Received: 20-11-2014; Revised: 25-12-2014; Accepted: 26-12-2014

DIAGNOSIS AND PROGNOSIS OF DIABETES: A SYSTEMIC REVIEW

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Keywords:
Type 1 diabetes, T2DM, a-HB, L-GPC, Children

ABSTRACT

Type 1 diabetes (T1D) is a chronic autoimmune disease which is related with numerous long term micro and macrovascular complications. The skill to identify individuals at high risk for type 1 diabetes using genetic and/or autoantibody pointers has been a long lasting goal of the diabetes research and medical community and a critical part in T1D prevention strategies. Research in this era reports progressively higher levels of a-HB and lower levels of L-GPC across quartiles of insulin resistance and in those with impaired glucose tolerance or T2DM. Obesity is toughly associated with insulin resistance, which, when attached with relative insulin deficiency, leads to the development of obvious T2DM. Children and adolescents with T2DM may experience the microvascular and macrovascular complications of this disease at earlier ages than individuals who develop diabetes in adulthood, counting atherosclerotic cardiovascular disease, stroke, myocardial infarction, and sudden death; renal insufficiency and chronic renal failure; limb-threatening neuropathy and vasculopathy; and retinopathy leading to blindness.
INTRODUCTION
Type 1 diabetes (T1D) is a chronic autoimmune disease in which the destruction of the insulin producing cells and resulting clinical indicators are headed by the onset of a number of islet-cell specific autoantibodies. Linkage[1] and association analyses have established that type 1 diabetes has a very strong genetic component, with specific alleles and haplotypes at the HLA class II genes, as well as HLA-A and -B alleles, convening either susceptibility to or protection from T1D [2–6]. The ability to identify individuals at high risk for type 1 diabetes using genetic and/or autoantibody markers [7–10] has been a long-standing goal of the diabetes research and clinical community and a critical element in T1D prevention strategies. The role of forecast in prevention is twofold: 1) Clinical trials to evaluate possible defensive interventions are more efficient if the recruited subjects are at high T1D risk, and 2) Interventions are likely to be more operative if administered early in disease development or during the prediabetic phase, a stage identified by autoantibody markers in individuals who carry genetic risk alleles.

There are many problems faced by the patients suffering with diabetes like Eye complications, Gum disease, Skin disorder, Ketoacidosis, Hypertension, Gastro paresis, Hearing loss, Foot complications, Mental health, Heart problems, Neuropathy, Nephropathy, Stroke, Infections, Erectile dysfunction and Healing of wounds.

There are some causes of diabetes similar to genetically disease it can be transferred from parents to the off spring. Diabetes mellitus is mainly caused by the insulin resistance. Obesity is the major cause of diabetes mellitus, age, to have blood vessels disease, Giving birth to a large baby, High blood pressure, High cholesterol level, Due to in conversion of sugar into energy, Due to large intake of sugar etc.

There are some symptoms of diabetes like frequent urine discharge, feel tired soon, loss of weight, mild thirst, fatigue, women face some vaginal infections. T2DM is characterized by insulin resistance, often associated with obesity, and insufficient insulin secretion to overcome the insulin resistance. As the occurrence of obesity increases, so too is the prevalence of T2DM. Still, depending upon their degree of obesity, only w25–40% of obese individuals develop T2DM [11]. Trials such as the Diabetes Prevention Program (DPP) have demonstrated that exhaustive lifestyle and pharmacologic interventions can prevent or delay the onset of T2DM. Knowing who is likely to progress to T2DM will help target these interventions.[12] Gestational
diabetes affects the females during pregnancy as well. In this diabetes the glucose level is more in the body but the body is unable to produce insulin. Thus the transport of glucose in the body becomes impossible. It raises the level of glucose.

