA.

Conclusion

The prevalence of diabetes mellitus is now a relatively common chronic condition. While it can be well-managed to prevent complications and like involvement in other systems of the body it needs close and consistent medical attention to prevent more serious developments in ENT diseases. It is advisable to seek out a supportive health care team that incorporates not only medicine and regular blood testing but also holistic treatments such as diet, exercise and stress management, all of which have been proven to be effective in treating diabetes and its association with various ENT diseases.

XXXXXX

Dr Karan Sharaf, MD, DM
Consultant Nephrologist & HOD, Nephrology
Excelcare Hospitals, Guwahati

DM having ESRD: Medical Management of both - Diabetes & CKD

Introduction

The definition of chronic kidney disease (CKD) has evolved over the years. It is defined as presence of kidney damage or estimated glomerular filtration rate (eGFR/1.73 m2) <60 mL/min for e" 3 months[1]. In 2013, Kidney Disease Improving Global Outcomes (KDIGO) CKD work group suggested a CGA system of diagnosis to include cause, GFR category and albuminuria category[2].

As GFR declines, the risk of death rises sharply. The mortality risk increases from 17% at an eGFR of 45-59 ml/min to a staggering 343% at eGFR <15 ml/min[3]. The most common driver of death is cardiovascular (CV) events. In the dialysis population, sudden cardiac death (SCD) most commonly due to ventricular arrhythmia is the common cause of death[4].

The following points must be considered while managing a patient with diabetic CKD: (i) screening and diagnosis of CKD **(Table 1)**[1], ((ii) staging (GFR category and albuminuria category; **Table 2)**[1], (iii) prognostication of risk of progression (**Table 3**)[2] and, (iv) stage appropriate interventions to delay progressive renal decline (**Table 4**). Once patients' GFR declines <45ml/min/1.73m^2, most drugs require dose modifications, long acting oral hypoglycemic drugs cause hypoglycemic episodes, specific complications of CKD start manifesting (anaemia, CKD mineral bone disease, acid and electrolyte imbalances) which requires early identification and management. With relentless decline in GFR to <30ml/min/1.73m^2 diuretics doses may need to be increased to achieve euvolumia and preparation for future renal replacement therapy **(Table 4)** should be started once GFR <20ml/min/1.73 m^2 to prolong survival.

Management of CKD in a person with diabetes

General protective strategies

Measures which are to be employed for all diabetic patients include- (i) preventing acute kidney injury (AKI) episodes by avoiding exposure to NSADIS, herbals, long term proton pump inhibitors

(PPI's), contrast; (ii) emphasizing the importance of smoking avoidance for retarding CKD progression(MRFIT)[5]; (iii) achieving near normal glycemia (KDIGO target<7.%) for preventing microvascular complication including diabetic kidney disease (DKD)[6]; (iv)controlling blood ressure to a renoprotective target ofd" 140/90 mmHg; (MDRD study[7]; AASK study[8]); (v) properly managing dysliporoteinimia for decreasing coronary revascularization, if not survival(SHARP)[9], though 'statin for all' with a 'fire and forget'. approach without adopting a target is also endorsed by KDOQI; (vi) screening an ruling out non diabetic kidney disease screening(glomerulopathies; urological diseases; pyelonephritis), and some other condition to which d iabetic patients are susceptible to such as papillary necrosis, recurrent urinary tract infections, post infectious GN.

Protein restriction

The largest trial, MDRD study[7] showed no effect of protein restricted diet. However, based on few meta-analysis KDIGO recommends a target of 0.8gram/kg/day protein intake for CKD patients. One needs to apply judgment when restricting protein intake, especially in malnourished CKD patients. Patients who are well-nourished at the start of dialysis have a better survival than their malnourished counterparts (reverse epidemiology), and restricting food choices always carries the risk of a worsening nutritional status. Close follow-up for any evidence of malnutrition, either by clinical parameters or by serum albumin, is essential. A dietitian should be monitoring such patients carefully. The recommended caloric intake is 30–35 kcal/kg per day. In stage 4 and 5 patients, evidence of failing nutritional status is one key determinant in the decision to begin dialysis therapy.

Uric acid lowering

Whether modest elevations of uric acid is detrimental to kidney is unclear. Evidence from small observational trials suggest a renoprotective effect of uric acid lowering with allopurinol or febuxostat and recommended for all patients with CKD having uric acid >7mg/dl.

Renin angiotensin system blockers (RAS)

Treatments that decrease urinary albumin excretion may slow the progression of DKD even in the absence of hypertension. KDIGO does not recomend angiotensin-converting enzyme inhibitor (ACE-I) or angiotensin receptor blocker (ARB) for the primary prevention of DKD in normotensive normoalbuminuric patients with diabetes. ACE-I or an ARB should be added even in normotensive

patients with diabetes and albuminuria levels >30 mg/g who are at high risk of DKD or its progression. In the absence of side effects or adverse events (e.g., hyperkalemia or acute kidney injury) the dose should be titrated up to the maximum tolerated. The use of a combination of ACE-Is and ARBs as a dual blockade of the RAS cannot be recommended at present. [10]

Management of complications of CKD

Anemia- The most common causes are erythropoietin deficiency, iron deficiency, and inflammation. The initial evaluation of anemia should include complete blood count (CBC), absolute reticulocyte count, serum ferritin level, serum transferrin saturation (TSAT) and preferably serum vitamin B12 and folate levels[11]. A trial of IV iron 1000mg (or in CKD non dialysis patients a 1–3 month trial of oral iron therapy) is recommended if TSAT is d—30% and ferritin is d—500 ng/ml[11]. Dosing strategies for oral iron aim at providing approximately 200 mg of elemental iron daily, which is equivalent to ferrous sulfate 325 mg three times daily; each pill providing 65 mg of elemental iron. After addressing all correctable causes of anemia and an iron trial, ESA therapy should be started once hemoglobin falls below 10gm/dl[11]. Caution should be exercised if patient has active malignancy or a history of stroke. KDIGO guidelines recommend maintaining the hemoglobin level between 9 and 11.5 g/dL.

Chronic Kidney Disease-Mineral and Bone Disorder (CKD-MBD)-An excellent review by Hruska et al.,lists the various pathogenic pathways of CKD-MBD[12]. The basic abnormalities seen as CKD progresses are progressive hyperphosphatemia, hypocalcemia and secondary hyperparathyroidism. Baseline evaluation should include serum calcium, albumin, phosphorous, intact parathormone levels (iPTH) and vitamin D levels. A high serum phosphorus level is associated with vascular calcification, left ventricular hyperthrophy and an increased risk for mortality and adverse cardiovascular outcomes in CKD patients. Managementincludes restrictedphosphorous intake to800–1,000mgperday (avoid dairyproducts, colasandprocessedmeats) and use of phosphate binders (either calcium based or sevelamer carbonate). Target phosphorous level should be towards upper limit of normal.[13] Previous recommendation to maintain serumcal ciumat the highend of the normal range to ensure PTH suppression have been replaced by astrategy of keeping serumcal cium to ward the middle or low range of normal to minimize the risk of vascular calcification (restricttotalcalciumintaketo about1, 500 mg per day). This means that if calcium salts are used as phosphorus binders,

theymayneedtobecombinedwithsevelamerorpossiblyoneofthenewerIron-containingphosphorus binders[14] (**Table 5**). When active vitamin D sterols(e.g. cinacalcet) are being given, the dose should be reduced in the presence of hypercalcemia or hyperphosphatemia.. [13]

Electrolyteandacid–basecomplications

Avarietyofelectrolyteabnormalitiesmaybecomeapparentaskidneyfunctiondeclines. Themostprominentishyperkalemia which usually results from high dietary potassium intake specially fruits, use of ACE-I/ARBs, mineralocorticoid receptor antagonists,NSAIDs, PPIs etc. The recent development of novel gastrointestinal sorbents (patiromer) to prevent absorption of ingested potassium may allow more wide spread use of renin–angiotensin aldosterone system (RAAS) antagonists. Sodium bicarbonate is is used to treat chronic metabolic acidosis.[2]

Preparation for future renal replacement therapy (RRT) - Although the primary aims of management in CKD is to prevent progression to stage 5 CKD, for many patients RRT is inevitable. While planning for the initiation of dialysis it must be ensuredappropriate modality with full knowledge of the patient is chosen in a supported environment in which adverse events will be minimized. Avoidance of pricks to one hand and consequently timely AV fistula construction, vaccinations and identification of suitable kidney donor within the family are of paramount importance. [2]

Predialysis care: referral to a nephrologist - Many guidelines have laid down criteria for referral to a nephrologist (**Table 6**). Internationally acceptedguidline[2] based on recent clinical trial data recommend that early referral (>12 months prior to RRT) was associated with reduced odds of mortality for upto 5 years. The potential benefits associated with timely speciality referral include: timely establishment of vascular access; improved diagnoses of treatable kidney disease; retarded CKD progression; enhanced CKD complications management such as CKD anemia, CKD MBD and refractory HTN.

Management of diabetes in End Stage Renal Disease

End stage renal disease (ESRD) is described as irreversible renal function loss subjecting the person to undergo renal replacement therapy, in the form of dialysis or renal transplantation, to sustain life. GFR value of 15 ml/min/1.73 m^2 or less correlates to ESRD. Diabetes mellitus is considered one of the most common causes of ESRD. Life time risk of developing diabetic kidney

disease is around 25-40% and 5-40% in type 1 diabetes and type 2 diabetes, respectively. [15,16] In developed countries, diabetes is the major cause in development of ESRD.[17] With progression of diabetic nephropathy to the ESRD, the attempts of strict glycemic control lead to higher hypoglycemic events and increased mortality. Various pharmacological agents are available for the management of diabetes mellitus, but insulin remains the most preferred agent for management of diabetes in ESRD.

