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To Dispel Darkness Of Diabetes

DIET MANAGEMENT

EXERCISE

MEDICATION

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CONTENTS

HYPERURICEMIA

*** Niti Desai**

Hyperuricemia has recently emerged as an independent risk factor in the development of type 2 diabetes mellitus and hypertension through several proposed mechanisms. Elevated serum uric acid has also been reported to predict development of nephropathy in type 1 diabetes. We are seeing increasing number of patients with elevated uric acid levels in our metro cities. It is one more of the `lifestyle disease' which may soon reach alarming numbers.

Hyperuricemia is a common biochemical aberration in which serum (or plasma) uric acid concentration exceeds the limit of solubility of urate (about 6.8mg/dL).Uric acid homeostasis is determined by the balance between production and renal excretion.

Hyperuricemia has been found to be associated with insulin resistance. Latest research suggests that elevated uric acid, by itself, is associated with higher rates of metabolic syndrome, diabetes and hypertension. It would therefore be advisable to get uric acid levels done during health check-ups and if elevated, control the same through diet and/or medication.

How does one get Uric acid in the body? Uric acid is a waste product of purine metabolism. Purines occur naturally in our bodies and they are also found in protein-rich foods. Endogenous production of uric acid from degradation of purines accounts for two-third of the body urate pool.The remainder is of dietary origin. Generally uric acid is eliminated from the body through urine.

A diet rich in purines from certain sources can raise uric acid levels.The principle would therefore be to reduce the intake of purine rich foods so that the body does not produce more uric acid. Generally, treatment focuses on eliminating all foods that are rich in purines. The list of foods to avoid becomes so long making the diet difficult to follow. However, recent research has given us a clearer picture of the role of diet in the treatment of hyperuricemia. It is more important to follow healthy eating guidelines, rather than just eliminating certain foods.

Some foods should be avoided, but not all foods with purines need to be eliminated, and in fact some foods should be included in the diet to control uric acid levels.

The most important dietary changes for the treatment of hyperuricemia would be

- To **lose weight** if one is overweight or obese. This is perhaps the single most important factor.
- Being overweight increases the risk of developing hyperuricemia and vice versa. Losing weight alone can reduce blood uric acid levels. It is important to avoid any type of crash dieting, as going without food for long periods and rapid loss of weight can increase uric acid levels.3
- **Fructose intake:** Consumption of fructose is known to raise the serum urate levels. Fruit and fruit juices may also increase the risk of gout. Eating an apple or orange a day, increases the risk by approximately 64%.

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Consumption of soft drinks, juices, candy and pastries will only add to the fructose load and therefore should be avoided more assiduously than restricting the protein intake.

- **Restrict fat intake:** Limit the intake of high fat foods such as – fried foods, namkeen, sweets, mithais, cakes, pastries, red meat and full fat dairy products.

What about protein intake?

Only hardcore nonvegetarians need to worry about limiting protein intake. In our country most nonvegetarians have non-vegetarian food few times a week.

Organ meats: One needs to avoid organ meats such as liver, kidney, brain, meat extracts and gravies which have high purine levels.

Select seafood: Avoid some types of seafood, which are very high in purines, e.g. herring, sardines, mackerel, tuna and shellfish such as mussels, crabs, shrimps, fish roe and caviar.

- Restrict nonvegetarian food to 100 -150 gms of fish and chicken daily.
- Avoid yeast extracts.
- Add protein to your diet with low-fat / fat-free dairy products, such as skimmed milk, skimmed milk curd and paneer. Low fat dairy products are in fact associated with reduced uric acid levels.

What about dals and pulses?

There is an increased risk associated with a higher consumption red meat (beef, pork and lamb) and seafood but NOT with consumption of vegetable protein. Diets high in purine rich vegetables (beans, pulses, lentils) do not increase the risk of gout.4One does not need to eliminate dals and pulses from the diet. Most of the time, our vegetarian diet is not meeting the daily protein requirements, so if one completely eliminates these foods from the diet, there will be even higher carbohydrate intake, increased insulin resistance, elevated blood sugar levels, increased serum triglycerides and weight loss becomes increasingly difficult, without lowering of uric acid levels. Dals and pulses can be included in moderate amounts in the daily diet.

- **Eat more fruits and vegetables:** Studies have shown that vegetables high in purines do not increase the risk of hyperuricemia. A healthy diet based on lots of fruits and vegetables can include high-purine vegetables such as spinach, peas, cauliflower or mushrooms. There is some evidence that eating cherries, celery and leeks is associated with a reduced risk of gout(symptomatic hyperuricemia) attacks.

  Some people find that certain foods such as strawberries, oranges, tomatoes and nuts trigger their gout even though they are not high in purines. Although there is no clear scientific evidence for this, it is probably best to avoid them if the patient has had this experience.

