Prevalence and risk factors of Type 2 diabetes in an urbanizing rural community of Bangladesh

Introduction:
It was reported that prevalence of type 2 diabetes is on the rise more in urban areas compared to rural population in Bangladesh. Bangladesh is one of the developing countries in the world, which is facing rapid urbanization in recent time. The purpose of the study was to observe the prevalence of type 2 diabetes and identify its’ risk factors in an urbanizing rural population in Bangladesh.

Methods:
Two villages were randomly selected from the rural areas of Gazipur district and total 975 subjects (>20 years), were included following simple random procedure. Capillary blood glucose levels, fasting blood glucose (FBG) levels and 2-hour after 75 g oral glucose load (OGTT) were measured. Height, weight, waist and hip circumferences and blood pressure were measured.

Results:
The study population was lean with mean body mass index (BMI) of 20.48. The total prevalence of type 2 diabetes was 8.5%, men showed higher prevalence (9.4%) compare to women (8.0%). Increasing age and higher BMI were found to be significant risk factors following both FBG and OGTT.

Conclusions:
In conclusion, the higher prevalence in the present study indicates the environmental factors may encompass a strong role for the rising prevalence of diabetes in urbanizing population in a developing country like Bangladesh. FBG showed higher prevalence compare to OGTT in our population. In this context FBG is suitable diagnostic tools for diagnosis of diabetes in epidemiological study rather than OGTT.

**Diabetescope**

**Metformin for Obesity in Children and Adolescents**

Metformin has been shown to reduce weight gain, hyperinsulinemia, and hyperglycemia in adults with type 2 diabetes and to reduce progression from impaired glucose tolerance to diabetes in those without diabetes. These benefits have led to an increase in the use of metformin in obese children with hyperinsulinemia. However, obesity is not a licensed indication for metformin and its use has proceeded faster than the evidence of its benefits. This systematic review of randomized controlled trials (RCTs) investigating the efficacy of metformin for reducing BMI and cardiometabolic risk in obese children without diabetes.

A systematic review and meta-analysis of randomized controlled trials (RCTs). Double-blind RCTs of >6 months duration in obese subjects age <19 years without diabetes were included. Primary outcomes of interest include changes in BMI and measures of insulin sensitivity.

Five trials met inclusion criteria (n = 320 individuals). Compared with placebo, metformin reduced BMI by 1.42 kg/m² and homeostasis model assessment insulin of resistance (HOMA-IR) score by 2.01.

The results of this review must be interpreted with caution: the studies were short-term and based on small samples; participants were mainly from the U.S., limiting the generalizability of findings; and the studies presented unadjusted measures without intention-to-treat analyses, which may have overestimated treatment effects.

Metformin may be efficacious in reducing BMI and insulin resistance among obese hyperinsulinemic children and adolescents in the short term. Larger, long-term studies across different populations are needed to establish the role of metformin as therapy for obesity and cardiometabolic risk in young people.

*Source: Diabetes Care 2008;31:2357-2361.*

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**Hypoglycemia Associated with the Use of Levofloxacin**

A case of hypoglycemia associated with levofloxacin is reported.

A 58-year-old Caucasian man was admitted to the hospital for a heart failure (HF) exacerbation with suspected community-acquired pneumonia (CAP). His medical history included HF (left ventricular ejection fraction, 25–35%), hypertension, and type 2 diabetes mellitus. Renal insufficiency was noted during hospitalization, with a serum creatinine concentration of 1.5 mg/dL. The patient’s only home medication was a self-reported “sugar pill,” later identified as glimepiride. A chest radiograph revealed consolidation in both lung bases and bilateral pleural effusions. Levofloxacin 750 mg was administered orally on hospital day 1 for the treatment of CAP and was ordered to be administered every 48 hours. On hospital day 3, glipizide 10 mg was administered with a sliding-scale regimen of regular insulin in preparation for discharge. On hospital day 4, glipizide 10 mg was given again with the second dose of levofloxacin, 65 hours after the first levofloxacin dose was administered. The patient also received furosemide 40 mg orally twice daily, lisinopril 20 mg orally daily, and metoprolol 25 mg twice daily. The patient was discharged on hospital day 4 and returned to the emergency department early the next morning with a serum glucose concentration of 20 mg/dL. An i.v. infusion of 10% dextrose injection and three ampuls of 50% dextrose injection were given to correct his hypoglycemia. Further glipizide doses were not administered.

