



COIN EFFECT OF TUBERCULOSIS AND DIABETES MELLITUS

Deepthy. B. Nair

Department of Microbiology, Saveetha Dental College, Chennai, Tamilnadu

Cite This Article: Deepthy. B. Nair, "Coin Effect of Tuberculosis and Diabetes Mellitus", International Journal of Multidisciplinary Research and Modern Education, Volume 3, Issue 2, Page Number 73-75, 2017.

Copy Right: © IJMRME, R&D Modern Research Publication, 2017 (All Rights Reserved). This is an Open Access Article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Abstract:

Tuberculosis is a common disease caused by various strains of mycobacterium, usually Mycobacterium tuberculosis [1]. The first reference to tuberculosis in non European civilization was found in Vedas. Diabetes mellitus is group of metabolic diseases where the person has high blood sugar level either because the pancreas does not produce insulin or because cells do not respond to insulin that is produced. It may eventually leads to polyuria, polyphagia and polydipsia. This review is to reveal the link between tuberculosis and diabetes mellitus

Tuberculosis:

Tuberculosis is a common disease caused by various strains of mycobacterium, usually Mycobacterium tuberculosis [1]. The first reference to tuberculosis in non European civilization was found in Vedas. The infections typically attack lungs, but also have the potential to attack other parts of body. It is an air borne disease when the infected person coughs, sneeze or transmit respiratory fluids through air. They are usually asymptomatic and latent. The classic symptoms of active TB infection are chronic cough with sputum containing blood, fever, night sweats, and weight loss [2]. Tuberculosis affecting lungs is known as pulmonary tuberculosis. Extra pulmonary tuberculosis may co exist with pulmonary tuberculosis as well. When the tuberculosis is active and commonly involves lungs, it is pulmonary [3]. Occasionally the person may cough up blood it may finally reach pulmonary artery resulting in massive bleeding. While in case of extra pulmonary tuberculosis the infection spreads outside lungs commonly seen in young children and immune suppressed persons. It may involve pleura, central nervous system, the lymphatic system, and bones and joints

Pathogenesis:

The main causative agent of tuberculosis is Mycobacterium tuberculosis. It falls under non motile bacillus which is aerobic and acid fast. It has an outer membrane. It can withstand weak disinfectants and survive in dry state for weeks. It needs a specific host for the growth. Tuberculosis infection is of two types; latent and active. Latent: TB germs are dormant in human body. This phase can last for a long years. This phase remains asymptomatic and will generally don't spread. The only manifestation of latent TB encounters in to tuberculin skin test or interferon gamma release assay [IGRA]. Active: In this phase there is multiple division happening causing tissue damage. When the bacteria reach the pulmonary alveoli, they invade and replicate within endosome of alveolar macrophages [1] [4]. The primary site of infection called as 'Ghon focus' is located in upper part of lower lobe [1]. The macrophages, T lymphocytes, B lymphocytes and fibroblast aggregate to form granuloma and thus it are called as granulomatous inflammatory disease. The granuloma with lymphocyte surrounding the infecting macrophages which prevents the dissemination of mycobacteria and provide a local environment for interacting with immune system.

Prevention, Diagnosis and Management:

BCG is the only available vaccine against TB in India. Skin test (Mantoux test or tuberculin test), sputum test, chest X-ray are commonly practiced. Infected patients required antibiotics for 6-9 months. Active tuberculosis, particularly for drug resistant strain, will require multiple drug therapy. The two antibiotics most commonly used are *isoniazid* and *rifampicin* or by combination of those drugs [5]. In drug resistant TB, combination of flouroquinolones and injectable medications are generally used for 20 to 30 months

Diabetes Mellitus:

It is group of metabolic diseases where the person has high blood sugar level either because the pancreas does not produce insulin or because cells do not respond to insulin that is produced. It may eventually leads to polyuria, polyphagia and polydipsia [6]. There are mainly two types of diabetes mellitus; type I and type II, gestational diabetes, congenital diabetes, etc. The classic symptoms of untreated diabetes are loss of weight, increased thirst, and increased hunger. Prolonged high blood glucose can cause glucose absorption in lens of eye, lead to change in shape of lens, resulting in vision changes. It is the main reason for the repeated complaint of blurred vision from patients suffering from diabetes mellitus. Diabetic dermadromes is visible in some cases.

Causes:

- Depending on type of diabetes, the causes also varies;
- ✓ Type I diabetes is partly inherited. It is unrelated to lifestyle

- ✓ Type II diabetes is mainly due to lifestyle factors and genetics [7]. Lack of physical activity, poor diet, stress, urbanization and obesity are the important lifestyle factors that lead to type ii diabetes [8]. Saturated fats and transfatty acids increases the risk and polyunsaturated and monosaturated fat decreases risk. Eating lot of white rice also may increase the risk

Pathophysiology:

Insulin is the principal hormone that regulate uptake of glucose from blood into most of cells. Insulin is released into blood by beta cells in islet of Langerhans in pancreas, in response to rising sugar levels. Insulin also aids in conversion of glucose to glycogen. Lowered glucose level result in reduced release of insulin and conversion to glycogen. Higher insulin level increases some anabolic processes such as growth and duplication, protein synthesis and fat storage. Insufficient insulin leads to turned off effect of glucose.