- Do not to skip meals or keep long intervals between meals.
- **Alcohol:** Alcohol can raise the level of uric acid in the blood in a number of ways. Alcohol stimulates the production of uric acid by liver. Alcohol is converted to lactic acid which interferes with the removal of uric acid by the kidneys. Alcohol consumption, particularly beer increases risk as beers contain large quantity of purines. Gout is two and half times more frequent in men

who drink two bottles of beer a day while two glasses of wine was not associated with increased risk. The effect of wine is not as well-understood. There is also a risk between binge drinking and gout attacks. Therefore alcohol should be completely avoided or at least reduced. Drinking alcohol can increase the risk of developing gout and can bring on a sudden attack in a gout sufferer.

- **Drink more water:** Increase fluid intake to 2-3litres, especially water intake. Drinking fluids flushes out uric acid from the body and prevents its deposition.

Making the above dietary changes will give one better results than just following the list of foods to avoid.

Foods to limit (very high in purines):

- Organ meats, such as liver, kidneys, sweetbreads, and brains
- Meats, including bacon, beef, pork, and lamb
- Any other meat in large amounts
- Rawas, sardines, mackerel and shellfish
- Meat extracts and broths
- Beer
- Yeast(baker's / brewer's)

Foods to eat occasionally (moderately high in purines, but may not raise your risk of gout):

- Fish and seafood (other than high purine seafood)
- Chicken
- Oatmeal and wheatbran
- Peas, brinjal
- Spinach, mushrooms
- Cauliflower
- Chickoo, custard apple

Foods that is safe to eat (low in purines):

- Green vegetables and tomatoes
- Fruits
- Breads and cereals
- Butter, cheese and eggs
- Chocolate and cocoa
- Coffee, tea, and aerated drinks
- Egg
- Nuts

Dairy products that may lower the risk of gout:

- skimmed milk
- curd
- paneer

Hyperuricemia should be managed with lifestyle modification, however if it is symptomatic, if the patient has had more than two attacks of gout a year or if the condition has been present long enough, then there may be a clear need for pharmacotherapy.

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SARCOPENIA IN DIABETES

***Ishitaa Bhatia**

A fairly new concept, sarcopenia is defined as *"generalized loss of muscle mass, muscle strength and muscle function and it is directly related to adverse outcomes in the population."* (Cederholm, 2017). Earlier, specifically defined for the aged population, recent studies also show the prevalence of sarcopenia in various disease conditions such as diabetes, liver cirrhosis and chronic kidney disease.

There are two components that define sarcopenia - the muscle mass and muscle function.

The most cost effective, simple and non-invasive way of assessing muscle mass is by estimating mid-arm muscle circumference (MAMC). It has been shown to be a surrogate marker for assessing muscle mass. To measure MAMC, the mid arm circumference (MAC) and the triceps skinfold (TSF) need to be measured. Using a measuring tape, the mid-point of the upper arm and MAC is determined. The MAC is estimated to be halfway between the olecranon process (bony process on elbow) and acromion process (bony process on top of the shoulder). Once the site is selected and marked with water-soluble ink, the examiner grasps the triceps skinfold between his/her thumb and index finger while the calliper is simultaneously placed perpendicular to the skinfold. The reading on the dial is then recorded. Thereafter, using the following equation the MAMC is calculated: MAMC= MAC- (TSF X 0.3142).

A majority of the definitions by various working groups include muscle mass index values established by dual-energy x-ray absorptiometry (DEXA). However, Cruz-Jentoft and colleagues argue that bioelectrical impedance analysis (BIA) could be a practical and portable alternative to DEXA. The BIA procedure is simple, short and inexpensive. One of the main advantages is that it can be used for bedridden and ambulatory patients. It measures the lean and fat mass in the patient.

The functional component of sarcopenia is measured by grip strength and gait speed test. Cruz-Jentoft and colleagues from the European Working Group on Sarcopenia in Older People define the cut off for sarcopenia <0.8m/s gait speed for men and women; <30 kgs and <20 kgs grip strength for men and women respectively.

Sarcopenia can also be assessed by a validated questionnaire called the SARC-F questionnaire. It measures functional activities such as, Strength, Assistance to walk, Rise from a chair, Climb stairs and Falls (SARC-F).

The scores range from 0-10, with a score of 4 or more being an indicator for sarcopenia.

SIGNIFICANCE AND PREVALENCE OF SARCOPENIA IN DIABETES

A review published in 2016 states that risk of mortality is greater in sarcopenic group versus non-sarcopenic group in the healthy population. Further, sarcopenia is a marker of frailty. Frailty can be defined as "vulnerability to adverse

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health outcomes" (Walston, 2015). Sarcopenia also reduces quality of life (QoL) as it reduces functional capacity. A review paper also found that sarcopenia and frailty leads to a decline in QoL.