ESRD and glucose metabolism alterations

ESRD patients are likely to have wide fluctuations in plasma glucose levels. Some of the responsible mechanisms are discussed here. ESRD is associated with uremia and it has been shown that uremia contributes to insulin resistance and subsequently can lead to hyperglycemia. Secondary hyperparathyroidism and vitamin D deficiency may lead to impaired insulin secretion. Correction of uremia with dialysis and vit D supplementation may lead to improved insulin secretion and sensitivity and reduces insulin requirements.[18, 19, 20]

Some patients with ESRD are seen to develop euglycemia and ongoing treatment may lead to frequent episodes of hypoglycemia. This has been described as 'burnt out diabetes'. Responsible mechanisms include the following: (i) reduced insulin clearance and increased plasma half life,[21] (ii) reduced renal gluconeogenesis,[22] (iii) malnutrition , protein energy wasting and gastroparesis,[23,24] (iv) uremic toxins acting like biguanide agents.[25]

Glycemic monitoring in ESRD

Monitoring of glycemic status include estimation of capillary glucose by point of care instruments (SMBG) which is an integral part of management as in other diabetic patients with no CKD or early stage of renal dysfunction. This helps in adjusting dose of insulin and early management of hypoglycemic events. It is recommended that patients on maintenance haemodialysis on active treatment of diabetes with insulin or oral hypoglycemic agent(s), should have capillary glucose assessed pre- and post-dialysis.[26] Patients with wide fluctuations and recurrent hypoglycemia are candidates for continuous glucose monitoring (CGM). CGM can be utilized to monitor fluctuation of the day of dialysis and accordingly insulin regimen can be individualized. HbA1c estimation may underestimate the actual levels due to various factors which lead to falsely low HbA1c. This includes

anemia, reduced RBC life span, RBC lysis during dialysis, erythropoietin treatment.[27] Fructosamine and glycated albumin can be used in place of HbA1c but clinical data is scanty.

Glycemic Targets in ESRD

Stringent glycemic control is associated with higher risk of more frequent hypoglycemic episodes. Various studies of shown that HbA1c <6% and >8% are associated with higher mortality rates. The HbA1c should be kept >7% in patients with co-morbidities, limited life expectancy, and those at risk for hypoglycemia (that includes patients with advanced CKD as well as those receiving dialysis). [28]

Treatment in ESRD

Insulin

Subcutaneous insulin remains the treatment of choice in patients with ESRD. Dose modification is required in ESRD. As per expert opinion, insulin dose should be reduced by 50% in patients with ESRD. Also those undergoing hemodialysis require lesser doses of insulin. Basal bolus regimen is the best for glycemic control; however, Short acting regular insulin pre meals may be adequate, without basal insulin, as there is reduced clearance of insulin and increased half life. Ultra short acting insulin analogues may be preferred over short acting insulin in patients who have nausea, vomiting, and reduced appetite, as these can be administered even after taking a meal. Patients undergoing peritoneal dialysis can be given intra peritoneal insulin as well, but long term safety data are lacking.

Other Anti diabetic agents(ADAs)

Due to impaired renal function there is alteration of pharmacokinetics of most of the oral agents used as anti diabetic drugs. Thus patients are at higher risk of developing hypoglycemia. And there is inadequate evidence with certain anti diabetic agents (which includes GLP 1 receptor agonist – Liraglutide).[26,29] The summary of recommendations for anti diabetic agents' usage in ESRD is narrated in **Table 7**.

Conclusion

With the burgeoningepidemic of T2DM across the globe, and universal shortcoming in achieving optimal glycemic control , a sizable portion of people with diabetes are already in different stages from diabetic CKD. An alert diabetologist can help decrease the menace of this progressively costly and irreversibly severe complication. It can be achieved by identifying and tackling the risk factors (primary prevention), and suitable intervention and timely referral to nephrologist to retard progression of CKD (secondary prevention). The article is expected to serve as a ready reference for our diabetologist colleagues in this regard.

References

1. Levey A S and Coresh J (2012). Chronic kidney disease. *Lancet*, 379, 165-80

2. Kidney disease: Improving global outcomes (KDIGO) CKD Work Group (2013). Clinical practice guidelines for evaluation and management of chronic kidney disease. *Kidney Int suppl,* 3, 1-150

3. Go, A. S., Chertow, G. M., Fan, D., et al. (2004). Chronic kidney disease and the risks of death, cardiovascular events, and hospitalisation. *N Engl J Med*, 351, 1296-305

4. United States Renal Data System (2011). *USRDS 2011 Annual Data Report: Atlas of end stage renal disease in the United States.* Bethseda, MD: National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases.

5. Ishani, A., Grandits, G. A., Grimm, R. H., et al. (2006) Association of Single Measurements of Dipstick Proteinuria, Estimated Glomerular Filtration Rate, and Hematocrit with 25-Year Incidence of End-Stage Renal Disease in the Multiple Risk Factor Intervention Trial. *J Am Soc Nephrol* 17, 1444-1452; DOI: 10.1681/ASN.2005091012

6. National Kidney Foundation. KDOQI Clinical Practice Guidelines and Clinical Practice Recommendations for Diabetes and Chronic Kidney Disease. *Am J Kidney Dis*. Feb 2007;49(2 Suppl 2):S1-S180.

7. Peterson JC, Adler S, Burkart JM et al. Blood pressure control, proteinuria, and the progression of renal disease. The Modification of Diet in Renal Disease Study. *Ann Intern Med* 1995; 123: 754–762

8. Appel LJ, Wright JT, Jr, Greene T et al. Intensive blood-pressure control in hypertensive chronic kidney disease. *N Engl J Med 2010*; 363: 918–929

9. Baigent C, Landray MJ, Reith C, et al. The effects of lowering LDL cholesterol with simvastatin plus ezetimibe in patients with chronic kidney disease (Study of Heart and Renal Protection): a randomised placebo-controlled trial. *Lancet*. Jun 25 2011;377(9784):2181-2192

10. National Kidney Foundation. KDOQI Clinical Practice Guideline for Diabetes and CKD: 2012 update. *Am J Kidney Dis*. 2012;60(5):850-886

11. Kidney disease: Improving global outcomes (KDIGO) Anemia Work Group (2012). Clinical practice guidelines for Anemia in chronic kidney disease. *Kidney Int suppl,* 2012;2:279-335

12. Hruska KA, Seifert M, Sugatani T. Pathophysiology of the chronic kidney disease-mineral bone disorder. *Curr Opin Nephrol Hypertens*. 2015;24(4):303–309.

13. Kidney disease: Improving global outcomes (KDIGO) CKD MBD Work Group (2009). Clinical practice guidelines for diagnosis, evaluation, prevention and management of chronic kidney disease - mineral and bone disease. *Kidney Int suppl,* 2009;76(suppl 113):S1-S130

14. Peter, W.L.S., Wazny, L.D., Weinhandl, E. et al. *Drugs* (2017) 77: 1155.

15. Ismail N, Becker B, Strzelczyk P, et al. Renal disease and hypertension in non–insulin-dependent diabetes mellitus. *Kidney Int*. 1999; 55: 1-28.

16. Parving HH, Hommel E, Mathiesen E, et al. Prevalence of microalbuminuria, arterial hypertension, retinopathy and neuropathy in patients with insulin dependent diabetes. *Br Med J (Clin Res Ed)*. 1988;296:156-160.

17 . U.S. Renal Data System. *USRDS 2006 Annual Data Report: Atlas of End-Stage Renal Disease in the United States*. Bethesda, MD: National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases; 2006.

18. Kovesdy CP, Sharma K, Kalantar-Zadeh K. Glycemic control in diabetic CKD patients: where do we stand? Am J Kidney Dis. 2008; 52(4):766–777. [PubMed: 18572289]

19. DeFronzo RA, Tobin JD, Rowe JW, Andres R. Glucose intolerance in uremia. Quantification of pancreatic beta cell sensitivity to glucose and tissue sensitivity to insulin. J Clin Invest. 1978; 62(2): 425–435. [PubMed: 353075]

20. Dzurik R, Spustova V, Lajdova I. Inhibition of glucose utilization in isolated rat soleus muscle by pseudouridine: implications for renal failure. Nephron. 1993; 65(1):108–110. [PubMed: 8413768]

21. Reilly JB, Berns JS. Selection and dosing of medications for management of diabetes in patients with advanced kidney disease. Semin Dial. 2010; 23(2):163–168. [PubMed: 20210915]

22. Arem R. Hypoglycemia associated with renal failure. Endocrinol Metab Clin North Am. 1989; 18(1):103–121. [PubMed: 2645122]

23. Kalantar-Zadeh K, Derose SF, Nicholas S, Benner D, Sharma K, Kovesdy CP. Burnt-out diabetes: impact of chronic kidney disease progression on the natural course of diabetes mellitus. J Ren Nutr. 2009; 19(1):33–37. [PubMed: 19121768]

24. Kovesdy CP, Park JC, Kalantar-Zadeh K. Glycemic control and burnt-out diabetes in ESRD. Semin Dial. 2010; 23(2):148–156. [PubMed: 20374552]

25. De Deyn PP, Vanholder R, Eloot S, Glorieux G. Guanidino compounds as uremic (neuro)toxins. Semin Dial. 2009; 22(4):340–345. [PubMed: 19708978]

26. Joint British Diabetes societies for inpatient care –Management of adults with diabetes on hemodialysis unit, April 2016.

