Diabetes Mellitus, Tuberculosis & Acquired Immune Deficiency Syndrome (AIDS); an Awful Triangle …?

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Diabetes mellitus (DM) and Human immune deficiency virus/ acquired immune deficiency syndrome (HIV/AIDS) are not only responsible for raising the incidence of tuberculosis (TB) but even make the treatment complicated and leads to morbidity and mortality [1]. DM, an auto-immune disorder affecting both cell mediated and humoral immunity, is known to reactivate latent tuberculosis (TB). Incidence of TB, more specifically pulmonary tuberculosis, among diabetic patients is 1.5–8 times higher when compared to the general population [2]. The World Health Organization (WHO) projections indicate that there will be 35 million cases of HIV infection and 30 million cases of DM in India by year 2025, and same picture will generalize on other AIDS epidemic countries [3]. In addition, extra pulmonary tuberculosis were found more frequent with HIV/AIDS patients. In a study of 109 tuberculous lymphadenitis cases, there was co-existence of DM or HIV infection [4]. Though there are some contradictory studies existing in this scenario.

HIV/AIDS induced immunosuppression provides a right atmosphere for opportunistic infection. Cytokine dysregulations have been observed, decreased production of interferon-γ (IFN-γ) caused by HIV, compromises the host’s ability to combat invasion by tubercle bacilli and thereby, facilitates reactivation of dormant foci of TB. Tumor necrosis factor-α (TNF-α), a stimulatory agent for some cell lines (e.g., fibroblasts), was noted to increase during the retrotroviral disease, which enhances HIV replication and progression of the AIDS disease process. This finding led to the speculation that damaged T-cells in AIDS patients are capable of destroying the beta cells of pancreas. Studies show that HIV seroprevalence was higher among diabetic patients when compared to the non-diabetic control group [5].

Smikle et al. [3] demonstrated that DM may be one of the manifestations of HIV disease process and it is the result of activated T-lymphocytes homing to anatomical sites that are not normally infected by HIV. This leads to apoptosis, increased cytotoxicity such as TNF-α, IFN-γ and interleukin-1 in pancreas, which in turn, may destruct the beta cells leading to development of DM. Furthermore, DM by compromising with the cell-mediated immunity reactivates latent TB infection and leads to dissemination of dormant organisms [6]. In addition, treatment of HIV is also linked with development of type 2 DM [8]. As people with HIV live longer with HAART, their care providers must be more vigilant about chronic conditions like diabetes and high blood pressure that can cause significant damage if not well controlled [9]. HIV and DM in the same patient with TB may suppress the immune system further as compared to TB-HIV or TB-DM cases and the chances of acquiring opportunistic infections is greater leading to higher morbidity and mortality. However, further research into this area is required to explain the actual causes of death as there is no literature existing on this issue.

References

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