Conclusion: A malnourished 58-year-old man with diabetes developed hypoglycemia after receiving levofloxacin in conjunction with glipizide.

*Source: American Journal of Health-System Pharmacy. 2009;66(11):1014-1019*

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Metformin is an oral hypoglycemic agent used commonly for the treatment of non-insulin dependent diabetes mellitus. Metformin is now shown in a laboratory study to have direct anti-tumor effect against endometrial cancer. Recent epidemiological evidence suggests that metformin may lower cancer risk and reduce rates of cancer deaths among diabetic patients. In general, patients with type 2 diabetes are at increased risk for cancer mortality. In a study from the Netherlands published last year, metformin use was associated with lower cancer mortality when compared to non-metformin use. Diabetic patients with breast cancer receiving metformin and neoadjuvant chemotherapy (i.e., chemotherapy administered before surgery) have a higher pathologic complete remission rate than do diabetics not receiving metformin. There is evidence to suggest that metformin has a positive impact on inflammation and endothelial dysfunction. Metformin was compared with another oral hypoglycaemic agent, repaglinide, in nonobese patients with type 2 diabetes and shown to be more effective in reducing levels of tumor necrosis factor alpha, plasminogen activator inhibitor-1 antigen, tissue-type plasminogen activator antigen, von Willebrand factor, soluble intercellular adhesion molecule-1, and soluble E-selectin. Metformin was also shown to be capable of producing a significant decrease in the levels of vascular endothelial growth factor and plasminogen activator inhibitor-1. Now a new study shows that in addition to its indirect effects, metformin can significantly and directly inhibit the growth of ovarian cancer cell lines. Metformin potently inhibited the growth of endometrial cancer cells in a dose-dependent manner.

Metformin may be of value as an adjunct to conventional chemotherapy, in a wide spectrum of malignant diseases, and not only in patients with diabetes.


The Diabetes Prevention Program (DPP) was a randomized clinical trial involving 3234 volunteers at high risk for diabetes. All participants had impaired glucose tolerance (IGT), defined as having a fasting plasma glucose level of 5.3-6.9 mmol/L and attaining a 2-hour glucose of 7.8-11.0 mmol/L during a 75-g oral glucose tolerance test. The study discussed here evaluated a subgroup of participants who also had impaired fasting glucose (IFG), defined by the American Diabetes Association as a baseline fasting glucose of > 5.6 mmol/L and < 7.0 mmol/L.

Regression from this combined IFG/IGT state to normal glucose regulation (NGR) (fasting glucose < 5.6 mmol/L and 2-hour glucose < 7.8 mmol/L) was the primary outcome measure, and regression to isolated IFG or isolated IGT was the secondary outcome measure. In addition, the study sought to identify predictors for regression to NGR, isolated IFG, or isolated IGT within each treatment group (intensive lifestyle modification or metformin vs placebo) using Cox regression analyses.

At the first annual examination, 16.5% of the 2528 study participants had regression to NGR. Of those who still had combined IFG/IGT, another 8% had regression to NGR at year 2, and among the remaining volunteers who still had IFG/IGT, another 4.4% had regression by year 3. Thus, a total of 600 (23.7%) participants had regression to NGR within 3 years. Lower baseline fasting and 2-hour glucose predicted regression to NGR, as did younger age and a greater insulin secretion to the oral glucose load. The intensive lifestyle intervention group was twice as likely to have regression as the placebo group (hazard ratio 2.05; 95% confidence interval 1.66-2.53), but the trend toward greater regression in the metformin group was not statistically significant (hazard ratio 1.25; 95% confidence interval 0.99-1.58). Greater weight loss also had a significant and independent effect on regression to NGR (hazard ratio 1.34; 95% confidence interval 1.21-1.49).

