diabetes NEWSLETTER

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Inside Bangladesh

Type 2 diabetes in rural and urban (slum) population: diverse prevalence and associated risk factors in Bangladesh

During the last two decades, the urban population of Dhaka city has grown rapidly because of urban migration and fast expanding urbanization. Most rural migrants settle in urban slums and are exposed to a number of unhealthy life conditions. It is likely that as an accumulated effect of all the adverse conditions, the slum dwellers suffer from a variety of health problems from communicable to non-communicable diseases. Under these hostile living settings it is less likely that the inhabitants will develop obesity which may result in the observed higher prevalence of chronic diseases like diabetes mellitus (DM). The purpose of this study was to investigate the differences in prevalence of T2DM and related risk factors between urban (slum) dwellers and a rural population.

Aims : To describe differences in prevalence of Type 2 diabetes mellitus with its associated risk factors between rural and urban populations in Bangladesh. Diagnostic criteria [fasting blood glucose (FBG) and oral glucose tolerance tests (OGTT)] were compared and reviewed for both populations.

Methods : A total of 1555 subjects from urban and 4757 from rural communities (age = 20 years) with similar cultural and ethnic backgrounds were randomly selected in a cross-sectional survey. FBG values were determined from all and 2-h post-glucose capillary blood samples were determined after a 75-g oral glucose load for a selected number (urban 476, rural 1046).

Results : A higher prevalence of diabetes was found in urban (8.1%) compared with rural populations (2.3%). Age, sex and waist-to-hip ratio for men were significant risk factors for both urban and rural subjects following fasting and 2-h post-glucose values adjusted for a number of confounding variables. Poor agreement was observed between FBG and OGTT for both urban (kappa 0.41) and rural (kappa 0.40) areas.

Conclusions: A higher prevalence of diabetes mellitus (DM) in the urban population was observed compared with rural subjects despite similar body mass indexes (BMI). Differences in obesity, waist/hip ratio or hypertension failed to explain the increasing occurrence of T2DM in the urban population.

Source: *A. Hussain, M. A. Rahim, A. K. Azad Khan, S. M. K. Ali, S. Vaaler (2005). Diabetic Medicine 22 (7), 931–936.*

Diabetescope

Risk of Schizophrenia is low in Type 1 Diabetics

The incidence of schizophrenia in patients with type 1 diabetes is less than half that seen in people without diabetes, according to findings published in the August issue of the Archives of General Psychiatry.

Patients with schizophrenia have an increased risk of type 2 diabetes mellitus. There are no conclusive studies, however, on the comorbidity of schizophrenia and type 1 diabetes."

The researchers examined the incidence of schizophrenia in a nationwide cohort of 896,175 people born in the decade 1950-1959 in Finland, with follow-up from 1969 to 1991. The cohort included 5009 patients with type 1 diabetes, and the incidence of schizophrenia was calculated using information from three health care registers.

The incidence of schizophrenia was 0.21 per 10,000 person-years in the group with type 1 diabetes compared with 0.56 per 10,000 person-years ($p < 0.001$) in the group without type 1 diabetes.

Several possible reasons may explain a negative association between two disorders with shared etiological features. One possibility is a linkage disequilibrium of protective and liability genes for schizophrenia and type 1 diabetes.

Another explanation is that early insults (e.g., prenatal and childhood infections and obstetric complications) evoke different responses among individuals with a genetic predisposition to type 1 diabetes or schizophrenia, leading to the development of type 1 diabetes in the former and schizophrenia in the latter.

A third possibility, according to the authors, is that some factors associated with type 1 diabetes could modify the phenotype or clinical picture of schizophrenia.

Arch Gen Psychiatry 2007;64:894-899.

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Chronic HCV Infection Linked to Glucose Intolerance

Chronic hepatitis C virus (HCV) infection is associated with glucose intolerance and, despite that, a favorable lipid pattern, according to findings published in the August issue of Gut. Furthermore, clearance of HCV seems to be facilitated by elevated triglycerides at the time of infection.

Infection with HCV has been associated with alterations in lipid metabolism in some studies and type 2 diabetes in others.

To investigate further, the researchers examined the association between lipid profiles and diabetes and past and chronic HCV infection among residents of a rural village in Egypt. A total of 765 subjects were included in the study. Overall, 113 (14.8%) participants had chronic HCV infection and 67 (8.8%) had past infection.

