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Study of level of vitamins and HbA1C status in patients suffered from tuberculosis along with diabetes from Bihar state

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Abstract
Exogenous and endogenous risk factors govern the risk of progression from exposure to the tuberculosis bacilli to the development of active disease. An exogenous factor makes the progression from exposure to infection more prominent among which the key factors are the bacillary load in the sputum and the proximity of an individual to an infectious TB case. And progression from infection to active TB disease is lead by endogenous factors. Emerging factors such as diabetes, indoor air pollution, alcohol, Immunosuppressive drugs, tobacco smoke also play a significant role along with well-established risk factors. Hence based on above reported findings the present study was planned for Study of Level of Vitamins and HbA1C Status in Patients Suffered from Tuberculosis Along with Diabetes from Bihar Region.
The present study was planned in Department of General Medicine, Nalanda Medical College and Hospital Patna, Bihar, India. The 20 cases of the diabetes diagnosed with the tuberculosis were evaluated as cases and 20 cases of the normal cases were evaluated as a control cases.
The data generated from the present study concludes that the levels of the HbA1C were increases in Diabetes patients with TB whereas the levels of the Vitamin D and Vitamin B12 were decreases in diabetic patients with TB.

Keywords: levels of vitamin and HbA1C status, tuberculosis, diabetes, etc

Introduction
Diabetes mellitus (DM), commonly known as diabetes, is a group of metabolic disorders characterized by a high blood sugar level over a prolonged period of time. Symptoms often include frequent urination, increased thirst, and increased hunger. If left untreated, diabetes can cause many complications. Acute complications can include diabetic ketoacidosis, hyperosmolar hyperglycemic state, or death. Serious long-term complications include cardiovascular disease, stroke, chronic kidney disease, foot ulcers, damage to the nerves, and damage to the eyes.
Diabetes is due to either the pancreas not producing enough insulin, or the cells of the body not responding properly to the insulin produced. There are three main types of diabetes mellitus:
Type 1 diabetes results from the pancreas's failure to produce enough insulin due to loss of beta cells. This form was previously referred to as "insulin-dependent diabetes mellitus" (IDDM) or "juvenile diabetes". The loss of beta cells is caused by an autoimmune response. The cause of this autoimmune response is unknown.
Type 2 diabetes begins with insulin resistance, a condition in which cells fail to respond to insulin properly. As the disease progresses, a lack of insulin may also develop. This form was previously referred to as "non-insulin-dependent diabetes mellitus" (NIDDM) or "adult-onset diabetes". The most common cause is a combination of excessive body weight and insufficient exercise. Gestational diabetes is the third main form, and occurs when pregnant women without a previous history of diabetes develop high blood sugar levels [1].
Prevention and treatment involve maintaining a healthy diet, regular physical exercise, a normal body weight, and avoiding use of tobacco. Control of blood pressure, maintaining proper foot care, and eye care are important for people with the disease. Type 1 diabetes must be managed with insulin injections. Type 2 diabetes may be treated with medications with or without insulin. Insulin and some oral medications can cause low blood sugar. Weight loss surgery in those with obesity is sometimes an effective measure in those with type 2 diabetes. Gestational diabetes usually resolves after the birth of the baby [2].
As of 2017, an estimated 425 million people had diabetes worldwide, with type 2 diabetes making up about 90% of the cases. This represents 8.8% of the adult population, with equal rates in both women and men. Trends suggest that rates will continue to rise. Diabetes at least doubles a person's risk of early death. In 2017, diabetes resulted in approximately 3.2 to 5.0 million deaths. The global economic cost of diabetes related health expenditure in 2017 was estimated at US$727 billion. In the United States, diabetes cost nearly US$245 billion in 2012. Average medical expenditures among people with diabetes are about 2.3 times higher [3].
The classic symptoms of untreated diabetes are unintended weight loss, polyuria (increased urination), polydipsia (increased thirst), and polyphagia (increased hunger). Symptoms may develop rapidly (weeks or months) in type 1 diabetes, while they usually develop much more slowly and may be subtle or absent in type 2 diabetes. Other symptoms of diabetes include weight loss and tiredness.
Several other signs and symptoms can mark the onset of

diabetes although they are not specific to the disease. In addition to the known ones above, they include blurred vision, headache, fatigue, slow healing of cuts, and itchy skin. Prolonged high blood glucose can cause glucose absorption in the lens of the eye, which leads to changes in its shape, resulting in vision changes. Long-term vision loss can also be caused by diabetic retinopathy. A number of skin rashes that can occur in diabetes are collectively known as diabetic dermadromes [4].

