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Journal of Diabetes Education

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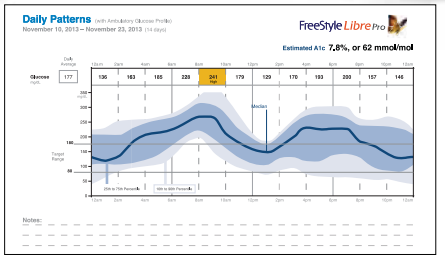

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JOURNAL OF DIABETES EDUCATION

To Dispel Darkness of Diabetes

Vol. 4 **Number 2** **April- June, 2016**

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CLOSED LOOP INSULIN PUMP

Dr. H. B. Chandalia*

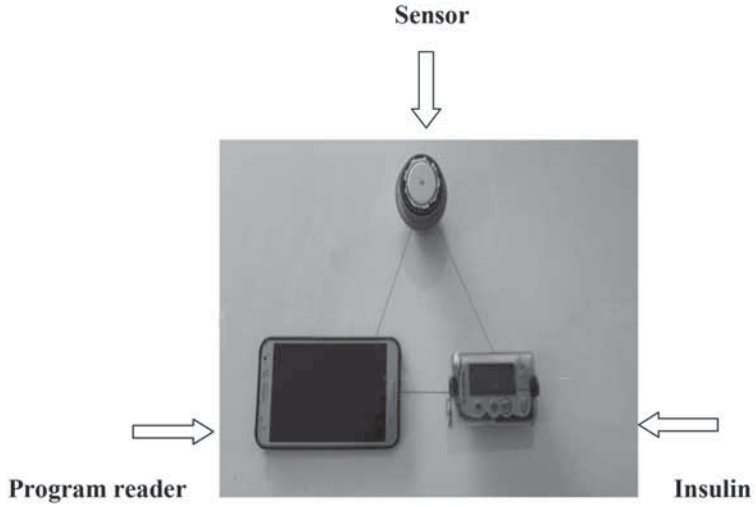
The basic function of all insulin pumps is to inject insulin round the clock to give basal supply and inject boluses at meal time. The basal as well as bolus need to be calculated by the user. These systems are called the open loop pumps, where the loop is closed by the user.

Efforts have been made to modify the system where the pump carries basic algorithms enabling it to calculate the dose and inject. For this purpose three differently functioning components have to be integrated, first - the pump itself, second - a system to sense the blood glucose on continuous basis and third - a system to calculate the insulin dose based on the algorithms built in the system. The third component can be a smart phone.

The earlier efforts in this direction saw the sensor - augmented pumps with mechanism to suspend the pump in the event of hypoglycemia. The suspension of pump can be based on a blood glucose threshold value or a better system based on predictive calculations. In another system Fuzzy logic is used, where the system learns by previous experiences.

It has been possible to design pumps with closed loop for providing the basal supply of insulin. Several well conducted studies have reported successful use of the basal modality of insulin infusion. However, at the present time, it is difficult to take care of boluses by using a closed loop system. Hence, the user has to do carb counting and inject the bolus dose as per the carbohydrate /insulin ratio. However, having basal supply fully automated is also a great advantage.

Subcutaneous injection therapy by multiple injections a day suffers from a great draw back. The coefficient of variation of insulin response is 15-40% with all insulins, which is a substantial variation, making users life miserable with wide swings in blood glucose. Some of the newer analogues (possibly insulin degludeg) has reduced the variation in the same subject to 15%. Human regular insulin and rapid acting analogues produce about 20% variability. The maximum variability is seen with basal insulins like NPH, and glargine. Use of insulin pump reduces the variability of response to about 3%. However, the studies reported on close-loop system report a 15% variability of response.



CLOSED LOOP INSULIN PUMP

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POLYURIA

*Tejal Shah**

Excessive amount of urine output (polyuria) does not always mean there is diabetes. There are many causes of polyuria.