27. Park J, Lertdumrongluk P, Molnar MZ, Kovesdy CP, Kalantar-Zadeh K. Glycemic control in diabetic dialysis patients and the burnt-out diabetes phenomenon. Curr Diab Rep. 2012; 12(4):432–439. [PubMed: 22638938]

28. KDIGO. Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. Kidney International Supplements 2013. 2012; 3(1):1–163.

29. Updates on the Management of Diabetes in Dialysis Patients. Connie M. Rhee, Angela M. Leung, Csaba P. Kovesdy et al, Semin Dial. 2014 March ; 27(2): 135–145. doi:10.1111/sdi.12198.

Table 1: Criteria for definition of chronic kidney disease (Modified from Levey and Coresh, *The Lancet* 2012)[1]

Criteria	Comment
Duration ≥ 3 months (documented or from inference)	Distinguishes acute from chronic kidney disease
GFR <60 ml/min/1.73 m^2	Can be calculated from serum creatinine by using CKD epi formula currently recommended by KDIGO (https://www.kidney.org/professionals/kdoqi/gfr_calculator) Decreased estimated GFR can be confirmed by measuring the GFR (DTPA scan) (not done routinely) GFR <60ml/min/1.73m^2 has been kept as cutoff because – • In normal young individuals with no damage to kidneys, GFR may vary between 60 to 120ml/min. It has no clinical consequence • However, if the GFR is <60ml/min/1.73m^2 it increases associated with CVS morbidity and mortality even in the absence of kidney damage markers.
Kidney damage (as defined by structural or functional abnormality other than decreased GFR)	**Albuminuria** as marker of kidney damage (dipstick/uACR findings) • uACR >30 mg/g indicates increased glomerular permeability. • A positive UACR test should be repeated at least twice over 3 months to exclude acute kidney injury and to confirm a positive test. • Higher levels are associated with adverse outcomes including progression of kidney disease and increased CVS morbidity • Therapies to decrease Albuminuria result in slowing the progression of diabetic kidney disease
	Urinary sediment as marker of kidney damage (urine routine findings) • Red-blood-cell casts (proliferative glomerulonephritis)

	• White-blood-cell casts (pyelonephritis or interstitial nephritis) • Oval fat bodies or fatty casts (proteinuric diseases) • Granular casts and renal tubular epithelial cells in many parenchymal diseases (non-specific)
	Imaging abnormalities as markers of kidney damage (ultrasound, CT, and MRI with or without contrast, isotope scans, angiography • Polycystic kidneys • Hydronephrosis (obstructive uropathy) • Cortical scarring (infarcts, pyelonephritis, VUR) • Renal masses • Renal artery stenosis • Small and echogenic kidneys (late stages of CKD)
	Pathological abnormalities as marker of kidney damage(kidney biopsy findings) • Glomerular/vascular/interstitial diseases
	Renal tubular syndromes as marker of kidney damage (electrolytes/ABG findings) • Renal tubular acidosis • Nephrogenic diabetes insipidus • Barrter and Gittelman syndromes

Table 2 :: Categories of CKD by level of GFR and albuminuria (KDIGO 2013)[2]

GFR Category	GFR levels (ml/min/1.73 m^2)	Albuminuria Category	uACR (mg/g)
G1#	>90	A1	<30
G2#	60-89	A2	30-299
G3a	45-59	A3	≥300
G3b	30-44		
G4	15-29		
G5	<15		

#GFR stage G1 and G2 without marker of kidney damage do not fulfill the criteria for CKD

Table 3: Prognosis of CKD by combining GFR and Albuminuria categories (with inputs from KDIGO 2013)[2]

• No CKD (-) • **Moderate-risk CKD (+)** • High-risk CKD (++) • Very high-risk CKD(+++)				**Albuminuria stages, description, and range(mg/g)**				
				A1		**A2**	**A3**	
				Optimum and high-normal		High	Very high and nephrotic range	
				<10	10-20	30-299	300-1999	>2000
GFR Stages, description, and range(mL/min er 1.73m^2	G1	High and optimum	>105	-	-	+	++	+++
			90-104	-	-	+	++	+++
	G2	Mild	75-80	-	-	+	++	+++
			60-74	-	-	+	++	+++
	G3a	Mild-moderate	45-59	+	+	++	+++	+++
	G3b	Moderate-severe	30-44	++	++	+++	+++	+++
	G4	Severe	15-29	+++	+++	+++	+++	+++
	G5	Kidney failure	<15	+++	+++	+++	+++	+++

Table 4 :Three-step guide for the detection and assessment of diabetic CKD along with clinical action plan summary (adapted and modified from Levey and Coresh, *The Lancet* 2012)[1]

Stepwise guide	Clinical action plan
STEP 1: Testing for CKD • serum creatinine to estimate GFR • urinary albumin (dipstick and uACR) • Search for other markers of kidney damage (urine sediments; imaging abnormalities, or renal tubular syndromes) • Kidney biopsy (if indicated to rule out non diabetic kidney disease)	At baseline and then according to CKD prognostic category
STEP 2: **Detection of CKD** • GFR <60ml/min/1.73m^2 (estimated or measured) • Kidney damage markers • Duration >3 months (documented or inferred)	Putting GFR, Albuminuria and damage markers together
STEP 3: **Prognosticate according to stage (Using inuts from Table 3)** • No CKD	*Protective strategies* • Avoid AKI episodes; Avoid exposure to NSADIS, herbals, long term PPI's, contrast, smoking, tobacco, • **Target HbA1c ≤ 7%; BP ≤ 140/90 mmHg** • Obesity management; LDL lowering • **Look out for non diabetic kidney disease (glomerulopathies; urological diseases; pyelonephritis)** • **Consider SGLT2 inhibitors as part management of diabetes (if not contraindicated)** (please refer to text)
• CKD G3a /	Protective strategies **ACE/ARB addition and titrate** **Protein restriction 0.8gm/kg/day** **Lower blood pressure goal to ≤130/80mmHg (if uACR >300mg/g)**
• CKD G3b	Protective strategies ACE/ARB continuation Protein restriction 0.8gm/kg/day Lower blood pressure goal to ≤130/80mmHg Other medications dose adjustment according toGFR Management of CKD complications • Anaemia correction (Iron + ESA optimization) • CKD – MBD (mineral bone disorder) assessment and management • Electrolyte and acid base optimization
• CKD G4	Protective strategies Protein restriction 0.8gm/kg/day Lower blood pressure goal to ≤130/80mmHg Other medications dose adjustment according to GFR Management of CKD complications ACE/ARB reassessment – may need discontinuation if hyperkalemia; rapid deterioration in GFR Discontinue SGLT2 inhibitors Volume optimization ; may need higher doses of diuretics Prepare for future renal replacement therapy (RRT) • Vaccination (Hepatitis B; Pneumococcal; Influenza; Varicella) • Preserve vascular access vessels in one hand (no pricks) • Counselling on different RRT options • AV Fistula construction once GFR <20ml/min/1.73m^2 • Avoid blood transfusions (may lead to HLA sensitization)
• CKD G5	All the above Monitor for indication of initiation of RRT **(signs and symptoms of uremia)** **May go for pre-emptive transplant (in absence of uremic symptoms) once GFR ≤ 10ml/min/m^2**

Table 5: Dosages of selected phosphate binders required to reach a phosphorus binder equivalent dose (PBED) Adapted and modified from: Peter et al., 2017)[14]

Phosphate binder	Tablet strength (mg)	Approximate number of tablets to reach PBED of 6 g/day	Grams of calcium in 6 g PBED dose
Calcium carbonate	750	8	2.4
Calcium acetate	667	9	1.5
Sevelamer carbonate	800	10	0
Ferric citrate	210	9	0
In US dialysis patients, PBED averages around 6 g/day. This means that patients require 6 g/day of calcium carbonate to control their serumPhosphorus			

Table 6: Red flags – Nephrology referral; relevant considerations for physiciansbased on current best racitces

1. eGFR <30ml/min/m^2
2. Suspicion of non diabetic kidney disease • Active urinary sediments • Rapidly declining GFR (>10ml/min/1.73m^2 in a year) • Recent onset nephrotic range proteinuria • Absence of diabetic retinopathy
3. Difficult to manage CKD complications
4. Difficult to control hypertension with atleast three drugs
5. Proteinuria plus hematuria in dipstick
6. Progressive decline in renal function
7. CKD with renal artery stenosis

Table 7. Status of anti- diabetic drug usage in end stage renal disease(ESRD)[#]

Anti-diabetic agent	Status in ESRD[#]
Biguanide – Metformin	Contraindicated
Sulfonylurea – Glipizide	Can be given, Not usually preferred due to hypoglycemia risk
Sulfonylureas (other than Glipizide)	Contraindicated
Meglitinide – Rapaglinide	Can be given , risk of hypoglycemia remains
Alpha glucosidase inhibitors	Contraindicated
Pioglitazone	Can be given in ESRD, risk of fluid overload , usually avoided in dialysis dependent patients
GLP1 receptor agonists	Not recommended at present
DPP4 Inhibitors	Can be given Dose modification(down titration) needed with sitagliptin , vildagliptin No dose modification with Linagliptin
SGLT 2 inhibitors	Deemed to be ineffective

[#]GFR value of 15 ml/min/1.73 m^2 or less correlates to ESRD

Dr. Rupam Das
MBBS; MD(PAED),FIAP
Tezpur

Diabetes Mellitus – In Children

Diabetes mellitus is a common disorder of fuel metabolism. It is found most frequently duo to an absolute deficiency insulin secretion, due to destruction of the Beta cells of the pancreatic islets. The disease is associated with a number of short and long term complications, many of which are linked with the degree of blood glucose control. Diabetes is best managed by a team of personnel including a diabetes nurse/educator, dietician and psychologist in addition to the physician. These children and their families need a lifetime intensive diabetic education, multiple daily insulin injection, daily blood glucose monitoring, prevention and handling of acute complication, screening for and managing chronic complication, safe disposal of sharps, psychological support and societal support rather than discrimination. This is thus a complex, expensive, exhausting disorder for a child and family to cope with. Given the enormous burden of controlling diabetes – as yet we cannot prevent or cure it – much greater attention and resources are needed by the health care personnel, community, administrations and industry.