All forms of diabetes increase the risk of long-term complications. These typically develop after many years (10–20) but may be the first symptom in those who have otherwise not received a diagnosis before that time.

The major long-term complications relate to damage to blood vessels. Diabetes doubles the risk of cardiovascular disease and about 75% of deaths in people with diabetes are due to coronary artery disease. Other macrovascular diseases include stroke, and peripheral artery disease.

The primary complications of diabetes due to damage in small blood vessels include damage to the eyes, kidneys, and nerves. Damage to the eyes, known as diabetic retinopathy, is caused by damage to the blood vessels in the retina of the eye, and can result in gradual vision loss and eventual blindness. Diabetes also increases the risk of having glaucoma, cataracts, and other eye problems. It is recommended that people with diabetes visit an eye doctor once a year. Damage to the kidneys, known as diabetic nephropathy, can lead to tissue scarring, urine protein loss, and eventually chronic kidney disease, sometimes requiring dialysis or kidney transplantation. Damage to the nerves of the body, known as diabetic neuropathy, is the most common complication of diabetes [5]. The symptoms can include numbness, tingling, pain, and altered pain sensation, which can lead to damage to the skin. Diabetes-related foot problems (such as diabetic foot ulcers) may occur, and can be difficult to treat, occasionally requiring amputation. Additionally, proximal diabetic neuropathy causes painful muscle atrophy and weakness.

There is a link between cognitive deficit and diabetes. Compared to those without diabetes, those with the disease have a 1.2 to 1.5-fold greater rate of decline in cognitive function. Having diabetes, especially when on insulin, increases the risk of falls in older people.

Type 2 diabetes is characterized by insulin resistance, which may be combined with relatively reduced insulin secretion. The defective responsiveness of body tissues to insulin is believed to involve the insulin receptor. However, the specific defects are not known. Diabetes mellitus cases due to a known defect are classified separately. Type 2 diabetes is the most common type of diabetes mellitus. Many people with type 2 diabetes have evidence of prediabetes (impaired fasting glucose and/or impaired glucose tolerance) before meeting the criteria for type 2 diabetes. The progression of prediabetes to overt type 2 diabetes can be slowed or reversed by lifestyle changes or medications that improve insulin sensitivity or reduce the liver's glucose production [6].

Type 2 diabetes is primarily due to lifestyle factors and genetics. A number of lifestyle factors are known to be important to the development of type 2 diabetes, including obesity (defined by a body mass index of greater than 30), lack of physical activity, poor diet, stress, and urbanization. Excess body fat is associated with 30% of cases in those of Chinese and Japanese descent, 60–80% of cases in those of European and African descent, and 100% of Pima Indians and Pacific Islanders. Even those who are not obese often have a high waist–hip ratio [7].

Dietary factors also influence the risk of developing type 2 diabetes. Consumption of sugar-sweetened drinks in excess is associated with an increased risk. The type of fats in the diet is also important, with saturated fat and trans fats increasing the risk and polyunsaturated and monounsaturated fat decreasing the risk. Eating lots of white rice also may increase the risk of diabetes, whereas substitution of brown rice or other whole grains for white rice may lower the risk of diabetes. A lack of physical activity is believed to cause 7% of cases [8].

Tuberculosis (TB) is an infectious disease usually caused by Mycobacterium tuberculosis (MTB) bacteria. Tuberculosis generally affects the lungs, but can also affect other parts of the body. Most infections do not have symptoms, in which case it is known as latent tuberculosis. About 10% of latent infections progress to active disease which, if left untreated, kills about half of those affected. The classic symptoms of active TB are a chronic cough with blood-containing mucus, fever, night sweats, and weight loss. It was historically called "consumption" due to the weight loss. Infection of other organs can cause a wide range of symptoms [9].

Tuberculosis is spread through the air when people who have active TB in their lungs cough, spit, speak, or sneeze. People with latent TB do not spread the disease. Active infection occurs more often in people with HIV/AIDS and in those who smoke. Diagnosis of active TB is based on chest X-rays, as well as microscopic examination and culture of body fluids. Diagnosis of latent TB relies on the tuberculin skin test (TST) or blood tests [9].