Source: Diabetes Care. 2009;32:1583-1588
Introduction

The association between diabetes mellitus and tuberculosis and their synergistic role in causing human disease has been recognised for centuries. Ancient works by Yugimahamuni, an Indian siddhar, describe the symptoms of patients with "meganoikal" (urinary disorders), which progressed from obesity to impotence, thirst, and glycosuria, and ultimately, to unconsciousness or tuberculosis. The introduction of insulin in the 1920s, the discovery of streptomycin in the 1940s, and the subsequent development of other antibiotics substantially lowered case fatality rates for individuals with diabetes mellitus or tuberculosis. Improved sanitation, better nutrition, and less crowding led to markedly diminished tuberculosis incidence. In recent decades, tuberculosis has increasingly become a problem in low-income countries, particularly those with HIV epidemics, and non-insulin-dependent diabetes mellitus (NIDDM) has emerged as a growing worldwide chronic health condition, as a consequence of increases in obesity, changing patterns of diet and physical activity, and aging populations. The effect of diabetes on the development and severity of tuberculosis, and the complex interrelations between nutrition, obesity, diabetes, and tuberculosis remain provocative issues in public health and clinical medicine. In the setting of the increasing overlap of populations at risk for both diseases, the combination of tuberculosis and diabetes mellitus represents a worldwide health threat.

Double burden of tuberculosis and diabetes

The burden of communicable diseases is concentrated in low-income countries. However, non-communicable diseases, which represented 47% of the disease burden in 1990 in low-income countries, have been predicted to rise to 69% by 2020. The number of people with diabetes, which was 171 million in 2000, is expected to grow to 366 million–440 million by 2030, with three-quarters of patients with diabetes living in low-income countries (figure). Diabetes poses a large financial burden in countries with limited resources. In many countries, insulin is expensive or availability is poor: a 1-month supply of insulin can cost up to 20 days’ wages. Thus, social and economic conditions heavily influence treatment options.

In these resource-limited settings, tuberculosis continues to be have high mortality. Whereas the most common causes of death in low-income and middle-income countries are ischaemic heart disease and cerebrovascular disease, HIV and tuberculosis are in the top five causes of death. Tuberculosis, poverty, and poor access to health services are closely linked, complicating provision of tuberculosis care. Comorbidities such as diabetes mellitus complicate tuberculosis care further. Several studies show that coaffliction with tuberculosis and diabetes mellitus is common, both in low-income and high-income countries.

Effect of diabetes on tuberculosis risk and severity

Historically, the incidence of tuberculosis in patients with diabetes has been high. In 1934, a treatise on the association between diabetes and tuberculosis was written by Howard Root (a physician at the Deaconess Hospital, USA), before the availability of antimycobacterial drugs. His lengthy tome described the epidemiology, pathology, and clinical course of dually affected patients. In his studies, tuberculosis in adults with diabetes was more common than expected, and risk was particularly high in school children and adolescents with diabetes. In his autopsy series of 126 patients, no pathological findings unique to "the tubercular diabetic" were discovered. Among a total of 245 tuberculosis cases in diabetic patients, he found "no special insidiousness" of signs and symptoms, and similar radiographic findings to those of non-diabetic patients. In the Philadelphia Diabetic Survey, Boucot and colleagues found a two-fold increase in prevalent tuberculosis by chest radiograph in 3106 diabetic patients compared with 70767 controls of similar demographics. Furthermore, they found that diabetic patients who needed more than 40 units of insulin per day were twice as likely to develop tuberculosis as those using lower doses, thus linking severity of diabetes mellitus with risk of tuberculosis.

In the past 20 years, the debate over whether diabetes mellitus causes increased susceptibility to tuberculosis, as well as differences in presentation, severity, and response to therapy, has been rekindled.

Tuberculosis incidence in patients with diabetes

The risk of developing active tuberculosis is a two-step process, beginning with initial exposure to and infection by Mycobacterium tuberculosis followed by subsequent progression to disease. Studies of diabetes mellitus and tuberculosis generally focus on active tuberculosis disease. However, in one study in Spain, 69(42%) of 163 diabetic patients had a positive tuberculin skin test, suggesting a high rate of latent tuberculosis in diabetic patients, although this could have been confounded by age and there was no control group. Several case–control studies have shown that the relative odds of developing tuberculosis in diabetic patients ranges from 2.44 to 8.33 compared with non-
Several largescale longitudinal cohort studies have shown similar findings. In Korea, a 3-year longitudinal study involving 800000 civil servants showed that the risk ratio of tuberculosis in diabetic patients versus nondiabetic controls was 3.47 (95% CI 2.98–4.03). In a study of the UK General Practice Research Database, which includes records from over 2 million patients, Jick and colleagues identified all cases of tuberculosis reported between 1990 and 2001 and compared them with controls, and found that the adjusted odds ratio for tuberculosis was 3.8 (95% CI 2.3–6.1) for diabetic patients compared with those without diabetes. In Hong Kong, in a 5-year study of 42000 elderly individuals, the adjusted hazard of active tuberculosis was higher in diabetic patients than in individuals without diabetes (1.77; 95% CI 1.41–2.24), but this increased risk was only present in those with haemoglobin A1c concentrations greater than 7%. These large studies involving thousands of participants provide convincing data that diabetes mellitus is a moderate-to-strong risk factor for the development of active tuberculosis.