It has been established that diabetic patients who are 65 years or older are more likely to be frail than their non-diabetic counterparts. The prevalence of frailty is 32-48% in diabetics who are 65 years or elder as compared to 5-10% prevalence in non-diabetic patients of the same age group. Leander and colleagues demonstrated that older diabetic patients had a reduced muscle mass, functional capacity and leg muscle strength than non-diabetic patients. Impaired muscular strength in older diabetics has been linked to intramuscular storage of fat - which is three times higher than in non-diabetics. A Korean study showed that sarcopenia was prevalent in 15.7% of the diabetics versus 6.9% of the control group.

PATHOPHYSIOLOGY: SARCOPENIA AND DIABETES

Skeletal muscle insulin resistance is the key link between sarcopenia and diabetes. Due to the reduced glucose availability in the muscle, gluconeogenesis takes place. This results in a catabolic effect i.e. the breaking down of muscle protein to provide a carbon skeleton for energy production. Hyperglycemia also leads to release of free fatty acids in the plasma. The increase in plasma free fatty acids encourages the production of pro-inflammatory cytokines- a common feature in diabetes. It is hypothesized that intramuscular inflammation can lead to proteolytic processes in the skeletal muscle. Further, lowered mitochondrial oxidative capacity leads to reduce fat oxidation activity. Thus muscle mitochondrial dysfunction causes accumulation of intramyocellular lipid metabolites producing marbling. Visser and colleagues point out that marbling in turn, results in lowered muscle quality and work performance.

Insulin also plays a role in protein anabolism. Insulin and amino acids have a synergistic effect on eliciting protein synthesis. Insulin can initiate protein synthesis only in the presence of amino acids intramuscularly, i.e. in a well-fed state (high insulin and amino acids). It has been shown that insulin deficiency leads to break down of protein, resulting in reduced muscle mass. Thus there is a clear correlation between insulin, protein intake and muscle anabolism.

Myostatin is a member of the growth factor family. It is an inhibitor of skeletal muscle growth. It is observed that type 2 diabetic patients have an elevated expression of muscular myostatin mRNA. Studies showed that the upregulation of myostatin was reduced by insulin. Mice with induced diabetes showed an increased expression of myostatin mRNA and its receptor. These findings establish the role of myostatin mediated muscle wasting in diabetics.

Anabolic hormones such as testosterone is shown to be lower in diabetic men. Reduced testosterone leads to a further decline in the synthesis of muscle protein and satellite cell activation.

Lastly, advanced glycation end products (AGEs) are markers of long-term glucose activity. In diabetics, the muscle accumulates AGEs. Along with being associated with lowered grip strength and gait speed, AGEs also increase the presence of Reactive Oxygen Species (ROS).

TREATMENT

The key treatment for sarcopenia is physical activity. Since resistance exercise improves muscle mass and endurance exercise increases functional capacity - a combination of both is recommended to improve sarcopenia. Exercise improves skeletal muscle synthesis by increasing capillary blood flow, nutrient delivery to the muscle and insulin sensitivity in diabetic patients. However, exercise regimes must be designed keeping in mind the diabetic patient's micro and macrovascular complications.

Sarcopenia has shown to increase mortality and reduced quality of life. Thus in addition to dietary screening it should be imperative to also check for muscle mass and function in a clinical set up. Detecting sarcopenia at an early stage has the potential to positively impact patient's daily activities and quality of life.

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TYPE 1 DIABETES AND ISLET CELL TRANSPLANTATION

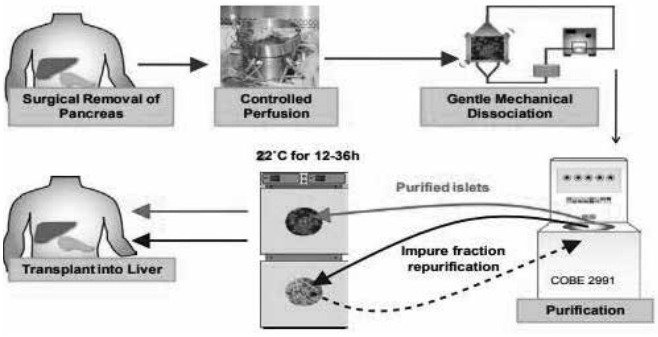
***Samina Burhanpurwala**

Pancreas, an exocrine gland, is a fish shaped organ. It is about the size of a hand, located behind the stomach. There are areas or clusters of cells called islets of Langerhans within the pancreas, which is predominantly occupied by beta cells. Beta cells are the one that secrete insulin and degeneration of these cells lead to type 1 diabetes. Normal subjects have around 1 million islet cells, of which 70% are beta cells.