POLYURIA: Polyuria is defined as the production of an abnormally large volume of dilute urine, more than 3 liters a day, as compared to normal daily urine output in an adult of one to two liters .Urine volume is considered excessive if it equals more than 2.5 - 3 liters/day.

CAUSES OF POLYURIA: Polyuria is usually the result of drinking excessive amount of fluids particularly water and fluids that contain caffeine or alcohol. Drinking excessive water or fluid more than 8 to 10 liters leads to polyuria. Some people are compulsive water drinkers and they can drink large amount of water which leads to polyuria.

There are some other causes of polyuria:

Diabetes Mellitus: Polyuria is the main symptom of Type 1 and Type 2 Diabetes. In fact, the word diabetes means “running through” as the excessive urine flow appears like opening of aqueducts. There are new forms of oral agents used in diabetes called SGLT-2 inhibitors, which act by increasing glucose excretion through the kidneys. This results into mild polyurea. Hence, these drugs are not used concomitantly with a diuretic and are taken with 2 glasses of water in the morning.

Diabetes Insipidus: This is caused by deficiency of an anti diuretic hormone, vasopressin, secreted by hypothalamus and posterior pituitary. This hormone increases permeability of collecting tubules in the renal medulla which leads to absorption of water. In the deficiency of this hormone, a profuse diuresis of 5 - 10 liters daily can occur. In case access to water is not available or sensorium is obtunded, it can result into severe dehydration.

Urinary diseases: There are many diseases of the kidney which produce polyuria. Acute tubular damage due to transiently reduced renal perfusion can lead to marked polyuria, so called polyuric form of acute renal failure. Any form of chronic kidney disease in a phase preceding the subtotal or total kidney damage can manifest polyuria. This includes all forms of nephritis, like glomerulonephritis or interstitial nephritis. Kidney damage caused by lithium, tetracyclines or sickle cell disease can result in polyuria. There are a few conditions where the solute load increases and polyuria occurs because the solute needs to be excreted with water. One such example is hypercalcemia. The polyuria is of 3 to 4 liter volume in this situation. There are often situations where a functional disturbance of kidney results in polyuria, for example, chronically low serum potassium, which can occur in chronic diarrheal diseases. This can result in the loss of concentrating ability of kidney and results in polyuria. This situation is reversible on correction of serum potassium level.

Other common causes: Use of diuretics will of course produce polyuria. In pregnancy mild polyuria is possible. In any edema state, like congestive heart failure or hypoalbuminemia, spontaneous diuresis occurs at night, thus increasing nocturia; this is due to a reduction of aldosterone and other hormones seen in recumbent position.

To summarise, if in a diabetic the glycemic control is near -optimal and there is polyuria, it is advisable to think of other causes of polyuria and institute appropriate diagnostic and therapeutic measures.

For Further Reference:

Alan G. Robinson, Joseph G. Verbalis, Posterior Pituitary. In Williams Text book of Endocrinology: 13th Edition; Elservier, Gurgoan; 2016: 300-313.

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DIET IN DIABETIC NEPHROPATHY

*Apeksha Ekbote**

Dietary factors have an important role to play in the development of diabetes and its various complications. Diabetes can affect the kidneys in a number of ways but one of the most important complications is diabetic nephropathy. This is a kidney condition that occurs only in people with diabetes mellitus and results in progressive damage to the small filtering units of the kidney (glomeruli). This eventually leads to large amounts of protein in the urine, high blood pressure and declining kidney function. Even when drugs and diet are able to control diabetes, the disease can lead to nephropathy and kidney failure.