After adjustment for age and sex, subjects with chronic HCV infection had lower levels of total and LDL cholesterol and lower triglycerides than those never infected. Compared to subjects never infected, those with past infections had higher triglyceride levels, which is an intriguing finding.

Diabetes was observed in 41 (5.4%) of the 754 participants. The long-term follow-up of this population will reveal whether HCV infected subjects have a different cardiovascular risk compared with the general population.

Gut 2007;56:1105-1110.

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IVIg May Ease Severe Diabetic Neuropathy

Intravenous immunoglobulin (IVIg) therapy can markedly reduce severe symptoms associated with diabetic neuropathy, according to a case report by Japanese researchers published in the August issue of the Journal of Neurology, Neurosurgery and Psychiatry.

IVIg was effective in improving severe pain symptoms and muscle weakness in a patient with proximal diabetic neuropathy.

Researchers at Nagoya University School of Medicine describe their patient as a wheelchair-bound 57-year-old man who had had type 2 diabetes mellitus for 10 years. He showed progressive loss of muscle strength in both legs, pain and muscle atrophy in the femoral region and significant weight loss.

The researchers note there is evidence that such neuropathy may involve an immune-associated mechanism. They therefore began treatment with IVIg 0.4g/kg daily for 5 days.

This led to a remarkable improvement in pain and muscle weakness, the team reports. Pain began to return over the following 3 weeks, but a repeat course of IVIg brought about a reduction to a level below that following the initial therapy.

The patient continued to have severe disturbance in the dorsal extension of the ankle. Nevertheless, following the IVIG sessions, he could walk with a cane. Previously he had been unable to stand, let alone walk.

Overall, pain, as assessed by a visual analogue scale, was reduced by 80% and muscle strength was greatly increased.

This observation would suggest that there are some diabetic patients or other painful neuropathic patients whose pain can be ameliorated and thereby possibly quality of life can be improved by IVIg therapy.

Source: J Neurol Neurosurg Psychiatry 2007;78:899-901.

Osteocalcin Enhances Insulin Secretion and Sensitivity

Osteocalcin, a protein produced by bone cells, influences beta-cell proliferation, insulin secretion, and insulin sensitivity in mice. This is a hormone acting upstream of insulin secretion and sensitivity, and it is made in bone. Researchers investigated whether bone exerts a feedback control of energy homeostasis by looking for genes expressed in mouse osteoblasts.

Mice lacking Esp, an osteoblast-expressed gene that encodes a receptor-like protein tyrosine phosphatase termed OST-PTP, showed increased beta-cell proliferation and insulin secretion resulting in hypoglycemia.

Despite having hyperinsulinemia, mice lacking Esp also showed significantly increased insulin sensitivity compared with wild-type mice. Esp-deficient mice were also protected from obesity and glucose intolerance.

In contrast, the report indicates, mice lacking osteocalcin (a protein under the control of Esp) showed decreased insulin secretion and sensitivity and decreased glucose tolerance, beta-cell mass, and adiponectin expression. Additional experiments suggested that osteocalcin regulates insulin sensitivity through adiponectin.

Mice lacking Esp that also lacked one allele of osteocalcin showed a significant reversal of their metabolic abnormalities, provides genetic evidence that Esp and osteocalcin lie in the same regulatory pathway and that Esp-/- mice metabolic phenotype is caused by a gain-of-activity of this hormone.

This finding raise the possibility that skeleton may contribute to the development of the metabolic syndrome since Esp-/- mice do not develop obesity or diabetes.

Dr. Karsenty suggested that osteocalcin might represent a new treatment for diabetes and indicated that further studies are planned to elucidate the mechanism of action of osteocalcin.

Source: Cell 2007.

Diabetes Management and Exercise in Pregnant

Pregnancy is a diabetogenic condition characterized by insulin resistance with a compensatory increase in β-cell response and hyperinsulinemia. The placental secretion of hormones (progesterone, cortisol, placental lactogen, prolactin, and growth hormone) is a major contributor to the insulin resistance, which likely plays a role in ensuring that the fetus has an adequate supply of glucose.