Management:

Diabetes mellitus is a chronic disease. Proper diet, exercise and use of appropriate medication can help in managing diabetes. Metformin is generally recommended as first line treatment for type 2 diabetes. Type I diabetes treated with combinations of NPH insulin or synthetic insulin [9]

Converging Epidemics:

There is major link between diabetes mellitus and tuberculosis over centuries. Tuberculosis is a specific morbidity associated with diabetes mellitus. The incidence of diabetes mellitus is increasing worldwide, especially in developing countries where tuberculosis is more prevalent [10]. Therefore the convergence of this disease is most likely to occur in places with least amount of healthcare resources. Diabetes is an independent risk factor for all lower respiratory tract infections [11]. The relationship between diabetes mellitus and tuberculosis is bi-directional [12] [13]. Tuberculosis induces glucose intolerance and worsen the glycemic control in people with diabetes. Active tuberculosis should be a differential diagnosis in patients with enlarged pancreas [14] and tuberculous pancreatitis might reveal itself only after development of diabetes. Moreover testing for diabetes mellitus in previously undiagnosed person before the appropriate treatment of tuberculosis may lead to over diagnosis of diabetes mellitus. Tuberculosis can lead to an infection- related hyperglycemia which may mimic diabetes mellitus. The hyperglycemia associated with tuberculosis often aggravates the glycemic control of diabetes. Diabetes is associated with decrease in cellular immunity. T lymphocyte and neutrophil and decreased neutrophil count in diabetes [15] [16]. Neutrophils from people with diabetes had reduced chemotaxis and oxidative killing potential. Leukocyte bactericidal activity was reduced in people with diabetes. They include those directly related to hyperglycemia and cellular insulinopenia, as well as indirect effects on macrophage and lymphocyte function. In a study of patients with tuberculosis alveolar macrophages were less activated. Insulin deficiency can cause impaired internalization Fc receptor mediated phagocytosis. Diabetes might adversely affect T cell production of interferon γ , T cell growth, function and proliferation. This is same as in case of immune responses to tuberculosis. In same way the drugs used to treat tuberculosis can cause glucose intolerance. Prevalence of pulmonary tuberculosis in patients with diabetes mellitus may be also due to hepatic dysfunction and consequent hypovitaminosis A and D. It is worthwhile to mention that both the disease may stimulate the symptoms of the other. Such common symptoms include lethargy, fatigue, weight loss, fever and loss of appetite.

Conclusion:

Tuberculosis is not a familial disease, it is curable. Diabetes and tuberculosis should be treated aggressively with insulin. Improved understanding about the bidirectional relationship of two diseases are important for proper diagnosis With increasing rates of obesity and diabetes worldwide and continued high rate of tuberculosis in low-income countries, there is a chance for increase in number of individuals who have both tuberculosis and diabetes mellitus in the coming decades.

References:

1. Kumar V, Abbas AK, Fausto N, Mitchell RN (2007). Robbins Basic Pathology (8th ed.). Saunders Elsevier. pp. 516–522. ISBN 978-1-4160-2973-1
2. Konstantinos A (2010). "Testing for tuberculosis". Australian Prescriber 33 (1): 12–18.
3. Dolin, [edited by] Gerald L. Mandell, John E. Bennett, Raphael (2010). Mandell, Douglas, and Bennett's principles and practice of infectious diseases (7th ed.). Philadelphia, PA: Churchill Livingstone/Elsevier. pp. Chapter 250. ISBN 978-0-443-06839-3.
4. Houben E, Nguyen L, Pieters J (2006). "Interaction of pathogenic mycobacteria with the host immune system". Curr Opin Microbiol 9 (1): 76–85. doi:10.1016/j.mib.2005.12.014.PMID 1640683
5. Ahmed N, Hasnain S (2011). "Molecular epidemiology of tuberculosis in India: Moving forward with a systems biology approach". Tuberculosis 91 (5): 407–3.
6. Cooke DW, Plotnick L (November 2008). "Type 1 diabetes mellitus in pediatrics". Pediatr Rev 29 (11): 374–84; quiz 385
7. Risérus U, Willet W (January 2009). "Dietary fats and prevention of type 2 diabetes". Progress in Lipid Research 48 (1): 44–51
8. Williams textbook of endocrinology (12th ed.). Philadelphia: Elsevier/Saunders. pp. 1371–1435

9. Ripsin CM, Kang, H, Urban, RJ (2009). "Management of blood glucose in type 2 diabetes mellitus". American family physician 79 (1): 29–36.
10. Nijland HMJ, Ruslami R, Stalenhoef JE, Nelwan EJ, Alisjahbana B, Nelwan RHH, et al.: Exposure to rifampicin is strongly reduced in patients with tuberculosis and type 2 diabetes.
11. Nichols GP: Diabetes among young tuberculous patients; a review of the association of the two diseases.
12. Winterbauer R, Bedon G, Ball W: Recurrent Pneumonia: Predisposing illness and clinical pattern of 158 patients. Ann Intern Med 1969, 70:689
13. Mugusi F, Swai A, Alberti K, Melarty G: Increased prevalence of diabetes mellitus in patients with pulmonary tuberculosis in Tanzania.
14. Geevarghese PJ: Pancreatic diabetes. Popular Prakashan: Bombay; 1967::26-28
15. Geerlings SC, Hopelman AI: Immune dysfunction in patients with diabetes mellitus (DM). FEMS Immunol Med Microbiol 1999, 26:259-65
16. Tsukaguchi K, Yoneda T, Yoshikawa M: Case study of interleukin-1 beta, tumor necrosis factor alpha and interleukin-6 production by peripheral blood monocytes in patients with diabetes mellitus complicated by pulmonary tuberculosis. Kekkaku 1992, 67:755-60.