Diabetes is the second most common cause of kidney failure in India. About 20% of the people who need dialysis (artificial kidney replacement) or kidney transplantation in India have usually diabetic nephropathy. (NKFI)

The kidneys are each made up of around 1 million nephrons that remove extra fluid and wastes out from the blood. These nephrons help regulate water, salts, glucose, urea, phosphorus and other minerals. Those with diabetes have a lot of glucose that comes out in their urine. High blood sugar levels can damage the tiny blood vessels in the nephrons by thickening and scarring them so that over time they are damaged. When this happens, protein leaks through the kidneys into the urine. The nephrons are no longer able to filter properly and this kidney damage can lead to kidney failure

SIGNS AND SYMPTOMS OF DIABETIC NEPHROPATHY

It is possible for individuals to have serious kidney damage without being aware of it. There may be no specific symptoms of diabetic kidney disease until the kidneys fail completely. Early signs or risk factors include:

- Albumin in the urine
- Tiredness
- Swollen ankles
- Fatigue
- Nausea and Vomiting
- Breathlessness

Symptoms related to kidney failure usually occur only in late stages of the disease, when kidney function has diminished to less than 25 percent of normal capacity. Damage to the kidney is reflected in the amount of protein in the urine and the estimated value of the glomerular filtration rate (eGFR). Initially, the amount of protein is very small, but gradually, over a number of years, the amount of protein in the urine increases.

DIETARY MANAGEMENT

The major dietary factors that have an impact on nephropathy are the protein intake, salt and fluid intake.

The concept of using one fixed dietary prescription for all patients is highly questionable. The diet of every patient needs to be individualised based on the tendency to retain or lose salt, serum potassium, serum protein and overall nutritional status and urine volume. The dietary guidelines for the management of diabetic nephropathy are as follows:

PROTEIN: Low protein diets have been promoted for people with chronic kidney disease to decrease the kidney's workload and perhaps delay progression of kidney failure. In diabetic nephropathy some protein is lost in the urine. Diabetics with poorly controlled blood sugar levels have increased protein breakdown and may need additional protein to prevent protein deficiency and muscle breakdown. Intake should be constituted to 10-15% of total calories which should

* Apeksha Ekbote, Senior Dietitian, HOSMAT Hospital, Bangalore.

include a mix of animal and vegetable protein of high biological value. Recommended protein in nephropathy is 0.6-0.8 gm/kg/day. If there is a heavy proteinuria and no other evidence of renal failure (e.g. raised creatinine) dietary protein will need to be increased.

ROLE OF BRANCH CHAIN AMINO ACIDS: The link between amino acids and insulin resistance has been known for decades but with the advent of comprehensive metabolomic profiling a more detailed picture of how amino acids participate in the progression of diabetes is being revealed. The pathogenesis of diabetic nephropathy is complex, and oxidative stress plays an important role in the development of diabetic nephropathy. Elevated reactive oxygen species (ROS) levels activate various signalling pathways and influence the activities of transforming growth factor-β (TGF-β) and matrix metalloproteinase-9 (MMP-9), which contribute to glomerular hypertrophy. Branched-chain amino acids (BCAAs) are widely used in clinical treatment, and BCAAs can reduce the oxidative stress associated with the diabetic pancreas.

CARBOHYDRATES: 55-60% of total calories should come from complex carbohydrates with high fibre content which may include whole grains, pulses, legumes, dried beans and fruits. This is to avoid protein catabolism, gluconeogenesis and subsequently uremia.

FATS: 25-35% of total calories preferably from MUFA and PUFA. It is best to avoid saturated fats. Avoid Trans fat which is found in processed foods, bakery items.

SALT INTAKE: The recommended salt intake in diabetic nephropathy is less than 5gm/day (<2gm of sodium per day) as per the ISC CKD guidelines of 2014. A low-sodium diet has been shown to further reduce BP and urine albumin or protein levels in the short term, in patients on ARBs and may be considered in those with high BP and poor response to ACE-Is or ARBs.

POTASSSIUM: Restricted to 1mmol/kg/day in stage III CKD. Avoid high potassium foods like ragi, coconut water, fruit juices, certain fruits which are high in potassium (citrus fruits), chocolates and nuts. It is best to use leached vegetables in order to eliminate the potassium present in it. Hypokalemia may result from excess diuretic use or extra renal losses, Hypokalemia leads to interstitial fibrosis and may accelerate progression of renal disease.