Pregnancy in patients with diabetes is associated with an increased incidence of congenital anomalies for the fetus and spontaneous abortions in women with poor glycemic control. For both type 1 and type 2 diabetes, hemoglobin A_{1C} (A_{1C}) values early in pregnancy are correlated with rates of spontaneous abortion and major congenital malformations. The effect of the increased glucose levels on the rate of spontaneous abortion occurs at the time of conception. Normalizing blood glucose concentrations before and early in pregnancy can reduce these risks to levels of the general population.

Treatment with close monitoring of glucose levels, oral agents (metformin and sulfonylureas), medical nutrition therapy, and insulin therapy if glucose levels are above goal may reduce maternal and fetal complications.

Table 1. Recommended glycemic targets preconception and during pregnancy*

	Glycemic targets
Pre-pregnancy	
A_{1C} (%)	≤ 7.0 (≤ 6.0, if possible)
Once pregnant	
FPG and preprandial PG (mmol/L / mg/L)	3.8-5.2 / 69-94
1-hour postprandial PG (mmol/L / mg/L) or 2-hour postprandial PG (mmol/L / mg/L)	5.5-7.7 / 99-139 5.0-6.6 / 90-119
Pre-bedtime snack PG (mmol/L / mg/L)	4.0-5.9 / 72-107
A_{1C} (%)	≤ 6.0 / 108 (normal)

*In women with type 1 diabetes, attempts to achieve these glycemic targets all the time may be associated with an unacceptable increase in severe hypoglycemic episodes.
Glycemic targets may need to be individualized.
A_{1C} = glycosylated hemoglobin, FPG = fasting plasma glucose, PG = plasma glucose

Monitoring Glucose Levels

Glucose is a potential for being a coteratogen or teratogen surrogate because there is a correlation between A_{1C} levels early in pregnancy and the predictive malformation risk for the fetus. However, there is a lack of a strong correlation between A_{1C} and glucose levels by continuous glucose monitoring. This may indicate that plasma blood glucose levels vary significantly day by day, and A_{1C} cannot adequately describe daily glucose profiles over an 8- to 10-week period.

Self-monitoring of blood glucose is mandatory during pregnancy, especially for patients with type 1 diabetes. Poor glycemic control leads to increased maternal-fetal transfer of glucose and amino acids as well as fetal hyperinsulinemia. These metabolic changes contribute to the development of macrosomia and can lead to difficult delivery, an increased rate of cesarean section, and an increase in fetal morbidity. However, preventing hypoglycemia is just as important. An elevated first morning blood glucose value may reflect an episode of hypoglycemia during the early morning hours because pregnancy is associated with an exaggerated rebound from hypoglycemia. Performing glucose checks at bedtime, 3 a.m., and in the morning can assist in the discovery of nocturnal hypoglycemia.

The goal of self-monitoring is to detect glucose levels elevated to concentrations that increase perinatal mortality.

Gestational diabetes mellitus (GDM) defined as any degree of glucose intoerane with onset of first onset or first recognition during pregnancy. The diagnostic criteria summarizes in table 2.

Fetal complications of GDM include macrosomia, neonatal hypoglycemia, perinatal mortality, congenital malformation, hyperbilirubinemia, polycythemia, hypocalcemia, and respiratory distress syndrome. Maternal complications associated with GDM include hypertension, preeclampsia, and an increased risk of cesarean delivery. Postprandial glucose also has a most important role in macrosomia.

Table - 2 CRITERIA FOR DIAGNOSIS OF GDM WITH THE 75 G OGTT

Organization	Fasting	1 h PG	2 h PG	Diagnostic Criteria for GDM
WHO	≥7.0 mmol/L (126 mg/dL)	≥Not measured 10.0 mmol/L	≥ 7.8 mmol/L (140 mg/dL)	One abnormal value Two or more
Fourth International Workshop/ ADA	(95 mg/dL) ≥5.3 mmol/L	(180 mg/dL)	8.6 mmol/L ≥ (155 mg/dL)	abnormal values

PG: Post glucose, ADA: American Diabetes Association.

Oral Agents

Among the drugs commonly used in the treatment of patients with diabetes, statins

are pregnancy category X and should be discontinued before conception if possible. ACE inhibitors and ARBs are category C in the first trimester (maternal benefit may outweigh fetal risk in certain situations) but category D in later pregnancy and should generally be discontinued before pregnancy. Among the oral antidiabetic agents, metformin and acarbose are classified as category B and all others as category C; potential risks and benefits of oral antidiabetic agents in the preconception period must be care-fully weighed, recognizing that sufficient data are not available to establish the safety of these agents in pregnancy. They should generally be discontinued in pregnancy.