Recently, Ferrannini and colleagues[^13] used a no targeted approach to identify plasma metabolites associated with insulin resistance and/or glucose intolerance. The two top-ranked metabolites were an organic acid, α-hydroxybutyrate (α-HB), and a lipid, 1-linoleoylglycerophosphocholine (L-GPC). To confirm and further explore these findings, they developed targeted assays to quantify the metabolites. In this issue of Diabetes, Ferrannini et al.[^14] report progressively higher levels of α-HB and lower levels of L-GPC across quartiles of insulin resistance and in individuals with impaired glucose tolerance or T2DM. They also demonstrate higher levels of α-HB and lower levels of L-GPC at baseline in those individuals with deteriorating as opposed to stable glucose tolerance after 3 years of follow-up and those individuals who progress to T2DM after 9.5 years of follow-up. When added to a model for predicting incident hyperglycemia or T2DM that included family history of diabetes, sex, age, BMI, and fasting glucose, the fasting levels of these two metabolites improved predictivity, similar to the addition of 2-h glucose. When the model included both fasting and 2-h glucose, the two metabolites had only minimal impact on predictively. With these results, Ferrannini et al. propose fasting α-HB and L-GPC levels as new biomarkers to help predict hyperglycemia and T2DM. Associations defined in metabolomics studies may be only correlative, but, in some cases, may redirect an underlying influence to pathogenesis. In addition to relations with insulin resistance, Ferrannini et al. also demonstrated an inverse relationship between α-HB levels and trials of b-cell function; a relationship with L-GPC levels was not observed. To discover a potential mechanism for this relationship, the effect of α-HB and L-GPC on glucose-inspired insulin secretion (GSIS) was examined. Using an immortalized rodent b-cell line, inhibition of GSIS by α-HB and stimulation by L-GPC were established, consistent with their association with disease development. Yet, the absence of a association of L-GPC with b-cell purpose, despite an effect on GSIS, relics unexplained, and the findings are yet to be established in human islets. Furthermore, although the results may explain, in part, association with the risk of progression, they do not clarify the observed associations with insulin match. Thus, much remains to be educated about the role of these metabolites in hyperglycemia T2DM.
The ability to predict type 1 diabetes on the root of immunologic, inherited, and metabolic indicators has led to numerous large studies intended to determine whether type 1 diabetes can be prohibited by intervening in individuals with recognized autoimmunity. Unfortunately, to date, such educations have been without pure success\textsuperscript{[15]}. In part, this can be qualified to the selection of interventions that force minimal risk to the topics participating in the hearings. Though, it also may be that once autoimmunity is established, the immuneprocedure progressively enlarges, involving more and more components of the immune organization and directed at an increasing number of islet auto antigens. Cell function is assessed by calculating C-peptide response to a provocative stimulus\textsuperscript{[16]}. A whole diversity of interventions has been discovered at this stage. \textsuperscript{[17]} Certain interventions have shown possible benefit but were limited by toxicity or by the advantage being of limited scale and or short duration of benefit.

Just, the popular of cases of diabetes mellitus between children and youths were immune-mediated type 1a diabetes. Obesity has led to a penetrating growth in the incidence of type 2 diabetes (T2DM) among children and adolescents over the past 2 eras. Obesity is strongly associated with insulin resistances when coupled with comparative insulin deficiency, hints to the development of overt T2DM. Children and adolescents with T2DM may experience the microvascular and macrovascular complications of this illness at younger ages than individuals who develop diabetes in maturity, including atherosclerotic cardiovascular disease, stroke, myocardial infarction, and sudden death; renal insufficiency and chronic renal failure; limb-threatening neuropathy and vasculopathy; and retinopathy leading to blindness. Health care professionals are advised to perform the suitable screening in children at risk for T2DM, diagnose the disorder as early as possible, and provide hard management of the disease.
FIG I Causes of Diabetes

Food with Animal fat

Not exercise

Obesity: Need extra insulin it becomes resistant

Fat deposits in pancreas cause it more damage

So body need more insulin it cannot produce result in Type 2 diabetes

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