PHOSPHORUS: Restricted to 800-1200—mg/day. Phosphate binders may be required to reduce absorption.

CALCIUM: Calcium supplements may be required in dosage of 1-2g/day to delay renal bone disease.

VITAMIN D3 & 1, 25 CHOLECALCIFEROL: Vitamin D influences the renin-angiotensin system, inflammation, and mineral bone disease, which may be associated with the cause and progression of CKD. There is increasing evidence that vitamin D deficiency may be a risk factor for DM and CKD. The Third National Health and Nutrition Examination Survey (NHANES III) found an increase in the prevalence of albuminuria with decreasing 25 (OH) D concentrations. The conventional rationale for vitamin D treatment in CKD is to slow the progression of secondary hyperparathyroidism, which begins early during the course of CKD, affects the majority of CKD patients, and will progress if left untreated.

IRON: Kidneys secrete a hormone called erythropoietin, which stimulates the bone marrow to produce red blood cells. In diabetic nephropathy, the tiny blood vessels that filter waste products from the body become damaged and start “leaking” substances (such as protein) into the urine. At the same time, the amount of erythropoietin produced by the kidneys is reduced, leading to anemia. Some studies have shown that reduced erythropoietin production and anemia happen earlier in people with diabetes and kidney disease than in those with kidney disease and no diabetes. Observational studies indicate that low Haemoglobin levels in such patients may increase risk for progression of kidney disease and cardiovascular morbidity and mortality. Iron

deficiency can be corrected by iron supplementation. If iron indices indicate absolute iron deficiency (Transferrin saturation ≤20 percent and serum ferritin <200 ng/mL), a sufficient amount of iron to correct the iron deficiency should be administered. In addition, among patients in whom an increase in haemoglobin concentration is desired, a sufficient amount of iron should be administered.

FLUID: As kidney disease progresses, the kidneys will clear less water. Excess fluid intake can lead to problems with fluid overload, including hypertension, shortness of breath, pulmonary oedema, and other cardiac problems. Fluid intake should be adjusted to the clinical state of the individual, taking into account the degree of reduced GFR, oedema, and hypertension management. Intake is dependent on the urine output and water balance. Fluid intake should be adequate to stimulate urine output for excretion of wastes but should avoid excess fluid retention at the same time. Urine output should be strictly recorded and an additional of 500ml can be given (output+500ml)

Table 1

DIET IN PATIENTS WITH STAGE 1 & 2 CKD DUE TO DIABETIC NEPHROPATHY (GFR >60ml/min)

Calories	35 kcal/kg; reduce in obese (BMI >30)
Protein	0.8-1g/kg/d, 10-15% of calories
Salt	5-6 gm/d
Phosphorus	Not Restricted
Potassium	Not Restricted

Table 2

DIET IN PATIENTS WITH STAGE 3 CKD DUE TO DIABETIC NEPHROPATHY (GFR - 30-60ml/min)

Calories	35 kcal/kg; reduce in obese (BMI >30)
Protein	0.8-1g/kg/d, 10-15% of calories
Salt	5-6 gm/d
Phosphorus	8-12 mg/kg/d
Potassium	Individualize, avoid ACEI

Table 3

DIET IN PATIENTS WITH STAGE 4 CKD DUE TO DIABETIC NEPHROPATHY (GFR-15-30ml/min)

Calories	35 kcal/kg; reduce in obese (BMI >30)
Protein	0.6-0.8g/kg/d, 10-15% of calories
Salt	5-6 gm/d
Phosphorus	8-12 mg/kg/d
Potassium	39.1 mg/kg/d

Table 4

DIET IN PATIENTS WITH DIABETIC NEPHROPATHY ON DIALYSIS

Calories	35 kcal/kg; reduce to 30 in elderly (>60yrs) & Obese (BMI >30), In Peritoneal dialysis subtract calories due to glucose absorption
Protein	1.2(on Hemodialysis) – 1.3g/kg/d (on PD), 10-15% of calories
Salt	5-6 gm/d
Phosphorus	17 mg/kg/d
Potassium	39.1 mg/kg/d
Fluids	750 ml/day + Urine Output
Vitamins	Water Soluble Vitamins

Table 5

DIET IN KIDNEY TRANSPLANT PATIENTS

Calories	35 kcal/kg; reduce in obese (BMI >30)
Protein	0.8g/kg/d
Salt	5-6 gm/d
Phosphorus	No Restriction
Potassium	No Restriction

SOURCE: Dietary Management of Diabetic Nephropathy-RK Sahay & Manisha Sahay

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WHAT'S NEW?

