IJNRD.ORG ISSN : 2456-4184
INTERNATIONAL JOURNAL OF NOVEL RESEARCH AND DEVELOPMENT (IJNRD) | IJNRD.ORG
An International Open Access, Peer-reviewed, Refereed Journal

A Review Article On- Tuberculosis In Diabetic Patients

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ABSTARCT

Abstract:

There have been claims that diabetes mellitus can change the way pulmonary tuberculosis manifests, however the evidence is conflicting, especially when it comes to the link to lower lung field involvement. The effects of diabetes on tuberculosis appeared to be both initiating and exacerbating. Diabetics are more likely than non-diabetics to develop tuberculosis. Diabetics exhibit more pronounced radiological TB symptoms. Death and treatment failure are also occurring more frequently. Compared to diabetics without tuberculosis, those with tuberculosis experience more complications and have worse diabetes. Since ancient times, people have understood that diabetes mellitus and TB are related. Recent years have seen a decrease in the frequency of tuberculosis in high-income nations, although incidence is still high in nations with high rates of HIV infection, malnutrition, congested living circumstances, or inadequate infrastructure for tuberculosis control. Likewise, obesity is a major contributor to the rise in diabetes mellitus prevalence that is occurring globally. There is mounting evidence that diabetes mellitus may have an impact on how the disease manifests and how well it responds to therapy, making it a significant risk factor for TB. Additionally, diabetes sufferers' glycaemic control may be harmed by TB, which can cause glucose intolerance. We also discuss the processes by which diabetes mellitus might result in tuberculosis as well as the implications of TB on diabetic management.

Keywords: Diabetes, Tuberculosis, Glycemic Control, Nutrition, Insulin.

MANUSCRIPT

INTRODUCTION

Introduction:

Diabetes mellitus and TB case fatality rates were significantly reduced thanks to the creation of insulin in the 1920s, streptomycin's discovery in the 1940s, and the subsequent invention of various antibiotics. The incidence of TB was significantly reduced as a result of better diet, sanitation, and reduced crowding. As a result of rising obesity rates, altered eating and physical activity habits, and ageing populations, non-insulin-dependent diabetes mellitus (NIDDM) has emerged as a chronic health condition that is spreading throughout the world in recent decades. Tuberculosis has also become a bigger issue in low-income countries, particularly those with HIV epidemics. (11).

The impact of diabetes on the severity and progression of TB as well as the intricate connections between nutrition, obesity, diabetes, and tuberculosis continue to be contentious problems in both clinical and public health. Together, TB and diabetes mellitus pose a global health issue due to the growing overlap of people who are at risk for both conditions (16, 17, 21& 20). Approximately 415 million people worldwide are currently estimated to have diabetes, according to the International Diabetes Federation (IDF), and by 2040, this number is expected to rise to 642 million (a 60% increase). The largest increases, primarily brought on by type 2 diabetes, are anticipated to occur in developing nations. The two situations' anticipated trajectory will also intensify their contact with one another (3).

Diabetes is becoming more widely acknowledged as an independent risk factor for TB, and the two frequently coexist (8, 6). These risk factors are in addition to the conventional ones, which include poverty, malnutrition, overcrowding, and immunosuppression, including HIV/AIDS. By noting that tuberculosis (or phthisis as it is known in Greek) frequently coexisted with diabetes, Avicenia, who lived around 1000 AD, recognised the link between the two diseases. In addition, an Indian saint by the name of Yugimahamuni defined a collection of symptoms for the TB/diabetes relationship that he named meganoikal. The following signs and symptoms are present: obesity, glycosuria, thirst, incontinence, respiratory problems, and unconsciousness. Therefore, this TB and diabetes relationship is becoming more and more understood and treated. As independent illnesses, diabetes and tuberculosis have an adverse effect on one another. While diabetes may cause a more severe type of TB and change how it presents, TB may result in reduced glucose tolerance and impede glycemic management. (1).

How might diabetes mellitus lead to tuberculosis?

Vascular disease, neuropathy, and heightened susceptibility to infection are just a few of the problems that can result from poorly managed diabetes (12). Multiple mechanisms suggest that diabetes may enhance susceptibility to M. tuberculosis illness. The processes include those directly linked to hyperglycemia and cellular insulinopenia as well as indirect impacts on lymphocyte and macrophage activity, resulting in decreased ability to confine the organism.

Does tuberculosis lead to diabetes?

If diabetes may put a person at risk for developing TB, can a tuberculosis infection cause diabetes mellitus? Patients with diabetes often experience lower glycemic control due to infections, such as TB, and infections themselves may become more severe as a result of poorly managed diabetes. (13).

The underlying pathophysiological mechanism:

Numerous theoretical pathways have been presented to explain why people with diabetes are more likely to get tuberculosis. Due to a low neutrophil count, decreased T-lymphocyte activity, and reduced neutrophil count, there is a decline in cellular immunity. When compared to those who don't have diabetes, diabetics produce less interleukin-1, interleukin-6, tumour necrosis factor (TNF-alpha and TNF-beta), and T-helper 1 (TH 1) cytokine response. Diabetes individuals are more vulnerable to TB because their T-lymphocyte counts and capacities are lower. Specifically Mycobacterium tuberculosis suppression by TH1 cytokines. Diabetes leads to macrophage malfunction, which affects phagocytic and chemotactic activity as well as the ability to produce reactive oxygen species. In individuals with diabetes, the chemotaxis of monocytes is similarly compromised, and insulin does not cure this abnormality. The ability of the respiratory burst to effectively remove germs is hypothesised to be impaired by hyperglycemia. Despite the plausibility of these hypothesised pathways, it is critical to do more mechanistic research to determine whether they are accurate. (18,23, 24& 15).