If diabetes is associated with tuberculosis, one might ask whether severity of diabetes is related to the magnitude of risk. Two studies have compared the incidence of active tuberculosis between insulin-dependent diabetes mellitus (IDDM) and NIDDM. In a cohort of 1529 diabetic individuals in Chile, who were followed prospectively from 1959 to 1982, the 10-year actuarial probability of developing tuberculosis was 24% in IDDM and 4.8% in NIDDM. In a prospective study of diabetic patients followed for 1–7 years in Tanzania, 9.0% of patients with IDDM and 2.7% of patients with NIDDM developed pulmonary tuberculosis. These two studies provide evidence that insulin dependence, as a marker for severity of disease, predicts increased tuberculosis risk. In a recent study of 4690 elderly diabetic patients in Hong Kong, those with haemoglobin A1c greater than 7% had a three times increased hazard of active tuberculosis compared with those with haemoglobin A1c less than 7% (hazard ratio 3.11; 95% CI 1.63–5.92). These data suggest that poor glycaemic control is a risk factor for tuberculosis.

Although there is no reason, a priori, to expect an association with diabetes mellitus and drug resistance, two studies have shown that diabetic patients are more likely to
develop multidrug-resistant tuberculosis than those without diabetes. However, four studies in disparate settings showed no significant increased risk. The mechanism by which diabetes mellitus would lead to preferential acquisition of multidrug-resistant tuberculosis is unclear.

Radiographic findings in tuberculous diabetic patients

The radiographic presentation of tuberculosis depends on many factors, including duration of illness and host immune status. In 1927, Sosman and Steidl reported that a large proportion of diabetic patients with tuberculosis had lower-lung involvement, whereas nondiabetic patients usually had upper-lobe infiltrates. Subsequent studies in the 1970s and 1980s corroborated this finding.49,50 and it was widely believed that pulmonary tuberculosis in diabetic patients presented with an atypical radiographic pattern and distribution, particularly lower-lung involvement. Clinically, this is important because lower-lobe tuberculosis might be misdiagnosed as community-acquired pneumonia or cancer. Also, patients with pulmonary tuberculosis that do not have upper-lobe involvement are less likely to have positive sputum smears and cultures. Whereas in one series, 20% of patients with diabetes mellitus presented with lower-lobe involvement, in other studies, lower-lobe involvement was only seen in 1.8% (8 of 438 patients) and 8.3% (1 of 12 patients). Subsequent studies have yielded mixed results.

Of note, older individuals are more likely to have lower-lobe involvement, and preferential changes in lower-lobe alveolar oxygen tension related to age or diabetes mellitus has been suggested to favour lower lobe disease in these groups. In most series, multilobar disease or the presence of multiple cavities was more common in diabetic patients, but lower-lung disease was rarely more common in diabetic patients than in controls, except, perhaps, in patients aged over 40 years. Results vary substantially between studies, and the frequency of unusual radiographic findings in diabetic patients has probably been overstated.

