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Journal of Diabetes Education

To Dispel Darkness Of Diabetes

DIET MANAGEMENT ▸

◂ EXERCISE

MEDICATION ▸

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CONTENTS

NUTRITIONAL MANAGEMENT OF HYPERGLYCEMIA POST CARDIAC SURGERY

* Meenakshi Bajaj

Glycemic abnormalities and diabetes are on the rise globally. Hyperglycemia is common after stressful events, such as myocardial infarction, stroke, and sepsis, or in the postoperative setting, after cardiac surgery. Stress-induced hyperglycemia is a transient phenomenon, distinct from the chronic glucose dysregulation brought about by diabetes. Studies have shown that stress hyperglycemia after cardiac surgery, which occurs in patients both with and without diabetes, is associated with a higher risk of complications, including major infections, and increased mortality.

Not all patients with stress hyperglycemia after cardiac surgery benefit from a "blanket approach" that aims to keep their maximum blood glucose below 180 mg/dL, a new study suggests. In fact, researchers found that these types of patients react differently to glucose control, depending on diabetes status and whether or not they use insulin.

Patients with insulin-treated diabetes had optimal clinical outcomes (less risk of infections and respiratory complications) and lower hospital costs and length of hospital stay if their maximum glucose in the 4 days after cardiac surgery was 180 to 240 mg/dL. Glucose levels <180 mg/dL are associated with better outcomes in most patients. Mortality and morbidity benefits are seen with overall control of hyperglycemia; however, the exact range is still not clearly defined.

Medical Nutrition Therapy (MNT) is an essential component of inpatient glycemic management programs. MNT is defined as a process of nutritional assessment and individualized meal planning in consultation with a Registered Dietician. Lack of attention to MNT in the hospital contributes to unfavorable changes in blood glucose. MNT is recommended as a component of the glycemic management program for all hospitalized patients with diabetes and hyperglycemia. The goals of inpatient MNT are to optimize glycemic control, to provide adequate calories to meet metabolic demands, and to create a discharge plan for follow-up care.

Evidence suggests that there is no optimum ratio described for percentage of calories from carbohydrate, protein, and fat for all people with diabetes (B); therefore, macronutrient distribution should be based on individualized assessment of current eating patterns, preferences, and metabolic goals.

Nutrition requirements often differ in the home vs. the hospital setting. The types of food may change or the route of administration may differ, e.g. enteral or parenteral feedings may be used instead of solid foods. Nutritional management in the hospital is further complicated by hospital routines characterized by abrupt discontinuation of meals in preparation for diagnostic studies or procedures, variability in appetite due to the underlying illness, limitations in food selections,

* Dietician at Tamil Nadu Govt. Multi Super Specialty Hospital, Chennai

and poor coordination between insulin administration and meal delivery that creates difficulties in predicting the efficacy of glycemic management strategies.

Several clinical trials have investigated the use of diabetes-specific formulas as a way of ameliorating the risk for hyperglycemia with Enteral Nutrition. These diabetes specific formulas differ from standard formulations by supplying a lower percentage of total calories as carbohydrate and substituting monounsaturated fatty acids for a major component of administered fat calories. A diabetes-specific enteral formula (50% carbohydrate) or a lower-carbohydrate (33–40% carbohydrate) formula may be used with least 30% of total energy as lipids.

Majority of hyperglycemic patients will still require insulin therapy for control of hyperglycemia while receiving this type of nutritional support.

Management of hyperglycemia in the non-critical care setting could be achieved by providing meals with a consistent amount of carbohydrate at each meal so that coordinating doses of rapid-acting insulin to carbohydrate ingestion is possible. A consistent carbohydrate meal-planning system may help to facilitate glycemic control in the hospital setting.

The system is based on the total amount of carbohydrate offered rather than on specific calorie content at each meal. An advantage to the use of consistent carbohydrate meal plans is that they facilitate matching the prandial insulin dose to the amount of carbohydrate consumed.

For patients on rapid acting insulin, low carbohydrate containing foods should be prescribed at mid-meals or patient must take rapid acting insulin for a carbohydrate containing snack based on his insulin to carbohydrate ratio. Carbohydrate intake from low Glycemic index and low Glycemic load vegetables, fruits, whole grains, legumes, and low fat dairy products should be advised over intake from other carbohydrate sources, especially those that contain added fats, sugars, or sodium.

Insulin to Carbohydrate Ratio (I: C factor):

Amount of carbohydrates (grams) covered by one unit of insulin

450 (Regular)/ 500 (Rapid acting) ÷ Total Daily Dose (TDD) = I:C Factor

For eg: TDD= 45

1 unit of insulin covers 10 grams of carbohydrate if on regular insulin and 11 grams of carbohydrate if on rapid acting insulin

It is important to remember that hypoglycemia is a well-recognized and feared complication in hospitalized patients with or without established diabetes. The risk for hypoglycemia is higher during periods of hospitalization due to variability in insulin sensitivity related to the underlying illness, changes in counter-regulatory hormonal responses to procedures or illness, and interruptions in usual nutritional intake. Frequent monitoring of BG levels allows for timely detection, prevention and treatment of hypoglycemia.

When the nutritional issues in the hospital are complex, a registered dietitian, knowledgeable and skilled in medical nutrition therapy, can serve as an individual inpatient team member. That person should be responsible for integrating information about the patient's clinical condition, meal planning, and lifestyle habits and for establishing realistic treatment goals after discharge. Orders should also indicate that the meal delivery and nutritional insulin coverage should be coordinated, as their variability often creates the possibility of hyperglycemic and hypoglycemic events.

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THE KETOGENIC DIET IN OBESITY

*** Priyangee Lahiry**

The ketogenic diet is a very low carbohydrate, high fat, adequate protein diet which had originally been developed in the 1920s for managing intractable epilepsy. Later, it has been indicated in many other conditions such as obesity, diabetes, cancer and Parkinson's disease. In the recent years, the ketogenic diet has become increasingly popular among people trying to lose weight.

The ketogenic diet aims at depriving the body of glucose and putting it in a metabolic state (ketosis) in which fats are burnt to produce ketone bodies. These ketones are utilized as a fuel for energy production instead of glucose. In order to create this state, the dietary carbohydrates are restricted to as low as fewer than 20-50g per day, and the fat to carbohydrate ratio is typically kept at 4:1 or 3:1 ratio by weight.

Ketosis:

Glucose is the preferred fuel especially for the brain. Glucose is also required for the complete oxidation of fats in the TCA cycle. In conditions of starvation or drastic carbohydrate restriction (below 20g/day), i.e. when glucose is not available, fats are broken down in an alternate pathway to produce ketone bodies. Acetoacetate is the primary ketone body which is eventually broken down to acetone and beta hydroxy butyrate. These ketone bodies are utilized by the brain and other tissues for energy production instead of glucose. This condition is called ketosis. Under non ketotic circumstances, very small amounts of ketones are produced, and normal blood levels of ketones are generally less than 0.5mM/dL, and only traces are excreted in the urine. But in ketosis, ketone bodies accumulate (up to 5mM/dL) in the blood and are excreted in the urine. Acetone being a volatile compound is eliminated through lungs while breathing, giving the characteristic fruity breath. This physiological ketosis is generally benign and is different from ketoacidosis, in which, the ketone bodies accumulate in the blood in dangerously high amounts (>10mM/dL) causing the pH of blood to drop, producing acidosis. In fact, physiological ketosis is associated with certain beneficial effects that can have potential therapeutic usages.

