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A Consortium Between Substance Abuse And Pulmonary Tuberculosis: A Literature Review

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Abstract

Background: Tuberculosis (TB) control measures are frequently ineffective in individuals who use illicit drugs or abuse substances. Patients who abuse substances are more contagious and remain contagious longer because treatment failure presumably extends periods of infectiousness. Even when hurdles to health care are addressed, protracted treatment regimens can be difficult to stick to, especially for drug users and alcoholics, who have been recognized as risk factors for treatment failure.

It is vital to establish the relationship and potential risk factors such as substance usage in tuberculosis patients to provide optimal care for overall status. The difficulty of maintaining high levels of adherence has obvious implications for tuberculosis control, and it may necessitate the provision and coordination of additional services for drug users, such as targeted testing and other possible pharmacological (ATT) and non-pharmacological (pulmonary rehabilitation) treatments.

Keywords: Alcohol abuse, drug abuse, pulmonary tuberculosis, smoking

Introduction

Tuberculosis (TB) remains one of the world's major causes of mortality and disability. While the present disease load is tremendous, tuberculosis is one of the top ten most deadly and disabling diseases. Even before the etiology of TB was discovered, there was a link between alcohol consumption and tuberculosis. Benjamin Rush identified tuberculosis and pneumonia as infectious sequelae of prolonged heavy drinking as early as 1785.(1) Several publications have been written since then that address the links between alcohol, alcohol use disorders (AUD), and tuberculosis (TB).(2)

In this work, we will attempt to review the evidence using established epidemiological criteria for causation, association, and strength of association, biological pathway plausibility between substance abuse and prevalence of pulmonary tuberculosis.

Methodology:

A literature search was conducted across patients with drug and alcohol abuse with the help of multiple electronic bibliographic databases such as PubMed, EMBASE, Web of Science, Google Scholar, and the Cochrane Database of Systematic Reviews. Up to and including September 2020, the available published and unpublished literature was combed.

The search included multiple variations of the key terms TB, alcohol, abuse, misuse, dependence, alcohol use disorders, prevalence, and incidence. A manual search of selected papers' bibliographic

pages, as well as the content pages of significant epidemiological journals. The search was not limited to a specific geographic location or English-language media.

Meta-analyses or systematic reviews, duplicate publications of the same study, and abstract-only papers were omitted from the analysis due to a lack of data on the prevalence of excessive alcohol use/AUD among TB patients or of TB among patients with AUD.

Drug Abuse And Pulmonary Tuberculosis:

According to scientists, if the world's smoking population continues to grow at its current rate, there would be more than 18 million tuberculosis cases and 40 million fatalities by 2050. They also predicted that between 2010 and 2050, smoking will result in a 7% increase in new tuberculosis cases (from 256 million to 274 million) and a 66% increase in tuberculosis-related deaths (from 61 million to 101 million), making meeting the WHO's tuberculosis control targets even more difficult.

The physiological effects of drug use, as well as the environment and drug users' risk behaviors, may all contribute to the high frequency of tuberculosis among drug users, according to a study conducted by Robert G et al. Drug use has been demonstrated to have deleterious effects on the immune system in several in vitro studies, with biochemical evidence showing opiates' direct impairment of the cell-mediated immune response.[3]

Although the therapeutic implications of this research are uncertain, drug use is typically linked to several epidemiological characteristics that increase the risk of tuberculosis, including cigarette use, homelessness, alcohol abuse, and incarceration. These physiological and epidemiological characteristics could all have a role in the reported outcomes, such as drug users being more likely to be infectious, taking longer to establish a negative culture, and having a higher chance of mortality.[4]

The link between tuberculosis and illicit drug use is strengthening, according to evidence from a study by Densine R et al, posing a public health threat due to illicit drug users' risky lifestyles, crowded housing conditions, the accumulation and isolation of people indoors for the consumption of illicit drugs, the sharing of materials like pipes, malnutrition, and severe cough.

Pulmonary edema, diffuse alveolar hemorrhage, acute asthma exacerbations, barotrauma, pulmonary eosinophilic infiltrates, nonspecific interstitial pneumonia, and bronchiolitis obliterans organizing pneumonia, as well as acute pulmonary infiltration and vasculitis, have all been linked to smoked cocaine.[5]

Cocaine reduces the activity of inducible nitric oxide synthase, which restricts the antibacterial activity of alveolar macrophages. Cocaine inhibits interferon, chemokine CCL2, and TNF all of which are critical in the immune response to tuberculosis. Overall, cocaine use reduces monocyte and alveolar macrophage protective capacity, failing to respond to a mycobacterial challenge and, as a result, a failure to prevent active tuberculosis.[6]

Long-term cocaine use, on the other hand, destroys the lungs, raising the risk of pulmonary tuberculosis. It has also been proven that cocaine inhibits the growth of alveolar macrophages and immunoregulatory cytokines, both of which are necessary for active tuberculosis resistance.[7]

Overall, excessive alcohol/drug abuse and TB have a substantial relationship. The link has been established in studies undertaken in several countries, each with its own set of conditions and methods.