BROWN ADIPOSE TISSUE

Our understanding of brown adipose tissue has increased for the past two decades. The adipose tissue we normally talk about in animals is white adipose tissue. In most mammals, brown adipose tissue is also present in variable amount in certain regions of the body. Most interestingly, certain metabolic changes can transform white adipose tissue to beige and brown and there are scattered cells of brown adipose tissue amongst the white adipose tissue in the whole body.

Brown adipose tissue is metabolically very active. Structurally, it has abundant mitochondria. On exposure to cold ambient temperature, humans generate immediate heat by the process of shivering. Here, the oxidation of glucose increases in the skeletal muscles. For a long term adaptation, nonshivering heat production occurs in the brown adipose tissue. The enzyme involved is uncoupling protein - 1 (UCP-1), Epinephrine stimulates the beta-3-adrenergic nerves to enhance lipolysis, thus making the fatty acid substrate available for this process.

There is some interesting research showing production of a protein, now labeled irisin, which arises from the exercising muscles and acts on the white adipose tissue to induce beiging or browning. This is one mechanism which explains the beneficial effect of exercise.

As we learn more about the physiology of brown adipose tissue and its contribution to heat generation, we expect to get smart clues to change the energy balance in obese subjects towards greater energy expenditure. We hope to solve at least a few riddles regarding etiological aspects of obesity.

Suggested further reading:

Cannon B, Nedergaard JA. Brown adipose tissue: function and physiological significance. Physiological reviews. 2004;84:277-359.

H.B.C.

QUESTION & ANSWERS

Q) When should you do your exercise when you have diabetes?

Exercise is beneficial to diabetic patients as it will help in maintaining good weight but also promote insulin sensitivity. Insulin sensitivity shows how sensitive one person's body is to insulin. If someone has good insulin sensitivity, it means that the body needs only a minimal amount of insulin to utilize the blood sugar released by the body in to the blood stream. Thirty minutes of exercise is recommended every day for maintaining a healthy life style.

However, one must always consult the doctor before taking up exercise. You should not have uncontrolled diabetes or Ketones in urine. During exercise our body needs more energy to be supplied to the muscles; hence the liver is triggered to release glucose into the body. However if one has low levels of insulin in the body then the blood sugar will not be utilized by the body and there would be a buildup of glucose in the system leading to hyperglycemia after exercise.

Therefore, it is a must to always check the blood sugar levels so as to determine the status of glycemic control. Always start with a low intensity exercise and then gradually increase the intensity so that the body is not put under physical stress.

Hypoglycemia can occur during or after exercise more often in type 1 patients or insulin requiring type 2 patients. In such cases the patients must always keep simple carbohydrates such as sugar or energy drinks with them so as to combat hypoglycemia when it occurs. Do not skip meal after exercise, have a snack or a normal meal within 30 minutes of exercise. Drink plenty of fluids so as to avoid dehydration. When you are sick do not exercise. Proper footwear is important so as to avoid any blisters or wound.

Walking, swimming, cycling, water aerobics, dancing are some of the examples of exercises that can be undertaken by diabetics. Increase daily physical activity levels during the course of the day for example, choose to use stairs instead of elevator such healthy life style modifications which can be made easily.