Effects of ketogenic diet on weight loss:

The ketogenic diet has been shown to have beneficial effects on weight loss in the short term and medium term. Several studies have compared the effects of low carbohydrate, high fat ketogenic diets with traditional low-fat diets for weight loss. Majority of randomized controlled trials comparing ad libitum ketogenic diets with low fat diets showed greater weight loss with the ketogenic diet in the short term. However, after 12 months the difference became insignificant. Studies done with energy restricted ketogenic diet vs isocaloric low fat diets also showed greater and more dramatic weight loss in the ketogenic group. In addition to weight loss, ketogenic diets have shown to produce greater total fat and truncal fat loss indicating its beneficial effect on body composition and hence metabolic advantage. Greater weight loss with low carbohydrate high fat diet has been shown in

* Registered Dietitian and Clinical Nutrition Consultant, N.G Medicare Clinic, Kolkata and Care Continuum Pvt. Ltd. Kolkata. Email: priyangee.lahiry@gmail.com.

both healthy obese subjects as well as in subjects with insulin resistance and glucose intolerance.

The effects of ketogenic diet on weight loss have been attributed to several possible factors:

1. Appetite suppressing action of ketone bodies
2. Reduction in lipogenesis and increased lipolysis
3. Increased metabolic cost of gluconeogenesis and thermic effect of proteins
4. High satiating effect of fats.

The anorexic effect of ketone bodies is of interest, and several studies have demonstrated the same, although the exact mechanism is not completely understood yet. Ketone bodies, particularly beta hydroxy butyrate is thought to have an appetite suppressing effect both directly and by modification of hormones regulating appetite.

Plasma free fatty acid, which is a peripheral modulator of appetite has been shown to increase with the administration of ketogenic diet. These free fatty acids may provide signal to the hypothalamus of nutrient abundance and hence have an anorexic effect.

Benefits of ketogenic diet beyond weight loss:

The beneficial effects of the ketogenic diet go beyond weight loss and might be helpful in managing conditions associated with obesity. Excess body fat is closely associated with insulin resistance. The metabolic disturbances associated with insulin resistance due to inefficient utilization of glucose are improved with low carbohydrate high fat diets When beta hydroxy butyrate is used as fuel instead of pyruvate (from glucose)- the normal mitochondrial fuel, the metabolic efficiency is altered and there are changes signaling functions. These changes beneficially affect gene expression, improve insulin resistant states, attenuate oxidative stress and inflammatory conditions.

Significant reductions in serum triglycerides, marked increase in HDL cholesterol and decrease in inflammatory markers have been observed with these diets. A 2-year long trial showed more weight loss and greater improvements in HDL cholesterol and serum triglyceride with ketogenic diet compared to low fat diet. A short-term trial of 8 weeks with type 2 DM patients showed greater decrease in liver fat percentage with a high fat diet than with a low fibre high fat diet. Insulin resistance is associated with increased hepatic lipogenesis and increased serum triglycerides as glucose is diverted for hepatic triglyceride synthesis at an increased rate. When carbohydrates are replaced with fats in the diet, metabolism shifts more towards fat oxidation and post prandial spikes in glucose and insulin decreases.

Potential risks of the ketogenic diet:

Despite the metabolic benefits of ketogenic diets, the severe carbohydrate restriction and the huge fat content of the diet raises question regarding its safety. Needless to say, that severe carbohydrate restriction calls for restrictions on a large number of foods which can give rise to several nutrient inadequacies.

One of the commonly noted deficiencies with the Ketogenic diet is selenium deficiency which has been shown to cause abnormalities in cardiac rhythm and impaired myocardial functions. Arterial stiffness, which is an early indicator of vascular damage, has been shown to increase with the ketogenic diet in the short term. In fact, the cardiovascular outcome of ketogenic diets is an obvious cause of concern due to its high fat content. Increase in LDL cholesterol occurs in about half the individuals on ketogenic diet. Although, ketogenic diets might

show improvements in certain cardiovascular parameters like increased HDL and lowered triglycerides, this mostly in the short term. Long term ketogenic diets have been shown to cause hepatic steatosis, dyslipidaemia and glucose intolerance in mice. The long-term effects of ketogenic diets have not been studied well in humans and studies have produced conflicting results.

Another safety concern of the ketogenic diet is its effect on calcium balance and bone health. Prolonged exposure to the acidic environment caused by the elevated ketones in blood can lead to progressive loss of bone minerals. Studies done on children with intractable epilepsy have shown the occurrence of osteopenia and osteoporosis despite supplementation with calcium and vitamin D.

In fact, nephrolithiasis, a very commonly noted complication of the ketogenic diet, can be attributed to increased urinary calcium excretion. Other factors that contribute to stone formation are decreased urinary citrate excretion and aciduria, both of are due to the acidic environment produced by the ketone bodies.

The high amounts of fat in the diet also give rise to gastrointestinal tolerance issues. Nausea, diarrhoea, and GERD are some of the commonly reported gastrointestinal side effects.

Another gastrointestinal side effect is constipation which is obvious due to the fact that with severe restriction of carbohydrate foods, fibre intake is minimal.

Other common side effects of ketogenic diet are dehydration, hypoglycemia, hyperuricaemia, vitamin and trace element deficiencies. Apart from this, acute pancreatitis, ketoacidosis, low serum sodium and magnesium have also been reported.

Till date there are very few studies which have evaluated the long-term outcomes of the ketogenic diets. Most of the long-term studies have been done on epileptic children where the ketogenic diet is used as a medical nutrition therapy and it is possible that some of the results might be confounded due to the use of anti-epileptic drugs.

Practical considerations and suggested recommendations:

In a true ketogenic diet, majority of the calories i.e.70-80% of calories are provided by fats, 10-20% from proteins and only 5-10% from carbohydrates. For a diet of 1600 Kcal for example, this corresponds to around 125-140g of fat, 20-40g of carbohydrates and around 60g of proteins. This might be very impractical especially in the context of Indian diets where cereals are the staple food and pulses and legumes serve as the major source of proteins. This would leave the patients, especially vegetarians with extremely limited food choices. Such diets are likely to be extremely unpalatable, monotonous and nutritionally inadequate.

In fact, many of the metabolic effects discussed above have been demonstrated with low carbohydrate high fat diets, where the exact proportion of carbohydrates have varied among studies. We still do not know whether extremely low carbohydrate ketogenic diets are superior to moderately low carbohydrate diets.

Thus, a more healthful as well as practical approach would be to modify the ketogenic diet to a moderately low carbohydrate diet with unprocessed whole grains, fruits and non-starchy vegetables as the sources of carbohydrates and moderate amounts of fats with a focus on unsaturated fats. Processed and refined carbohydrate food like biscuits and cakes should

be restricted and can be replaced with natural high fat foods like nuts and whole eggs.

It is also important to consider the total calorie intake especially in context of weight loss. Reduction in total calorie intake is always important and not just replacing carbohydrate calories with fats. One of the main reasons for weight loss that has been demonstrated with ad libitum ketogenic diets is the spontaneous reduction in calorie intake which might be in part due to the monotony of the diet.

