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Editorial

Insulin Plays the Role of a Double-Edged Sword for the Pathogenesis of Tuberculosis in the Context of Diabetes Mellitus

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More than one-third of the world population is infected with Mycobacterium tuberculosis [1]. Many of them are suffering from diabetes mellitus [2]. In fact, diabetes mellitus is recognized as a potential risk factor for tuberculosis [3]. Prediabetes is an early stage in the course of type 2 diabetes mellitus. It is associated with glucose intolerance and hyperinsulinemia. Recent reports suggest that the pre-diabetes stage is also an independent risk factor for tuberculosis [4-6]. However, in prediabetes, the glycated hemoglobin (HbA1C) is around 5.7 - 6.4% [7], but authorities still believe that HbA1C below 7% is not enough to increase the risk of tuberculosis [8]. Therefore, the increased association of tuberculosis with prediabetes may be caused by some factors other than the glycation state (HbA1c). Hyperinsulinemia may be one such factor. It is one of the hallmark features of the prediabetes state [4,9,10]. This aspect requires explicit confirmation by observation-based studies since if hyperinsulinemia is proved to be a risk factor for tuberculosis; it requires a specific understanding to avoid insulin supplementation in prediabetes. At the present moment, there are some experiences of aggressive insulin therapy in prediabetes to reduce HbA1C without considering the insulin status [11-13]. The benefit or harm of such studies is still not understood. Moreover, currently no definitive view on the matter is existing in the literature. If insulin per se in hyperinsulinemic state is proved to be causative of tuberculosis by observational studies, such practice of insulin administration in prediabetes has to be done with caution and appropriate guidelines should be framed for the purpose. Interestingly, advanced diabetic state, characterised by persistent hyperglycemia and hypoinsulinemia due to extensive destruction of insulin-producing pancreatic beta cell mass is also associated with tuberculosis infection [14-16]. There are established mechanisms in the body to cause hypoinsulinemia as a result of chronic hyperglycemia [16] where tuberculosis infection is more frequent than the usual host. So, both hyperinsulinemia state in prediabetes and hypoinsulinemia state in advanced diabetes seem to be risk factors for the development of tuberculosis. It appears that both high and low concentration of insulin in the natural course of pathogenesis of diabetes in the context of existing hyperglycemia is favourable for the tuberculosis bacterium to cause the disease. Therefore, the presented view regarding altered insulin level and occurrence of tuberculosis should be verified on an urgent basis to understand whether insulin concentration is critical for predisposition to tuberculosis or not.

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Conflict of Interest

The authors declare that there is no conflict of interest associated with this manuscript.

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