The incidence and prevalence of Type – I diabetes is suspected to be high in India, but in the absence of a nationwide registry, it is not possible to be sure of the numbers. The Diabetes Atlas 2017 estimates that there are 1,28,500 children and adolescents with diabetes in India. Type-I diabetes makeup an estimated 5-10% of all diabetes case or 11-12 million worldwide. In 2006 it affected 4,40,000 children under 14 years of age and was the primary cause of diabetes in those less than 10 years of age. The incidence of Type – I diabetes has been increasing by about 3% per year. Rates vary widely by country. In Finland the incidence is a high of 57 per 100,000per year, in Japan and China a low 1 to 3 per 100000 per year and in Northern Europe and the USA an intermediate of 8 to 17 per 100,000 per year.

In the United States, type – I diabetes affected about 208,000 youth under age of 20 in 2015. Over 18,000 youth are diagnosed with Type-I diabetes every year. Every year about 2,34,051, American die due to diabetes (Type – I or II) or diabetes related complications, with 69,071 having it as primary Couse of death.

Type-II Diabetes Mellitus for merely known as adult onset diabetes mellitus or non-insulin dependent diabetes mellitus. Type-II Diabetes Mellitus develops as a result of insulin resistance and progressive non-auto immune B cell failure. While Type-II Diabetes Mellitus has long been the most

prevalent form of diabetes in adult, the dramatic rise in childhood obesity over the past few decades has led to a markedly increase incidence of this disease in children and adolescent. Pediatric Type-II Diabetes Mellitus may account for up to 80% of the new cases of diabetes in high risk populations such as obese population. It is now apparent that childhood onset Type-II Diabetes Mellitus differs from adult disease in that it is associated with a more rapid decline in B cell function and earlier development of Type-II Diabetes Mellitus related complication. Type-II Diabetes Mellitus is typically more insidious than that with Type-II Diabetes Mellitus. In contrast to patient with Type-II Diabetes Mellitus who are usually ill at the time of diagnosis and whose spans more than a few weeks.

Etiological Classification of Diabetes Mellitus:

- Type-I diabetes:
  - Immune mediated
  - Idiopathic
- Type –II diabetes
- Other specific types:

- Genetic defects of beta- cell function:

- HNF-1 alpha, 4 alpha and glucokinase(formerly MODY 3,1 and 2)
- Mitochondrial DNA
- Neonatal diabetes.

- Genetic defects in insulin action:

-Type- A insulin resistance, leprechaunism, Rabson-Mendenhall syndrome,lipoatrophic diabetes.

- Diseases of exocrine pancreas:

- Fibrocalcificpancreatopathy, cystic fibrosis, hemochromatosis

-Endocrinopathies:

- Cushing syndrome, pheochromocytoma, hyperthyroidism, gigantism/acromegaly

- Drug or chemical induced:

- Pentamidine, glucocorticoids,phenytoin,thiazides,diazoxide

-Infections:

- Congenital rubella, cytomegalovirus

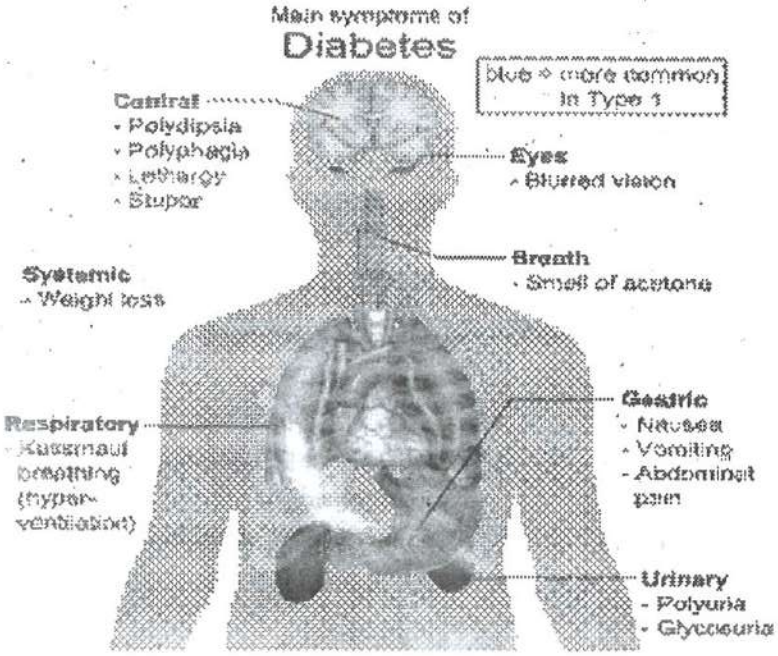
- Uncommon forms of immune-mediated diabetes:

- Stiffman syndrome, anti – insulin receptor antibodies

-Other genetic syndromes associated with diabetes:

- Down, Klinefelter, Turner, Wolfram, Friedreich ataxia,
- Laurence –Moon-Biedl, Prader-Willi, myotonic dystrophy
- Gestational diabetes mellitus.

Signs and Symptoms:-



Diagnostic criteria for diabetes mellitus:

- Symptoms of diabetes plus random plasma glucose e" 200mg/dl(on two separate occasions if symptoms not typical or hyperglycemia not unequivocal)or
- Fasting plasma glucose e" 126 mg/dl on two occasions or
- Two- hour plasma glucose e" 200 mg/dl during oral glucose tolerance test on two occasions (glucose load 1.75g/kg) or
- HbA1c >6.5%(taking care the laboratory reporting the HbA1c is NGSP certified and it is standardized to the diabetes control and complications trial)along with one of the above criteria.

Diabetes criteria for Dysglycemia and Diabetes Mellitus:-

DYSGLYCEMIA	DIABETES MELLITUS
Impaired fasting glucose: Fasting(at least 8 hr) plasma glucose 100-125 mg/dl (5.6-7.0mmol/L	Fasting(at least8 hr) plasma glucose e"126mg/dL(7.0 mmol/L
Or	
Impaired glucose tolerance: 2 hr plasma glucose during OGTT e" 140mg/dL(7.8 mmol/L), but <200 mg/dL (11.1mmol/L)	2 hr plasma glucose during OGTT e" 200mg/dL(11.1mmol/L) (11.1mmol/L)
	Or
Prediabetes: Hemoglobin A1c 5.7- 6.4% (39-47 mmol/mol)	Hemoglobin A1c e" 6.5% (48 mmol/mol)
Or	Symptoms* of diabetes mellitus plus random or casual plasma glucose e" 200 mg/dL (11.1 mmol/L)

Key features of Diabetes in Pediatric patients:

	TYPE 1 DIABETES	TYPE 2 DIABETES	MATURITY - ONSET DIABETES IN THE YOUNG	NEOMATAL DIABETES
Age at diagnosis	6 mo-18 yr	Puberty;rarely younger than 10 yr	Younger than 25	Younger than 6 mo
Causes and genetic factors	Autoimmune;genetic predisposition (HLA and other genes)	Obesity: genetic and ethnic predisposition	Autosomal dominant: HNF1A, HNF4A,GCK,HNF1B(rare)	KCNJ11,ABCC8,INS,and other genes
Associated features	Lean or weight loss at diagnosis; thyroid autoimmunity celiac disease	Obesity; acanthosisnigricans;polycystic ovarian syndrome; hypertension; hyperlipidemia; fatty liver disease;family history	Lean or weight loss at diagnosis; GCK mutations are asymptomatic	Failure to thrive
Diabetic ketoacidosis at presentation	Yes; about 25%	Yes; 5-20%	No	Yes
Treatment	Insulin	Lifestyle modification; metformin; insulin	Sulfonylurea; no treatment for GCK mutations	Sulfonylurea for KCJN11 and ABCC8 mutations; insulin for other mutations

Management :

Treatment of ketoacidosis(Just outlined)

- Fluid and electrolytes balance
- Alkali therapy
- Insulin therapy
- Monitoring and management of complications
- Education of the patient and family

Complications of Diabetes:

1. Hypoglycemia reaction :

   Mismatch between insulin dose on the one hand, and meal and exercise on the other, result in hypoglycemia quite frequently in the life of a child with diabetes.

2. Long Term Complications :

   (a) Micro vascular affecting the eye, kidneys and nerves.

   (b) Macro vascular causing cerebrovascular and coronary heart disease.

   (c) Growth retardation and pubertal delay.

   (d) Hypertension.

   (e) Anxiety and depression

   (f) Poor Cognitive function

   (g) Eating disorders

   (h) Skeletal defect

(i) Mauriac Syndrome

(j) Sexual dysfunction

Complication of diabetes usually do not set in before a duration of diabetes of at least3-5 years. Background retinopathy occurs in almost 90% after 15 years duration, but vision threatening (proliferating)retinopathy occurs in only 25% after 25 years. Similarly end stage renal disease occurs in 15 – 20% patients after a similar duration.