With the above considerations, a modified ketogenic diet can serve as an effective tool for weight loss with certain added metabolic advantage and can be advised to obese individuals for a limited period. This should however be followed up with a healthy and more balanced eating plan on the long term.

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REAL TIME CGM (RTCGM) VERSUS INTERMITTENTLY VIEWED CGM (ICGM)

*** Sakina Lanewala**

Continuous glucose monitors (CGMs) are electronic devices that measure and display glucose levels in the body throughout day and night. CGM provides immediate, ongoing feedback that the patient can apply towards reaching desired goals.

Technology is developing and changing all the time by introducing improvements in accuracy, reliability, and ease of use. Today, we can choose between real-time and intermittently scanned CGMs. Both provide data about patient's current glucose level and the trend about changing glucose levels. However there are some specific differences between these approaches that we may want to consider when choosing a system that's right for our patient. Here's what we need to know:

There are two types of CGM:

- Real time: One can check his/her sugar levels at any time, as well as being able to download them.
- Intermittent: One can't see his/her sugar levels in real time but can look back at results by downloading them.

Real-time CGM

It displays updated glucose every few minutes from subcutaneous interstitial fluid using sensor. These systems are made up of three components: the sensor that just sits underneath the skin and measures sugar levels; a transmitter that attaches to the sensor; and a smartphone that displays the glucose data. It automatically transfers real-time data to a receiver or mobile device and features alarms that warns the user about immediate and impending low and high glucose. And all offer the ability to share real-time data with family members, caregivers, and health care providers.

The advantage with real-time CGM systems is that the user is always informed about his/her diabetes management. They take over the job of constantly checking glucose, offer around-the-clock vigilance, and provide data at times when the user can use it.

Real-Time CGM : Upsides

1. Offers Alerts

   The most valuable aspect of real time CGM system is their ability to have audible alarms that can warn the user of high or low blood sugar. It can provide early warning than most of us would detect them on our own. User can have enough time to make adjustments that could blunt the effect of high or low blood glucose. Most importantly, it is more beneficial for older people as they can't detect the changes in their glucose as they used to do when they were younger. Some time, blood glucose can get pretty low before patients are fully aware that they are getting into trouble. In this regard, the real-time CGM is exceptionally valuable and potentially lifesaving tool, especially when the patient is in sleep.

2. Offers Predictive Alerts

   Some systems even have predictive alerts that are easy for the user to know if blood sugar is expected to cross the high or low threshold based on the current level, rate and direction of change. Low alerts are helpful and safer for exercise where as high alerts allow the user to be more aggressive at managing post meal blood glucose spike. The ability to forecast the blood sugar for the next couple of hours can help patients to stay in their desired blood glucose range.

* Dietician and Diabetes Educator at Dr. Chandalia's Diabetes Endocrine Nutrition Management & Research Centre

3. Transmits Data Continuously

   With real-time CGM devices, it can measure the interstitial fluid continuously and sends this directly to the compatible device every 5 minutes. Patient can constantly get their blood sugar readings on their smart phones/ receiver. They don't have to do any other task such as finger stick, the data are always right there in the receiver.

4. Shares Data

   The ability to share data with five other family members is another important feature of CGM. It acts as a safety net. For e.g. If patient is travelling or doesn't wake-up to lower glucose alarm during night, they can get a call from the family member informing them about the situation.

Real-Time CGM : Downsides

1. Requires Setup

   To use the alarm and alert features, the user has to program the settings, such as their low glucose threshold and target ranges. This can get a little complicated, especially if one doesn't read the instructions. However, the manufacturers offer online video tutorials to guide patients through the process.

2. Compatibility

   For rtCGM like that of Medtronic, The Guardian Connect app is intended for use only by patients using a compatible mobile device.

3. Calibration

   Yes, calibration is required at least 2 times a day. After the first two start-up calibrations, calibrations are needed every 12 hour to maintain accuracy. However, it gives the option to recalibrate if the sensor values don't match the symptoms or a confirmatory finger-stick check.

4. Lifespan

   The sensor can be used one time, and it has a maximum life of 170hours (seven days). The 170 hour life span of the sensor begins when the sensor is connected to the transmitter.

5. Devices Can Be Expensive

   Although they are covered by most insurance companies, they may not be affordable if one has to pay out of pocket.

Intermittently Scanned CGM

Abbott's FreeStyle Libre takes another approach. Known as an intermittently scanned CGM, the system requires the user to scan the device to obtain the glucose data. It uses two components: sensor/transmitter (inserted in upper arm) and a separate reader device. The sensor continuously samples and measures glucose levels, generates a new glucose value every minute, and records the reading every 15 minutes for 10 to 14 days of sensor wear time, depending on the model. It provides the same glucose data as real-time CGM systems, but it doesn't automatically "push" data to the receiver or mobile device. To get their glucose trends, one needs to wave the reader over the transmitter.

Intermittently Scanned CGM: Upsides

1. Eliminates Finger Sticks

   With this system, glucose data can be used to make treatment adjustments without the need for finger-stick confirmation. Additionally, its factory calibrated, eliminating the hassle and pain of daily calibration with finger sticks.

2. Its Convenient and Easy to Use

   The sensor, which is about the size of two stacked quarters, is painless to apply, comfortable to wear, and easy to use. And user can scan the transmitter through his/her clothes, a real benefit when one wants to be discreet.

3. Its Affordable

   They are much less expensive than real-time CGM devices, and it is covered by most insurance companies.

Intermittently Scanned CGM: Downsides

1. Offers No Alerts

The biggest drawback is the lack of alerts to warn about glucose fluctuations. It is difficult for especially older people to sense low blood sugar. People with hypoglycemia unawareness especially may not know their blood sugar is low until their judgement is impaired. It is a downside for anyone concerned about overnight laws.

2. Real-Time Data Isn't Shared

   Unlike with other CGMs, the glucose values can't be shared in real time with family or caregivers. This doesn't provide a safety net if the patient is going low, travelling or distracted by other things.

3. Lifespan

   The sensor can be used one time, and it has a maximum life of 14 days.

4. One Version Takes 12 Hours to Warm Up

   When a new sensor is inserted, it will not show any glucose data for the first 12 hours. During this time, the user needs to do finger-stick checks. Thankfully, the latest model takes only an hour to warm up.

5. Offers No Option to Calibrate

   This does not allow one to recalibrate the sensor when glucose values don't match their finger-stick results which may indicate that the meter or CGM sensor is inaccurate. Without the ability to recalibrate the sensor, the user may need to insert a new sensor long before its indicated wear time has expired.

The Bottom Line

Whether real-time or intermittently scanned, a CGM is a major technological breakthrough in diabetes self-management. Both approaches offer opportunities to improve diabetes management. It can serve as a valuable learning tool showing the immediate impact of lifestyle and medication decision. Research suggests that consistent use of CGM can reduce HbA1c levels, glycemic variability and the frequency, duration and magnitude of hypoglycemic events.

Growing evidence supports the benefits of using CGM: the studies and clinical trials suggest that adults with type 1 diabetes (T1D) who wear a CGM device most days can improve glycemic control without increasing risk of hypoglycemia, while those already close to target HbA1c can maintain control while reducing risk of hypoglycemia. In children and adolescents, achieving adequate adherence remains a significant barrier, although usability has improved with current-generation CGM devices in this age-group.