Vanditha Mohanan

PROBLEMS & SOLUTIONS IN DIABETES EDUCATION

DETERIORATION OF GLYCEMIC CONTROL IN TYPE 2 DIABETES MELLITUS

*Hemraj B. Chandalia**

Ms. K M, a long standing type 2 diabetic has been on a full basal bolus regime of insulin plus metformin. Her last HbA1c a few months ago was 7.2%. However, for the past 2 weeks, upon return from a trip to Singapore, her glycemic control deteriorated, as most SMBG values premeal were 150-200 mg/dL and post meal 200-300 mg/dL. She has been on Aspart insulin 8, 6, 6 units before breakfast, lunch and dinner respectively and Glargine insulin 10 units at 10 PM. She was also on Sitagliptin plus metformin (100 g + 500 g) once a day. Her diet and activities remained almost same for the past 6 months, although there has been a family discord lately causing some mental stress.

An inspection of insulin injection sites did not reveal any lipohypertrophy. She was rotating the injection sites regularly. An inspection of insulin pens revealed normal delivery based upon daily consumption. There was no flocculation of insulin or deposit of insulin on the cartridge wall. A direct question was asked to her which led to the discovery of the cause of deterioration of metabolic control.

Question:

How did you transport your insulin pens during travel?

Ms. K. M had put her insulin pens in the check- in bag properly stored in a cool insulin pouch. The bags had arrived on time and transported in the car trunk. She usually stores the pens in use at ambient temperature at home, which is usually cool.

The insulin has been rendered ineffective as it was transported in the check-in bag. The temperature in the baggage - hold in all aircrafts is not uniformly controlled. Hence, at high altitude it can occasionally go very low. This freezes the insulin. Upon landing, the insulin is unfrozen by the time one reaches home. Thus, no obvious problem is noticed by the user.

Recommendation:

It is recommended that insulin be carried in the carry-on cabin bag, where ambient temperature is usually around 70°F. It is not necessary to carry it in a cool insulin pouch or cool-gel pack; however, this can be a good precaution if there is long ground travel on arrival and the ambient temperature is high.

Resolution of problem:

Ms. K. M was advised to buy new insulin cartridges and continue her usual insulin dose. Her glycemic control improved in almost a week's time.

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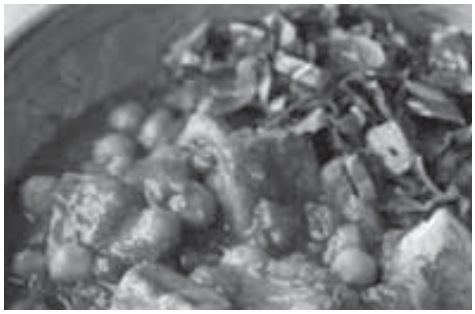
WHAT'S COOKING ?

TOFU & CHICKPEA CURRY WITH SPRING GREENS



INGREDIENTS	AMOUNT
Onion	25 Gm
Garlic	2 Cloves
Tofu	75 Gm
Spring Greens	50 Gm
Vegetable Oil	3 Tsp
Coriander	For garnishing
Cumin	1/2 tsp
Red Chilli Powder	1/2 tsp
Turmeric	1/4 Tsp
Tomato	25 Gm
Chickpeas	50 Gm
Garam Masala	1/4 Tsp
Lemon Juice	1 Tsp
Cumin Seeds	1/2 Tsp
Salt	To Taste

METHOD OF PREPARATION:

- Peel and finely slice the onion. Peel the garlic, and then finely chop the cloves. divide the chopped garlic into half portions.
- Cut the tofu into cubes.
- Finely chop the spring greens.
- Heat 1 tsp of vegetable oil in a pan, add the onion and chopped garlic, and cook gently until the onion begins to brown.
- Add the ground coriander and cumin, and turmeric. Cook for 1 minute. Stir in the tomatoes and half a cup of water. Simmer for 10 minutes.
- Meanwhile, heat 1 tsp of oil in a frying pan and cook the tofu until golden brown. Drain on kitchen paper if needed.
- Drain, rinse and stir the chickpeas into the tomato curry, heat through, then add the chilli powder, garam masala, lemon juice and seasoning to taste. Add more water if it looks too dry. Add the tofu.
- Heat 1 tsp of vegetable oil in a pan, add the chopped garlic. When the garlic begins to brown, add the spring greens and stir-fry for 2 to 3 minutes, until just cooked. Serve with the tofu and chickpea curry.