Healthy Life style like, healthy eating, regular physical activity, no smoking and regular monitoring and insulintherapy reduces the risk of complication of Diabetes Mellitus.

Reference:-

1. IAP Textbook of Pediatrics(6th Edition)
2. Nelson Textbook of Pediatrics (21st edition)
3. Journal Of Indian Pediatrics, Vol – 56;Number – 3; 3/2019

:::::::::::::::::::

Dr. S. Gogoi. MBBS,
DGO, M.S. (Obs)
.Past State President
IMA, ASB

GESTATIONAL DIABETES

Diabetes mellitus is one of the commonest medical problems encountered, both in pregnancy and out of it .Apart from those who are diagnosed and under treatment, there are those who are affected by the condition but not yet diagnosed. In this disease, almost more than any other, "coming events cast their shadow before".

Increased awareness and the widespread use of screening methods have led to increased recognition. The incidence varies considerably, from 1 to 14 % depending on ethnicity, selection criteria and the diagnostic tests performed, Asians being somewhere midway with incidences around 5-8%. Ninety per cent of these are cases of gestational diabetes.

Gestational diabetes is **one of the most common health issues that can occur during pregnancy.** It happens when the mother is diagnosed with diabetes for the first time partway through the pregnancy. The key to minimizing the effects of gestational diabetes is **diagnosing it early** through the use of an oral glucose tolerance test. If gestational diabetes is not diagnosed correctly, it can lead to macrosomia in the baby (abnormally large fetal size), which puts the baby at risk for neonatal hypoglycemia, trauma, and other complications. It can also lead to jaundice, premature birth, birth asphyxia, and reduced uteroplacental perfusion (RUPP), which harms the child by reducing oxygen flow to the brain.

What is gestational diabetes?

During pregnancy, the body produces a larger amount of certain hormones that impact the placenta, and help to maintain a healthy pregnancy. This increase in hormones leads to insulin resistance, which increases the amount of glucose in the blood stream. This is normal in pregnancy, as this extra glucose is needed to support the baby. However, when insulin resistance becomes too great, and the amount of glucose in the bloodstream is very high, gestational diabetes can result (10).

Causes of gestational diabetes

During pregnancy, certain hormones are released that can lead to a mass amount of glucose in the blood. In addition, pregnancy hormones like placental lactogen can interfere with susceptible insulin receptors, which further increases blood glucose levels. When the amount of insulin produced is less than the amount needed to handle blood glucose levels, gestational diabetes can arise (1).

Risk factors for gestational diabetes

Risk for gestational diabetes increases if the patient has any of the following conditions (1):

- Age greater than 25 years
- A family history of type 2 diabetes
- Prior birth of a baby that weighed more than 9 pounds or had a birth defect
- Previous poor obstetric history
- High blood pressure and preeclampsia
- Too much amniotic fluid (polyhydramnios)
- An unexplained miscarriage or stillbirth
- Overweight before pregnancy
- Polycystic ovary syndrome
- A previous diagnosis of gestational diabetes or prediabetes, impaired glucose tolerance, or impaired fasting glycemia

Signs and symptoms of gestational diabetes

Gestational diabetes can be diagnosed through prenatal testing and screening. Signs and symptoms for gestational diabetes may include

- Blurred vision
- Fatigue
- Frequent infections, including those of the bladder, vagina, and skin

- Increased thirst
- Increased urination
- Nausea and vomiting
- Weight loss despite increased appetite

Diagnosing gestational diabetes through blood tests

The American Congress of Obstetrics and Gynecologists (ACOG) recommends that all pregnant women receive an oral glucose tolerance test between the 24th and 28th week of pregnancy to screen for gestational diabetes. Women who have risk factors for it may have this test earlier in the pregnancy, typically around the 18th week of pregnancy .

Diagnosis is by the glucose tolerance test but the amount of glucose load used varies from 75g as recommended by WHO to 100g used by most other authorities the 100g test however has been better validated in clinical trials than the former.

The values accepted as diagnostic of gestational diabetes are also not universally accepted. 'O' Sullivan and mahan in 1964 suggested the use of 100g oral glucose tolerance test to diagnose GDM and proposed the normal values for the test based on blood glucose measurement in whole blood . Over the years , most laboratories switched over to analyzing venous plasma rather than whole blood for measuring blood glucose , therefore the National Diabetets Data Group (NDDG) in 1979 revised the values accordingly . But of course , as change is an inevitable part of life , new superior enzymatic methods have replaced old techniques , and Carpenter and Coustan published yet another set of values for the GTT , which have lower plasma glucose threshold and are widely in use since 2000.

The procedure for the 100g GTT is as follows : For at least three days prior to the test the patient should consume her normal unrestricted diet containing a minimum of 150 g of carbohydrate . After an overnight fast (8 to 14 hours) , a fasting blood sample is drawn, following which she drinks a solution of 100g glucose dissolved in a glass of about 300ml of water to which the juice of half a lemon can be added . This simple precaution protects against the vomiting that so often follows the rapid ingestion of so large a quantity of glucose on an empty stomach . Thereafter plasma glucose levels are

estimated every hour for 3 hours . She should remain at rest for the entire duration of the test and refrain from smoking .

The cut – off values for diagnosing gestational diabetes as given by NDDG and Carpenter and Coustan are given in Table below.

Table . Criteria for diagnosis of gestational diabetes mellitus

	Carpenter and Coustan	National Diabetes Data Group
Fasting	95mg/dl	105mg/dl
1 hour	180mg/dl	190mg/dl
2 hour	155mg/dl	165mg/dl
3 hour	140mg/dl	145mg/dl

The glucose tolerance test is considered abnormal, i.e., gestational diabetes is diagnosed, if any two values are met or exceeded. If only one value is abnormal, it is termed gestational impaired glucose tolerance. As can be seen from the table, the cut-off values proposed by the National Diabetes Data Group are higher . Using the lower values of Carpenter and Coustan , the number of women diagnosed as gestational diabetes increased from3.2 to 4.8% and women who were diagnosed as GDM with Carpenter and Coustan criteria and normal with NDDG had higher rates of perinatal complications like macrosomia , hypoglycaemia and hyperbilirubinaemia .

The Who diagnostic criteria , which is used in many parts of the world is based on a 75 g glucose tolerance test . A fasting plasma glucose 126mg/dl or two hour value after the glucose load 200mg/dl is diagnostic of gestational diabetes .

If fasting plasma glucose is 126mg/dl or a casual (random) plasma glucose is 200mg/dl , glucose administration is unnecessary as the criteria for the diagnosis of diabetes mellitus are met . If in doubt , the levels should be confirmed on a subsequent day .

estimated every hour for 3 hours . She should remain at rest for the entire duration of the test and refrain from smoking .

The cut – off values for diagnosing gestational diabetes as given by NDDG and Carpenter and Coustan are given in Table below.

Table . Criteria for diagnosis of gestational diabetes mellitus

	Carpenter and Coustan	National Diabetes Data Group
Fasting	95mg/dl	105mg/dl
1 hour	180mg/dl	190mg/dl
2 hour	155mg/dl	165mg/dl
3 hour	140mg/dl	145mg/dl

The glucose tolerance test is considered abnormal, i.e., gestational diabetes is diagnosed, if any two values are met or exceeded. If only one value is abnormal, it is termed gestational impaired glucose tolerance. As can be seen from the table, the cut-off values proposed by the National Diabetes Data Group are higher . Using the lower values of Carpenter and Coustan , the number of women diagnosed as gestational diabetes increased from3.2 to 4.8% and women who were diagnosed as GDM with Carpenter and Coustan criteria and normal with NDDG had higher rates of perinatal complications like macrosomia , hypoglycaemia and hyperbilirubinaemia .

The Who diagnostic criteria , which is used in many parts of the world is based on a 75 g glucose tolerance test . A fasting plasma glucose 126mg/dl or two hour value after the glucose load 200mg/dl is diagnostic of gestational diabetes .

If fasting plasma glucose is 126mg/dl or a casual (random) plasma glucose is 200mg/dl , glucose administration is unnecessary as the criteria for the diagnosis of diabetes mellitus are met . If in doubt , the levels should be confirmed on a subsequent day .

Treatment and management for gestational diabetes

Once a patient is diagnosed with gestational diabetes, the physician will have her monitor her diabetes by testing her glucose level at home. The most common way involves obtaining a drop of blood from a finger and putting it on a device that will give a glucose reading. The goals of treatment are to keep blood glucose levels within normal limits during the pregnancy and to make sure the growing baby is healthy.

With gestational diabetes, frequent check-ups by the physician are essential, especially during the last three months of pregnancy. The physician will carefully monitor the patient's blood glucose levels.

During the Antenatal Check up the medical team will also perform other tests to monitor the baby's health. Tests used to monitor and protect the baby include: (By USG)

- Fetal monitoring to check the size and health of the fetus.
- A **nonstress test (NST)**, which measures the baby's heart rate. The heart rate should increase when the baby moves. If the baby's heart doesn't beat faster during movement, the baby may not be getting enough oxygen.
- **Amniotic fluid index (AFI)**, a measurement, calculated by measuring the depth of the amniotic fluid in four sections of the womb and adding them together.
- A **biophysical profile (BPP),** which combines a nonstress test with an ultrasound study of the baby. There is a scoring system that enables the physician to evaluate the baby's heartbeat, movements, breathing, and overall muscle tone, and determine whether the baby is surrounded by a normal amount of amniotic fluid. The baby's scores on heartbeat, breathing, and movement help the physician determine if the baby is getting enough oxygen. When the amniotic fluid index (AFI) is low, it may mean that the baby hasn't been urinating enough. This could indicate that over time, the placenta has not been working as well as it should.
- **Doppler flow studies** test how well blood is flowing to the baby's brain, organs and other parts of the body.