If a patient is taking any medication that puts him at risk for hypoglycemia (such as insulin or sulfonylureas), then a real-time CGM is probably the best choice because it will alert him, when the glucose is dropping so one can prevent or stop a low before it becomes critical. It's particularly important for children and adolescents who would benefit from remote monitoring through the data-sharing feature.

The right CGMs for a patient can help to interpret and use data to make appropriate treatment decisions and achieve desired blood glucose goals.

Ready to choose a CGM?

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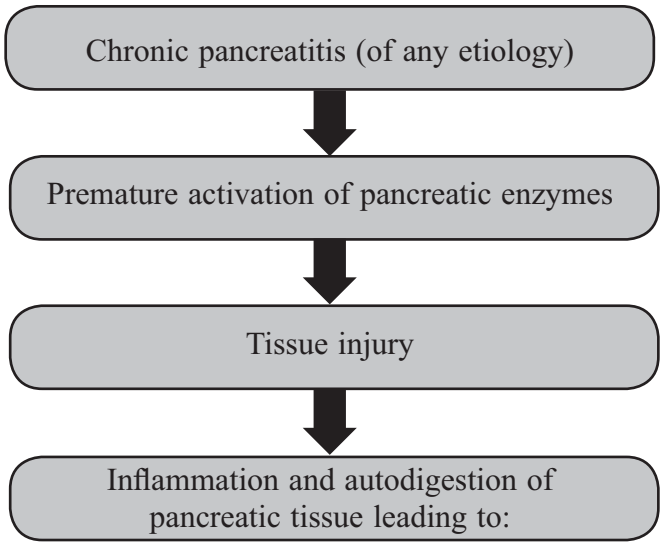
PANCREATIC DISEASE AND SECONDARY DIABETES

*** Samina Burhanpurwala**

The most prevalent form of diabetes is type 2 diabetes, which is characterized by impaired insulin sensitivity, inadequate insulin response and insulin deficiency. However, diabetes can also develop as a consequence to other disease, such as an exocrine disease of pancreas. According to ADA and WHO, diabetes occurring secondary to pancreatic diseases is commonly referred to as *Pancreatogenic diabetes or type 3c diabetes mellitus*. It is due to impairment in pancreatic endocrine function related to pancreatic exocrine damage due to acute, relapsing and chronic pancreatitis, cystic fibrosis, hemochromatosis, pancreatic cancer, fibrocalculous pancreatopathy and pancreatectomy. Chronic pancreatitis is the most common disease of exocrine pancreas associated with development of diabetes.

On the basis of the pathophysiology of chronic pancreatitis, recently guidelines have been established which support the specific diagnostic criteria and treatment algorithm.

Pathogenesis:



Clinical Characteristics:

Clinical features are similar to those with diabetes due to other reasons. Along with the predominant symptoms of polyuria, polydipsia and polyphagia, patients with type 3C diabetes or pancreatic diabetes present the following characteristics:

* Dietician and Diabetes Educator at Dr.Chandalia's Diabetes Endocrine Nutrition Management & Research Centre. saminab0708@gmail.com

1. Have a history of pancreatitis.
2. Pain in abdomen
3. Dyspepsia
4. Steatorrhea and
5. Malabsorptive symptoms

Diagnosis:

Talking about diabetes, correctly diagnosing and classifying a patient is the most crucial part before deciding the algorithm of treatment and/or management. Since, there is very poor awareness of this type of diabetes, correct classification of type 3c diabetes is often missed and patients are misclassified.

Currently, there is no universally accepted criteria for pancreatogenic diabetes. However, initially the diagnosis remains the same as for type 1 and type 2 diabetes i.e measurement of FBS and HbA1c. Both these tests are to be repeated annually and impairment in these calls for further action. Impairment in either should be further evaluated by a standard 75 g oral glucose tolerance test (OGTT). The challenging part here is discriminating type 2 diabetes from type 3c, since T2DM is present in 8-10% of general population, which again is so very common in patients with pancreatic disease. When suspected, clinical or biochemical evidence of insulin resistance i.e. by presence of acanthosis nigricans or hyperinsulinemia can confirm T2DM. Similarly, destruction of the islet cells by pancreatic inflammation differs from that of type 1 diabetes, since there is also loss of glucagon from alpha cells and loss of pancreatic polypeptide (PP) from PP cells in type 3C DM.. In addition to this, there is nutrient malabsorption and maldigestion which leads to impaired incretin secretion.

Hence, to improve recognition of pancreatogenic diabetes, Ewald and Bretzel subsequently proposed useful major diagnostic criteria (all must be present) and minor criteria (Table 1) for T3cDM.

An additional diagnostic criterion of interest can be the evaluation of pancreatic polypeptide (PP) response to a mixed nutrient ingestion. The absence or reduced PP secretion is easily able to distinguish between type 3c diabetes from early type 1 and also from type 2 diabetes. However, routine testing of PP response in everyday practice doesn't seem feasible.

Management:

Primary goals to the management of pancreatic diabetes includes:

a) Control of hyperglycemia (HbA1c < 7%)

b) Prevention of hypoglycemia

c) Managing malnutrition and abdominal symptoms

Table 1: Diagnostic criteria for Type 3C Diabetes Mellitus
Major Criteria (all must be fulfilled):
• Presence of exocrine pancreatic insufficiency (according to monoclonal fecal elastase 1 or direct function test)
• Pathological pancreatic imaging (by endoscopic ultrasound, MRI or CT)
• Absence of Type 1 DM associated autoimmune markers.
Minor Criteria:
• Impaired beta cell function (as measured by HOMA-B, C-peptide/ glucose ratio)
• No excessive insulin resistance (measured by HOMA-IR)
• Impaired incretin (e.g. GIP) or pancreatic polypeptide secretion
• Low serum levels of lipid soluble vitamins (A, D, E and K)

Adapted from Ewald and Bretzel [4]

Management here is problematic as there is both carbohydrate and lipid malabsorption. There is also unstable blood glucose control due to loss of glucagon response to hypoglycemia and abdominal pain and/or nausea which leads to irregular eating pattern. Astonishingly, large clinical trials conducted by The Diabetes Control and Complications Trial (DCCT, 1986) and UK Prospective Diabetes Study (UKPDS, 1991) have specifically excluded patients with type 3c diabetes. Hence there is no definite guidelines established for the management of type 3c diabetes. So the consensus follows treatment guidelines for type 2 diabetes with few modifications when required.

Pharmacological therapy: ADA and EASD have recommended metformin as the 1st line of treatment for type 2 diabetes. Therefore, many patients with type 3c diabetes are initially treated with metformin as the 1st choice. This can prove to be good choice if the patient has mild hyperglycemia and insulin resistance is suspected. However, its side effects such as nausea, abdominal pain and weight loss should be taken into consideration, as it might not be tolerated by all patients. Additionally, in view of pathological changes in exocrine and endocrine pancreas, insulin secretagogues and insulin sensitizers have very limited role to play in management of type 3c diabetes.

Incretin based therapies such as GLP-1 analogues and DPP-IV inhibitors also enhance insulin secretion. Yet, they both are associated with gastrointestinal side effects such as nausea, delayed gastric emptying and weight loss. Therefore, it's best to avoid its use until their safety is confirmed.

So the mainstay of treatment what remains is insulin therapy. It can be the primary choice of treatment for patients with severe malnutrition.

