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Evaluation of Pulmonary Function Parameters in Children with Type 1 Diabetes Mellitus

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ABSTRACT

Introduction

A class of illnesses known as diabetes is defined by elevated blood glucose levels.. It results in protein and lipid metabolic abnormalities and is caused by a lack of insulin, either in its synthesis or activity, or both. Many factors can cause this. People with type-1 diabetes (T1D), a chronic condition, have little or no insulin produced by their pancreas. More than a million children and teenagers are anticipated to have T1D in 2019. Around the world, the prevalence of T1D is rising. Type 1 diabetes mellitus (T1DM) is among the most prevalent endocrine disorders in children. T1D patients die four to five times as often as the normal population, and chronic complications account for about 30% of all fatalities . Reduced lung volumes, changed alveolocapillary diffusion, and decreased elastic lung recoil are all reported in studies involving adult T1DM patients.

However, There is a lack of information on the prevalence of T1DM in children.

The lungs' antioxidant defenses are known to be secondarily reduced by hyperglycemia, making the body more vulnerable to oxidative stressors and ultimately leading to a decrease of respiratory function. Data on these parameters in children with type 1 diabetes are few. The purpose of this study is to evaluate lung function parameters in children with type 1 diabetes.

Aims and Objectives

Aim:

To assess pulmonary function changes in children with Type 1 Diabetes Mellitus (T1DM) and explore their potential association with glycemic control and other influencing factors.

Objectives:

1. To evaluate and compare pulmonary function test (PFT) parameters, including Forced Vital Capacity (FVC), Forced Expiratory Volume in 1 second (FEV1), Diffusion Capacity for Carbon Monoxide (DLCO), and Peak Expiratory Flow Rate (PEFR), in children with T1DM versus healthy controls.
2. To analyze the impact of glycemic control, as measured by HbA1c levels, on pulmonary function parameters in children with T1DM.
3. To determine whether chronic hyperglycemia contributes to restrictive or obstructive lung function changes in pediatric diabetic patients.
4. To investigate the role of disease duration in the progression of pulmonary function changes in children with T1DM.
5. To examine potential genetic and environmental influences, such as family history of diabetes and lifestyle factors, on lung function abnormalities in this population.
6. To assess the correlation between HBsAg status and pulmonary function changes, if any, in the study cohort.
7. To provide clinical recommendations regarding the necessity of routine pulmonary function monitoring in pediatric T1DM patients for early detection of respiratory complications.

Results ;

Type 1 Diabetes mellitus group and control group and found that 56.25%(18) were males and 43.7%(n-14) were females among cases and among control group 63.3%(n-19) were males and 36.6%(n-11) were females, 12.5%(n-4) had history of maternal history of DM. The mean and SD of FVC,FEV,FEV/FVC Is 2.03(0.474), 1.72(0.523) and 80.41 (16.71) respectively. It is found to be significant

Conclusion

This study aimed to evaluate pulmonary function parameters in children with Type 1 Diabetes Mellitus (T1DM) and analyze potential impairments associated with the disease. The findings reveal significant alterations in pulmonary function in children with T1DM compared to healthy controls, highlighting the need for regular respiratory monitoring in diabetic children.

The results indicate that key spirometry parameters, including **Forced Vital Capacity (FVC),** and **Diffusion Capacity for Carbon Monoxide (DLCO),** were significantly reduced in children with T1DM. These findings suggest that diabetes-related metabolic changes may **contribute to restrictive lung patterns and decreased lung compliance**.

Additionally, the FEV1/FVC ratio was lower in diabetic children, pointing towards potential airway involvement. However, parameters such as Peak Expiratory Flow Rate (PEFR) did not show a significant difference, **suggesting that obstructive lung disease may not be a primary concern in this population**.

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Abbreviation

ADA	American Diabetes Association
DK	diabetic ketoacidosis
COPD	chronic obstructive pulmonary disease
GLP	glucagon-like peptide-
FEV	forced expiratory volume
FVC	forced vital capacity
HLA	human leukocyte antigen
NO	nitric oxide
OSAP	obstructive sleep apnea syndrome
PFT	Pulmonary function test
T1DM	type-1 diabetes
TNFA	tumor necrosis factor-alpha
TGF	transforming growth factor-beta
VEGF	vascular endothelial growth factor

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Evaluation of Pulmonary Function Parameters in Children with Type 1 Diabetes Mellitus

Introduction

The term "diabetes" refers to a collection of illnesses marked by elevated blood glucose levels. It is brought on by insufficient insulin, either in its manufacture or activity, or both, and causes anomalies in the metabolism of proteins and fats. This can be caused by a variety of circumstances. People with type-1 diabetes (T1D), a chronic condition, have little or no insulin produced by their pancreas[1].

Although it can manifest at any age, it is the main cause of diabetes in children. In 2019, it is projected that over a million children and adolescents will develop T1D.

"The prevalence of T1D is increasing globally. In children, type 1 diabetes mellitus (T1DM) is one of the most common endocrine conditions. 2. Over half of them live in developing nations; in India, there are an estimated 97,700 children with type 1 diabetes." Type 1 diabetes mellitus accounts for just 5–10% of all cases of diabetes. T1DM is becoming more common worldwide and has serious short- and long-term effects. "The Karnataka state T1DM registry revealed an incidence of 3.7/100,000 for males and 4.0/100,000 for females after 13 years of data collection.[2]"Geographical differences in T1D incidence and prevalence are significant. The microvascular consequences of type 1 diabetes include retinopathy, nephropathy, and neuropathy. Amputation of lower limbs, kidney failure, blindness, and cardiovascular disease are all primarily caused by diabetes.

One major source of mortality and disability is T1D. T1D patients die four to five times as often as the normal population, and chronic complications account for about 30% of all fatalities.[3]

All of the body's organs are impacted by T1D. In patients with diabetes, several studies have revealed fibrotic alterations and problems with pulmonary microcirculation. Diabetes and compromised lung function have been linked.[4]

Diabetes-related pulmonary dysfunction is primarily caused by two factors: diabetic microangiopathy and changes in collagen and elastin connective tissue.

The lung is believed to be a target organ for diabetes because of its extensive microvascular circulation and high amount of connective tissue.

The integrity of the lung's connective tissue and microcirculation are essential for gas exchange and pulmonary function, therefore conditions affecting Reduced gas exchange and mechanical lung dysfunction may be the outcome of these factors.[5, 6, 7]

Reduced lung volumes, changed alveolocapillary diffusion, and decreased elastic lung recoil are all reported in studies involving adult T1DM patients.

In people with diabetes, pulmonary dysfunction is brought on by alterations in collagen and elastin connective tissue as well as diabetic microangiopathy.

Diabetic illness is believed to target the lung because of its widespread microvascular circulation and abundance of connective tissue.[8] Abnormal connections between the

connective tissue and microcirculation in the lung might result in mechanical lung dysfunction and poor blood gas exchange since these components are essential to pulmonary function and gas exchange. Furthermore, diabetes's elevated systemic inflammation may cause pulmonary inflammation and, consequently, airway damage.