Heavy alcohol consumption/AUD has been shown in studies to be a risk factor for a weaker immune system, making a person more vulnerable to active tuberculosis infection and the reactivation of latent disease. Alcoholics are classed as "Immunocompromised" because the prevalence and severity of infectious illnesses are higher among them than among abstainers. Those who abuse alcohol are more likely to get lung infections such as TB and pneumonia.[8]

Inhaled Mycobacterium TB is generally eliminated by alveolar macrophages in greater than 90% of cases. If alveolar macrophages fail to kill M. tuberculosis, the bacteria grow inside macrophages, resulting in tuberculosis in the lungs. Alcohol promotes M. tuberculosis intracellular survival and proliferation in human macrophages, according to in vitro testing. In mice with pulmonary tuberculosis, alcohol usage has also been linked to changes in

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region-specific CD4+ and CD8+ lymphocyte responses, as well as faulty lung granuloma development.[9]

Alcoholism is one of the top five causes of sickness, disability, and mortality around the globe, as well as the cause of over 200 diseases and injuries, including tuberculosis. Alcohol is thought to be the cause of ten percent of all TB cases.[10]

Alcohol use, both acute and chronic, may decrease the macrophage's ability to transfer mycobacterial antigen to lymphocytes, lowering antigen-specific T cell activation. Alcohol has also been shown to impair macrophage reactivity to immune system modifiers (e.g., cytokines such as interleukin-6 (IL-6) and interleukin-1 (IL-1), TNF-, and IL-8) as well as reduce their protective effects. 16

Alcohol may prevent antigen-specific T-cell activation, resulting in a Th2 (humoral immunity) population that outnumbers Th1 (innate immunity) (cell-mediated immunity, responsible for overcoming TB infection). The immune system's balance is upset as a result of this shift, reducing immunological defense and increasing vulnerability to tuberculosis as a result of alcohol usage.[11]

Low to moderate alcohol consumption is not connected to an increased risk of TB disease, according to a study by Knut L et al. People who consume more than 40 grams of alcohol per day and/or have an alcohol use disorder, on the other hand, appear to be at a significantly increased risk. Alcohol may have a direct negative impact on the immune system, making the host more vulnerable to tuberculosis (TB) infection. Only pulmonary tuberculosis patients were included in the research, which had greater odds ratios than studies that included all tuberculosis cases.

It's been difficult to separate the role of alcohol in TB through its immune system effects from other risk factors because it's still unclear whether alcohol or other consequences of heavy drinking and alcohol dependence, such as liver damage, nutritional deficiency, or hygienic factors, are primarily responsible for the impaired immunity associated with alcoholism. Regardless of the cause for consumption, alcohol depresses the immune system, increasing the risk of active tuberculosis.

Specific social mixing patterns in locations like bars, homeless shelters, jails, and social institutions, which may increase the risk of exposure to people with infectious TB disease, possibly explain the link between alcohol intake and TB. These findings have significant implications for tuberculosis (TB) management programs, especially in locations where alcohol use accounts for a high proportion of TB cases.

Passive and active cigarette smoke exposure has been linked to an elevated risk of M. tuberculosis infection and active tuberculosis development. Cigarette smoke is connected to ciliary dysfunction, a reduced immunological response, and abnormalities in macrophage immune responses, all of which enhance susceptibility to infection with Mycobacterium TB, with or without a drop in CD4 count.[12]

Cigarette smoke raises low levels of interleukin-12 and TNF-, which prevents the formation of granulomas, which would limit infection in immune-competent people at this time. As a result, smoking fosters the growth of active tuberculosis. Compared to nonsmokers, smokers have a far greater rate of tuberculosis-related mortality. In those without a history of tuberculosis, smokers have a nine-fold greater risk of dying from tuberculosis than never-smokers. [13]

In a qualitative systematic analysis published in 2007, researchers observed a significant correlation between smoking and active tuberculosis, as well as a moderate link between passive smoking and active tuberculosis and the need for retreatment. When examining bouts of respiratory infection in children, a history of parental smoking is already taken into account. A recent study found that children living in tuberculosis-endemic areas had a higher chance of M. tuberculosis infection and that parental smoking was strongly associated with the risk of active tuberculosis, even after other factors were taken into account. As a result, the effects of passive smoking on active tuberculosis are a matter of concern, and tuberculosis smokers should be conscious of the harm their addiction can do to others, particularly their contacts, who are at a higher risk of getting active tuberculosis. [14]

The association between substance abuse and increased M. tuberculosis transmission can be explained in several ways, some of which are

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indirect, such as delayed diagnosis, difficulty identifying at-risk contacts, screening them for TB, and treating patients with positive results. Substance abusers may have less access to basic medical care, which could lead to a diagnosis delay. As the disease worsens, patients become more contagious.

Conclusion:

This study looked at the prevalence of substance use among pulmonary TB patients. Tuberculosis (TB) may be becoming more common among drug and alcohol addicts. Increased transmission is consistent with our finding that substance-abusing patients are more likely to be part of a localized genotype cluster, indicating recent transfer.

Poor adherence to treatment and limited access to care are significant barriers to treatment for drug users, but they are also modifiable risk factors that should be the focus of future interventions. The importance of TB control among drug users is evident, and it involves the provision of additional services aimed at sustaining positive outcomes.

Increased attention to high-risk groups, such as drug users, is an important part of a bigger strategy that has likely contributed to the TB the prevalence drop witnessed in many countries over the previous decade. To maintain present gains and help stop TB epidemics around the world, continued attention must be given to those at high risks, such as drug users and IDUs.

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