Nutrition therapy: Preventing further malnutrition and reducing meal-induced hyperglycemia are the primary goals of medical nutrition therapy. Patients are advised to eat foods rich in soluble fiber and low in fat.

Exocrine pancreatic deficiency calls for oral enzyme replacement therapy such as lipase along with fat soluble vitamin (A, D, E, K) and other nutritional supplements. Supplementation with enzyme is particularly important for fat digestion and absorption of nutrients. This helps to control symptoms of steatorrhea and prevent malabsorption of fat-soluble vitamins. In addition to this, it plays vital role in maintaining incretin hormone secretion and thereby improving glucose tolerance.

Lifestyle modification: Abstinence from possible contributors of chronic pancreatitis such as alcohol and smoking is highly recommended as both exacerbate the underlying pancreatic inflammation and increase the pain. Abstinence from alcohol is also beneficial in diabetes, knowing that it inhibits hepatic gluconeogenesis and cause alcohol-induced hypoglycemia, especially if patient is on insulin. Alcohol ingestion is also likely to precipitate an attack of pancreatitis.

In a nutshell, we can say that most patients with type 3c diabetes suffer from chronic pancreatitis as the underlying disease. However, in a patient first presenting Diabetes, chronic pancreatitis is seldom considered and patients are misdiagnosed.

References for further reading:

1. Choudhuri G, Lakshmi C and Goel A. Pancreatic Diabetes. *Tropical Gastroenterology 2009; 2:71-75*
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3. Das S, Tripathy S and Panda B. Pancreatogenic Diabetes. *API Medicine Update 2017; 3. (accessed 5th Feb, 2019).*
4. Ewald N, Bretzel RG. Diabetes mellitus secondary to pancreatic diseases (Type 3c) - Are we neglecting an important disease? Eur J Intern Med 2013; 24:203-206. PMID: 23375619.

WHAT'S NEW?
FIASP

*** Sakina Lanewala**

Novo Nordisk's new Fiasp insulin has hit the market in various parts of the world. Fiasp is the latest edition to meal time insulin. It is a faster acting insulin Aspart formulation that is absorbed more quickly than insulin Aspart (Novo Rapid), attaining peak concentration of insulin in the blood earlier than aspart. This is predicted to improve PPG control in patients with Type 1 and Type 2 Diabetes.

Managing patient's blood sugar at meal time is an important aspect of reaching ones A1c goals. It is designed to more closely mimic the natural physiology of human insulin produced by a person without diabetes to prevent those brisk post-meal blood sugar spikes. In non-diabetics, early phase insulin response is almost instatenous which the Fiasp is able to mimic closely. By getting into the system faster, it helps to metabolise the glucose from the digested meal quickly.

While most fast acting insulins would need to be dosed 20 min before eating to prevent that post meal spike, Fiasp could be dosed just before or upto 20 min after eating and still reduces the post meal spike. It can be taken 0-2 minutes before a meal or upto 20min after starting a meal.

It is marketed as 3ml cartridges. 1 ml solution contains 100units of insulin Aspart. One pre filled pen contains 300 units of insulin Aspart in 3 ml solution.

What Fiasp Contains:

The active substance is insulin Aspart.

The other ingredients are phenol, meta cresol, glycerol, zinc acetate, disodium phosphate dehydrate, arginine hydrochloride, niacinamide (Vit B3), hydrochloric acid and sodium hydroxide (for ph adjustment).

Niacinamide helps to increase the speed of initial insulin absorption which means Fiasp enters the blood stream in approximately 2 ½ minutes. It also contains arginine, a naturally occurring amino acid. Arginine provides increased stability to insulin formulations and has been shown to effectively inhibit insulin aggregation which may lead to loss of activity and even trigger an immune response. Niacinamide increases the rate of absorption and L-arginine maintains stability in the insulin.

How much faster is Fiasp as compared to normal insulin?

The onset of action was 5 minutes earlier and time to maximum glucose infusion rate was 11 minutes earlier with Fiasp than with Novorapid. The maximum glucose lowering effect of Fiasp occurred between 1 and 3 hours after injection. The glucose lowering effect was 74% larger during the 1st 30 minutes with Fiasp than with Novo rapid.

The duration of action was shorter and the late glucose lowering effect was 10% smaller for Fiasp compared to that of Novorapid.

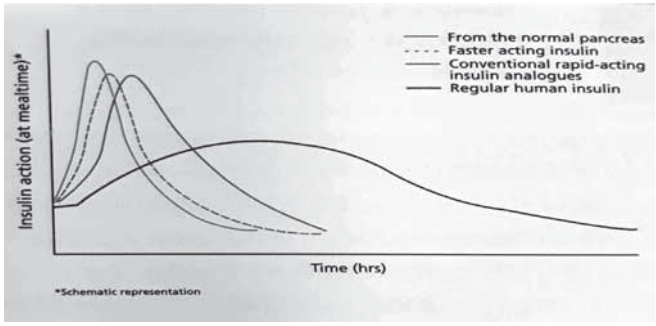

Figure 1: Schematic of insulin action

As seen in figure 1, faster insulin approaches the physiological insulin secretion in people with diabetes. Additionally, faster acting insulin would

* Dietician and Diabetes Educator at Dr. Chandalia's Diabetes Endocrine Nutrition Management & Research Centre

have a better profile for pump therapy because faster acting insulin has a decreased time to onset of action and a shortened duration of insulin action, which better mimics the insulin release in people without diabetes.

Drug Interactions

Table below includes clinically significant drug interactions with FIASP

Drugs	Interventions
Drugs that may increase the risk of hypoglycemia	
Antidiabetic agents, ACE inhibitors, angiotensin II receptor blocking agents, disopyramide, fibrates, fluoxetine, monoamine oxidase inhibitors, pentoxifylline, pramlintide, salicylates, somatostatin analogs and sulphonamide antibiotics.	Dose reductions and increased frequency of glucose monitoring may be required when FIASP is co-administered with these drugs.
Drugs that may decrease the blood glucose lowering effect of FIASP	
Atypical antipsychotics, corticosteroids, danazol, diuretics, estrogens, glucagon, isoniazid, niacin, oral contraceptives, phenothiazines, progesterones, protease inhibitors, somatropin, sympathomimetic agents and thyroid hormones	Dose increases and increased frequency of glucose monitoring may be required when FIASP is co-administered with these drugs
Drugs that may increase or decrease the blood glucose lowering effects of FIASP	
Alcohol, beta-blockers, clonidine, and lithium salts. Pentamidine may cause hypoglycaemia, which may sometimes be followed by hyperglycemia	Dose adjustment and increased frequency of glucose monitoring may be required when FIASP is co-administered with these drugs.

Fiasp can be a great option for many, but it also may not necessarily be the best option for everyone or every type of meal. “It works really fast”, but for some type of meals, it’s too fast. From a dietary perspective if a person is eating a low carb-diet, Fiasp probably isn’t going to be a great choice for their meal time insulin because the type of meals they are eating will be digested more slowly.

If you think about the way you currently dose insulin for something like pizza, which is high carb but digested slow due to the high fat content, Fiasp could actually lead to some very rough bouts of hypoglycaemia because it’s going to start working so soon. On the other hand, it could lead to high blood sugars during the few hours after eating because Fiasp insulin is out of your blood stream for sooner than other insulin.

