



A Brief Study on Diabetes Mellitus: Pathophysiology and Diagnosis

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

Background: This article reviews published articles and existing diagnostic and therapeutic techniques relevant to adolescents with obesity and diabetes mellitus.

Obesity, in addition to DIABETES MELLITUS, is a crucial risk factor for the occurrence of future chronic and noncommunicable diseases.

Obese and overweight teenagers are more prone to acquire Adult Onset Diabetes, formerly rare among the younger population. However, a global trend of Adult Onset Diabetes was noted in the late 1990s and early 2000s. In the United States and the United Kingdom, and other developing and developed countries, this is particularly true. Adult Onset Diabetes is nearly as common as Insulin Dependent Diabetes in some locations. There has been a significant surge in the prevalence and severity of obesity in several population groups in teenagers.

Objective and Methodology: To read and review the existing literature on the prevalence of Diabetes Mellitus and obesity in adolescence. About 15 articles and literature were studied using the PubMed and Google Scholar search engine to produce a detailed review article on the topic of interest so chosen.

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Results and Conclusion: After reviewing the articles, we can agree that TYPE-2 DIABETES MELLITUS is linked easily to young adults who are obese, that is, having a Body Mass Index of more than 30 or more than 30. TYPE-2 DIABETES MELLITUS causes destruction of receptors for insulin which results in an increased level of glucose, causing various diseases. Obesity in young adults is a critical factor in the occurrence of Adult Onset Diabetes which makes them at risk of developing severe diseases in the 3rd or 4th decade of life.

Keywords: Obesity; adolescents; hyperinsulinemia; adult onset diabetes; hyperglycemia.

1. INTRODUCTION

Adult Onset Diabetes was not considered widespread in the pediatric age group or young adults. However, increased cases of Adult Onset Diabetes have been observed worldwide since the late 1990s to early 2000s. This is particularly the case in countries like the US, UK and has been reported in other countries, both developing and developed [1,2]. In some countries, the prevalence of Adult Onset Diabetes can be as common as Insulin Dependent Diabetes. A striking increase has been observed in the generality of Adult Onset Diabetes amongst the overweight young adult population [1,3].

Obesity in both industrialized and emerging countries, in children and adults, is the most typical type of malnutrition to be seen globally [4]. It is the sixth most crucial worldwide mortality risk factor [3]. Obesity can be considered a significant factor responsible for this increasing trend of Adult Onset Diabetes in young adults. This attributes to the fact that the universality of the obese population is not increasing, but the effect it has on children and adolescents is causing problems. Obesity is the leading cause of 5% of all global morbidities [5]. Dietary factors are the primary cause of obesity in young adults.

Obesity predisposes an individual to many comorbidities, not only Type 2 DM but also to chronic heart diseases, hyper-insulinemia, hypertension, polycystic ovarian syndrome, hyper-androgenesis, and many others [5].

Diabetes Mellitus type 2 is a dangerous condition long-standing doing case of Diabetes Mellitus can result in chronic consequences such as accelerated cardiovascular disease, chronic renal disease, retinopathy, and limb amputations due to gangrene's development following an injury to the limb, neuropathic changes, and many more. These problems present an increased risk of morbidity and mortality in people with this disease [1].

We as a society are going to see several young adults with complications if the rate at which Type 2 DM keeps on increasing amongst the adolescent population of the country. The effect we will have on DIABETES MELLITUS and its complications will change the lives of our coming generation if the situation is not handled carefully [1].

The critical factor for the disease mentioned is obesity. The majority of youngsters with Adult Onset Diabetes are obese with BMI higher than 30 or severely overweight at the time of clinical symptoms, with reducing sugar in urine but no ketone bodies, MILD POLYURIA and POLYDISPIA, and almost no loss of weight [6]. Young adolescents in the early phases of puberty are currently diagnosed as TYPE-2 DIABETES MELLITUS [1,6].