In addition to close monitoring, it is recommended that mothers with gestational diabetes be treated with dietary counseling, oral hypoglycemic medications, or insulin .

Currently, the American College of Obstetricians and Gynecologists (ACOG) and the American Diabetes Association recommend screening all pregnant women for gestational diabetes. Additionally, ACOG states that women who develop pregnancy-related diabetes should be re-tested 6 to 12 weeks after delivering their babies (1).

However, according to a 2010 study of one million patient records, only about two-thirds of pregnant women undergo screening tests for gestational diabetes. Among the 5% of women who tested positive for gestational diabetes, just 1 in 5 were screened again within six months of giving birth (2).

The findings are particularly concerning given that a recent large study, the Hyperglycemia and Adverse Pregnancy Outcome (HAPO) trial, found that even subtle defects in maternal glucose metabolism during pregnancy led to health problems for mother and baby

High-risk pregnancy

Gestational diabetes places mothers in the high-risk pregnancy category, as it poses an increased risk for complications during pregnancy, labor, and delivery. With proper monitoring and care, however, many of the risks associated with gestational diabetes can be mitigated, resulting in a healthy baby.

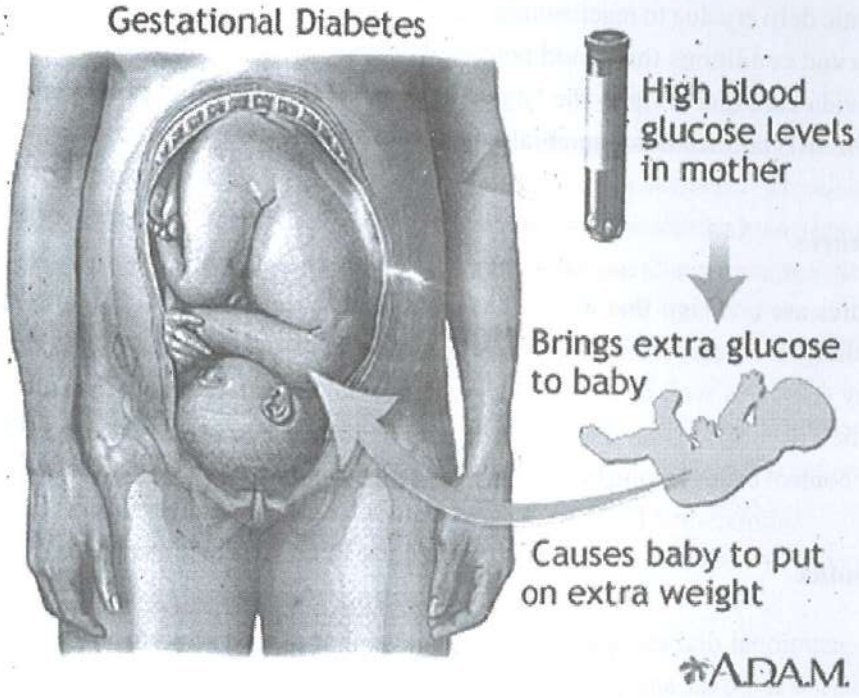
Mothers who have a high-risk pregnancy are referred to maternal-fetal specialists and require more frequent **prenatal testing**. In addition, these mothers are often advised to have a scheduled, early delivery in order to minimize the risks associated with their specific high-risk conditions.

Premature birth

In many cases, it is safest for babies of mothers with gestational diabetes to be delivered in a scheduled delivery prior to 40 weeks (3). If a mother's diabetes is poorly controlled, delivery is recommended as early as week 36 (4). Although premature birth does have associated risks, in the case of diabetes, the benefits of early delivery outweigh the risks. With proper postnatal care, babies born at 36+ weekly typically do well.

Macrosomia

Mothers with diabetes have high blood sugar levels, which are often passed on to the baby through the placenta (5). This large amount of energy is put towards growth, and can result in a **baby being too large** (macrosomic), which increases the baby's risk of a traumatic birth. Babies with macrosomia are most safely delivered via C-section. If macrosomia goes undetected, this may result in a prolonged or arrested labor or in traumatic injury to the baby's head or brain (6).



Neonatal hypoglycemia

While in utero, babies are used to having a certain supply of glucose from the mother. Once they are born, they begin to produce their own energy stores. While in utero, babies produce larger quantities of insulin to process the existing larger supply of blood sugar. When babies of mothers with gestational diabetes are born, the amount of insulin they produce does not drop as quickly as their glucose supply does, since they are accustomed to a larger supply of glucose in utero (7). If they don't take in enough energy as newborns, their blood sugars drop dangerously low. This is extremely problematic

because blood sugar is the sole energy source for the brain. If low blood sugar (hypoglycemia) continues, brain cells begin to die, which can cause seizures and permanent brain damage. After birth, its very important to monitor the baby's blood sugar, especially if their mother had diabetes, in order to prevent it from dipping too low (7).

Hypoxic-ischemic encephalopathy and cerebral palsy

Hypoxic-ischemic encephalopathy (HIE) is a brain injury caused by a lack of blood and oxygen flow to the brain. This can happen due to complications associated with diabetes, such as prolonged labor or traumatic delivery due to macrosomia (1, 3, 7). Medical practitioners are trained to recognize sentinel events and conditions that could potentially cause injury – and should be trained to avoid injury and provide care that is up to the 'standard of care.' If they do not provide proper care, the baby may suffer HIE and resultant cerebral palsy.

Neonatal seizures

Neonatal seizures are one sign that a baby has neurological abnormalities. Seizures are abnormal electrical discharges in the brain and are very common in the presence of hypoxic-ischemic encephalopathy (HIE), as well as in neonatal hypoglycemia. Neonatal hypoglycemia can damage brain cells, which in turn can cause seizures. It is very important that medical professionals bring seizures under control because continuing seizures can make underlying brain damage worse.

Neonatal jaundice

Mothers with gestational diabetes can sometimes have poor placental perfusion, which can cause chronic intrauterine hypoxia and placental insufficiency. These conditions can cause the baby to produce too many red blood cells (7). Babies' livers break down these red blood cells, releasing a substance called **bilirubin** into the blood. When too much bilirubin is released (**hyperbilirubinemia**), the baby's skin and eyes can turn yellowish. This condition is called **jaundice**.

Jaundice is a sign that should be reported to a medical professional for immediate treatment. In some babies, jaundice goes away on its own, but in other cases, babies may need some help. When babies need help eliminating bilirubin, they are put under blue phototherapy lights. If jaundice isn't treated properly, bilirubin can continue to accumulate to toxic levels and cross the blood-brain barrier. If

bilirubin reaches the brain, it causes a form of brain damage called **kernicterus** – a condition which is always avoidable (8).

Gestational diabetes: Steps for a healthy pregnancy, labor, and delivery

Starting at 32 weeks of gestation, the mother should have prenatal testing done at least every two weeks. This includes a nonstress test with and ultrasound to assess the amniotic fluid index (AFI). An ultrasound to estimate fetal weight should be done at 36 to 39 weeks as well. If a mother with gestational diabetes has other complicating factors, such as obesity or preeclampsia, prenatal testing should be more frequent and a planned delivery may need to occur at an earlier date (3, 5).

Labor induction and gestational diabetes

It is common practice to induce labor prior to 40 weeks for women with gestational diabetes as a strategy to reduce complications, especially those related to macrosomia. Labor induction is often planned at 38 ½ to 39 weeks' gestation and can occur when the baby's lungs are mature, antepartum tests are normal, diabetes is under control, and the mother doesn't have vascular disease (1).

ACOG recommends consideration of C-section delivery in diabetic women when estimated fetal weight exceeds 4500 grams. Some researchers recommend considering C-section delivery when the estimated weight is between 4000 and 4500 grams, after evaluating obstetrical history and clinical measurements of the pelvis. Physicians should be alerted to the possibility of cephalopelvic disproportion if dilation or descent stops during labor, and the baby's estimated weight exceeds 4000 grams. These researchers also suggest consideration of C-section delivery in diabetic women who demonstrate significant protracted labor and failure of descent (1).

Physicians have to consider a number of factors when caring for a woman with gestational diabetes and her baby. Due to the variety and complexity of these issues, it is essential that medical professionals monitor and treat the mother and baby very closely throughout the pregnancy, labor, and delivery.

Screening

Historical risk factors suggestive of gestational diabetes have been known for a long time . Unexplained stillbirth , macrosomia and preterm births have been consistently found to be significantly increased in both gestational and pre-gestational diabetic pregnancies, more so in women who require insulin for their treatment. Congenital malformations are increased much more in known diabetics , for obvious reasons , because true gestational diabetes usually manifests in the second half of pregnancy

, by which time fetal organogenesis is nearly complete . A history of gestational diabetes in a previous pregnancy , or diabetes in a first degree relative also increase the risk of GDM in subsequent pregnancies , Besides these , age , race and body mass index have been found to be independent clinical predictors of gestational glucose intolerance .

There has been a debate whether screening for diabetes should be universal or selective based on risk factors , or whether it is required at all. Nevertheless , the policy of universal screening in populations with high prevalence of type 2 disease is not only justified but certainly simpler to execute.

