

## Pathophysiology and Management of Type 1 Diabetes Patients

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### Abstract

The incidence of diabetes mellitus (DM) in developing countries like Asia has increased over the past few years. This is secondary to the changing development along with lifestyle of the younger generation who prefer to take up the western style of eating high calorie fast food with minimum exercise. Previously, the rate of DM was very low but is increasing at significant rates which are a cause of concern in Asia. There is not much literature available regarding T1D in Asian countries in view of lack of lower field surveys and absence of quantitative data. However in USA approximately 30% of subjects having T1D don't achieve the aimed glycaemic targets and lot of comorbidities exist which suggest some novel treatment avenues which include lifestyle modifications. Recent nutritional guidelines implicate a flexible approach in carbohydrate intake which is in relation to intensive insulin therapy. Basically these guidelines are meant to get more freedom in nutritional choices but may result in greater calorie intakes and unhealthy eating patterns which are resulting in higher prevalence of obesity and Metabolic Syndrome in people living with T1D, a phenomenon observed worldwide, so that recently the term diabetes got coined to understand the importance of treating the 2 together. Low carbohydrate/day (LCD<130g/day) might be a better way of getting a glycaemic control and metabolic health in subjects with T1D. Recreational exercise on a regular basis or getting a high athletic performance might be of importance in many people living with T1D. Work done on subjects not having T1D implicates that if athletes do training using decreased carbohydrates enhance adaptation in contrast to that with normal/high carbohydrates. Yet this train low aspect has not been checked in T1D subjects. Further use of low restricted/periodised carbohydrate diets in T1D athletes is discussed. Type 1 diabetes mellitus (T1D) reflects a heterogenous autoimmune disorder which involves millions of subject's worldwide. The basic characteristics of T1D are damage of the insulin generating  $\beta$  cells which takes place in view of abnormal activation of various immune effector cells. At present T1D treatment is done by lifelong delivery of novel kinds of insulin which have been synthesized recently. The aim of T1D care, that has been guided by the Diabetes Control and Complications Trial (DCCT) are to get good glycaemic control, to avoid hyperglycaemia (as it is correlated with long-term microvascular as well as macrovascular complications) along with preventing recurrent hypoglycaemic episodes (as they adversely influence cognitive function). But even following repeated optimization of insulin therapy regimens, the hormonal substituted treatment only works to treat the symptoms without influencing the etiopathogenesis. New approaches which could influence the underlying modes responsible for  $\beta$  cells destruction have been evaluated in detail. These methods on the basis of immunotherapies have got incorporated within a panel of existing therapies for T1D, to block T cell responses against  $\beta$  cells antigens which are quite common at the time of initiation as well as development of T1D. But a total preservation of  $\beta$  cells mass along with insulin independency is not getting achieved despite massive exploration. Due to that right now no existing targeted immunotherapies are capable of replacing the standard insulin delivery. Currently a lot of interest has been concentrated on

preventive methods in high risk subjects, on the basis of the posit that a therapeutic intervention once applied the early stage of disease, might aid in sustaining the endogenous  $\beta$  cell function by preserving the residual  $\beta$  cells reserve by the autoimmune attack. In this review we try to evaluate the present status of immunotherapies in T1D by emphasizing on the most significant studies in this field and detailing on novel methods that might get utilized to treat T1D in the future a death warning became a disease that could be controlled although still remaining a chronic disease, with insulin not curing the disease process, rather only its consequences i.e. blood sugars. Noticeably insulin does not achieve normoglycaemia. Despite maximum sophisticated, practically 'near closed loops' ways, glucose homeostasis does not get back to normal. Both short as well as long term complications are incurred by T1DM patients, with hypoglycaemic as well as hyperglycaemic events as well as long term effects of enhanced glycosylation of proteins resulting in eye, renal, central nervous system (CNS) as well as other complications. These sequelae are correlated with marked morbidity as well as mortality despite following aggressive insulin treatment. Practically a century after insulin discovery, we still battle with the hurdle of addressing disease process by itself, just to make the life of these patients better. Lot of work have been done to be able to totally arrest the autoimmune mechanism damaging the insulin synthesizing cells within the pancreas, or minimum at least reduce the speed of the process for blunting as well as delaying short as well as long term complications. Basic idea is to discuss a method that might aid in quantitative result measurements by particular therapies, short or clinical cure might be contrasted and exact advantage of their help in DM treatment might get assessed by the T1DM metabolic recovery index (DMMRI).

Type 1 diabetes mellitus (T1D) represents roughly 5-10% of all diabetic patients. Prevalence of this pathology points that >500,000 children suffer from T1D globally, that are located mostly in North America as well as Europe [1]. But the epidemiology points that the incidence of T1D has escalated rapidly in recent yrs. In 2017, the International Diabetes Federation (IDF) declared 132,600 newly diagnosed T1D cases all over the world. It is predicted that incidence of T1D in  $\leq 5$ yr age will increase 2 fold in <than 20yrs. But a total preservation of  $\beta$  cells mass along with insulin independency is not getting achieved despite massive exploration.