2. PATHOPHYSIOLOGY

Adult Onset Diabetes, also known as type-2 diabetes mellitus, arises from insulin resistance and relative insulin insufficiency in the absence of damaged beta cells. The illness has a substantial hereditary (possibly multigenic) component, important being GENETIC MAKEUP highlighted with disparities about Adult Onset Diabetes in different races is taken into account [1,6]. Significant progress has been made in our understanding of the familial basis of the disease-related; these new findings in the prevalence of the disease discussed are too rapid due to increasing gene frequency and a changed gene pool, highlighting the role of environmental influences [1].

Beta-cells of the pancreas secretes insulin, and INSULIN action must be in equilibrium for glucose homeostasis to occur. Insulin resistance to glucose absorption driven by insulin is a problem—a well-known feature of patients with Adult Onset Diabetes and impairment of glucose metabolism. The progression from good glucose tolerance to IMPAIRED GLUCOSE

TOLERANCE (IGT) is linked to increased insulin resistance [1].

Impaired glucose endurance is a stage linked with an increased risk of being diagnosed with the disease discussed and cardiovascular disease. Insufficient insulin is not enough to cause the onset of the disease discussed; insufficient beta-cell insulin production is also required [1,7].

In children and young adults with diminished endurance of insulin, however, there will be a significant rate of quick and spontaneous conversion from diminishing glucose endurance to standard glucose tolerance in the coming few years [8]. Changes in insulin resistance around adolescence have been blamed for this normalization [1].

Insulin secretory failure and reduced insulin action are common in TYPE-2 DIABETIC INDIVIDUALS. Hyperglycemia is being demonstrated to worsen insulin resistance and its secretion irregularities, hastening the transition from glucose resistance into DIABETES [1,9].

Adolescence plays a significant influence on the occurrence of the disease mentioned [9]. In adolescence, there is a spike in insulin tolerance, culminating in an increased glucose level in the blood [1].

Insulin responses, both basal and stimulated, decrease during puberty. Insulin-mediated glucose disposal is 30 percent poorer in teenagers between Tanner stages II and IV compared to prepubertal children and adolescents, according to hyperinsulinemic-euglycemic clamp studies. Insulin resistance during puberty is hypothetically linked with increased GROWTH HORMONE released during puberty [1,10].

Adult-onset diabetes is marked by obesity [11]. A large proportion of children with the disease mentioned and discussed are obese or have excessively increased body mass index at being diagnosed, with a high BMI, sugars in urine but no ketone bodies, absence or minimal POLYURIA and POLYDISPIA, and essentially no loss of weight. Adult Onset Diabetes is currently identified in young adolescent girls and boys above the age of 10 who are still in the early phases of puberty [11,12].

3. DIAGNOSTIC CRITERIA FOR ADULT-ONSET DIABETES IN YOUNG ADULTS

Even the absence of beta-cell autoantibodies is what defines Type 2 DM. Positive beta-cell antibodies in insulin-independent adolescents and young adults may indicate the presence of a latent self-destructing diabetes mellitus comparable to that seen in adults (LADA: LATENT AUTOIMMUNE DIABETES MELLITUS of Adults). According to a global survey, beta-cell autoantibodies occur in 10% to 20% of adult diabetics who do not use insulin. [1,12]. Insulin resistance is similar in Latent Autoimmune Diabetes Mellitus of Adult patients to type 2 diabetes patients, but their -cell capacity is drastically diminished [1].

Clinical features of DIABETES MELLITUS, such as POLYDISPIA, increased urine output, and loss of weight without any explanation, are combined with a casual glucose concentration of 200 MG/DL (11.1 mmol/L) in venous plasma, fasting glucose, of 126 mg/dL (7.0 mmol/L) in venous or capillary plasma, or two-hours glucose during LGBT of 200 MG/DL (11.1 mmol/L) in The American Diabetes Association (ADA) has changed its criteria to allow for the use of hemoglobin A1c (HbA1c) levels of less than 6.5 percent in the diagnosis of disease mentioned [1]. FASTING GLUCOSE, HBA1C, GTT tests must be performed on a different day in the case of asymptomatic manifestation [1,13].