Prevention of TB involves screening those at high risk, early detection and treatment of cases, and vaccination with the bacillus Calmette-Guérin (BCG) vaccine. Those at high risk include household, workplace, and social contacts of people with active TB. Treatment requires the use of multiple antibiotics over a long period of time. Antibiotic resistance is a growing problem with increasing rates of multiple drug-resistant tuberculosis (MDR-TB) and extensively drug-resistant tuberculosis (XDR-TB).

A number of factors make people more susceptible to TB infections. The most important risk factor globally is HIV; 13% of all people with TB are infected by the virus. This is a particular problem in sub-Saharan Africa, where rates of HIV are high. Of people without HIV who are infected with tuberculosis, about 5–10% develop active disease during their lifetimes; in contrast, 30% of those coinfected with HIV develop the active disease [10].

Tuberculosis is closely linked to both overcrowding and malnutrition, making it one of the principal diseases of poverty. Those at high risk thus include: people who inject illicit drugs, inhabitants and employees of locales where vulnerable people gather (e.g. prisons and homeless shelters), medically underprivileged and resource-poor communities, high-risk ethnic minorities, children in close contact with high-risk category patients, and health-care providers serving these patients. Chronic lung disease is another significant risk factor. Silicosis increases the risk about 30-fold. Those who smoke cigarettes have nearly twice the risk of TB compared to nonsmokers. Other disease states can also increase the risk of developing tuberculosis. These include alcoholism and diabetes mellitus (three-fold increase) [11].

Certain medications, such as corticosteroids and infliximab (an anti-αTNF monoclonal antibody), are other important risk factors, especially in the developed world. Genetic susceptibility also exists, for which the overall importance remains undefined.

Diabetes is a chronic (long-lasting) disease that affects how the body turns food into energy. Tuberculosis (TB) is a serious health threat, especially for people living with diabetes. Two TB-related conditions exist: latent TB infection and TB disease. People with latent TB infection are not sick because the body is able to fight the bacteria to stop them from growing. People with TB disease are sick and have active TB because the body cannot stop the bacteria from growing. People living with diabetes who are also infected with TB are more likely to develop TB disease and become sick with TB. Someone with untreated latent TB infection and diabetes is more likely to develop TB disease than someone without diabetes. Without proper treatment, diabetes and TB can increase health complications.

Untreated latent TB infection can progress to TB disease. TB disease, without treatment, can progress from sickness to death. Fortunately, treatment options are available for people with diabetes who also have either latent TB infection or TB disease. If a person is diagnosed with TB infection, further testing is required to rule out TB disease. People with either latent TB infection or TB disease can be effectively treated. Before beginning treatment for TB disease or for latent TB infection, TB patients should talk to their doctor about any other medication they are taking, including medicine for diabetes. Some medications used to treat TB might interact with medicine used treat diabetes [12].

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 [13]. 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 [14]. 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.6–8 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 [15].

Exogenous and endogenous risk factors govern the risk of progression from exposure to the tuberculosis bacilli to the development of active disease. An exogenous factor makes the progression from exposure to infection more prominent among which the key factors are the bacillary load in the sputum and the proximity of an individual to an infectious TB case. And progression from infection to active TB disease is lead by endogenous factors. Emerging factors such as diabetes, indoor air pollution, alcohol, Immunosuppressive drugs, tobacco smoke also play a significant role along with well-established risk factors. Hence based on above reported findings the present study was planned for Study of Levels of Vitamin and HbA1C Status in Patients Suffered from Tuberculosis Patients Along with Diabetes from Bihar Region.

Methodology

The present study was planned in Department of General Medicine, Nalanda Medical College and Hospital Patna, Bihar, India. The 20 cases of the diabetes diagnosed with the tuberculosis were evaluated as cases and 20 cases of the normal cases were evaluated as a control cases.

All the patients were informed consents. The aim and the objective of the present study were conveyed to them. Approval of the institutional ethical committee was taken prior to conduct of this study.

Following was the inclusion and exclusion criteria for the present study.

Inclusion criteria: Participants aged 30–65 years with T2DM and on oral anti diabetics and Tuberculosis (TB) Participants who gave informed consent; T2DM participants with Vitamin D deficiency and poor glycemic control as evidenced by HbA1c > 6.5%.