1) It is **compatible** with most needles.
2) The pens are **colour-coded** for easy identification
3) It is **easy to use**, with a non-extending dose button that is specially designed to make it easier to inject
4) An **audible ‘click’** sound lets you know that your full insulin dose has been delivered
5) It has an easy-to-read dose display

QUESTION AND ANSWERS

Q. How does anemia affect HbA1c levels?

Haemoglobin A1C is the fraction of haemoglobin which is covalently bound to glucose. It is used as an indicator of patient's glycemic status over the last 3 months. The test is limited to a 3 month average because the lifespan of red blood cells is 120 days.

According to ADA guidelines, HbA1c levels should be maintained below 7% to prevent further complications. Other than blood glucose, the 3 major factors on which HbA1c levels depend are:

- HbA1c in reticulocytes, when released from bone marrow.
- Hb glycation rate as RBCs become older and
- The mean age of RBCs in the circulation

In general, HbA1C changes with a change in erythrokinetics, i.e. a change in the life span of red blood cells. Once a steady state is reached, the HbA1C is not affected significantly.

Other factors that may affect HbA1c levels are: age, smoking, hemolytic anemias, haemoglobinopathies (HbAS, HbAC, HbD and HbE), blood loss, pregnancy and uremia. Folate, Vitamin B12 and iron deficiency has also shown to affect HbA1c levels.

Iron deficiency anemia (IDA) is the most common form of anemia observed in Indians. It becomes imperative to find out the relationship between iron deficiency anemia and HbA1c, since it's a common observation in diabetic patients.

It has been observed that iron deficiency anemia causes longer red blood cell survival, which leads to falsely increased HbA1c levels. In addition, malondialdehyde is increased in iron deficiency anemia and this enhances the glycation of haemoglobin. Combination of these two mechanism results in false increase in HbA1c levels in patients with iron deficiency anemia. Though the exact mechanism of how iron deficiency anemia (IDA) causes increase in HbA1c levels is unclear, it has been suggested that the quaternary structure of the Hb molecule may be altered and that the glycation of the beta-globin chains occurs more readily in patients with IDA.

In contrast to this, conditions such as hemolytic anemia, blood transfusion, increased hemolysis from splenomegaly or blood loss, has been shown to lower HbA1c levels than the actual value, no matter what assay is used to measure. This is due to hemolysis and short survival of red cells.

Similarly, vitamin B12 deficiency, renal failure, and bone marrow suppression in alcoholism inhibit erythropoiesis and increase the mean survival duration of erythrocyte, leading to increase in HbA1c levels. Chronic renal failure is generally associated with shortened survival of HbA1c and hence low HbA1c. However, when talking about CRF, there are other mechanisms that are also in play, which may instead raise the HbA1c levels. These include decreased levels of erythropoietin, increased glycation, higher levels of carbamylated hemoglobin, and variable exposure to higher levels of glucose during dialysis.

Samina Burhanpurwala

RECIPES

RAGI-RAVA DHOKLA

Ingredients:

- Ragi flour 20 gm (1½ tbsp)
- Rava/ Sooji 60 gm (½ cup)
- Low fat curd 50 gm (3 tbsp)
- Oil 10 gm (2 tbsp)

Method:

- For this, first prepare a steamer filled with water and grease the dhokla plate with a teaspoon of oil. Keep this aside.
- Combine ragi flour, rava, curd and salt in a mixing bowl. Add water accordingly to make a thick instant dhokla batter.
- Let the dhokla batter rest for 5 minutes.
- Add Eno or baking powder to the batter. Mix well and pour the ragi dhokla batter into the greased plate.
- Keep the steamer for preheating with water. Place the dhokla plate in the steamer for steaming.
- Steam the dhokla on high heat for about 10 minutes.
- Insert a toothpick or knife in the center to check if it comes out clean.
- Once steamed, remove the dhokla from the steamer and keep aside.
- For seasoning, heat oil in a pan. Add in mustard seeds, sesame seeds, curry leaves and allow it to crackle.
- Turn off the heat and pour the seasoning over ragi dhokla and cut them into squares or diamond.

Nutritional info :

Energy (Kcals)	Protein (gms)	Carbohydrates (gms)	Fats (gms)	GI
380	10	55	12	Medium

Note:

- A healthy preparation of sooji dhokla
- A high protein, high calcium recipe
- Medium GI food suitable for breakfast or snacks for diabetics

PALAK-OATS-RAGI- WHEAT THEPLA

Ingredients

- Wheat flour 50 gm (½ cup)
- Oats flour 10 gm (2 tsp)
- Ragi flour 20 gm (1½ tbsp)
- Spinach leaves 50 gm (finely chopped)
- Curd 45 gm (3 tbsp)
- Oil 1 tbsp

Method:

- To begin making Palak Ragi Oats Wheat Thepla, in a large bowl combine - whole wheat flour, ragi flour, oat flour, flax seed powder, turmeric powder, chilli powder, asafoetida powder and the spinach leaves. Add the yogurt to the flour mixture and knead into a firm smooth dough adding water only if required.
- Finally add two tablespoons of oil to coat the dough and knead until it is firm and smooth. Cover and set aside for 15 minutes. Preheat the iron skillet on medium heat. Divide the ragi oats thepla dough into 15 portions.
- Roll the portions into balls; flatten them with the palm of your hand. Toss the thepla dough on flour and roll them out into thin circles to approximately 6 inches in diameter.
- As you roll them out, you can keep tossing the dough in dry flour to prevent sticking.

Nutritional info :

Energy (Kcals)	Protein (gms)	Carbohydrates (gms)	Fats (gms)	GI
440	11.06	57.35	17.8	Medium

Note:

- A healthy preparation of usual wheat thepla
- Adequate fiber, high protein, high calcium recipe
- Medium GI food suitable for breakfast or snacks for diabetics

HOW KNOWLEDGEABLE ARE YOU?

1. Which one of the drugs causes hypokalemia?
   a) ACE inhibitors
   b) Furosemide
   c) K-sparing diuretics
   d) Beta blockers

2. Which one of the medications requires a dose reduction in the setting of reduced GFR < 45 ml/min per 1.73 m^2?
   a) Linagliptin
   b) Vildagliptin
   c) Tenegliptin
   d) Atorvastatin

3. All are typical infections in diabetes except
   a) Malignant otitis externa
   b) Necrotizing fasciitis
   c) Lobar pneumonia
   d) Renal papillary necrosis

4. In diabetic nephropathy with vitamin D3 deficiency one should use
   a) Vitamin D3
   b) One-aplha D3
   c) Calcium
   d) All of the above

5. Growth is most likely to be affected in children suffering from
   a) Type 1 diabetes
   b) Type 2 diabetes
   c) Pancreatic diabetes
   d) None of the above

6. Normal ejection fraction on a 2 D-echo of heart is
   a) 60%
   b) 80%
   c) 100%
   d) None of the above

7. Which of the following is not a risk factors for progression of nephropathy?
   a) Poor glucose control
   b) Low blood pressure
   c) Higher urinary albumin excretion
   d) Higher triglycerides

8. Female sexual dysfunction can be caused by a range of physical or psychological factors including:
   a) Hormonal imbalance
   b) Diabetes
   c) Medications being taken for other disorders
   d) All of the above