Severity of disease and outcomes in diabetic patients with tuberculosis

Mycobacterial burden, culture conversion, and relapse

If diabetes alters immunity to tuberculosis, leading to higher baseline mycobacterial burdens and longer times to culture conversion with treatment, a higher rate of relapse might result. Three small retrospective studies suggest that baseline mycobacterial burdens might be higher in diabetic patients than in controls. However, results of studies assessing sputum-culture conversion show mixed results depending on the outcome variable used. In studies that assessed sputum-culture conversion after at least 2 months of treatment (a common surrogate marker used to predict tuberculosis relapse), conversion proportions were similar in diabetic patients and controls. For example, in a study in Indonesia, diabetes was not a risk factor for sputum smear or sputum-culture positivity at 2 months after adjustment for age, sex, body–mass index, study site, chest radiographic findings, and baseline sputum mycobacterial load. Similarly, among 692 smear-positive tuberculosis patients in Saudi Arabia, 98.9% of diabetic patients and 94.7% of controls had negative sputum cultures at 3 months. However, in studies assessing time to sputum-culture conversion, diabetic patients seem to take longer to achieve culture negativity. In one study in Turkey, patients with diabetes who received tuberculosis treatment had longer sputum-culture conversion times than non-diabetic patients (67 vs 55 days; p=0.02). In a study that used survival analysis to measure time to culture conversion, median time to culture negativity was significantly longer in diabetic patients than in controls (42 vs 37 days; p=0.03). Using similar techniques, a third study also found a trend toward increased median time to culture conversion in diabetic patients (49 vs 39 days; p=0.09). Together, these data suggest that although bacillary burden might be higher at presentation in diabetic patients, leading to modestly longer times to sputum-culture conversion, rates of sputum-culture conversion are similar to those of non-diabetic patients by 2–3 months of treatment. Whether increased time to culture conversion in diabetic patients leads to higher risk of relapse has not been adequately studied.

Treatment failure and death

In one study in Egypt, which compared 119 patients with treatment failure to 119 controls, diabetes conferred a 3.9 times increased risk of treatment failure in patients receiving directly observed short-course therapy. In a study in Indonesia in patients with high adherence to treatment, 6-month sputum cultures were positive in 22.2% of patients with diabetes mellitus and in 6.9% of controls. Importantly, drug resistance was lower, and medication adherence was higher in diabetic patients; so increased failure was not due to resistance or non-adherence to treatment. In a descriptive case–control study by Mboussa and colleagues, treatment failure or death was seen in 41% of the patients with tuberculosis and diabetes mellitus, but in only 13% of those with tuberculosis alone. Of the eight patients who died in the tuberculosis and diabetes group, seven patients died of respiratory failure related to tuberculosis whereas one patient died of diabetic coma.

Two retrospective cohort studies of patients with pulmonary tuberculosis in Maryland, USA, have shown a 6.5–6.7 times increased risk of death in diabetic patients compared to...
non-diabetic controls. In a recent study by Wang and colleagues, 1-year all-cause mortality was 17.6% in diabetic patients versus 7.7% in non-diabetic controls, and death specifically attributable to pulmonary tuberculosis was significantly more common in diabetic patients (12.2% vs 4.2%). These studies suggest that treatment failure and death are more frequent in diabetic patients. However, whether aggressive management of diabetes mellitus would improve treatment response remains unclear. Furthermore, because causes of death are not reported in most studies, we do not know whether excess mortality is explained by increased severity of tuberculosis in diabetic patients or by the existence of comorbidities attributable to diabetes mellitus compounded by more advanced age.

How might diabetes mellitus lead to tuberculosis?

Diabetes might also lead to increased susceptibility to disease caused by M tuberculosis via multiple mechanisms. The mechanisms include those directly related to hyperglycaemia and cellular insulinopenia, as well as indirect effects on macrophage and lymphocyte function, leading to diminished ability to contain the organism. Studies point to depressed immunological function in IDDM and NIDDM that might predispose a patient to infections for which cell mediated immunity has a pivotal role, such as tuberculosis. Decreased phagocyte and T-cell function are likely contributors. The implications of diabetes related differences in the immune response to tuberculosis are being investigated. The relative contribution of genetics, vitamin deficiencies, and other factors to increased risk of tuberculosis in diabetic patients remains to be established.

Does tuberculosis lead to diabetes?

Infections often worsen glycaemic control in diabetic patients, and poorly controlled diabetes might in turn augment the severity of infections. Some studies suggest that tuberculosis can even cause diabetes. Many studies have used oral glucose tolerance testing to show that patients with tuberculosis have higher rates of glucose intolerance than community controls. Whereas the high incidence of abnormal oral glucose tolerance found in tuberculosis patients is of concern, it is unclear whether glucose intolerance or diabetes mellitus was truly incident, or whether prevalent diabetes mellitus was being newly diagnosed in patients receiving expanded medical services related to tuberculosis treatment. Also, the implications of these findings depend on whether diabetes persists in these patients, and whether its presence is substantially more common with tuberculosis than with other infectious diseases.