Most people suffering from this disease can be effectively classified based on their symptoms, presentation, and course. Other tests, such as preprandial GLUCOSE or c-peptide determinations and, on rare occasions, cell autoantibodies measures, may be required in the event of an uncommon scenario that necessitates a specific classification. A set of tests is required to reach a high degree of sensitivity, which considerably raises the cost of categorization [1].

In patients with Adult Onset Diabetes, C-peptide levels were higher than those with insulin-dependent diabetes or MODY diabetes. Individuals with immune-mediated insulin-dependent diabetes have autoantibodies against INSULIN, GAD-II, or the TYROSINE PHOSPHATASES antibodies (IA)-2 and IA-2b at the time of diagnosis. HLA typing is not a useful marker for insulin-dependent diabetes, with a

tremendous HUMAN LEUKOCYTE ANTIGEN connection [14].

The diagnosis of Adult Onset Diabetes in its mildest form is achieved during a routine medical check-up in an asymptomatic child by detecting hyperglycemia or glycosuria [1,15]. Urinalysis is used to diagnose one-third of patients during standard physical examinations [1,15]. Increased urine output, polydipsia, and loss of weight are symptoms of this condition in its most severe form. At diagnosis, ketonuria affects up to 33% of people in some racial groups, while ketoacidosis affects 5% to 25% of people at presentation [15].

4. EPIDEMIOLOGY

Most juvenile persons diagnosed with Adult Onset Diabetes were from certain racial minority groups such as Black-Americans, Hispanics, south Asian Islanders, and American-Indians, with Pima Indians having the most significant prevalence (22.3/1000 in 10- to 14-year-old population). In addition, most of the subjects found to be overweight. According to screening studies, overweight adolescents aged 12 years had a prevalence of Adult Onset Diabetes ranging from 0.4 percent to 1% [1]. Compared to TYPE-1 DIABETES, the overall occurrence of Adult Onset Diabetes in the entire pediatric age surveyed remained low. Even though there is universal agreement that Adult Onset Diabetes in young considered to be becoming a severe medical problem [1,15], this has led some researchers to dispute the assertions of an "EPIDEMIC" of pediatric Adult Onset Diabetes [1].

5. COMPLICATIONS IN YOUNG ADULTS WITH TYPES 2 DIABETES MELLITUS

Heart-related complications are common in Adult Onset Diabetes adolescents than insulin-dependent diabetes adolescents. According to a TODAY study, 14 % of adolescents suffering from Adult Onset Diabetes had a high blood pressure level, 80% had reduced HDL levels, and 10% had high levels of TRIGLYCERIDES [15].

Contrasted to Adults with DIABETES, the pediatric age group and young adults with the disease mentioned and discussed have a higher risk of complications. As a result, Adult Onset Diabetes diagnosed at a juvenile period of life is linked to a substantially increased chance of developing into heart-related disease than Adult

Onset Diabetes diagnosed at 30-45 years [15] junior persons with TYPE-2 DIABETES tend to have a greater chance of acquiring DIABETES-RELATED problems early in life than those with TYPE-1 DIABETES. This increased possibility was linked to the incidence of hypertension and dyslipidemia rather than overall glycemic control or disease duration [16].

Microvascular diabetic diseases like retinopathy and microalbuminuria can be diagnosed early and are the hallmark of hyperlocal

6. DISCUSSION

Several research have been conducted in recent years and discovered that increased weight increases the risk of Adult Onset Diabetes by over 40%. The rate at which obesity has become more common among teenagers and the obesity-related co-morbidities is concerning.

Even though current research suggests that Diabetes Mellitus is mostly a hereditary disease, its link to obesity must not be neglected [1,16].