Medical Nutrition Therapy

Medical nutrition therapy includes not only the total daily caloric intake and its carbohydrate content, but also the number of meals throughout the day. Generally, there is not an increased energy requirement during the first trimester, and a normal-weight woman will need an additional 300 kcal/day in the second and third trimesters.

Normal-weight women with GDM need a daily caloric intake of 30 kcal/ kg/ day based on their present pregnant weight. The composition of the diet should be 40% carbohydrate, 40% fat, and 20% protein divided among three meals and three snacks.

In addition, folate (400µg) should be prescribed to all pregnant patients early in the first trimester to decrease the incidence of neural tube defects. Ideally, folate should be given in the preconception phase. Women with a previously affected infant with a neural tube defect and those taking anticonvulsant medications need folate 4 mg/day.

Obese women and women with type 2 diabetes are characterized by insulin resistance that is aggravated during pregnancy. Insulin resistance has been associated with pregnancy complications. Maternal overweight and obesity in normoglycemic women is associated with an increased rate of complications (stillbirth, operative delivery, hypertensive complications, and congenital malformations). For obese women (BMI > 30 kg/m2), a 30-33% calorie restriction (to~25 kcal/ kg actual weight/ day) has been shown to reduce hyperglycemia and plasma triglycerides with no increase in ketonuria. Restriction of carbohydrate to 35-40% of calories has been shown to decrease maternal glucose levels and improve maternal and fetal outcomes.

Insulin Therapy

Insulin requirements vary during pregnancy. There is an increase in requirement during weeks 3-7, a decreased need during weeks 7-15, and then an increase again during weeks 15-40. The total daily insulin requirement for type 1 diabetes during pregnancy is 0.7 units/kg in the first trimester, progressing over time to 1.0 units/kg as the patient nears term. Pregnant type 2 diabetic patients may need higher doses (1-2 units/kg) to deal with insulin resistance and the requirements of the pregnancy. Avoiding excessive weight gain and incorporating an exercise program can improve glycemic control and decrease insulin resistance.

Exercise

The American College of Obstetricians and Gynecologists (ACOG) guidelines recommends that pregnant women perform 30 minutes or more of moderate exercise daily. The American Diabetes Association recommends starting or continuing a program of moderate exercise in women without medical or obstetrical contraindications (Table 3).

Recommended forms of exercise include walking, stationary bicycling, low-impact aerobics, and swimming. Each exercise session should begin with a 5- to 10-minute warm-up period involving some flexibility exercises (stretching) to reduce the risk of musculoskeletal injury during the workout, followed by a cool-down period. In sedentary women who decide to exercise during pregnancy, ACOG recommends that exercise heart rates should not exceed 140 bpm (~ 60-70% of VO2max).

Table 3. Absolute Contraindications to Exercise During Pregnancy

- Preterm labor
- Premature rupture of membranes
- Incompetent cervix
- Persistent second- or third-trimester-bleeding
- Intrauterine growth reduction
- Placenta previa beyond week 26
- Pregnancy-induced hypertension

Exercise increases insulin sensitivity of muscle glucose transport and also enhances insulin action in extramuscular tissues. The three most important endocrine responses to exercise include 1) a decrease in plasma insulin, 2) an increase in

sympathethic nervous activity causing changes in insulin counterregulatory hormones, and 3) hormones affecting sodium and water balance. The autonomic nervous system cannot adjust the plasma insulin concentration in patients with type 1 diabetes and relies on the pharmacodynamics of the injected insulin. There are changes in metabolism resulting from exercise in these patients based on the dose of insulin taken.

There is a potential increase in insulin levels during exercise (because of an increase in the absorption of insulin) causing inhibition of the normal increase in hepatic glucose production and increased muscle glucose uptake. Both of these effects can result in hypoglycemia.

For patients with type 2 diabetes, there is some insulin secretory capacity, so these patients do not have the same metabolic risks for hypoglycemia with exercise as their type 1 diabetic counterparts. Even though these patients have insulin resistance, exercise-mediated glucose uptake is not reduced. This means that diet-treated individuals are able to perform prolonged mild to moderate exercise and still have a marked reduction in hyperglycemia.