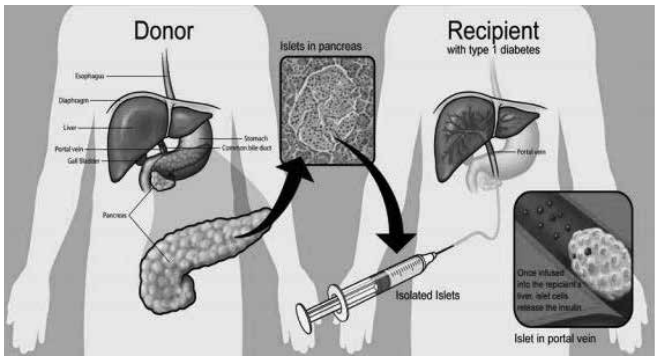
ISLET CELL TRANSPLANTATION

Till date, insulin therapy, given by a pen or pump, is the best treatment any type 1 diabetic can get. But some people with type 1 diabetes have what doctors call as brittle diabetes, with too many fluctuations in their blood sugar levels. This leads to complications such as diabetic retinopathy, nephropathy and recurrent hypoglycemia. For such people, islet transplantation can prove to be a life-saving therapy. Transplant experiments initially conducted in mice in around 1970s were highly successful. However, initial trials in human showed dissatisfactory results. Transplanted islets had failed after few months of transplantation in recipients. After a gap of few years, Edmonton protocol was followed for implantation of islets in type 1 diabetics. However, with speedy advances in transplant technology, researchers have come with modified version in the Edmonton protocol to treat type 1 diabetes.

Islet cell transplantation is an experimental treatment and was first performed by University of Chicago Medicine. Since it is an experimental procedure, it can only be performed as part of U.S FDA-permitted clinical trial or permission of appropriate regulatory bodies in India. Islet cell transplantation is used to treat type 1 diabetes, whereby the doctors take the islets from the healthy cells of pancreas of a deceased organ donor. Islets from a deceased person are removed using mixture of enzymes called liberase (blend of highly purified enzymes which improve quality and reproducibility of tissue dissociation). They are then purified, processed and then transferred by injecting it into the portal vein that carries blood to the liver.



Purification of Islet cells



Islet Transplant procedure

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

With the help of a percutaneous transhepatic portal vein catheter, islets are infused into the liver. Portal pressure is monitored throughout the procedure. Since the islets are fragile, transplantation occurs soon after the islets are removed from deceased donor.

To be insulin independent, the recipients usually have to undergo 2 transplants. Any patient undergoing transplant typically needs 10000 islet "equivalent" per kg body weight (IE/ kg) per infusion from 2 deceased donors. After transplantation, beta cells begin to make and release insulin. Till the time islets are fully functional, recipients are kept on insulin and a strict blood sugar monitoring is done. To make sure that the immune system doesn't reject the transplanted islets, recipients are kept on 2 immunosuppressants- sirolimus (rapamycin) and tacrolimus (fujimycin/ FK506). They are also kept on a drug called daclizumab (a monoclonal antibody made by identical immune cells that bind to a part of antigen recognized by the antibody).

DONOR SELECTION

There are several parameters for donor selection such as donor age, BMI. These parameters are strongly associated with successful transplant. It is generally observed that higher the BMI, higher is the yield of islets. There are certain compatibility tests to be carried out for any successful transplantation.

1. **Blood type:**

   It is of foremost important that recipient's blood type should match with the donor's. Occasionally, if a recipient's blood group doesn't match with donor's, a special blood "washing" technique can be used to match the transplant with its donor.

2. **Human Leukocyte antigen:**

   HLA system or complex is major histocompatibility complex protein found on surface of all cells. They are responsible for regulation of our immune system. In this blood test, donor's antigens are matched with the recipients'. HLA is considered to be genetic marker, inherited from the parents. Hence, first degree relatives are the best match.

3. **Crossmatch:**

   In this blood test, a sample of donor's blood is mixed the intended recipient's and checked for compatibility. A positive crossmatch means the recipient has developed antibodies or immune response to donor's blood or tissue. So the immune system will attack and destroy the organ from that of donor's.

CRITERIA FOR TRANSPLANT

For patients undergoing Islet Transplant Alone (ITA)

- Type 1 diabetes for more than 5 years with a negative or negligible C-peptide
- Retinopathy
- Age > 20 years
- Complicated diabetes with poor glycemic control and elevated HbA1c
- Severe hypoglycemic unawareness (severe drop in blood sugar levels to < 54 mg/dl where person can't recognize hypoglycemic symptoms)

For patients undergoing Islet after Kidney Transplant (IAK)

- Type 1 diabetes between the age of 18- 60 years

- Hypoglycemic unawareness
- One year post renal transplant, with no graft rejection
- Tolerating maintenance immunosuppression
- On prednisone 5mg/day

BENEFITS

- Improve blood sugar levels
- Reduced or no need for insulin for at least 5 years post transplant: Restoration of beta cell function with improved insulin secretion, in response to IV glucose is observed. As well as, a positive C-peptide or increase in C-peptide in response to oral glucose is seen.
- Reduced risk of hypoglycemia: Since the main goal of islet transplantation is to be insulin independent, the most phenomenal effect of transplantation is suspension of hypoglycemia.