Exclusion criteria: Participants below 30 years or above 65 years; Participants with T1DM T2DM participants on insulin (due to influence of insulin antibodies on serum insulin assay); pregnant women (serum Vitamin D levels are generally low in pregnancy).

Participants with chronic diseases including renal insufficiency (glomerular filtration rate <30 ml/min), history of chronic liver disease or alanine transferase >5 times upper reference limit, tuberculosis, diarrheal, or malabsorption state.

Results and Discussion

Individuals in lower income countries, where the majority of the world's TB burden is located, are more likely to report symptoms of active TB disease if they also reported a prior diagnosis of T2DM. At the population level, between the 1990s and early 2000s, TB prevalence and incidence were more likely to increase in countries in which diabetes prevalence increased, conditioning on base year, percapita gross domestic product (GDP) [16]. It has been seen that patients with DM and severe vitamin D deficiency are more susceptible to develop PMT infection than those having normal or low vitamin D status [17].

On the other hand, the increased prevalence of T2DM in countries endemic for TB poses a serious complication in the clinical management of this major infectious disease. Moreover, patients with coincident TB-T2DM exhibited increased plasma levels of tissue inhibitor of metalloproteinase-4 (TIMP-4) and elevated peripheral blood neutrophil counts which when considered together with heme oxygenase-1 (HO-1) resulted in increased power to discriminate diabetic from non-diabetic individuals with active TB [18].

On the basis of recent study bi-directional screening and care of TB and DM patients, since both entities adversely affect one another and there is currently no plausible evidence supporting the strong association between DM and

TB [19]. Diabetic patients suffering from cell-mediated immunity, renal failure, micronutrient deficiency and pulmonary microangiopathy, all there increases their propensity to develop TB. Requirement of Some research as well as knowledge gap on the association between these two diseases has recently been shown in some studies [20]. The increasing co-occurrence of TB and DM is a clear case in point, especially in countries with rapidly emerging economies such as India and China, and that has resulted in the confluence of two pandemicsone communicable and another non-communicable [21]. The union of these two epidemics may lead to an increased occurrence of TB, especially in low and middle-income countries with increasing numbers of people with DM and associated TB. DM almost triples the risk of developing TB [22] in areas such as the border population of South Texas and Mexico known for its high prevalence of DM, and where self-reported DM is the most common risk factor associated with TB development [23].

Faurholt-Jepsen *et al*., [24] in a prospective cohort study of the same study population also demonstrated that DM is a strong predictor of early mortality during anti TB treatment. In the initial 100 days, diabetes was associated with a fivefold increased risk of mortality [relative ratio (RR): 5.09, 95% CI: 2.36-11.02, P < 0.001] among HIV uninfected, and a twofold increase among HIV co-infected patients (RR: 2.33 95% CI: 1.20-4.53, P = 0.012). However, it was not associated with long-term mortality.

A study in Ethiopia resulted in prevalence of smear positive Pulmonary TB was 6.2% in TB suspected diabetic patients, which is higher compared with the general population (0.39%). It was also found that Patients with a previous history of contact with TB patients, as well as those who had prolonged diabetes, were more prone to have PTB [25]. A retrospective study in Texas and Mexico found 27.8% of Texan and 17.8% of Mexican TB patients suffered from Diabetes. It has been reported that patients with TB and diabetes were more likely to be smear positive at diagnosis, and remain positive at the end of the first or second month of treatment [26].

Table 1: Age group and No. of cases

Group	Group I	Group II
Group of	**Diabetes with TB**	**Normal Cases**
Sex		
Males	12	10
Females	8	10
Age in Years		
31 – 40 years	3	2
41 – 50 years	7	9
51 – 60 years	10	9
Total	20	20

Table 2: Levels of Vitamins and HbA1C+

Group	Group I	Group II
Group of	**Diabetes with TB**	**Normal Cases**
No. of Cases	**20**	**20**
HbA1C (%)	7.4 – 8.9	4.8 – 6.1
Vit B12 (pmol/L)	181.5 – 263.8	350.1 – 384.3
Vit D (ng/ml)	14.21 – 19.85	18.23 – 25.40
BMI	24.7 – 30.5	19.4 – 26.7

HbA1c reflects average blood glucose levels over a period of 2–3 months and may be less affected by infection related hyperglycaemia [27]. HbA1c ≥10% represents poor glycaemic control and is an indicator for administering insulin treatment in DM patients with TB as recommended by the Union and World Diabetes Foundation. A recent systematic review highlighted that HbA1c is a reliable risk factor of all-cause mortality and cardiovascular mortality in both DM and non-DM populations [35]. In patients with DM, the risk of all-cause mortality increased when HbA1c levels were above 8.0% and were highest when levels were above 9.0% [28].