Furthermore, hyperglycemia may cause the lungs' antioxidant defences to deteriorate related to increased susceptibility to oxidative environmental assaults, ultimately leading to a loss of respiratory function.[9,10.] The results of research on pulmonary function impairment in T1D patients, however, show a great deal of diversity.

Van den Borst et al. (2010)[8] conducted a meta-analysis which revealed a correlation between T1D and a restricted pattern. This meta-analysis presented information on the lungs' carbon monoxide (DLCO) diffusing capacity, forced expiratory volume in one second (FEV1), forced vital capacity (FVC), and FEV1/FVC ratio.

However, There is a lack of information on the prevalence of T1DM in children. The lungs' antioxidant defenses are known to be secondarily reduced by hyperglycemia, making the body more vulnerable to oxidative stressors and ultimately leading to a decrease of respiratory function. There is a lack of data on these parameters in kids with type 1 diabetes. Assessing lung function parameters in kids with type 1 diabetes is the aim of this study.

AIMS AND OBJECTIVES OF THE STUDY

Aim:

"To assess pulmonary function changes in children with Type 1 Diabetes Mellitus (T1DM) and explore their potential association with glycemic control and other influencing factors."

Objectives:

1. **To evaluate and compare pulmonary function test (PFT) parameters, including Forced Vital Capacity (FVC), Forced Expiratory Volume in 1 second (FEV1), Diffusion Capacity for Carbon Monoxide (DLCO), and Peak Expiratory Flow Rate (PEFR), in children with T1DM versus healthy controls.**
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7. **To provide clinical recommendations regarding the necessity of routine pulmonary function monitoring in pediatric T1DM patients for early detection of respiratory complications.**

Review of literature

Type 1 diabetes (T1D) is caused by the immune system attacking the insulin-producing β-cells of the pancreas, leading to total insulin insufficiency. Because of the age-related variations in the metabolic, genetic, and immunogenetic features of T1D, each person requires a customized treatment plan. Many patients with the illness have underlying genetic risk. Therefore, the American Diabetes Association (ADA) suggests screening and providing T1D autoantibody testing to first- and second-degree relatives of people with T1D.[11,12] Multiple autoantibodies linked to T1D eventually cause clinical illness in affected individuals. It is possible for insulin secretion to decrease gradually or quickly. Individual differences exist in the clinical presentation, but the classic beginning symptoms include polyuria, polydipsia, and inadvertent weight loss. Although their symptoms may develop more gradually, adults with new-onset T1D typically exhibit symptoms that are comparable to those of children. Young patients with newly diagnosed type 1 diabetes are more likely to develop diabetic ketoacidosis.[2] In order to postpone the onset of clinical diabetes, disease-modifying medication has now been authorized in the early preclinical stages of type 1 diabetes.[13]

Etiology[14]

T1D, which leads to total insulin insufficiency, is caused by the autoimmune destruction of the β-cells in the Langerhans pancreatic islets. [5]. In genetically susceptible people, one or more environmental stressors initiate the autoimmune process, which results in immune-mediated β-cell death. Over the course of months to years, the affected person will gradually lose their β-cell function while remaining asymptomatic. When there is a considerable level of β-cell malfunction, symptomatic hyperglycemia begins.

Genetic Associations of Type 1 Diabetes[15]

The precise cause of type 1 diabetes is still unknown. The human leukocyte antigen (HLA) alleles DR and DQ, however, are significantly linked to a hereditary predisposition. A person's human leukocyte antigen (HLA) haplotype determines between 30% and 50% of their genetic risk. “A process that recruits antigen-presenting cells to transport beta cell self-antigens to autoreactive T cells is suspected to be initiated by a "triggering" insult (such as the maternal and intrauterine environment, exposure to viruses, host microbiome, diet, and many other factors that are thought to contribute to disease susceptibility). Reports indicate that HLA genes are responsible for over 40% of T1D familial aggregation. T1D is most strongly associated with the HLA class II DRB1, -DQA1, -DQB1 genotypes. Approximately 90% of children with T1D have been shown to have HLA DR4-DQ8 and3-DQ2.“ T1D patients' close relatives have a markedly elevated lifetime chance of getting the disease.

The majority of cases, meanwhile, happen to people who have no family history of T1D or another autoimmune condition.

This correlation is stronger in T1D that develops in youth as opposed to adulthood. A number of additional genes also play a role in heritability. To find those who could be at risk, family members must be screened, particularly first-degree relatives of those with T1D.[15]

Environmental Risk Factors

It is commonly accepted that in genetically vulnerable individuals, environmental conditions cause the demise of autoimmune β-cells. A higher incidence of T1D has been associated in certain studies with infections caused by the coxsackie virus, enteroviruses, cytomegalovirus, rubella virus, influenza B, mumps virus, and more recently, SARS-CoV-2 (COVID-19). Other environmental variables that may increase risk include childhood vaccines, pregnancy and postpartum conditions, and dietary factors such as exposure to cow's milk and cereal. The precise involvement of these environmental factors in the etiology of T1D is still being investigated.[16]

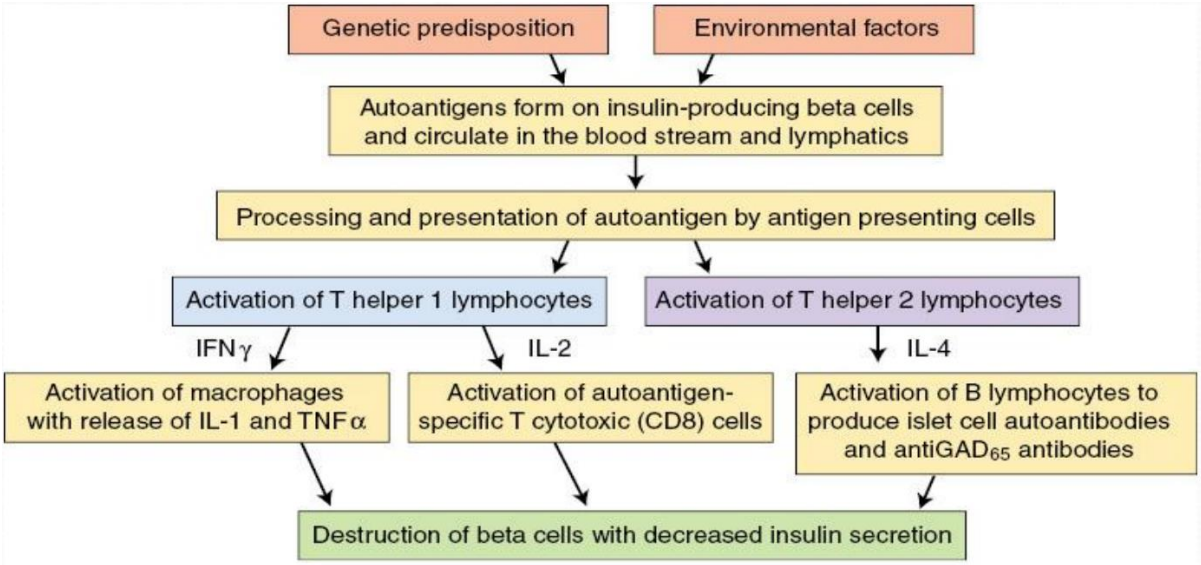

Figure 21-13 Pathophysiology of type 1 diabetes mellitus. *GAD_{65}*, glutamic acid decarboxylase; *INF*-γ Interferon-gamma; *IL*, interleukin; *TNF*-α, tumor necrosis factor-alpha.