RISK

- ***Rejection***

  It is one of the biggest issues after any transplant. There is no way as of now to monitor if there's rejection post islet transplant. If there is any rejection, it's usually recognized too late to interpose any changes. The usual signs are high blood sugar level, which explains why frequent and close monitoring of blood sugar is an integral part of post transplant care. Hence, large doses of immuno suppressants are given to stop the immune system from rejecting the transplanted organ.

- ***Infection***

  Recipients will be on immuno suppresants, a possible side effect of which is infection. Some of the common infections are Candida, Herpes simplex virus infection, Pneumocystis carinii pneumonia. Taking immunosuppressant can also increase risk of cancer.

CLINICAL TRIALS

- From year 1999-2013, 1011 patients underwent islet transplantation. Out of 1011, 819 had islet transplantation alone (ITA) and 192 had islet after kidney transplant (IAK). These transplants were performed at research centers in Europe, Australia, Asia and North America. It was observed that approximately 30 percent of patients with type 1 diabetes who underwent islet transplantation alone and 20 percent islet after kidney were insulin independent at five years.
- In phase 3 clinical trials, conducted by National Institute of Health, which included type 1 diabetics with poor glycemic control, hypoglycemia unawareness and severe hypoglycemia. It was observed that 1 year post islet transplantation, approx 9 out of 10 recipients had an HbA1C level below 7 percent with no episodes of severe hypoglycemia. Following year, 7 out of 10 recipients had an A1C below 7 percent and 4 out of 10 were insulin independent.
- A few studies also showed that there was restoration of insulin production in the body with a good blood glucose control. However, they were not completely free of insulin.

NUTRITION THERAPY POST TRANSPLANT

For successful recovery from any transplant, nutrition plays a key role. Post surgery, adequate

intake of calories and protein should be there to support wound healing. As for the patient will be on anti-rejection medication including corticosteroids, a strict control of diet has to be maintained.

Protein:

To recover from the stress post surgery, enough protein has to be part of diet. Also if the patients are on prednisone, which causes hypercatabolism, their protein requirements are increased. About half of protein should comprise first class protein foods such as low fat dairy products, egg whites, and fish (twice a week).

Carbohydrates:

Some medications such as prednisone can decrease body's ability to utilize blood sugars. This can cause hyperglycemia. Also, the patients will be on insulin for some period of time till the islets are fully functional. This explains why the diet should focus on more of complex carbohydrates and low in simple sugars.

Potassium:

Patients on Calcineurin inhibitors such as Tacrolimus can raise postassium levels. Hence, fruits and vegetables containing low potassium are to be advised.

Salt:

Patients are on corticosteroids post transplant which leads to sodium and water retention, thereby increasing BP. In order to avoid water retention, a low-salt diet has to be advised. Depending on the need of the patients, salt intake of up to 5gm can be allowed.

In a country like India or any other developing countries, the biggest hurdle for islet transplantation will be the lack of number of donated pancreas/islets and the cost for the procedure. The cost of islet transplantation alone in India is about 18,000-30,000 USD and pancreas after kidney (PAK) or simultaneous pancreas-kidney transplant (SPK) is about 30,000-70,000 USD.

However, islet transplant is not the end of treatment. Even if the patients are insulin free, they are on immunosuppressant which suppresses immunity. Immune suppression might increase risk of infection or cancer. With limited organs available, research is being done to find out new sources for islet cell isolation. Researchers are also trying to cultivate human islet cells in laboratory, while some are trying to develop cell lines that will produce insulin. For the time being, we will have to rely on donated organ.

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QUESTION AND ANSWERS

Q. In what ways can diabetics manage nocturnal hypoglycemia?

Nocturnal hypoglycemia also called as night time hypoglycemia is very common in patients treated with insulin. It is one of the main concerns in type 1 diabetics. Type 2 diabetics that are treated with long acting sulfonylureas or insulin combined with OHAs, are also susceptible to nocturnal hypoglycemia. A person will only realize about sugars going low at night, if he wakes up from a hypoglycemic episode with symptoms such as morning headache, tiredness, restlessness during sleep or bed sheets and clothes soaked in sweat. The feeling of damp and sweaty skin is one of a clear indication of night time hypoglycemia.

Following these symptoms, it is advisable to check the blood sugar levels between 2am-3am, which may provide a confirmation. An abnormally low blood sugar level of < 68 mg/dl occurring at night time is defined as nocturnal hypoglycemia.

Nocturnal hypoglycemia is more daunting than daytime hypoglycemia. During daytime the body provides warning against hypoglycemia by sympatho-adrenal responses which causes profuse sweating, shivering and rapid heartbeat. These responses are suppressed during sleep and therefore it is unlikely that the patient will wake up during the episode and have a snack. Undetected nocturnal hypoglycemia can lead to hypoglycemia unawareness, anxiety and hence poor quality of life. Severe nocturnal hypoglycemia can cause seizures, coma and also neuronal cell death, requiring emergency care.