Low risk category

According to the recommendations of the American diabetes Association , women are considered low risk and do not require screening provided they fulfil all the following criteria .

25years of age

Normal body weight

No family history (i.e, first degree relative) of diabetes

No history of abnormal glucose metabolism.

No history of poor obstetric outcome

Not members of an ethnic/racial group with a high prevalence of diabetes.

High risk category

Certain ethnic groups are at high risk of type 2 diabetes , and therefore of gestational diabetes as well . These include the first generation Hispanics , South Asians and Middle – Eastern women . Therefore , all pregnant women from these regions should ideally undergo screening at 24 to 28 weeks gestation . Women with any of the following factors are at highest risk of developing impaired glucose tolerance in pregnancy :

1. markedly obese (120% ideal body weight or BMI 27kg/m)

2. history of diabetes in a first degree relative .

3. previous abnormal glucose tolerance .

4. previous large baby (4kg).

5. previous bad obstetric history , unexplained stillbirth or congenitally malformed babies .

6. Those with persistent glycosuria. This group of patients should undergo blood glucose testing at the first visit and if found normal , screening should be repeated between 24 to 28 weeks or at any other time that clinical features are suggestive of high blood glucose levels. These include obstetric complications like macrosomia , hydramnios and pre- eclampsia . Other features developing during the current pregnancy that should cause concern include recurrent urinary tract infection , especially a history of pyelonephritis and recurrent vulvovaginitis.

Test for screening

The next question which arises is that , which test should be performed for screening of GDM. Random blood sugars or timed samples in relation to meals, serum fructosamine and HBA1c are poor tests for screening of GDM . Fasting plasma glucose also has a high false positive rate at threshold levels which would offer reasonable sensitivity . Besides , there are women who have normal fasting and postprandial levels but show exaggerated response to glucose challenge . Milder from of the disease may be therefore missed if testing is done without administering a glucose load which helps borderline glucose intolerance to become overt .

So far the test which has been found to be most cost - effective is the 75g glucose challenge test .75g glucose load is administered , irrespective of the time of the last meal and plasma glucose is measured one hour later . All women with a level of 140mg/dl are tested by the 100 g oral glucose tolerance test (GTT) . Using this cut – off , about 80 % of gestational diabetics can be detected and 15% of patients will be required to undergo GTT . Although using a lower cut-off of 130mg/dl would increase the sensitivity of the test to nearly 90 % , it would also mean a corresponding increase in the number of women who would need GTT, with the attendant logistic problems .

Screeening can be done in one or two stages . A one – stage approach implies that a diagnostic test , i. e. 100g GTT is performed without prior screening . This approach may be used at the discretion of the obstetrician in high – risk pregnancy , but would be certainly too cumbersome and not cost – effective in low – risk women , where many more unnecessary GTT would be done . The two – stage approach is used in all others , in whom a glucose challenge test is first performed and GTT requested if the result is abnormal to make a diagnosis .

Gestational Diabetes diet

Eating complex carbs rather than simple carbs is recommended.Spacing meals and snacks containing carbohydrates evenly throughout the day can help avoid spikes in blood sugar. The American Diabetes Association recommend that women with gestational diabetes should eat three small-to-moderate meals and two to four snacks per day.

Other ways to help regulate blood sugar include:

- avoiding eating too many carbohydrates at one time
- sticking to complex carbohydrates that are high in fiber
- combining carbohydrates with protein or healthy fat
- not skipping meals
- eating a protein-rich and fibrous carbohydrate breakfast

Eating low glycemic index foods

Eating foods that have a low glycemic load is another crucial factor in a gestational diabetes diet.

The glycemic load is calculated by multiplying the grams of carbohydrate in a serving of a particular type of food by that food's glycemic index (GI). This number gives a more accurate picture of a food's real impact on blood sugar.

Foods with a low glycemic load are broken down more slowly than simple carbohydrates, which are typically considered high GI foods.

A glycemic load of 10 or below is considered low and is ideal for those with gestational diabetes who are trying to manage blood sugar.

Low glycemic load foods to eat include:

- 100 percent wholegrain breads and cereals
- non-starchy vegetables
- some starchy vegetables, such as peas and carrots

It is customary to advice screening Test for GDM at the Gestational age of 24-28 weeks, routinely, especially those who have a family history of diabetes and by age 25 years or above.

Proper dietary management is one of the essential criteria for Glycemic control.

- women who develop pregnancy-related diabetes should be re -tested 6 to 12 weeks after delivering their babies
- Metformin and Insulin are the drugs of choice for Medical management of GDM.

Sources:

1. Durnwald, C. (2018, September). Diabetes mellitus in pregnancy: Screening and diagnosis. Retrieved from https://www.uptodate.com/contents/diabetes-mellitus-in-pregnancy-screening-and-diagnosis.
2. Gestational Diabetes. (n.d.). Retrieved from http://www.questdiagnostics.com/home/physicians/health-trends/trends/gestational-diabetes.html.
3. Caughey, A. B. (2018, July). Gestational diabetes mellitus: Obstetrical issues and management. Retrieved from https://www.uptodate.com/contents/gestational-diabetes-mellitus-obstetrical-issues-and-management.
4. Kalra, B., Gupta, Y., & Kalra, S. (2016, June). Timing of Delivery in Gestational Diabetes Mellitus: Need for Person-Centered, Shared Decision-Making. Retrieved from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4900972/.
5. Durnwald, C. (2018, October). Gestational diabetes mellitus: Glycemic control and maternal prognosis. Retrieved from https://www.uptodate.com/contents/gestational-diabetes-mellitus-glycemic-control-and-maternal-prognosis.
6. Mandy, G. T. (2018, August). Large for gestational age newborn. Retrieved from https://www.uptodate.com/contents/large-for-gestational-age-newborn.
7. Riskin, A., & Garcia-Prats, J. A. (2016, August). Infant of a diabetic mother. Retrieved from https://www.uptodate.com/contents/infant-of-a-diabetic-mother.

ACHIEVEMENT BY IMA CGP ASSAM STATE FACULTY

It is my proud privilege to mentioned that after establishment of the IMA College of General Practitioners Assam State Faculty under Assam State Branch IMA for the first time able to achieved recognition and appreciation in the National Level IMA and CGP H.Q's in different categories by its members in the year 2020.

The year 2020 will be the memorable year in the history of IMA CGP Assam State Faculty.

I offered my heartiest thanks and gratitude from my core of the heart on behalf of the members of IMA AGP Assam State Faculty to the Honourable National Leaders of IMA and IMA CGP H.Qs

Followings are the categories received recognition and appreciation

1. **LIFE TIME ACHIEVEMENT FOR**

   a. **Dr. V. Parameshivara Award** for Life Time Achievement in Medicines and Commitment to the art of Medicine by **Dr. Pramatheswar Borooah** Tezpur.He was Past National President of IMA, ASB.

   b. **Dr. Tapan Deka, Nalbari** : IMA National President Appreciation Award for Life Long Service to IMA. He was Past Vice President and Past President of IMA ASB.

   c. **Dr. Hiranmoy Adhikary**, Past President of IMA ASB and Dean CGP for IMA Dr. Jyotiprasad Ganguli Memorial Award.

   d. **IMA Dr. C.L. Jagga** Award for best Faculty of IMA CGP.

"Assam State Faculty"

2. **APPRECIATION CERTIFICATE CATEGORY**

   1. **IMA CGP Life Membership Enrolment 2019-2020** to IMA Assam given on 08-11-2020 virtual IMA CGP Conference – Chennai.

   2. Best State Chapter IMA Assam for **CGP NEWS JOURNAL** given on 08-11-2020 virtual IMA CGP H.Qs conference, Chennai.

3. **IMA CGP Honorary Fellowship (FCGP) Conferred during its VIRTUAL NATIONAL GPCON 2020 held on 8th November'2020 conference of IMA CGP at Chennai**

to

   a. **Dr. HiranmoyAdhikary**, Bangaigaon Branch. He was Dean, CGP – 2020

   b. **Dr. Hemendra Kumar Borah**, Tezpur Branch, Director of Studies, Assam State Faculty.

   c. **Dr. Nayan Kumar Phukan**, Life Member of Tezpur Branch, CGP.

4. **IMA NATIONAL PRESIDENT APPRECIATION AWARD FOR :**

Best Adjudged President of State

   a. Dr. Satyajit Borah, President IMA ASB

Best Adjudged President of Local Branch

   b. Dr. Atul Kumar Kalita, President, IMA Tezpur Local Branch

   c. Dr. Apurba Kumar Sarma, President, IMA Nagaon Local Branch

Secretary Level

d. **Dr. HemangaBaishya**, Secretary, IMA Assam State Branch

e. **Dr. Sahad Ullah**, Secretary, IMA Jorhat Branch

5. **COVID CARE AND CONTROL CATEGORY**

a. **Dr. Tul Tul Das**, Tezpur Branch for **IMA National President Appreciation Award** for COVID care and control.

b. **DOCTORS DAY AWARD – 1st July, 2020**

Dr. Pradip Kumar Sarma, Tezpur Branch, for **National Doctors Day Award -2020** towards COVID-19 care and control, in recognition of the sacrifice in line of National Duty.

6. **ELECTION / SELECTION**

a. **Dr. Satyajit Borah**, Tezpur Branch, elected **Hony. Joint Secretary IMA CGP H.Qs 2020-22**

b. **Dr. Sikha Sarma**, Guwahati Branch, selected member of the **National Pink Health**.