Figure 1: Genetic and environmental cause for type 1 DM

Autoimmunity

The loss of β-cells is caused by immune-mediated T1D-related autoantibodies that target pancreatic β-cell autoantigens, in addition to hereditary and environmental variablesAutoantibodies target a variety of substances, including zinc transporter isoform 8 (ZnT8), insulin (IAA), insulinoma antigen 2/islet tyrosine phosphatase 2 (IA-2), glutamic acid decarboxylase isoform 65 (GAD65), and antigens in the cytoplasm of islet cells (ICA). The majority of IAAs are found in youngsters. The most prevalent autoantibody seen in adults is GAD65.[13] It is crucial to test for autoantibodies to pancreatic β-cell autoantigens in order to confirm the diagnosis and differentiate type 1 diabetes from other types of diabetes, primarily type 2 diabetes (T2D). An increased risk of T1D is associated with both the quantity of detectable antibodies and their titers. [11]

Epidemiology of Type 1 Diabetes mellitus [11]

Every age group can be impacted by T1D, however it is one of the most common chronic disorders among children. Symptomatic extreme hyperglycemia or diabetic ketoacidosis (DKA) are among the more severe clinical manifestations that are typically seen in children with T1D. New-onset T1D in adults can be mistaken for T2D, however T1D in children is more prevalent than in adults. While T1D seems to be slightly more common in men, autoimmune illness is generally more common in women.

With a steady rise in incidence and prevalence, T1D now accounts for between 5% and 10% of all cases of diabetes. According to a meta-analysis and comprehensive review, the incidence of T1D was 15 per 100,000 individuals, and the global prevalence was 9.5%.Globally, the regional incidence of T1D varies significantly. China and Venezuela are said to have the lowest prevalence, whereas Finland and other Northern European countries have the greatest reported instances, with rates over 400 times higher.

Of all cases of diabetes, only 5–10% are type 1 diabetes mellitus. Globally, the prevalence of type 1 diabetes is still rising, and it has major immediate and long-term effects. The prevalence of type 1 diabetes is increasing by 3% year, and over 90,000 children in Europe alone currently have the disease. Approximately 75,000 new children are diagnosed with type 1 diabetes each year. A larger prevalence is anticipated soon as a result of this rise in incidence, improved access to insulin, and improved survival rates. [2]

According to the Karnataka state T1DM registry, throughout the 13 years of data collecting, the incidence was 3.7/1 lakh for males and 4.0/1 lakh for girls. [18]T1DM prevalence in Karnal, Haryana, is 26.6/1 lakh in urban areas and 4.27/1 lakh in rural regions, for an average prevalence of 10.20/100,000 people. The prevalence of T1DM in Karnal city is quite high (31.9/100,000).According to a government sample survey carried out in schools across three cities, approximately one percent of Indian youngsters suffer from diabetes.

In Nainital (Uttarakhand), Ratlam (Madhya Pradesh), and Bhilwara (Rajasthan), 92,047 schoolchildren participated in a study on cancer, diabetes, cardiovascular diseases, and stroke as part of the National Program for Prevention and Control of these conditions[19]In this study, 1,351 pupils (1.467%) were suspected of having diabetes. In 2011, it is estimated 18,000 children below the age of 15 in the aforementioned regions received a new diagnosis of type 1 diabetes. An International Diabetes Federation report for the South-East Asian Region from the World Health Organization states that 1,11,500 children have type 1 diabetes.

Pathophysiology of type 1 Diabetes mellitus

There are three stages in the natural history and development of T1D in genetically vulnerable people.

Stage 1- known as the preclinical stage, autoimmune β-cell death and immune-mediated insulitis begin to manifest. The presence of at least two pancreatic autoantibodies, normal fasting glucose, and normal glucose tolerance are the hallmarks of this asymptomatic stage. **Stage 2**- Dysglycemia has already been brought on by a considerable level of β-cell malfunction.

Stage 3 -People who have symptomatic hyperglycemia at the clinical beginning of the disease.

Hyperglycemia, hyperglucagonemia, glucosuria, and, if left untreated, ketosis, acidosis, dehydration, and mortality are caused by insufficient endogenous insulin. Despite intensive therapy, diabetic ketoacidosis (DKA), which has a mortality rate of 0.3-0.5%, is present in around one-third of individuals with newly diagnosed type 1 diabetes.

The landmark 1993 study known as the Diabetes Control and problems Trial established a direct link between long-term microvascular problems like retinopathy, neuropathy, and microalbuminuria (a proxy for nephropathy) and persistent hyperglycemia. Chronic hyperglycemia has been linked in follow-up studies to both macrovascular problems and all-cause death. The main obstacle to strict glucose management, however, was found to be iatrogenic hypoglycemia.[20]

Over the past few decades, treatment has centered on bringing blood sugar levels back to normal while reducing the chance of hypoglycemia. It also acknowledges the significant psychosocial elements that impact a child's growth and development when they have a chronic illness and monitors for long-term problems. Hyperglycemia is the hallmark sign of type 1 diabetes, particularly in youngsters. Polydipsia, polyuria, polyphagia, inadvertent weight loss, exhaustion, and weakness are typical signs of classic new-onset type 1 diabetes. If T1D is not assessed and treated right away, life-threatening DKA may develop.[21]