Hypoglycemia is usually caused by an imbalance between factors that tend to increase blood sugars (food, stress) and factors that lower blood sugar (insulin, exercise and few OHAs) levels. Nocturnal hypoglycemia most commonly occurs due to:

- Use of NPH insulin in evening
- Very high dose of basal insulin
- Drinking alcohol in evening
- Exercising too close to a bedtime
- Skipping dinner
- Slow clearance of OHAs due to hepato-renal disease
- Falling sick after an increase in basal insulin dose
- Increasing rapid acting insulin dose to cover bedtime snacks
- Increasing rapid acting insulin to correct high blood sugars at bedtime

Treatment for nighttime lows is same as general advice given for hypoglycemia i.e. to take a quick acting 10-15 grams carbohydrate rich food. For example:

- 1 tbsp honey/ sugar
- 3-4 glucose tablets
- 1/2 cup of any fruit
- 1/2 cup of any regular soft drink / fruit juice
- 5-6 pieces of hard candies (mango bite)

This is followed by some wholesome snack to maintain the blood sugar level.

One of the best preventive measures is to check blood glucose levels before going to bed. Other ways to ward off the night time hypo are:

- If there is any doubt, check the sugars at 2 am. In young children especially, the parents may want to wake up and check blood sugars for a few nights.

- If the patient is on NPH at dinner time, it can be switched to peakless basal insulin like glargine or degludec. If NPH has to be continued, a bedtime snack has to be introduced with complex carbohydrates for slow and steady absorption of glucose.
- Basal insulin dosage can be reduced if blood sugar drops more than 2 mmol/l in the period from bedtime to waking up.
- If having alcohol in evening or in dinner, consider having a snack at bedtime or lower the dose of basal insulin, since alcohol suppresses hepatic gluconeogenesis.
- If doing exhaustive exercise close to bedtime, consider having a snack or reduce the dose of basal insulin.
- Do not miss out on evening snack or dinner regardless of what the sugar levels are.
- Long acting OHAs can be replaced with short acting ones.
- Real-time blood sugars can also be monitored with the help of continuous glucose monitoring system (CGMS). It senses the glucose present in the interstitial fluid and thereby warns a patient if going into hypo or hyperglycemia.
- In severe episodes, if the patient is asleep and cannot be awakened, subcutaneous administration of glucagon (an anti-insulin hormone that stimulates hepatic gluconeogenesis and glycolysis) can be given. It will elevate the blood sugars rapidly if the glycogen stores are not depleted. But a family member of the patient has to be trained for this.

SAMINA BURHANPURWALA

Q. How to optimize glycemic control in diabetic on enteral feeding?

Enteral feeding in diabetes and corresponding glucose control has been a matter of debate and research for a long time. Providing the required amount of nutrients while still maintaining appropriate glycemic control is important in situations demanding enteral feeding for short as well as long duration. Enteral feeding is usually covered with insulin therapy and not oral hypoglycemic agents. This is because crushing tablets and giving them via the feeding tube is not recommended. It is easier to manage hyperglycemia on enteral feeds rather than parenteral feeds as feeding via the gut provides the incretin effect thus aiding glycemic control.

Circumstances of enteral feeding: Patients may require enteral feeding for short-term or long-term. Examples of circumstance where enteral feeding may be required transitionally for a short period are post-gastrectomy, partial glossectomy, intensive care, inadequate oral intake, burns. Conditions like mechanical obstruction, pancreatitis, gastroparesis, post-vagotomy, CVA or neurological impairment like Parkinson's Disease require more long-standing enteral nutrition support.

Feeding schedule: Bolus feeding pattern is most widely used and advocated as it closely mimics the person's usual eating habits. In patients with excessive regurgitation, vomiting or aspiration, a continuous drip feeding method may be opted for. Some hospitals may even follow overnight feeding.

Use of insulin: If a patient is on basal insulin prior to admission, it is recommended to

continue with the same and add prandial doses according to the feeding method. With overnight feeding, a combination of intermediate and regular insulin may be used to achieve appropriate glycemic control. NPH insulin suits better as the action duration is similar to the duration of the feeding. In case of continuous drip feeds, 50% daily requirement of insulin is provided as basal insulin and the rest is provided every 4-6 hours depending on the action profile of the prandial insulin (short or rapid acting insulin). If the patient is provided only with basal insulin, and feeds are suddenly discontinued, s/he is at high risk of developing hypoglycemia. Thus, a basal-prandial insulin schedule is followed even if feeds are provided at a steady rate. On 3-4 hourly bolus feeding, prandial, rapid-acting insulin is provided at the start of each feed or short acting insulin for 6 hourly feeds is used. Incase fasting blood glucose levels are found to be consistently high, a basal dose may be added. Prandial insulin doses are titrated according to the blood glucose levels. An IV infusion of insulin must be avoided unless called for in extremely critical conditions and severe hyperglycemia. One must perform capillary glucose check every 4-6 hours when on enteral feeding, connected with the feeds if bolus feeding is undertaken.