The pathogenesis of type 2 diabetes involves both beta cell dysfunction and insulin resistance. However insulin resistance is the major culprit which is defined as the common pathologic state in which target cells (muscle, liver and adipose tissue) fails to respond to normal levels of circulating insulin. The following are the mechanisms by which vitamin D deficiency has been linked to insulin resistance and type 2 diabetes: ™ optimal concentrations of intracellular calcium (140-370nm) is needed for insulin to mediate its effect on target tissues. Vitamin D deficiency leads to elevated parathyroid hormone levels (PTH). This hyperparathyroidism cause a paradoxical rise in intracellular calcium level (‘calcium paradox’) more than the optimal level that results in diminished cellular response to insulin. ™

Vitamin D regulates the transcription & expression of insulin receptors in peripheral tissues thereby enhancing insulin responsiveness for glucose transport. ™ Vitamin D decreases the expression of various inflammatory cytokines thereby keeping systemic inflammation in check. ™ The mechanism of insulin secretion in beta cells is critically dependent on changes in intracellular calcium concentration. Vitamin D regulates extracellular calcium levels and calcium flux through beta cell membranes. Either vitamin D or calcium deficiency alter the physiological balance between intracellular and extracellular calcium in beta cells interfering with insulin synthesis and secretion.

Conclusion

The data generated from the present study concludes that the levels of the HbA1C were increases in Diabetes patients with TB whereas the levels of the Vitamin D and Vitamin B12 were decreases in diabetic patients with TB.

References

1. "Diabetes Fact sheet N°312". WHO. October 2013. Archived from the original on 26 August, 2013. Retrieved 25 March 2014.
2. Cash Jill. Family Practice Guidelines (3rd ed.). Springer, 2014, p. 396. ISBN 978-0-8261-6875-7. Archived from the original on 31 October 2015.
3. "Deaths and Cost | Data & Statistics | Diabetes | CDC". Www.cdc.gov. 20 February, 2019. Retrieved 2 July 2019.
4. Rockefeller JD. Diabetes: Symptoms, Causes, Treatment and Prevention, 2015. ISBN 978-1-5146-0305-5.
5. "Diabetes Programme". World Health Organization. Archived from the original on 26 April 2014. Retrieved 22 April, 2014.
6. Carris NW, Magness RR, Labovitz AJ. "Prevention of Diabetes Mellitus in Patients with Prediabetes". The American Journal of Cardiology. 2019; 123(3):507-512. doi:10.1016/j.amjcard.2018.10.032. PMC 635089.