9. A diagnosis of diabetic autonomic neuropathy can be confirmed by:
   a) Abnormal heart rate response to Valsalva 's manoeuvre
   b) Abnormal expired to inspired heart rate ratio
   c) Abnormal blood pressure response to standing
   d) None of the above

10. The ideal anti diabetic drug in a pregnant type 2 diabetic is
    a) Glibenclamide
    b) Metformin
    c) Human insulin short and intermediate acting
    d) Glargine insulin

ANSWERS:

1) d 2) b 3) c
4) b 5) c 6) a
7) b 8) d 9) a
10) c

DIABETES TODAY

Dr. Chandalia's DENMARC in collaboration with Help Defeat Diabetes Trust and Association for Diabetes Care and Prevention (ADCP) presents to you Diabetes Today Magazine

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BOOK REVIEW

RSSDI text book of Diabetes Mellitus; Editor-in-Chief: H B Chandalia, Executive Editor: G R Sridhar, Editors: A K Das, S V Madhu, V Mohan, P V Rao

Jaypee Brothers Medical Publishers; New Delhi; 2014; pages 1457; Price Rs 2995

The third edition of RSSDI Text Book of Diabetes Mellitus (D M) has been published six years after the second edition. It is authored and edited by those clinicians and professors who have been teaching and practising diabetes over many years within the country. A few chapters are contributed by Non-resident Indians. As pointed out by the editor-in-chief, this edition has undergone considerable revision. The material published both within the country and outside till the end of 2013 has been critically analysed and included. A few topics which are paid scant attention in other books, like-the complexity of insulin resistance, the criteria applicable to metabolic syndrome in Asians,challenges in the management of children and elderly with diabetes,musculoskeletal manifestation of diabetes, malnutrition modulated diabetes, Latent Autoimmune Diabetes in Adults (LADA), neonatal diabetes and the role of Yoga and relaxation techniques are unique to this book.

The flow chart on the management of diabetic ketoacidosis available in this book should be in possession of all ICUs. The colour pictures of retinopathy, foot lesions, skin diseases and musculoskeletal manifestation are well presented. The role of alternate therapy is extensively discussed. The guidelines for the beginner to organise a diabetic clinic and optimal health care for diabetes amidst diversity of social, economic and regional food habits is noteworthy. The limitation of stem cell therapy as of now is a good reminder. Some controversial issues are discussed in individual chapters. Much alike the chapter on A Glimpse in the Future, I wish a full chapter was devoted to controversies in diabetes. New chapters added in this edition are valuable and discuss important current issues. These include Sleep and Type 2 diabetes-mellitus, Early-onset Type 2 DM, Nutrient blockers and Bromocriptine, Insulin Pump Therapy, Glycemic Management in Hospitalized Patients, Continuous Glucose Monitoring System, Vitamin D and DM, HIV in Diabetes, Diabetes and Cancer.

The appendix is retained from the previous edition and gives a wealth of information applicable to Indian subjects like BMI and waist circumference and laboratory values in S I and conventional units.The index has attained perfection.The novel feature of this edition is mentioning the chapter number on the right edge of each page.

The book will prove to be valuable to students, physicians, diabetologists, endocrinologists and providers of diabetes care. It should be on the shelf of every medical library. The availability of this book has made the Western text books redundant. The single volume covering so many topics is bulky and heavy. I wish it was brought out in two volumes.

C. Munichoodappa. F.R.C.P.C.
Diplomate, American Board in Internal Medicine
Bangalore
Email id: dr.munichoodappa@gmail.com

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CHALLENGES IN DIABETES EDUCATION

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- **Lower risk of hypoglycaemia*** including during the titration phase, in people with T2DM[1]
- The advantage of **dosing flexibility** (±3 hours)when needed[2,3]

* Confirmed ≤3.9 mmol/L (≤70 mg/dL) or severe hypoglycaemic events (24 hours). ^ Better glycaemic control and less hypoglycaemia with insulin glargine 300 U/mL vs glargine 100 U/Ml. Ritzel R et al. Diabetes Obes Metab. 2017 Sep 1. (Epub ahead of print) 1. Bolli GB, et al. Diabetes Obes and Metab. 2015;17(4):386–394. 2. Becker RH, et al. Diabetes Care. 2015;38:637-643. 3. Toujeo™ prescribing information 4. Strong J et al. Curr Med Res Opin.2017 Apr;33(4):785-793

INSULIN GLARGINE INJECTION
TOUJEO™ Solostar® **Abridged Prescribing Information**
COMPOSITION: Insulin glargine 300 U/ml. 1 ml contains 10.91 mg insulin glargine I.P., corresponding to 300 U of insulin glargine. **INDICATION:** For the treatment of diabetes mellitus in adults. **DOSAGE AND ADMINISTRATION:** Toujeo™ is given subcutaneously.Toujeo™ is administered once daily, at any time during the day, preferably at the same time every day. The recommended daily starting dose is 0.2 U/kg once daily followed by individual dosage adjustments. When needed, patients can administer their injections up to 3 hours before or after their usual time of administration. The desired blood glucose levels as well as the doses and timing of anti-hyperglycaemic medications must be determined and adjusted individually. Toujeo™ is not the insulin of choice for the treatment of diabetic ketoacidosis. Changing from once-daily basal insulin products to once-daily Toujeo™ can be done unit-to-unit based on the previous basal insulin dose. Changing from twice-daily basal insulin products to once-daily Toujeo™ , the recommended initial Toujeo™ dose is 80% of the total daily dose of the basal insulin that is being discontinued. Toujeo™ must not be mixed with any other insulin products. Toujeo™ must not be diluted. The safety and effectiveness of Toujeo™ has not been established in paediatric patients (under 18 years of age). Toujeo™ can be used in elderly patients, in patients with renal impairment and in patients with hepatic impairment. Close glucose monitoring is recommended. **SAFETY-RELATED INFORMATION Contraindications:**Toujeo™ must not be used in patients hypersensitive to insulin glargine or any of the excipients. **Warnings:** No Core Safety Information **Precautions: General** :Insulin treatment generally requires appropriate diabetes self-management skills including glucose monitoring, proper injection technique and hypo and hyperglycaemia management. Patients and their relatives must know what steps to take if hyperglycaemia or hypoglycaemia occurs or is suspected, and they must know when to inform a physician. Hypoglycemia: The time of occurrence of hypoglycaemia depends on the action profile of the insulins used and may, therefore, change when the treatment regimen is changed. As with all insulins, particular caution should be exercised, and intensified blood glucose monitoring is advisable, in patients in whom sequelae of hypoglycaemic episodes might be of particular clinical relevance. The prolonged effect of subcutaneous Toujeo™ may delay recovery from hypoglycaemia. In patients with renal impairment or severe hepatic impairment, insulin requirements may be diminished. In the elderly, progressive deterioration of renal function may lead to a steady decrease in insulin requirements. Hypoglycaemia can generally be corrected by immediate carbohydrate intake. So that initial corrective action can be taken immediately, patients must carry a minimum of 20 grams of carbohydrates with them at all times. Intercurrent illness: Requires intensified metabolic monitoring. In many cases urine tests for ketones are indicated, and often it is necessary to adjust the insulin dose. **Medication errors prevention:** Insulin label must always be checked before each injection to avoid medication errors between Toujeo™ and other insulins. The patients must also be instructed to never use a syringe to remove Toujeo™ from the SoloStar pre-filled pen into a syringe and not to re-use the needles. **Pregnancy & Lactation:** It is essential for patients with pre-existing or gestational diabetes to maintain good metabolic control throughout pregnancy to prevent adverse outcomes associated with hyperglycaemia. Toujeo can be used during pregnancy, if clinically needed. Insulin requirements may decrease during the first trimester and generally increase during the second and third trimesters. Immediately after delivery, insulin requirements decline rapidly. Careful monitoring of glucose control, is essential in such patients. Patients with diabetes must inform their doctor if they are pregnant or are contemplating pregnancy. **Adverse Reactions:** Hypoglycaemia is most frequent and may occur if the insulin dose is too high in relation to the insulin requirement. A marked change in glycaemic control may cause temporary visual impairment. Lipodystrophy may occur at the injection site. Allergic reactions at the injection site includes redness, pain, itching, hives, swelling or inflammation. Immediate type allergic reactions are rare
For full prescribing information please write to Sanofi India Ltd., Sanofi House, CT Survey No 117-B, L&T Business Park, Saki Vihar Road, Powai, Mumbai 400072
Dated: June 2017 **Source:** CCDS Version 1.1 dated June 2016