Dysglycemia may also be a result of the stress reaction to infection, which is mediated by the actions of IL-1, IL-6, and TNF-alpha (8, 28). This temporal association has been shown in certain studies where between 19 and 42.6% of active TB patients were found to have IGT or diabetes, with a considerable reduction or full regression in the rates following therapy (26, 19). One of the studies also supported the notion of a stress reaction to infection since the control group in that study who had community-acquired pneumonia experienced a comparable rate of glucose intolerance. (19).

Management of comorbid and diabetes:

The dose and duration of anti-TB medications remain the same for those with or without TB, despite claims that diabetes may increase the risk of mortality, serious illness, and relapse(4,10). A two-month intensive phase of rifampicin, isoniazid, pyrazinamide, and ethambutol is followed by a four-month maintenance phase of rifampicin and isoniazid in the majority of centres' traditional six-month TB treatment regimens.(4) It has been suggested that antiglycemic medicines and anti-TB medications interact pharmacokinetically and pharmacodynamically. By accelerating the metabolism of sulphonylureas and biguanides by enzyme induction, rifampicin—a crucial component of the cocktail of anti-TB medications—decreases their plasma levels, causing hyperglycemia (8, 7, and 27). Sulphonylureas are inhibited by isoniazid, which makes glycemic control worse.

Cytochrome P2C9 (CYP2C9), which is involved in the metabolism of sulphonylureas, is one oral antiglycemic agent whose metabolism is decreased by isoniazid and whose plasma levels are increased in some circumstances. However, it is believed that rifampicin's inducing effect outweighs this inhibitory effect (25). Once more, it has the potential to impede insulin release in people who are not diabetics, leading to hyperglycemia. (27)

The use of DPP IV inhibitors for the treatment of tuberculosis has the potential to decrease treatment results and, theoretically, to produce immunological paresis (8). Careful consideration must go into selecting the right antiglycemic drug for the management of diabetes with concurrent TB infection. (4, 8).

In order to reconcile glycemic management with the nutritional requirements of those who are primarily underweight and malnourished, appropriate food counselling is required. The primary anti-hyperglycemic medicine is still metformin, which is also reasonably inexpensive, safe, and has a low risk of hypoglycemia. The following medications should also be taken into account: sulphonylureas, meglitinides, alpha-glucosidase inhibitors, dipeptidyl peptidase (DPP) IV inhibitors, glucagon-like peptide (GLP) 1 analogues, thiazolidinediones, and insulin. The specific drug selection must take into account the patient's features, the medication's cost, availability, and side effect profileTrue, each patient needs a unique approach to treatment. If necessary, the course of treatment can be changed to reach the desired level of glycemic control, such as by raising the dosage or frequency of a certain class or by adding one or more classes (2).

When type 2 diabetes is present and a TB infection is active, insulin is frequently the drug of choice [3]. The severe TB infection, body tissue loss, increased anabolism required, pancreatic hypofunction, interactions between oral antidiabetic medications and some antituberculous medications as mentioned above, and the potential for associated liver disease that would preclude the use of oral agents are all factors that led to the decision to use insulin. (8, 22).

For the aforementioned reasons, after a diagnosis of active TB is obtained, or if the patient is already receiving insulin therapy, changes may need to be made to account for poor glycemic control. Patients with preexisting diabetes who are already on oral medications may be transferred to insulin therapy. The amount of insulin required might decrease if glucotoxicity and infection are managed. But if hunger returns and food consumption rises, the amount needed could also. Based on patient characteristics, cost, safety, and efficacy, the best insulin should be chosen. To be clear, though, oral antidiabetic medications may be carefully taken into consideration when the illness is under control (8).

Regular glucose monitoring is necessary for best management. This aids in the early detection of potential adverse effects, such as hypoglycemia from various antiglycemic drugs like sulphonylurea and insulin, as well as the geographic trend of glucose profiles that may require dose modifications. However, there aren't any reimbursement or compensation programmes for glucometers or glucose strips in many regions of the world, including underdeveloped nations. This makes it more difficult to meet objectives and identify and confirm hypoglycemia. Patient education is crucial in explaining the nature of the illness (both TB and diabetes), the length of therapy, drug side effects, and disease consequences, as well as in promoting healthy lifestyle choices (2,5&4) .

CONCLUSION

Conclusion:

Diabetes is becoming better acknowledged as a risk factor for TB and may impact how it manifests, whereas TB may decrease glycemic control or cause IGT in TB patients. Because of this connection, it is necessary to modify the course of therapy and, as necessary, use insulin to manage hyperglycemia while the TB infection is active. Once TB treatment is over, an evaluation of the antiglycemic drug(s) is also necessary.

The impact of diabetes on tuberculosis will likely increase in the next years due to the anticipated increase in diabetes cases in developing nations (mostly type 2 diabetes), which also bear the burden of the disease. It is intended that this analysis would provide further light on whether routine testing for dysglycemia should be performed on all TB patients, particularly at the time of diagnosis. A greater argument may be made for routine TB screening, or at the very least, suspicion, among diabetic patients. Patient referrals to TB centres for treatment should be made as soon as a patient is diagnosed. It is important to consistently emphasise TB preventive methods.

ETHICAL STATEMENT

This study has no cruelty to humans or animals. Since, it is a review article, we did not took a subjects in this study.

CONFLICT OF INTEREST

The above study describes the how tuberculosis occurs in diabetic patients, what is the underlying mechanism and how to manage the comarbity of diabetes.

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