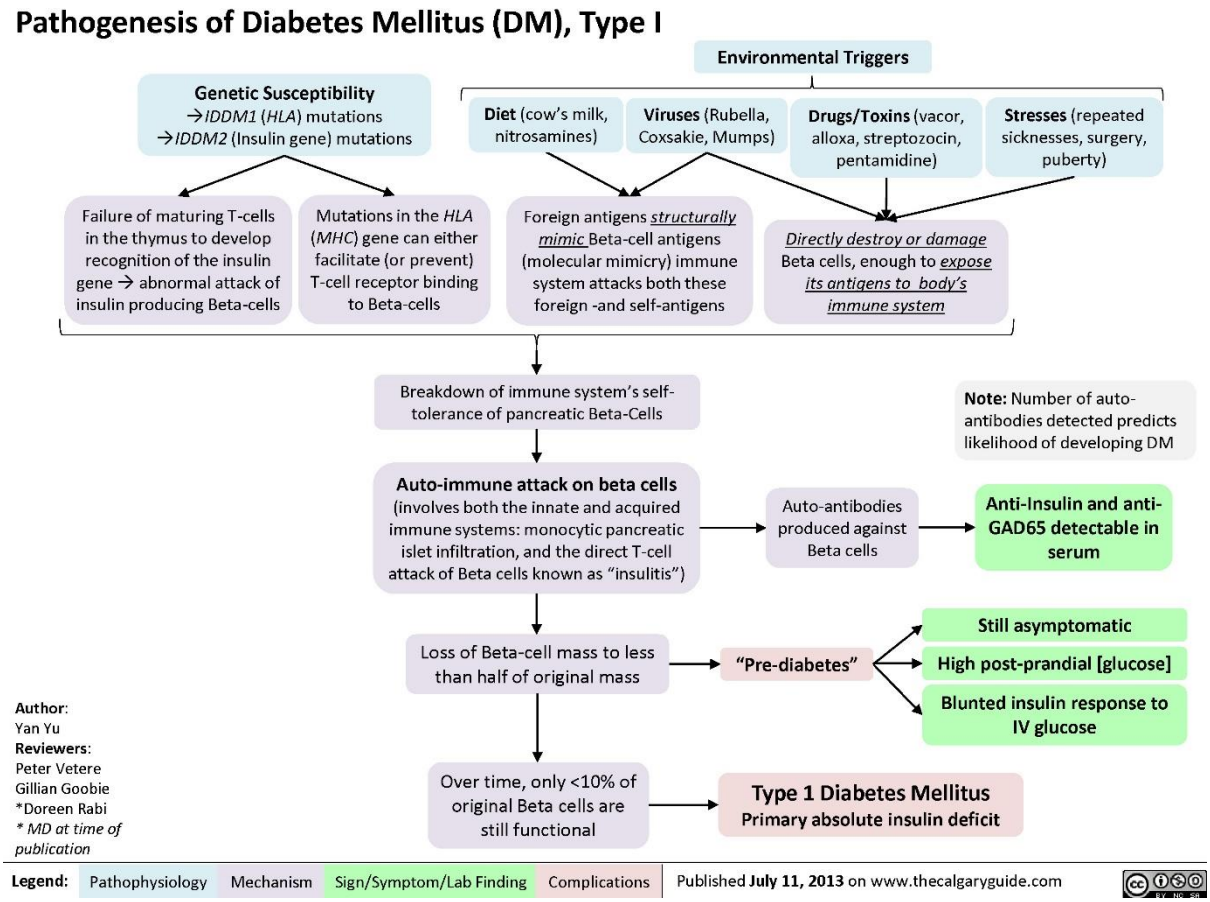

Figure 2: Pathophysiology of type 1 Diabetes mellitus

Signs and symptoms

Children typically come with a history of weight loss ranging from days to months, polyuria, and polydipsia. Dehydration, acidosis, vomiting, lethargy, and altered mental status could occur if the diagnosis is postponed. A pediatric endocrinologist usually coordinates follow-up management on a roughly quarterly basis following diagnosis and the start of insulin medication.

The following characteristics are linked to ketoacidosis in certain children:

- Ketone smell
- Dehydration
- Pain in the abdomen
- Kussmaul breathing
- Vomiting
- Coma
- Modified mental state

Pulmonary Changes in Type 1 diabetes mellitus

"Diabetes mellitus is a chronic illness that damages and incapacitates several organs, including the lungs. Coronary artery disease, cerebrovascular accidents, retinopathy, nephropathy, and neuropathy are among the vascular disorders that increase morbidity and death in diabetics. Furthermore, obstructive sleep apnea syndrome (OSA), asthma, chronic obstructive pulmonary disease (COPD), acute lung damage, and respiratory infections are also more common in diabetics. Additionally, a number of studies have shown that respiratory conditions coexist with a range of metabolic disorders, and the pathophysiological mechanisms that determine the main degenerative effects of diabetes may also be the main cause of lung function impairments.". [22, 23, 24]

Diabetes mellitus is a long-term, crippling condition that damages the lungs among other organs. Every day, the prevalence and incidence of diabetes mellitus and its consequences rise. Various pulmonary problems in persons with diabetes have been documented in numerous investigations. Non-enzymatic glycation of the collagen protein in the bronchial tree and chest wall due to chronic hyperglycemia results in the production of fibrous tissue. The respiratory muscles are weaker as a result of increased protein catabolism. Diaphragmatic paralysis can be caused by phrenic nerve neuropathy. Thus, diabetes mellitus results in decreased ventilation.

Along with lowering the gas diffusing ability, the illness also thickens the basal lamina by glycation. Additionally, it raises the risk of both acute and chronic pulmonary infections, which results in lung parenchymal fibrosis and, ultimately, a decrease in lung mechanics. It also induces immunoglobulin to glycate.

The lungs' antioxidant defense is diminished and local biochemical alterations are brought on by hyperglycemia. Reduced lung volumes, pulmonary elastic rebound, and decreased bronchodilatation are the results of local oxidative stress. The development of pulmonary complications is therefore significantly influenced by diabetes mellitus, and the lungs are the target organ in this condition, just like other organs.24.

Schuyler et al.'s 1976 study was the first to raise the possibility that diabetic problems could affect the lung. Unsurprisingly, there were a lot of contradictory findings, which could be explained by the fact that diabetes and associated metabolic comorbidities are diverse, as well as the many methods that were employed to describe the pulmonary physiology of individuals with type 1 diabetes. A clinically demonstrated rapid deterioration in lung function in individuals with diabetes Alveolar capillary anomalies that have been pathologically demonstrated and the part played by pulmonary autonomic dysfunction .[25]

Numerous factors can lead to endothelial dysfunction in people with diabetes. Some of the common theories include PKC activation," increased expression of transforming growth factor-beta (TGF-β) and vascular endothelial growth factor (VEGF), oxidative stress, non-enzymatic glycation, activation of the coagulation cascade, increased expression of tumour necrosis factor-alpha (TNF-α), increased expression of insulin and insulin precursor molecules, and hyperglycemic pseudohypoxia." These are all potential causes of endothelial dysfunction. However, there has been no evaluation of the importance of these proposed pathways in relation to lung disease and diabetes.

Potential mechanisms of diabetes related lung disease

Traditionally, blood arteries have been used to transport essential nutrients and oxygen to other tissues. Endothelial cells are the main core lesion cells in the majority of these vascular diseases. Previously believed to be controlled exclusively by angiogenic growth factors like VEGF and other signals like Notch, research indicates that the metabolic switch in endothelial cells also affects the angiogenic switch. Type 2 diabetes impacts the vascular wall through a metabolic milieu that includes endothelial dysfunction, platelet overactivity, oxidative stress, inflammation, insulin resistance, hyperglycemia, high levels of free fatty acids, and other metabolic abnormalities.. The activation of these pathways accelerates thrombosis and intensifies vasoconstriction.