Before initiating a regular exercise program, all patients with diabetes should undergo a medical evaluation, be educated on the benefits and risks of exercise, and understand the potential effects exercise can have on their glucose levels (the interaction between food, insulin, and exercise). During any physical activity, muscles consume glucose at a rate of 2-3 mg/kg of body wt/minute of exercise. An exercise program should be started only when glucose levels are adequately controlled.

Exercise regimens differ for each type of diabetes and according to patients' preconception exercise habits.

Type 1 diabetic patients need to be watched closely for exercise-induced hypoglycemia. If they were exercising regularly before pregnancy, they should be able to continue an exercise program during pregnancy. Type 2 diabetic patients may either start or continue an exercise program depending on their preconception exercise habits. Sedentary individuals who wish to start exercising should do so after being evaluated by a physician. They should start on a low-intensity program, progressing in intensity and duration slowly and as tolerated. The progression should not exceed a 10% increase per week. Patients need to be advised of the warning signs to stop exercising and seek medical attention (Table 4). Appropriate exercises during pregnancy and the postpartum period include low-impact aerobics, swimming, stationary bicycling, walking, 65-cm Swiss ball exercises, yoga, light weights, and resistant band exercises.

Programs of moderate physical exercise have been shown to lower maternal glucose concentrations in women with GDM. The impact of exercise on any neonatal complications awaits clinical trials. The beneficial glucose-lowering effects of exercise warrant the encouragement of women without medical or obstetrical contraindications to start or continue a program of moderate exercise as a part of their treatment for GDM. Patients should be taught to palpate their uterus during exercise to detect contractions and to discontinue the exercise if contractions occur.

Physiological changes of pregnancy produce an alteration in the point of gravity (lordosis), an increase in elastin (joint and ligament laxity), and an accumulation of interstitial fluids (edema). These changes increase the risk for soft tissue injuries during exercise. The exercise prescription should take into account these potential injuries and be designed to prevent them as well as to strengthen other areas, such as the abdominal and lower back muscles. To prevent aortocaval compression and hypotension, exercises should be avoided in the supine position after the first trimester. Exercise heart rates should not exceed 140 bpm or 60-70% of VO2max.

During exercise there is preferential carbohydrate utilization, and this is most noticeable in non-weight-bearing exercises (bicycling and swimming). Exercise is safe when the glucose levels are between 90 and 140 mg/dl. The exercise program should be for < 45 minutes, and a meal should be consumed 1-3 hours before the exercise. Ideally, insulin should be given in the abdomen and should not be injected into active muscles (extremities). The insulin injection should occur ~ 1 hour before

Table 4. Warning Signs to Stop Exercising and Seek Medical Evaluation

- Vaginal bleeding
- Faintness
- Decreased fetal activity
- Generalized edema
- Low back pain

exercise. In addition, one should decrease the bolus insulin if its peak activity is going to coincide with the exercise period. Over time, the basal insulin may need adjustment.

Patients should avoid exercising at the time their insulin is peaking. It is imperative to monitor glucose levels, checking the level before exercise (if < 100 mg/dl, then take a supplemental snack), during exercise (if > 45 minutes), and after exercise (being aware of postexercise hypoglycemia). If the glucose level is > 250 mg/dl, then the urine should be checked for ketones. If the urine is positive for ketones, exercise should be delayed until the glucose is better controlled and ketones have resolved. If ketones are negative but the glucose level is > 300 mg/dl, then be cautious with exercise. Some authors recommend avoidance of exercise with hyperglycemia. Volume depletion from osmotic diuresis may cause orthostatic changes and predispose patients to injury. Patients should always carry some readily digestible carbohydrate and injectable glucagon in case of hypoglycemia.

Preconception Counseling

Because the planning of a pregnancy and early admittance for antenatal care are associated with a reduction in complications, every woman with diabetes in the reproductive age group must be counseled about contraception and family planning. At each office visit, these women should be asked if there is the slightest chance they could be pregnant. They should be counseled before becoming pregnant (preconception counseling) about optimizing metabolic control and planning their pregnancy. They should be aware of the risks of spontaneous abortion and birth defects associated with poor metabolic control.

Despite the risks involved, women of reproductive age with diabetes can become pregnant and give birth to healthy infants.

In addition, patients should be screened for risk factors for GDM at their initial visit (Table 5).