Feeding considerations: Factors to be considered while planning an enteral feeding regimen include stress, steroid use, weight status, inappropriate carbohydrate content, gut functioning, gut motility, presence of gastroparesis, risk of refeeding and pre-existing glycemic control of the individual. Research has shown that using polymeric, diabetes-specific formulas is associated with better glycemic control as compared to standard enteral formulas. In the presence of gastroparesis, one may use a feeding pump with a steady infusion rate and prepare a hypertonic feed (upto 1.5 kcal/ml of feed). Carbohydrates must be evenly distributed throughout the day, much like an oral feeding plan has 3 main meals and 2-3 snacks which have a lower glycemic load. This mimics the person's usual eating habits and may relate well to the physiologic profile of insulin secretion as well. Very often it is observed that calories are restricted for fear of hyperglycemia. Also because of the fine-bore NG tubes – many high fibre foods cannot be given and fibre intake is restricted, leading to hyperglycaemia. There may have to be upward revision of insulin dosage in order to maintain caloric and nutrient requirements. In case feeding is suddenly discontinued, one must add 10% IV dextrose at the rate providing the same carbohydrate calories as the enteral feeding regimen.

Risk of hypoglycemia: Patients who receive enteral nutrition are at higher risk for hypoglycemia as clinical manifestations of declining blood glucose levels are often blunted in these patients. Hypoglycemia can develop due to excess of insulin dose, abrupt discontinuation of nutrition support, recovery from acute illness, reduction in dose of glucocorticosteroids or vasopressors and progressive organ failure. The development of hypoglycemia in critically ill patients has been shown to be associated with increased risk of complications, length of hospital stay, and mortality. Thus, one must be very cautious when restricting carbohydrates in enteral feeding.

SHAMBHAVI KAMAT

WHAT'S COOKING?

DALIA TIKKIS

Ingredients:

- Broken wheat (Bulgur, Dalia, Lapsi) 10 gm
- Chickpea 10 gm
- Mixed vegetables (grated carrots, potato, capsicum) 30 gm
- Onion 10 gm
- Wheat flour/ bread crumbs 10 gm (2tsp)
- Oil 10 gm (2tsp)

Method:

1. Take 1 cup of water and bring to a boil.
2. Once the water boils, add some salt. Reduce the heat and add the broken wheat to it while constantly stirring.
3. After couple of minutes switch off and keep it covered for 5 minutes. The broken wheat will get cooked with the steam. If you are making with leftover broken wheat upma, use it instead.
4. In the meantime, grate or finely chop all the vegetables. Take all these raw vegetables in a mixing bowl. You can partially cook them in the microwave, but here they are added as such.
5. Add the cooked broken wheat, turmeric powder, cumin seed powder, red chilli powder and green chili (optional) to this. Mix everything together.
6. If there is excess moisture, add some bread crumbs or plain wheat flour and mix.
7. Check for seasoning and adjust if needed. Take small portions of the mixture and shape it into cutlets.
8. Heat a griddle and drizzle about 2 tsps of oil. Place these cutlets and shallow fry them till they turn golden brown on both sides.

Benefits

- A good snack providing complementary protein (combination of cereal + pulse)
- Nutritious and easy to prepare
- Low Glycemic index

Nutrition profile per serving size

Energy (Kcals)	Protein (gms)	Carbohydrates (gms)	Fats (gms)	GI
203	4.8	22.7	10.7	Low

Samina Burhanpurwala

HUMMUS KHAKHRA

Ingredients

- Bajra 30 gm
- Wheat flour 15 gm
- Fenugreek leaves 25 gm
- Chick peas 45 gm
- Sesame seeds 15 gm

Pre preparation

1. Soak chick peas overnight n cook to soft
2. Grind 1 teaspoons jeera/ cumin seeds, 2 teaspoons sesame seeds
3. Grind green chillies ginger n garlic

Method:

1. Blend the cooked chick peas after draining the water (collect the water in a glass, to b used later if required).
2. Add all d remaining ingredients n blend further to a smooth paste.
3. Garnish with oregano/ red chilli flakes
4. Enjoy the tasty hummus with nutritious bajra methi khakhra and methi masala khakhra

Benefits

- Convenient easy to prepare nutritious meal
- Lente carbohydrates delays the rise in blood sugar
- Good amount of soluble and insoluble fibre, calcium, iron and zinc
- Apple cider vinegar helps in insulin resistance issues with diabesity

Nutrition profile per serving size

Energy (kcal)	Protein (gm)	CHO (gm)	Fats (gm)	Total Fiber	GI
345	14	49.75	9.96	18.29	Medium

Bina Chheda
- Dietician

MYTHS AND FACTS

Myth: Fat people always get diabetes

Fact: That's not true. Being overweight or obese increases the risk of developing diabetes. It's a risk factor, that doesn't mean they will certainly turn diabetic. A small percentage of obese people are now classified as "healthy obesity" as well. Many diabetics belong to normal BMI category too.