One important early stage in the development of vascular issues that cannot be ignored is diabetes-induced endothelial dysfunction. In microvascular issues, endothelial dysfunction is frequently manifested by reduced nitric oxide (NO) release, increased oxidative stress, increased generation of inflammatory agents, aberrant angiogenesis, and impaired endothelium repair.

Reduced lung capacity, pulmonary elastic recoil, and pulmonary diffusion impairment—all caused by decreased capillary blood volume—are the most prevalent problems in both young and adult diabetic people. In clinical practice, some diabetic patients exhibit abnormal pulmonary function. In addition to demonstrating thickening of the pulmonary capillaries' basal lamina, the histological data also suggests the presence of pulmonary microangiopathy . [22,27]The presence of underlying pulmonary microangiopathy is the most likely explanation for impaired pulmonary microangiopathy in diabetic subjects; aberrant endothelial cell function is another common mechanism.[28]

Common etiology of T2DM related endothelial microangiopathy[22,29]

There is significant variation in the responses of endothelial cells in different organ systems and even within the same arterial bed. Apart from the differences, there are signaling pathways that are common. Nitric oxide (NO) research has concentrated on two important endothelial cell byproducts that are essential for maintaining vascular homeostasis: prostacyclin and NO. Endothelial NO synthase (eNOS) is an enzyme that generates NO and is essential for controlling endothelial function. Abnormal NADPH oxidases (NOX) activation causes endothelial dysfunction and eNOS, according to studies conducted in T2DM models[29]. According to recent research, the pathophysiology of treating diabetes and pulmonary disorders by targeting vascular endothelial cells is similar.

For example, studies have shown that glucagon-like peptide-1 (GLP-1) is related to the activity of vascular endothelial cells and is widely employed in the treatment of diabetes. In a receptor-dependent manner, GLP-1 inhibited the senescence caused by reactive oxygen species in human umbilical vein endothelial cells (HUVECs) through downstream Protein kinase cAMP-dependent (PKA) signalling and the activation of antioxidant genes. GLP-1 receptor agonists (GLP-1RAs) were shown to decrease reactive oxygen species-induced senescence in vitro on HUVECs in a receptor-dependent way via activating antioxidant genes and downstream PKA signalling.[30]

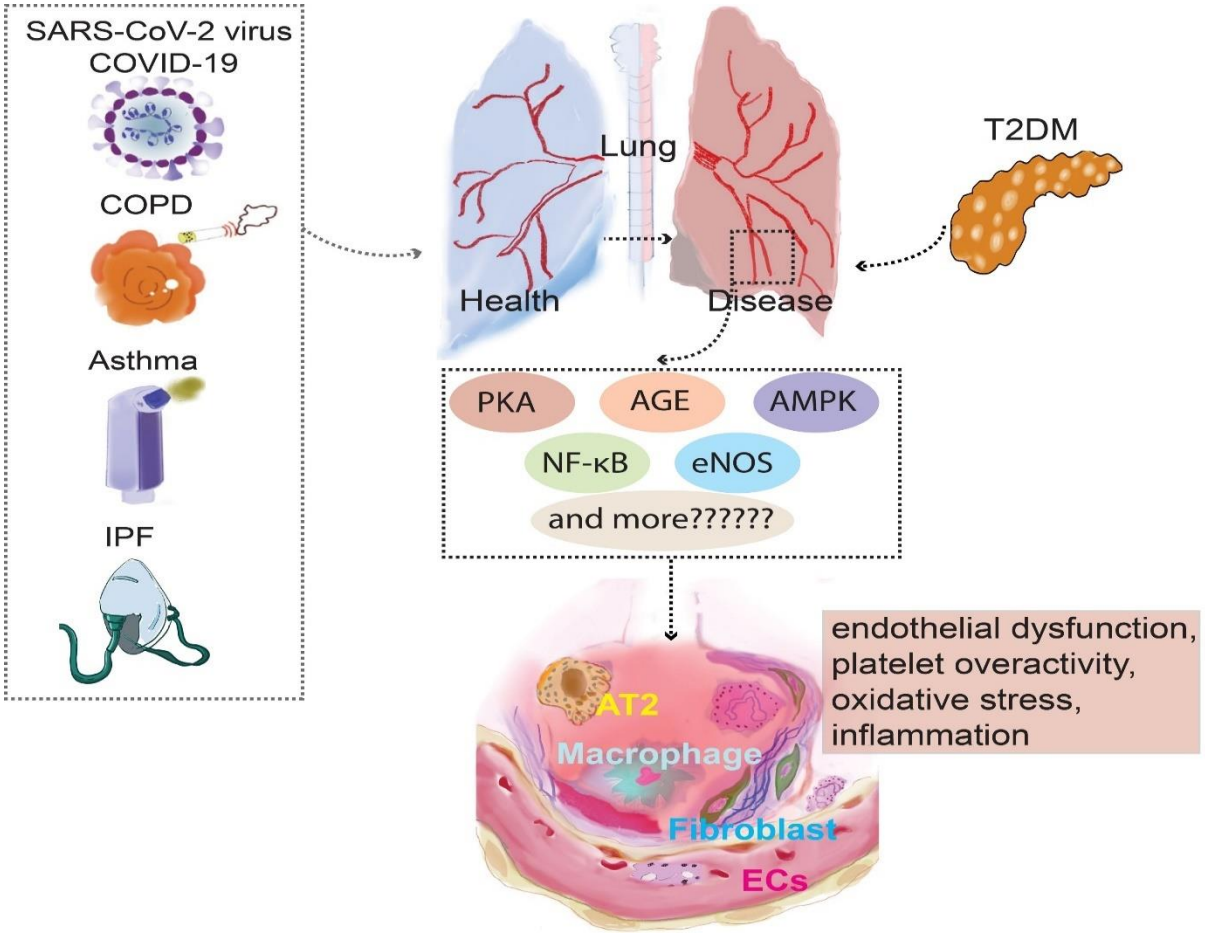

Figure 3: Pathophysiology of pulmonary changes in Diabetes mellitus

“Advanced glycation end products (AGEs) increased endothelial cells; however, GLP-1-RAs suppressed these cells by increasing VCAM-1 mRNA levels and lowering the expression of the AGE receptor (RAGE). By blocking PKC-α, NADPH oxidase, and NF-κB signalling and upregulating antioxidant and anti-inflammatory enzymes, GLP-1RAs significantly reduced inflammation and had antioxidant effects on endothelial cells. GLP-1RAs significantly reduced the inflammation of human aortic endothelial cells. It has also been shown to raise intracellular Ca2+ and activate calcium/calmodulin-dependent protein kinase kinase (CAMKK), which leads to the activation of AMPKGLP-1RAs inhibit the production of endothelin-1, which is also present in endothelial cells, by preventing nuclear factor kappa B from activating. Advanced glycation end products (AGEs) increased endothelial cells, although GLP-1-RAs suppressed RAGE expression and increased VCAM-1 mRNA levels to block these endothelial cells (42). Through the inhibition of PKC-α, NADPH oxidase, and NF-κB signalling, as well as the upregulation of antioxidant and anti-inflammatory enzymes, GLP-1RAs demonstrated antioxidant effects on endothelial cells and significantly reduced inflammation. GLP-1RAs significantly reduced inflammation of the human aortic endothelial cells during the procedure. It has also been shown to raise intracellular Ca2+ and switch on CAMKK (calcium/calmodulin-dependent protein kinase kinase), which turns on AMPK. Additionally present in endothelial cells, GLP-1RAs inhibit endothelin-1 by preventing nuclear factor kappa B from activating”.[31,32]