PMID 30528418.
7. Shoback DG, Gardner D, eds. "Chapter 17". Greenspan's basic & clinical endocrinology (9th ed.). New York: McGraw-Hill Medical, 2011. ISBN 978-0-07-162243-1.
8. Lee IM, Shiroma EJ, Lobelo F, Puska P, Blair SN, Katzmarzyk PT. *et al*. "Effect of physical inactivity on major non-communicable diseases worldwide: an analysis of burden of disease and life expectancy". Lancet. 2012; 380(9838):219-29. doi:10.1016/S0140-6736(12)61031-9. PMC 3645500. PMID 22818936.
9. Konstantinos A. "Testing for tuberculosis". Australian Prescriber. 2010; 33(1):12-18. doi:10.18773/austprescr.2010.005.
10. Gibson Peter G. (ed.); Abramson, Michael (ed.); Wood-Baker, Richard (ed.); Volmink, Jimmy (ed.); Hensley, Michael (ed.); Costabel, Ulrich (ed.). Evidence-Based Respiratory Medicine (1st ed.). BMJ Books, 2005, p. 321. ISBN 978-0-7279-1605-1. Archived from the original on 8 December 2015.
11. Restrepo BI. "Convergence of the tuberculosis and diabetes epidemics: renewal of old acquaintances". Clinical Infectious Diseases. 2007; 45(4):436-38. doi:10.1086/519939. PMC 2900315. PMID 17638190.
12. https://www.cdc.gov/tb/topic/basics/tb-and-diabetes.html
13. Rajalakshmi S, Veluchamy G. Yugi's pramegam and diebetes mellitus: an analogue. Bull Indian Inst Hist Med Hyderabad, 1999; 29:83-87.
14. Hu FB, Manson JE, Stampfer MJ, *et al*. Diet, lifestyle, and the risk of type 2 diabetes mellitus in women. N Engl J Med, 2001; 345:790-97.
15. Leung CC, Lam TH, Chan WM, *et al*. Lower risk of tuberculosis in obesity. Arch Intern Med, 2007; 167:1297-304.
16. Goldhaber-Fiebert JD, Jeon CY, Cohen T, Murray MB. Diabetes mellitus and tuberculosis in countries with high tuberculosis burdens: individual risks and social determinants. Int J Epidemiol, 2011; 40:417-428.
17. Chaudhary S, Thukral A, Tiwari S, Pratyush DD, Singh SK. Vitamin D status of patients with type 2 diabetes and sputum positive pulmonary tuberculosis. Indian J Endocrinol Metab. 2013; 17(Suppl 3):S670-673.
18. Andrade BB, Pavan Kumar N, Sridhar R, Banurekha VV, Jawahar MS, *et al*. Heightened plasma levels of heme oxygenase-1 and tissue inhibitor of metalloproteinase-4 as well as elevated peripheral neutrophil counts are associated with tuberculosis-diabetes comorbidity. Chest, 2014.
19. Faurholt-Jepsen D, Range N, Praygod G, Jeremiah K, Faurholt-Jepsen M, *et al*. Diabetes is a risk factor for pulmonary tuberculosis: a casecontrol study from Mwanza, Tanzania. PLoSOne, 2011; 6:e24215.
20. Baker MA, Harries AD, Jeon CY, Hart JE, Kapur A, *et al*. The impact of diabetes on tuberculosis treatment outcomes: a systematic review. BMC Med, 2011; 9:81.
21. Magee MJ, Blumberg HM, Narayan KM. Commentary: Co-occurrence of tuberculosis and diabetes: new paradigm of epidemiological transition. Int J Epidemiol, 2011; 40:428-431.
22. Stevenson CR, Forouhi NG, Roglic G, Williams BG, Lauer JA, *et al*. Diabetes and tuberculosis: the impact of the diabetes epidemicon tuberculosis incidence. BMC Public Health, 2007; 7:234.
23. Restrepo BI. Convergence of the tuberculosis and diabetes epidemics: renewal of old acquaintances. Clin Infect Dis, 2007; 45:436-438.
24. Faurholt-Jepsen D, Range N, PrayGod G, Jeremiah K, Faurholt-Jepsen M, Aabye MG, *et al*. Diabetes is a strong predictor of mortality during tuberculosis treatment: A prospective cohort study among tuberculosis patients from Mwanza, Tanzania. Trop Med Int Health, 2013; 18:822-9.
25. Amare H, Gelaw A, Anagaw B, Gelaw B. Smear positive pulmonary tuberculosis among diabetic patients at the Dessiereferral hospital, Northeast Ethiopia. Infectious Diseases of Poverty, 2013; 2:6.
26. Restrepo BI, Fisher-Hoch SP, Crespo JG, Whitney E, Perez A, *et al*. Type 2 diabetes and tuberculosis in a dynamic bi-national border population. Epidemiol Infect, 2007; 135:483-491.
27. World Health Organization. Use of Glycated Haemoglobin (HbA1c) in the Diagnosis of Diabetes Mellitus Abbreviated Report of a WHO Consultation. WHO, 2011. WHO/NMH/CHP/CPM/11.1. Report available. http://www.who.int/diabetes/publications/report-hba1c_2011.pdf [PubMed] [Google Scholar]
28. Cavero-Redondo I, Peleteiro B, Alvarez-Bueno C, Rodriguez-Artalejo F, Martinez-Vizcaino V. Glycated haemoglobin A1c as a risk factor of cardiovascular outcomes and all-cause mortality in diabetic and non-diabetic population: a systematic review and meta-analysis. BMJ Open, 2017; 7:e015949. 10.1136/bmjopen-2017-015949