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Infectious Pathoetiology of Type I Diabetes and its Interventional Implications

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Introduction

As per the American Diabetes Association, in 2015 approximately 30.3 million (9.4%) of Americans had diabetes with 1.25 million children and adults affected by type I diabetes [1]. The recommendations for treatment of Type I Diabetes (T1D) have changed over the years, but have remained largely dependent on the development of tools for measuring of blood glucose and adjustment of the insulin regimens. Historically, the exact pathoetiology of type I diabetes has remained largely unknown, and genetic predisposition is thought to be one key contributor to the T1D development. Recent findings are shedding light on infection as a possible pathoetiology of T1D.

Discussion

The congenital and childhood infections may possibly be an etiology of T1D. It is hypothesized that in certain genetically predisposed individuals, persistence of an initial viral infection in pancreatic tissue can cause chronic local inflammation and activation of autoimmunity through molecular mimicry, bystander activation, or both [2]. In the BABYDIET study, it was found that the early childhood respiratory infections are a potential risk factor for the development of T1D [3]. In a three-year follow-up study done in human leukocyte antigen (HLA) conferred T1D susceptible children of European descent, it was found that infections occurred more often at age of 1-3 years in children who developed islet cell auto-immunity and became T1D progressors [4]. Furthermore, the children who developed islet cell auto-immunity and became T1D progressors developed infections at a younger age compared to the non-diabetic children [4].

In a recent report on TEDDY study involving 7,869 newborns followed till age of 4 years and having HLA genotypes conferred T1D risk, it was found that respiratory infections which occurred in a 9-month period were associated with a significant risk of developing islet cell autoimmunity. For each one per year rate increase in infections, the hazard ratio for islet cell autoimmunity increased by 5.6% [5]. Furthermore, the respiratory infections independently associated with autoimmunity were common cold, influenza-like illness, sinusitis, and laryngitis/tracheitis with influenza-like illness and sinusitis holding highest hazard ratio of 2.37 and 2.63, respectively [5]. In another large study done in Germany where infants were followed till 8 years of age, there was an association with T1D development and recurrent viral respiratory infections during the first 6 months [6].

The enterovirus (EV) infection may play a role in increasing risk of T1D development. A large systemic review and metanalysis has shown that there is a clinically significant correlation between the enterovirus infection and the development of T1D/auto-immunity [7]. In a Taiwanese retrospective study on children aged up to 18 years, incidence of T1D was higher in EV than in the non-EV infection cohort [8]. In Egyptian children with T1D, 26.2% had EV RNA positivity versus 0% of healthy controls [9]. Overall, larger prospective studies are needed to show age group stratified incidence of EV infection, type of EV strain involved, and to draw a strong association between EV infection and T1D.

Conclusion

Multiple recent research studies have shown a positive association between the respiratory or EV infections and the development of T1D making preventative interventions a key. Larger prospective studies are needed to define the types of respiratory pathogens and EV involved. Based on the pathogenic findings, preventative interventions such as vaccinations, immunoglobulins, and guidelines targeted to provide appropriate care of respiratory or EV pathogens can be developed. Furthermore, these preventative interventions can be used not only in children, but may also be used in pregnant women. Overall, infections as a possible pathoetiology of T1D development opens up new interventional prospects.

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