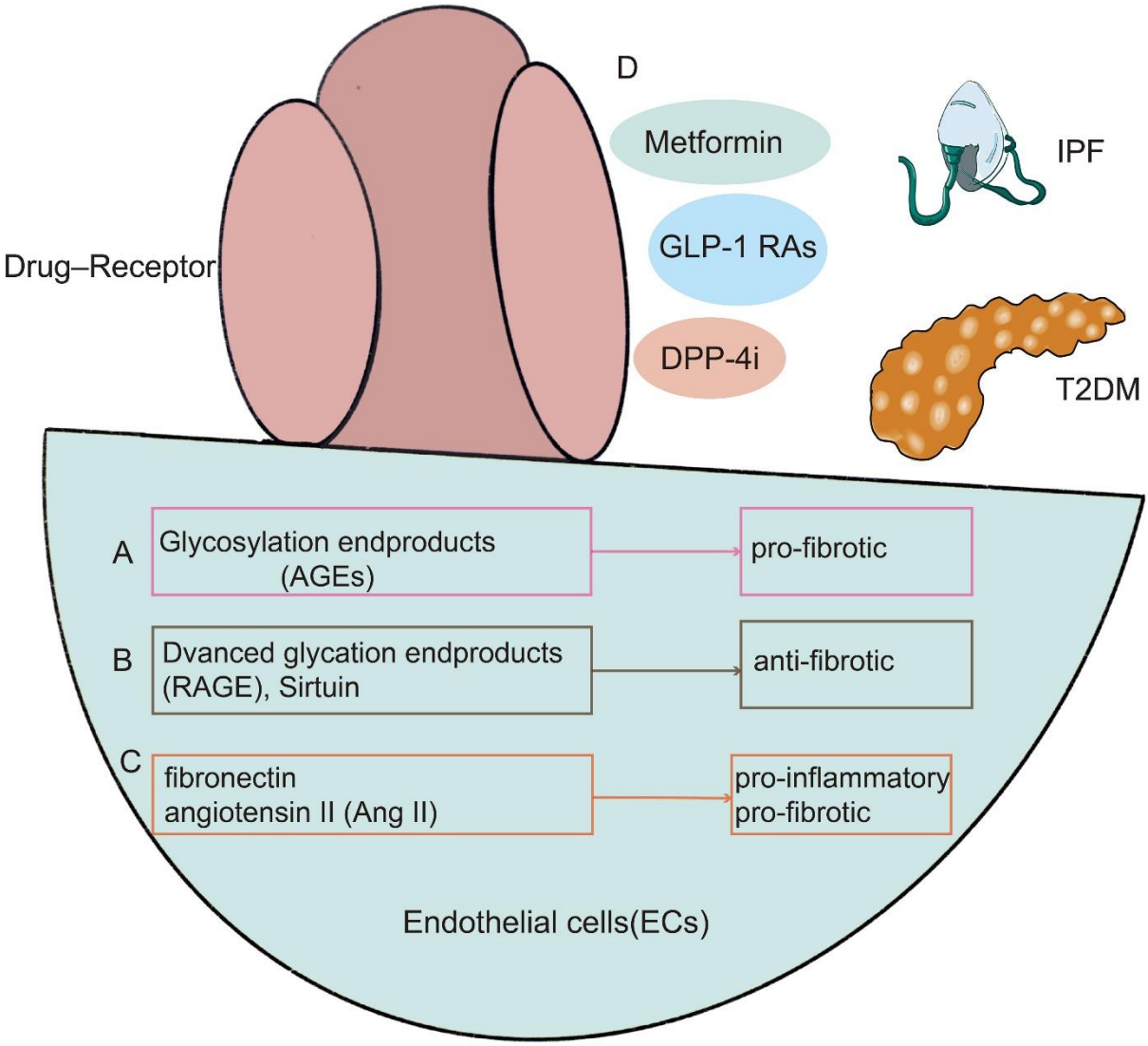

Figure 4: Pathophysiology of pulmonary changes in Diabetes mellitus

Pulmonary Function test changes in Type 1 DM

Pulmonary function tests conducted on Type 1 Diabetes Mellitus patients revealed substantial test results modifications indicating that diabetes might affect lung organs.

1.Diminished Spirometric Measurements:

Multiple studies demonstrate that type 1 diabetes patients perform worse than healthy children in their measured spirometric results which include FVC and FEV_1.[10]

2. Restrictive Lung Patterns:

The research indicates that type 1 diabetes children have restrictive lung patterns that become worse when blood glucose stays uncontrolled[33]

3. **Increased Airway Resistance:** Some medical studies identify type 1 diabetes patients as having elevated airway resistance although they do not show clinical respiratory indications.[34]

4. **The Effect of Glycemic Management:** The study shows that proper blood glucose management is essential for lung health because inadequate diabetes control leads to worse pulmonary impairments.[10]

These findings establish routine pulmonary function examinations essential for people with type 1 diabetes because immediate therapy can help protect their lung health when pulmonary problems are identified early.

Epidemiology of Type 1 Dm and pulmonary function parameters

Multiple pulmonary function abnormalities exist between children who receive type 1 diabetes mellitus (T1DM) treatment. Research findings show that T1DM patients experience diminished FVC and FEV_1 measurements as their lungs follow restrictive patterns. Research shows that children with T1DM tend to exhibit an elevated FEV_1/FVC ratio which provides evidence of possible changes in their airway functioning. [35]

Children with T1DM show decreased amounts of carbon monoxide transfer in their lungs which suggests the possible involvement of early microvascular pathology. [36]

Diabetes duration & metabolic control level together affect the nature of pulmonary change development. Long-duration diabetes combined with poor blood sugar management leads children to show greater deficits in lung function evaluation data according to research studies.[10]

The evaluation of lung functions in children with T1DM needs to consider their current stage of puberty development because this factor influences assessment results. The assessment process should take pubertal status into account because it ensures proper interpretation of pulmonary function tests. [36]

When comparing diabetic children to healthy control children, **Ismail L. et al.'s** study revealed a substantial decrease in all spirometeric parameters. Compared to children with good glycemic control, those with poor glycemic control showed a marked reduction in lung functioning. Lung function is impaired in children with type 1 diabetes, and this impairment worsens with inadequate glucose control.[10]

The clinical research by **Pachampalayam et al.** shows that diabetic patients exhibit diminished values of FEV1, FVC, FEV1/FVC% and PEFR when compared to healthy non-diabetic controls. The measured mean FVC value of 3.161 is significantly lower according to our study analysis (p-value <0.001). Diabetic patients display restrictive patterns in pulmonary function tests even in symptom-free situations. A longer duration of diabetes results in an increasing restriction in this pattern. The requirement for routine spirometry tests as an inexpensive non-invasive procedure exists because diabetics need regular monitoring of their lung functions.[37]

According to results from a study by **van Gent**, children with type 1 diabetes do experience an increase in airway resistance.The encouraging outcomes of treatment with intrabronchial injection of insulin may be hampered by progressive impairments in lung function.[38]

In conclusion, inadequate glycemic control exacerbates lung function impairment linked to type 1 diabetes in children. Children with type 1 diabetes do experience an increase in airway resistance.