STATE LEVEL

a. **Dr. Lakheswar Bhuyan**, Tezpur Branch, elected **Chairman, Association of Surgeons of Assam Chapter of Association of Surgeons of India-2021-2022**.

b. **Dr. Bibhu Priyo Das**, Tezpur Branch, elected **Joint Secretary, Association of Surgeons of Assam Chapter of Association of Surgeons of India-2021-2022.**

Out of **15 (fifteen) Nos**. awardee **9 (nine) Nos**. from IMA CGP Tezpur Branch Life member.

I being the Director of IMA CGP Assam State Faculty honestly acknowledged with sincere thanks and gratitude to our Honourable National President Dr. RajanSarma, Hony Secretary General Dr. R.V. Asokan and all other respected members of the Award Committee, without their recommendation and selection it would not be possible to achieved the recognition and appreciation by our members of IMA CGP Assam State Faculty in National Level IMA.

It will be my shortcoming if I don't name of the Hony Secretary IMA CGP HQ Dr. L. Yosuda and Dean CGP Dr. Hiranmoy Adhikari, without their support and help it would not be possible to achieved the National Level recognition and appreciation.

I am very much grateful to them and offer sincere thanks.

With deep sense of appreciation all these have been possible due to collective efforts of the esteemed members of IMA CGP Assam State Faculty and IMA ASB.

I am grateful to State President Dr. Satyajit Borah and Hony. State Secretary Dr. HemangaBaishya for their active support.

My special thanks goes to **Hony. Secretary, IMA CGP, Assam State Faculty, Dr. Jagadish Basumatary** for meticulous and systematic editorial works of this 'CGP NEWS' amidst his extensive '**COVID DUTY**' as Head of the Department of Critical Care, Anaesthesiology Department of Tezpur Medical College Hospital during the entire COVID-19 pandemic period.

Lastly congratulation and greetings to all Awardee from the IMA CGP Assam State Faculty for their recognisable and appreciable works during the COVID-19 pandemic period.

Long Live IMA!!!
Long Live CGP!!!
Long Live ASB!!!

(Dr. H.K. Borah)
Director of Studies
IMA, CGP, Assam State Faculty

for COVID

20 towards
y.

s 2020-22
lth.

geons of

rgeons of

mber.

sincere thanks and
al Dr. R.V. Asokan
on and selection it
f IMA CGP Assam

da and Dean CGP
the National Level

s of the esteemed

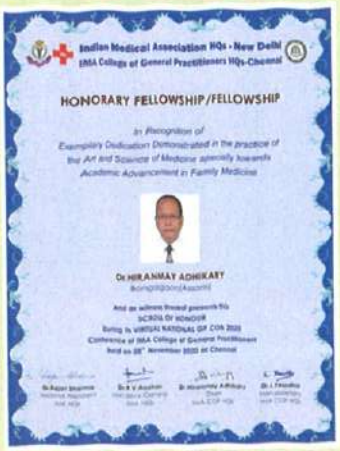
mangaBaishya for

adish Basumatary
'COVID DUTY' as
l College Hospital

e Faculty for their

Borah)
f Studies
m State Faculty

ACHIVEMENT OF IMA CGP ASF DURING THE YEAR 2020



Dr. Hiranmay Adhikary
CGP Member, Bongaigaon

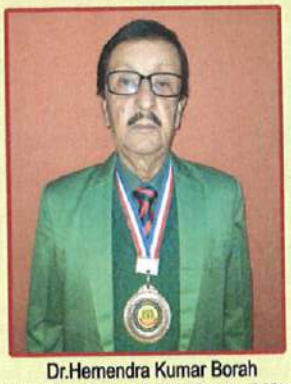
Dr. Hemendra Kumar Borah
CGP Member, Tezpur

Dr. Nayan Kumar Phukan
CGP Member, Tezpur

AWARDED with Honorary Felloship (FCGP) During the VIRTUAL National GPCON-2020 Conference of College of GeneralPrectioners held on 8th November-2020 at CHENNAI

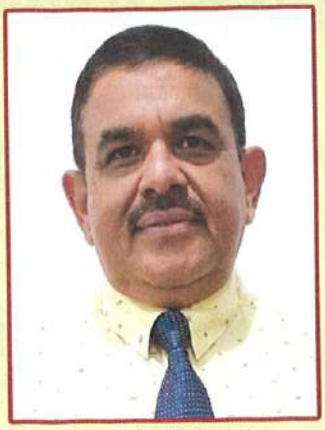
Dr. Atul Chandra Saikia
CGP Member, Tezpur

Dr. Rupam Das
CGP Member, Tezpur



Dr.Hemendra Kumar Borah
awarded Hon. Fellowship (FCGP)of IMA CGP and present the SCROLL OF HONOUR during VIRTUAL NATIONAL GP CON 2020 Conference of IMA CGP held on 8th November -2020 at Chennai

Dr. Laksheswar Bhuyan
Chairman IMA AMS ASB
CGP Member, Tezpur
Elected Chairman Assam Chepter
ASI for the Association year 2021-22



Dr. Satyajit Borah
President IMA ASB
CGP Member, Tezpur
Elected Joint Secretary
IMA CGP HQ. for the Association year 2020-22

Dr. Bibhu Priyo Das
CGP Member, Tezpur
Elected Joint Secretary
Assam Chepter of
ASI for the Association year 2021-22

R 2020

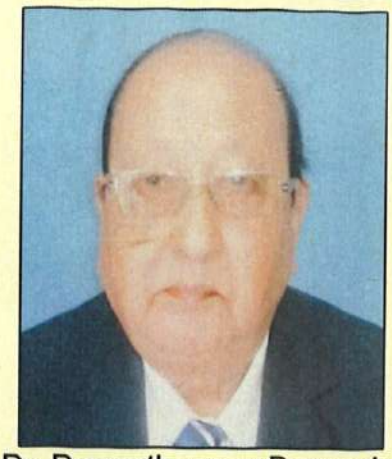
umar Phukan
ber, Tezpur

Conference of

a Kumar Borah
ip (FCGP)of IMA CGP and
HONOUR during VIRTUAL
20 Conference of IMA CGP
nber -2020 at Chennai

u Priyo Das
mber, Tezpur
oint Secretary
Chepter of
ociation year 2021-22

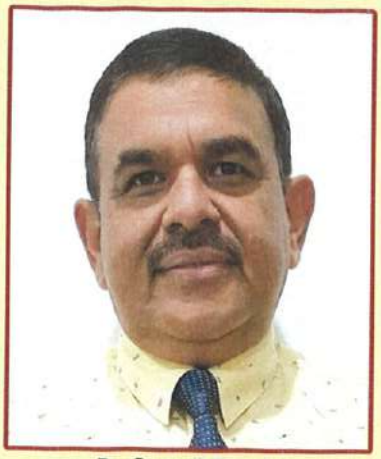
ACHIVEMENT OF IMA CGP ASF DURING THE YEAR 2020



Dr. Pramatheswar Barooah
Past President IMA ASB

Dr. Tapan Deka
Past National Vice President and
Past President IMA ASB

Both honourd for Life time Achivement Award by IMA National H.Qs 2020



Dr. Satyajit Borah
President IMA, ASB

Dr. Atul Kr. Kalita
President IMA, Tezpur Local Branch

Dr. Apurba Kr. Sarma
President IMA, Nagaon Local Branch

National president appreciation Award for best Adjudged State President and Local Branch President

Dr. Hemenga Baishya
Secretary IMA, ASB

Dr. Sahad Ullah
Secretary IMA,
Jorhat Local Branch

Dr. Tul Tul Das
IMA National president
appreciation Award for
Covid Care and Control

Dr. Sikha Sarma
Selected Member of the
National Pink Health

National president appreciation Award for
best Adjudged State Secretary and Local Branch Secretary

ACHIVEMENT OF IMA CGP ASF DURING THE YEAR 2020

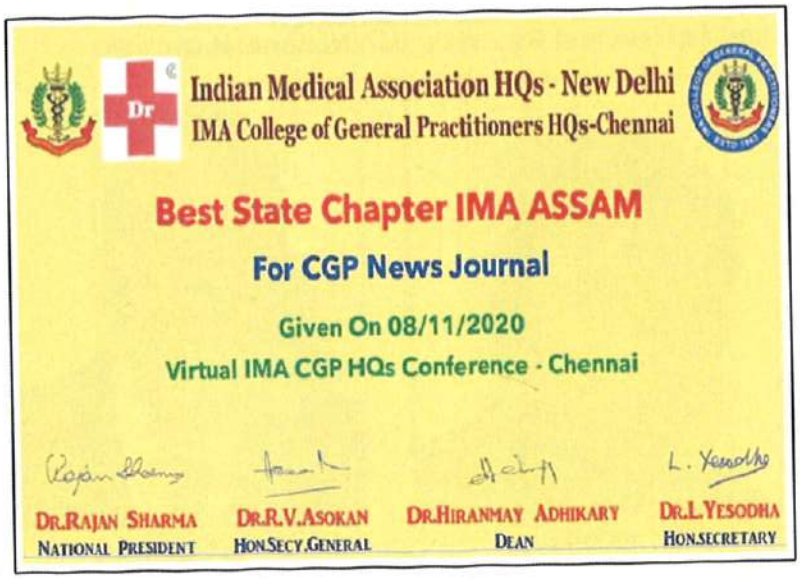


Photo Galary













Photo Galary

Printed and Published by – Dr. Jagadish Basumatary on behalf of IMA CGP Assam State faculty and
Printed at- M/s Extensile, Anwar Complex, Tezpur -784001, Dist.-Sonitpur (Assam)