Serves: 1

Nutritive value for 1 serving:

Total Energy (Kcal)	CHO (gm)	Protein (gm)	Fat (gm)	TDF (gm)
296	33.8	16.8	14.6	2.8

BARLEY IDLI

INGREDIENTS	AMOUNT
Barley (Cooked)	50 Gms
Parboiled Rice	50 Gms
Urad Dal	50 Gms
Fenugreek Seeds	1/4 Tsp
Salt	To Taste
Carrot and French Beans (Mix Veg)	50 Gms

METHOD OF PREPARATION:

- Wash and soak the parboiled rice, urad dal and fenugreek seeds in a deep bowl in enough water for 2 hours. Drain and keep aside.
- Combine the parboiled rice, urad dal, fenugreek seeds and barley in a mixer to a smooth paste using approx. 1½ cups of water.
- Transfer the paste into a deep bowl, add the salt and mix well.
- Cover it with a lid and keep the batter aside to ferment for 4 hours.
- After fermentation, mix the batter well once again.
- Wet the idli moulds with a little water and pour spoonfuls of the batter into each of the idli moulds.
- Sprinkle a few mixed vegetables over each idli.
- Steam in an idli steamer for about 12 minutes or till they are cooked

Serves: 2

Nutritive value for 1 serving:

Total Energy (Kcal)	CHO (gm)	Protein (gm)	Fat (gm)	TDF (gm)
141.5	26.5	6.3	0.5	1.1

MEMBERSHIP FORM

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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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JOIN US IN DIABETES PREVENTION PLAN

IF YOU HAVE A FAMILY MEMBER WITH TYPE 2 DIABETES, IT PUTS YOU AT RISK OF DEVELOPING IT TOO

WHY NOT ACT TOWARDS PREVENTING IT BEFORE ITS TOO LATE

GET YOUR FASTING BLOOD SUGAR LEVELS TESTED FREE OF COST AT OUR CLINIC

IF DETECTED WITH BORDERLINE DIABETES, WE WILL PUT YOU ON A PREVENTION PLAN FOR THREE YEARS COMPLETELY FREE OF CHARGE

Age Limit – 30 – 70 years

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Charni Road – 022-23634320
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Prof (Dr) H B Chandalia's creative writing abilities & practical acumen has always been illustrated by his multiple contributions as an author of chapters in various textbooks. One such outstanding example is the book **'Conquest of Diabetes- by diet & exercise'** which is running its fourth edition in the English language and an edition in Hindi as well as in Gujarati. The Marathi version of the book is under preparation. It is a comprehensive, extensively illustrated two color book which is characterized by its brevity, clarity and offers a systematic approach towards the management of diabetes by diet and exercise.

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This book is directed to persons suffering from diabetes, health-care practitioners like doctors, nutritionists and diabetes educators and other health professionals involved in the care of diabetics.

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References :
1. ISO 15197:2013 standard
2. Goldy et.al.International conference on Advanced technologies and Treatments for Diabetes 2013
3. Bayer. Data on file

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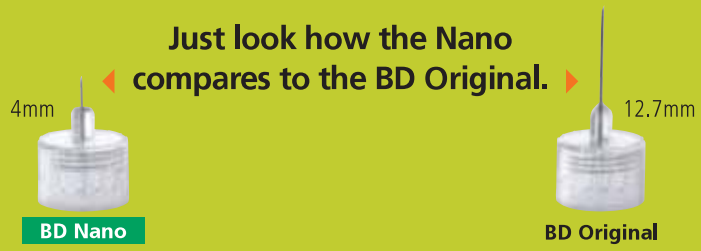

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7. As of April, 2010.

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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.

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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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