METHODOLOGY

Materials and methods

Source of data

All patients admitted to Paediatric Intensive Care Unit ,ward and opd basis , BLDE (Deemed to be University, Shri B. M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPUR, KARNATAKA who meet the inclusion criteria for a period from to May 2023 to December 2024.

Study design: Comparative Study

Duration of study: For a period of 18 months, from May 2023 to December 2024.

Inclusion criteria

- Patients with type 1 DM with age ranging from 6 to 16 years.
- The patients will be matched with non type 1 DM children(control group) ranging from 6 to 16 .

Exclusion criteria

Participants who have undergone major abdominal or chest surgery, have a history of acute or chronic respiratory infections, neuromuscular disease, cancer, or cardiopulmonary disease, or have gross abnormalities of the vertebral column or thoracic cage are not allowed to participate in the study. Patients who have diabetic neuropathy, nephropathy, or retinopathy are also not allowed.

Data analysis

Sample Size

The anticipated Mean±SD of VC in Type 1 Diabetics group in children 1.86±0.81 and in control group 2.45±0.63 resp. [10] the required minimum sample size is 30 per group (i.e. a total sample size of 60, assuming equal group sizes) to achieve a power of 90% and a level of significance of 5% (two sided), for detecting a true difference in means between two groups.

$$N = 2\left[\frac{\left(Z_{\propto} + z_{\beta}\right) * S}{d}\right]^{2}$$

$Z_{\propto}$ Level of significance=95%

Z_{β}--power of the study=90%

d=clinically significant difference between two parameters

SD= Common standard deviation

Sample size calculation

To achieve a power of 90% and a level of significance of 5% (two sided), the study would need a sample size of 30 for each group, or 60 total, assuming equal group sizes and a pooled standard deviation of 0.7 units. This would allow for the detection of a true difference in means between the test and the reference group of 0.5900000000000001 (i.e. 2.45 - 1.86 units).

Method of collection of Data

Healthy people between the ages of six and sixteen are chosen at random. In terms of age, height, weight, body mass index (BMI), and socioeconomic level, each of them will be matched with a group of diabetes participants. Two categories of T1DM patients were additionally created: (1) well-managed cases and (2) poorly controlled diabetic mellitus. All subjects underwent standard spirometry using a fully functional computerised system. Before the test, each participant will be instructed on the entire manoeuvre and urged to practice it. The device will function in the 20–25°C ambient temperature range and be calibrated every day.

Statistical Analysis

● The acquired data will be input into a Microsoft Excel document, and the statistical package for the social sciences (version 20 JMP SAS) will be used to conduct statistical analysis.

● The results will be displayed as graphs, counts, percentages, mean±SD, and interquartile range.

● “The Independent t test will be used to compare two groups' normally distributed continuous variables. The Mann Whitney U test will be used for variables that are not normally distributed; a p-value of less than 0.05 will be regarded as statistically significant. All statistical tests will performed two tailed”.

RESULTS

Table 1: Gender wise distribution of Children with Type 1 Diabetes mellitus group and control group

Sl no	Gender	Children with type 1 DM (n=32)	Control group (n=30)
1	Male	18 (56.25%)	19 (63.3%)
2	Female	14 (43.7%)	11 (36.6%)
3	Total	32 (100%)	30 (100%)

This table represents Gender wise distribution of Children with Type 1 Diabetes mellitus group and control group and found that 56.25%(18) were males and 43.7%(n-14) were females among cases and among control group 63.3%(n-19) were males and 36.6%(n-11) were females and it is shown in bar diagram

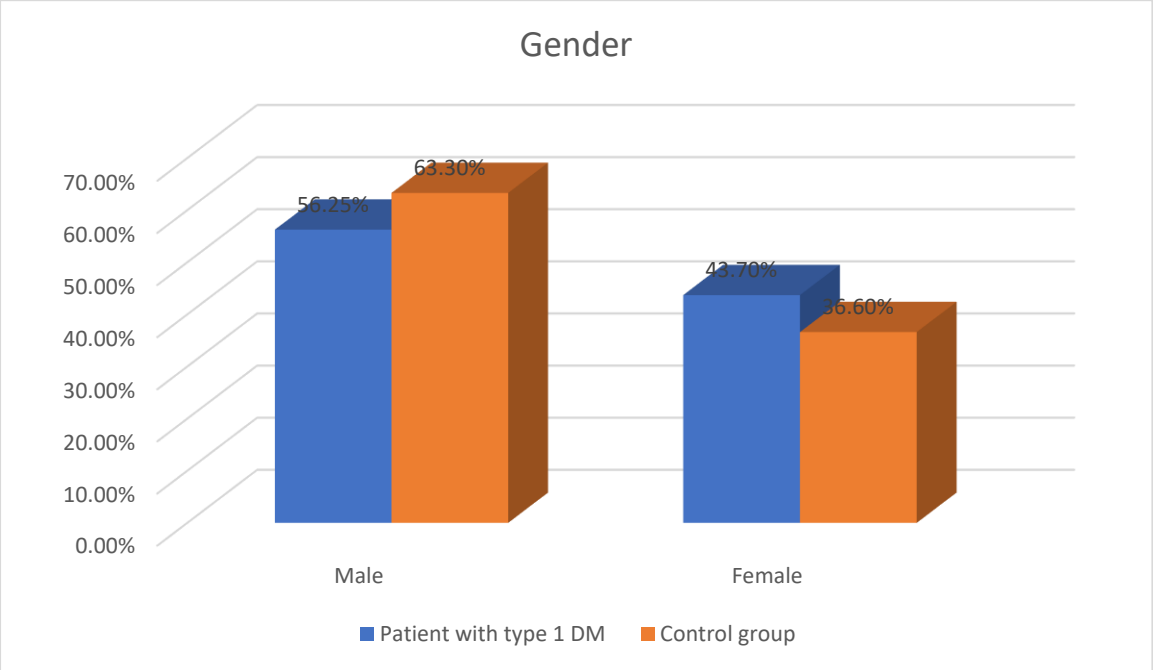

Figure 5: Gender wise distribution of Children with Type 1 Diabetes mellitus group and control group

Table 2: Age wise distribution of Children with Type 1 Diabetes mellitus group and control group