For the use of a registered medical practitioner or a hospital or a laboratory only. SAN.GLA.17.12.1565. 04/18

SANOFI

My **NEW** Diabetes Therapy
helps me Control HbA1c & lose weight

NOVEL ß-CELL INDEPENDENT MOA[1]

UNSURPASSED EFFICACY
Compared to Glimepiride and Sitagliptin,
INVOKANA® 100mg is non-inferior[2,3]
INVOKANA® 300mg is superior[2,3]

SUSTAINED & SIGNIFICANT WEIGHT LOSS[3,4]

HYPOGLYCEMIA COMPARABLE TO PLACEBO[5]

Invokana®
canagliflozin tablets

A CLASS APART

References:

1. INVOKANA® India Prescribing Information (January 2014) **2.** Lavalle-González FJ et al. Diabetologia. 2013;56(12):2582-92 **3.** Cefalu WT et al. Lancet 2013;382(9896):941-50 **4.** Leiter LA et al. Diabetes Care. 2014. **5.** Stenlöf K et al. Diabetes Obes Metab. 2013;15(4):372-82.

For the use of a Registered Medical Practitioner or a Hospital or Laboratory Canagliflozin tablets 100mg / 300mg

INVOKANA®

Composition and Strength: Canagliflozin 100 mg / 300mg. Each 100 mg tablet contains 102 mg Canagliflozin hemihydrate, equivalent to 100 mg Canagliflozin. Each 300 mg tablet contains 306 mg Canagliflozin hemihydrate, equivalent to 300 mg of Canagliflozin. **Pharmaceutical form:** 100 mg - The tablet is yellow, capsule-shaped, immediate-release and film-coated, with "CFZ" on one side and "100" on the other side. 300 mg - The tablet is white, capsule-shaped, immediate-release and film-coated, with "CFZ" on one side and "300" on the other side. **Therapeutic Indications:** INVOKANA® is indicated as an adjunct to diet and exercises to improve glycemic control in adults with type 2 diabetes mellitus as monotherapy and combination therapy. **Dosage and Administration:** The recommended starting dose for adult > 18 years is 100 mg or 300 mg once daily orally preferably before the first meal of the day. A starting dose of 100 mg once daily should be used in patients on loop diuretics and patients ≥ 75 years of age. In patients with an eGFR 45 mL/min/1.73 m2 to < 60 mL/min/1.73 m2, the dose of INVOKANA® is limited to 100 mg once daily. The 300 mg dose may be considered for patients with an eGFR > 60 mL/min/1.73 m2, who need tighter glycemic control and who have a low risk of adverse reactions associated with reduced intravascular volume with INVOKANA® treatment. **Contraindications:** Hypersensitivity to the active substance or to any of the excipients. **Warnings and Precautions:** INVOKANA® has not been studied in pediatric patients (< 18 years), patients with type 1 diabetes and is therefore not recommended for use. INVOKANA® should not be used for the treatment of diabetic ketoacidosis or in patients with an eGFR < 45 mL/min/1.73 m2 [CrCl < 45 mL/min], as it would not be effective in these settings. In patients with evidence of reduced intravascular volume, correcting this condition prior to initiation of INVOKANA® is recommended. **Drug Interactions:** The metabolism of INVOKANA® is primarily via glucuronide conjugation mediated by UDP glucuronosyl transferase 1A9 (UGT1A9) and 2B4. If a combined inducer of these UGTs and drug transport systems (e.g., rifampicin, phenytoin, barbituates, phenobarbitol, ritonavir, carbamazepine, efavirenz) must be co-administered with INVOKANA®, monitor HbA1c in patients receiving INVOKANA® 100 mg once daily with consideration to increasing the dose to 300 mg once daily if additional glycemic control is needed. INVOKANA® neither inhibits cytochrome P450 CYP1A2, CYP2A6, CYP2C19, CYP2D6, or CYP2E1, CYP2B6, CYP2C8, CYP2C9, nor induces CYP1A2, CYP2C19, CYP2B6, CYP3A4 at higher than therapeutic concentrations. INVOKANA® is a P-glycoprotein (P-gp) substrate, and inhibits P-glycoprotein mediated transport of digoxin with low potency. Patients taking digoxin or other cardiac glycosides (e.g., digitoxin) should be monitored appropriately. **Pregnancy, Breast-feeding and Fertility:** There are no adequate and well-controlled studies in pregnant women. INVOKANA® should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus. It is not known if INVOKANA® is excreted in human milk. A risk to the breast-fed child cannot be excluded. The effect of INVOKANA® on fertility in humans has not been studied. **Adverse reactions:** In clinical studies of INVOKANA® the most commonly reported adverse reactions during treatment (≥ 5%) were vulvovaginal candidiasis, urinary tract infection, and polyuria or pollakiuria. Other adverse reactions in clinical studies of INVOKANA® that occurred at a rate < 2% in placebo-controlled studies were adverse reactions related to reduced intravascular volume (postural dizziness, orthostatic hypotension, hypotension, dehydration, and syncope), skin rash, and urticaria. In the event of an overdose, it is reasonable to employ the usual supportive measures, including monitoring of vital signs and observation of clinical conditions. **Overdose:** Single doses up to 1600 mg of INVOKANA® in healthy subjects and INVOKANA® 300 mg twice daily for 12 weeks in patients with type 2 diabetes were generally well-tolerated. In the event of an overdose, it is reasonable to employ the usual supportive measures, e.g., remove unabsorbed material from the gastrointestinal tract, employ clinical monitoring, and institute supportive treatment as dictated by the patient's clinical status. Canagliflozin was negligibly removed during a 4-hour hemodialysis session. Canagliflozin is not expected to be dialyzable by peritoneal dialysis.
Storage: Store below 30°C and in dry place. Protect from light. Keep out of reach of children.

Warning: To be sold by retail on the prescription of Registered Medical Practitioner only. Version: CCDS 09 Jan 2014
For complete prescribing information, please contact: Johnson & Johnson Private Limited, Arena Space, Behind Majas Depot, Off J.V. Link Road, Jogeshwari (E), Mumbai 400060

INV/33/LBL/0515/Q2.15-Q4.15

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