Table 5 . Risk Factors for GDM at the Initial Visit

- Marked obesity
- Personal history of GDM
- Glucose intolerance or glycosuria
- Strong family history of type 2 diabetes

Exercise Beliefs and Behaviors in Women

The unique physical changes of pregnancy coupled with inaccurate perceptions (e.g., exercise may be harmful to the fetus and recommendations are not appropriate or feasible) may keep many pregnant women from being active. For example, it was found that the physical limitations (e.g., nausea and vomiting) and a lack of time were the strongest barriers to exercise behavior during pregnancy and postpartum behavior. Because women at risk for GDM are likely to be less physically fit than women in general and are known to be more overweight and obese than pregnant women in general, the determinants of their exercise behavior may in fact be different from women without GDM.

In understanding exercise beliefs and behaviors in women with gestational diabetes mellitus it was found that ,

1) the strongest perceived advantage of exercise during pregnancy was controlling blood glucose and postpartum it was controlling weight,

2) the most common barrier to exercise during pregnancy was fatigue and postpartum it was a lack of time,

3) women's husband/partner most strongly influenced their exercise during pregnancy and postpartum,

4) women exercised more during the postpartum period than before or during pregnancy, and

5) the number of exercise advantages was positively associated with women's pregnancy and postpartum exercise behavior**.**

To increase exercise behavior and reduce the risk of type 2 diabetes in women with GDM, researchers and health care professionals are encouraged to use women's exercise beliefs, that is, advantages, social influences, and perceived barriers to exercise, as a framework for designing effective diabetes treatment.

Summary

To improve outcomes, pregnant women with type 2 diabetes should plan their pregnancy, maintain good metabolic control of their diabetes, exercise, and take folate daily. Ideally, an interdisciplinary team approach with centralized care offers the best outcomes.

Refereces:

1.Diabetes Management and Exercise in Pregnant Patients With Diabetes. George D. Harris, MD, MS, and Russell D. White, MD. Clinical Diabetes • Volume 23, Number 4, 2005

2.Understanding Exercise Beliefs and Behaviors in Women With Gestational Diabetes Mellitus. Danielle Symons Downs, PhD,Jan S. Ulbrecht, MD. Diabetes Care, Volume 29, Number 2, February 2006

Diabetes Myths

Continue .....

Myth #6 People with diabetes are more likely to get colds and other illnesses.

No. You are no more likely to get a cold or another illness if you have diabetes. However, people with diabetes are advised to get flu shots. This is because any infection interferes with your blood glucose management, putting you at risk of high blood glucose levels and, for those with type 1 diabetes, an increased risk of ketoacidosis.

Myth #7 Insulin causes atherosclerosis (hardening of the arteries) and high blood pressure.

No, insulin does not cause atherosclerosis. In the laboratory, there is evidence that insulin can initiate some of the early processes associated with atherosclerosis. Therefore, some physicians were fearful that insulin might aggravate the development of high blood pressure and hardening of the arteries. But it doesn't.

Myth #8 Insulin causes weight gain, and because obesity is bad for you, insulin should not be taken.

Both the UKPDS (United Kingdom Prospective Diabetes Study) and the DCCT (Diabetes Control & Complications Trial) have shown that the benefit of glucose management with insulin far outweighs (no pun intended) the risk of weight gain.

Myth #9 Fruit is a healthy food. Therefore, it is ok to eat as much of it as you wish.

Fruit is a healthy food. It contains fiber and lots of vitamins and minerals. Because fruit contains carbohydrate, it needs to be included in your meal plan. Talk to your dietitian about the amount, frequency and types of fruits you should eat.

Myth #10 You don't need to change your diabetes regimen unless your A_{1C} is greater than 8 percent.

The better your glucose control, the less likely you are to develop complications of diabetes. An A_{1C} in the sevens (7s), however, does not represent good control. The ADA goal is less than 7 percent. The closer your A_{1C} is to the normal range (less than 6 percent), the lower your chances of complications. However, you increase your risk of hypoglycemia, especially if you have type 1 diabetes. Talk with your health care provider about the best goal for you.

Congratulations !

The Winners of

diabetes Quiz Competition

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2. **Dr. S. M. Hasan Murad**
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3. **Dr. Md. Atiqul Islam**
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Editorial Note:

Dear Doctor, This issue of your *diabetes newsletter is* focused on *"Diabetes Management and Exercise in Pregnant"*. We appreciate your comments and queries. Please participate in quiz competition & win prizes.

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