Pharmacological issues in the co-management of diabetes mellitus and tuberculosis

Infections are known to worsen diabetic control, and tuberculosis is no exception. Although tuberculosis can cause glucose intolerance and might predispose patients to diabetes mellitus, the drugs used to treat tuberculosis might also worsen glycaemic control in patients with diabetes. Overlapping toxicities must also be considered when co-managing tuberculosis and diabetes, such as peripheral neuropathy caused by treatment with isoniazid. Given the risk of peripheral neuropathy, pyridoxine should be given with isoniazid during tuberculosis treatment in diabetic patients. In addition, treatment with rifampicin can cause hyperglycaemia directly or indirectly via interactions with oral hypoglycaemic drugs.

Rifampicin is a powerful inducer of a host of metabolising enzymes, including cytochrome P450 system enzymes and phase II enzymes. Induction of these enzymes can lead to accelerated metabolism of drugs given with rifampicin and reduced treatment effect. The sulfonylureas are among the most commonly used oral hypoglycaemic drugs for patients with NIDDM. Glyburide and glipizide are both substrates of cytochrome P450 isoenzyme 2C9 (CYP2C9), and pharmacokinetic studies show that serum concentrations of these drugs are decreased by 39% and 22%, respectively, when given with rifampicin. Pharmacodynamic data further show that glyburide’s hypoglycaemic effect is reduced when given with rifampicin. Thiazolidinediones are often used as substrates for the cytochrome P450 enzymes. Rosiglitazone is metabolised largely by CYP2C8, and rifampicin decreases concentrations of rosiglitazone by 54–65% and of the related drug pioglitazone by 54%. Nateglinide, a short-acting insulin secretagogue given to prevent postprandial hyperglycaemia, is metabolised by oxidative bio transformation, with involvement from CYP2C9 and CYP3A4; its area under the curve is reduced by only 24% with no appreciable glycaemic effect when given with rifampicin. Repaglinide, another related drug, had an area under the curve that was decreased by 31–57% when given with rifampicin, although its glucose-lowering effect was reduced in one study and unchanged in another. In patients with IDDM, insulin requirements might increase when on rifampicin. Rifampicin has been shown to cause early-phase hyperglycaemia with associated hyperinsulinaemia even in non-diabetic patients. Rifampicin’s direct and indirect effects on glycaemic control make careful monitoring with appropriate dose adjustment of diabetic agents essential in diabetic patients with tuberculosis.
Just as tuberculosis drug treatment affects diabetes treatment, diabetes might alter the pharmacokinetics of antituberculosis drugs. In one study in Indonesia, diabetic patients with tuberculosis had rifampicin serum concentrations that were 53% lower than in non-diabetic patients with tuberculosis, and there was an indirect relation between fasting glucose and rifampicin concentrations. Given that low concentrations of antituberculosis drugs have been linked to treatment failure or resistance, this finding is of particular concern. Diabetes can also cause changes in oral absorption, decreased protein binding of drugs, and renal insufficiency or fatty liver with impaired drug clearance. Its effect on tuberculosis drug concentrations has not been formally studied; in cases of poor response to treatment in diabetic patients with tuberculosis, therapeutic drug monitoring might be considered.


Congratulations!

The Winners of diabetes Quiz Competition

Vol.7, No.4, Nov 09- Jan 10

1. Dr. Shafiqul Hasan
   MBBS, FCPS (Med)
   PDC Hospital, Pabna

2. Dr. Iqbal Hossain Sagor
   MBBS, FCPS
   Medical Officer, Porsha UHC, Naogaon

3. Dr. Kazi Mustafizur Rahman
   Senior Medical Officer
   Ahad Diabetic & Health Complex, Jessore

4. Dr. S. M. A. Habib
   MBBS, FCGP, MCPS
   Hossain Pharmacy, Kalirbazar, Naryangonj

5. Dr. Elias Chowdhury
   MBBS, CCD
   Jononi Diagnostic, Nazirhat, Chittagong