In youngsters, puberty appears to impact the development of the disease mentioned and discussed significantly. During adolescence, INSULIN RESISTANCE increases, eventually leading to increased glucose levels in the blood. Both basal and stimulated insulin responses decrease during adolescence. INSULIN-MEDIATED GLUCOSE clearance is thirty percent worse in teenagers between TANNER STAGES II, and IV seen in contrast to primary children and youngsters, according to hyperinsulinemic-euglycemic clamp studies. Insulin resistance during puberty is hypothesized to be caused by increased GROWTH HORMONE production throughout puberty [1,16].

INSULIN RESISTANCE and inadequate β -cell INSULIN production were causes of diabetes mellitus. INSULIN secretory failure and decreased insulin action are frequent in patients with the disease mentioned and discussed. Hyperglycemia has been shown to exacerbate intolerance of insulin and aberrant INSULIN production, hastening the establishment of diabetes mellitus due to decreased glucose tolerance [16].

7. RATIONALE

There is a physiological increase in insulin levels throughout puberty triggered by fat, resulting in

hyperinsulinemia, which invariably leads to insulin intolerance, leading to adolescent non-insulin-dependent diabetes. Insulin receptors become immune to insulin due to the development of insulin resistance. Beta cells secrete more insulin at the pre-clinical stage of the disease, resulting in hyperinsulinemia [17-21].

Obesity hurts glucose metabolism starting in childhood. Obese children are hyperinsulinemic, with insulin-stimulated glucose levels 40 percent lower than non-obese children. Most diabetes mellitus can be classified based on their clinical presentation and progress. If an uncommon scenario justifies a specific classification, additional tests, such as preprandial insulin or C-peptide readings, and, in rare cases, cell autoantibody measures may be required. A succession of tests is required to reach a high level of sensitivity, which considerably raises the cost of classification. Adversity is more likely in children and young adults with the disease mentioned and discussed [22-24].

As a result, TYPE-2 DIABETES diagnosed at an even more young are linked to a higher risk of long-term heart-related disease than Adult-onset diabetes detected later in life. Adolescents with non-insulin-dependent diabetes are considerably more likely than those with insulin-dependent diabetes to have DIABETES-related problems early in life. This increased risk appears to be linked to the onset of hypertension and dyslipidemia rather than overall glycemic control or the length of time the condition has been present.

8. CONCLUSION

Many factors influence the clinical symptoms of Diabetes Mellitus. Obesity is a significant contributor to the presence and severity of this illness, although it is neither sufficient nor necessary for Diabetes Mellitus to develop.

Adult Onset Diabetes is still uncommon in youth and puberty, but current research suggests that its incidence is increasing over the world, possibly as a result of rising childhood and teenage obesity rates. This is particularly true in the United States, but it has also been observed in Asia and Europe.

If they show clinical symptoms of intolerance to insulin (blackening of neck and axilla, DYSLIPIDEMIA, increased blood pressure,

POLYCYSTIC OVARIAN SYNDROME), family history of Adult Onset Diabetes, or come from specific race-related groups, obese children and adolescents over the age of 10 should be evaluated for Adult Onset Diabetes (Asian, American Indian, African-Americans, Hispanics). Preventing and treating the disease mentioned and discussed should focus on public health intervention programs. Prevention and the development of preventative strategies should be prioritized much earlier in our society.

Beginning in childhood, obesity has a harmful impact on glucose metabolism. Overweight early adolescents are HYPERINSULINEMIC and have a 40% reduced GLUCOSE METABOLISM activated by insulin than non-obese children. In addition, visceral abdominal fat has a stronger negative relationship with insulin sensitivity than subcutaneous fat.

In conclusion, and probably more required, increasing public awareness of the epidemic's mounting health and economic consequences is vital. If not taken into consideration and adequate measures in controlling the incidence of the disease so highlighted, in near time, there can be a steep rise in the adolescent type 2 Diabetes rate, making the future population of the country susceptible to various other co-morbidities associated with it. Physicians should inform the public about the pediatric obesity pandemic and its severe consequences, including the disease mentioned and discussed. Appropriate steps should be taken for the timely diagnosis of this disease, thus mentioned and discussed, to prevent any further future risks of the disease.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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