Myth: People with diabetes cannot eat rice and bread

Fact: People with diabetes can eat starchy foods when their blood sugars are in control. However, portion size is the key to being healthy. Also, if one has an option, brown rice can be chosen over white rice and whole wheat or multigrain breads are preferred over white bread.

Myth: People with diabetes cannot eat chocolates

Fact: White chocolate and milk chocolate are high in calories and sugar than dark chocolates. Diabetic chocolates are made with fructose and sorbitol/xylitol/maltitol and do not contain sugar. It is also high in fat i.e. cocoa butter rich which might affect blood sugars. However, dark chocolates on the other side are polyphenol rich. Flavonols in it has been shown to improve insulin sensitivity which increases endothelium derived nitric oxide synthase activity and hence more vasodilation. The cocoa content with 70% or more of the dark chocolate is advisable to diabetics.

Myth: Being on insulin doesn't require lifestyle changes

Fact: On diagnosis of diabetes, blood sugars may be controlled with help of diet, exercise and oral medication (in T2DM). However, if the medications are not effective enough as they were, insulin is initiated. Managing diet and exercise with insulin is also important since one of the side effects of insulin is weight gain. It is also required to minimize hypoglycemia.

Myth: A patient following healthy diet and taking medication, need not exercise

Fact: Glycemic control can be achieved if the triad of diet, exercise and medication is followed thoroughly. If glycemic control is well maintained with a healthy lifestyle, the physician might reduce or stop the diabetic medication. However, the patients need to keep a regular check of their blood sugars.

HOW KNOWLEDGEABLE ARE YOU?

1. Which of the following OHAs cannot be used along with insulin therapy in type 2 diabetics?
   a) Metformin
   b) α-Glucosidase inhibitors
   c) Gliptins (DPP-4 inhibitors)
   d) None of the above

2. Clinical weight gain is associated with:
   a) Sibutramine
   b) Pioglitazone
   c) Metformin
   d) None of the above

3. Which of the following statement is true? Diabetic ketoacidosis:
   a) May present as an "acute abdomen"
   b) Is associated with rapid shallow breathing
   c) Develops primarily a result of insulin resistance
   d) Ketonuria is usually absent

4. Which of the following statement is true? During pregnancy:
   a) Presentation with osmotic symptoms is more likely to indicate pre-gestational diabetes than gestational diabetes
   b) Insulin requirement increases by 40% during the first trimester
   c) After one pregnancy affected by gestational diabetes, it is likely to recur in 80% of subsequent pregnancies
   d) Glucose renal threshold is reduced meaning that screening by urinalysis is inappropriate

5. Hypoglycemia in type 2 diabetes can be caused by treatment with:
   a) Metformin
   b) Metformin plus exenatide
   c) Metformin plus sitagliptin
   d) Metformin plus glibenclamide

6. Which of the following diabetes treatments increases risk of fracture?
   a) Thiazolidinediones
   b) Sulfonylureas
   c) Acarbose
   d) Metformin

7. When should post-prandial blood sugars be checked in pregnancy?
   a) They are unreliable and should not be checked
   b) 2 hours after a meal
   c) 4 hours after a meal
   d) 1 hour after a meal

8. Which of the following does not slow progression of nephropathy?
   a) Angiotensin II receptor blocker
   b) Decreased dietary protein intake
   c) Starting insulin therapy
   d) None of the above

9. Impaired glucose counter-regulation in type 1 diabetes is the result of:
   a) An attenuated increase in epinephrine with a drop in blood sugars
   b) No increase in glucagon with a drop in blood sugars
   c) No decrease in insulin with a drop in blood sugars
   d) All of the above

10. Which of the following does not induce or precipitate diabetes?
    a) Beta-blockers
    b) Hepatitis C
    c) Angiotensin-converting enzyme inhibitors
    d) HIV/ AIDS

ANSWERS:

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

Invitation to write in the

JOURNAL OF DIABETES EDUCATION

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Please include biographical information, including affiliation of all authors and email of corresponding author.

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Manuscripts should be in English and submitted electronically to ademembers@gmail.com. Interested candidates can e-mail their topics for approval. Please note that your targeted readership consists of diabetes educators, diabetologists, nutritionist, nurses and pharmacists.

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About 2000 words is optimum, but this can change if required.

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Please do not cite references in the text. About 5-15 key references at the end of article are needed. Use Vancouver style of references.

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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.

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Bangalore
Email id: dr.munichoodappa@gmail.com

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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

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