Sl no	Age (Years)	Children with type 1 DM (n=32)	Control group (n=30)
1	<10 years	20 (62.5%)	15 (50%)
2	≥10 years	12 (37.5%)	15(50%)
3	Total	32 (100%)	30 (100%)

This table represents the Age wise distribution of Children with Type 1 Diabetes mellitus group and control group and found that among case group 62.5%(n-20) were <10 years and followed by 37.5%(12) were >10 years. But in Control group it is equally distributed and it is shown in bar diagram

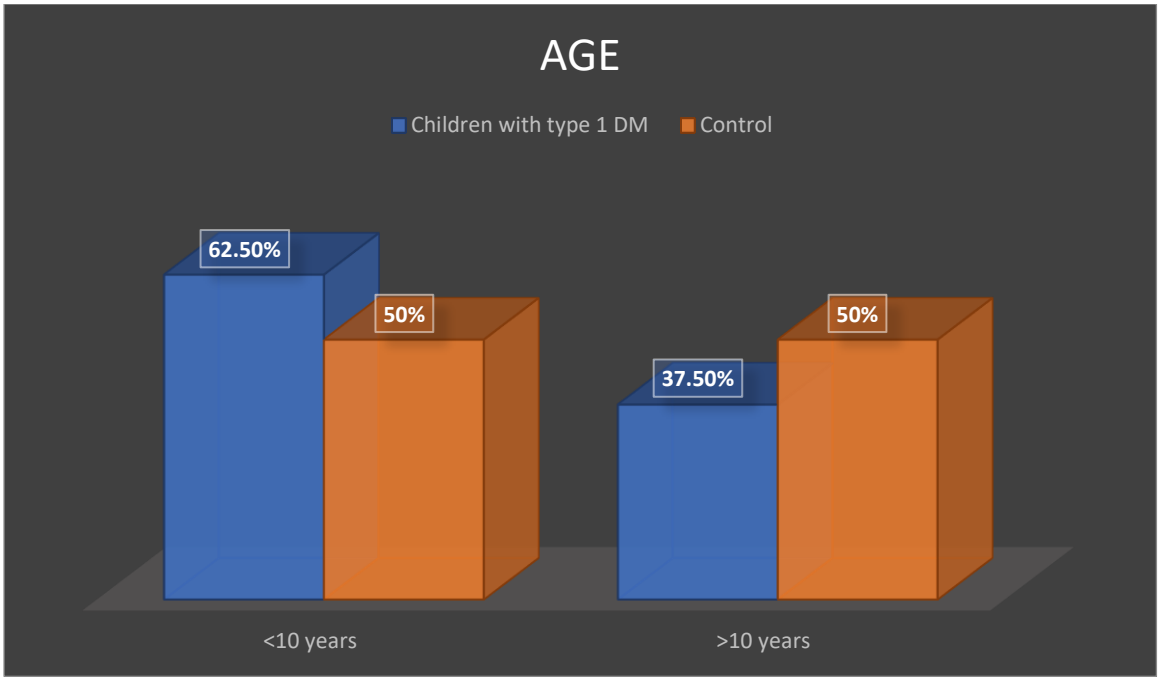

Figure 6: Age wise distribution of Children with Type 1 Diabetes mellitus group and control group

group and control group

Table 3: Family history wise distribution of Children with Type 1 Diabetes mellitus group and control group

Sl no	**FAMILY HISTORY**	Children with type 1 DM (n=32)	Control group (n=30)
1	Yes	4 (12.5%)	0
2	No	28 (87.5%)	30 (100%)
3	Total	32 (100%)	30 (100%)

This table represents the Family history wise distribution of Children with Type 1 Diabetes mellitus group and control group and found that among cases 87.5%(n-28) had no Family history of diabetes but 12.5%(n-4) had history of Family history of DM . Among control group None of them had Family history of diabetes and it is shown in bar diagram

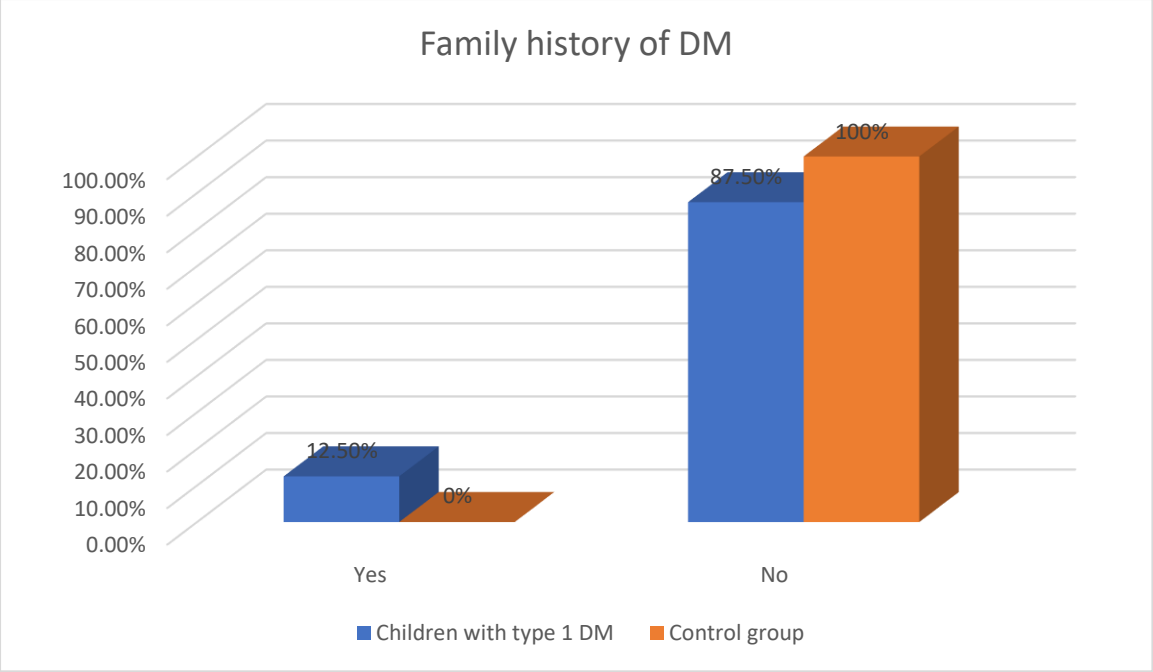

Figure 8: Family history wise distribution of Children with Type 1 Diabetes mellitus group and control group

Table 4: Chest X ray findings wise distribution of Children with Type 1 Diabetes mellitus group and control group

Sl no	**Chest-X ray finding**	Children with type 1 DM (n=32)	Control group (n=30)
1	Normal	29 (90.6%)	29 (96.6%)
2	Abnormal	3 (9.3%)	1 (3.3%)
3	Total	32 (100%)	30 (100%)

This table presents the chest X ray among Study participants and found that 9.3%(n-3) had abnormal x ray among cases and 3.3%(n-1) had abnormal Xray and it is shown in bar diagram

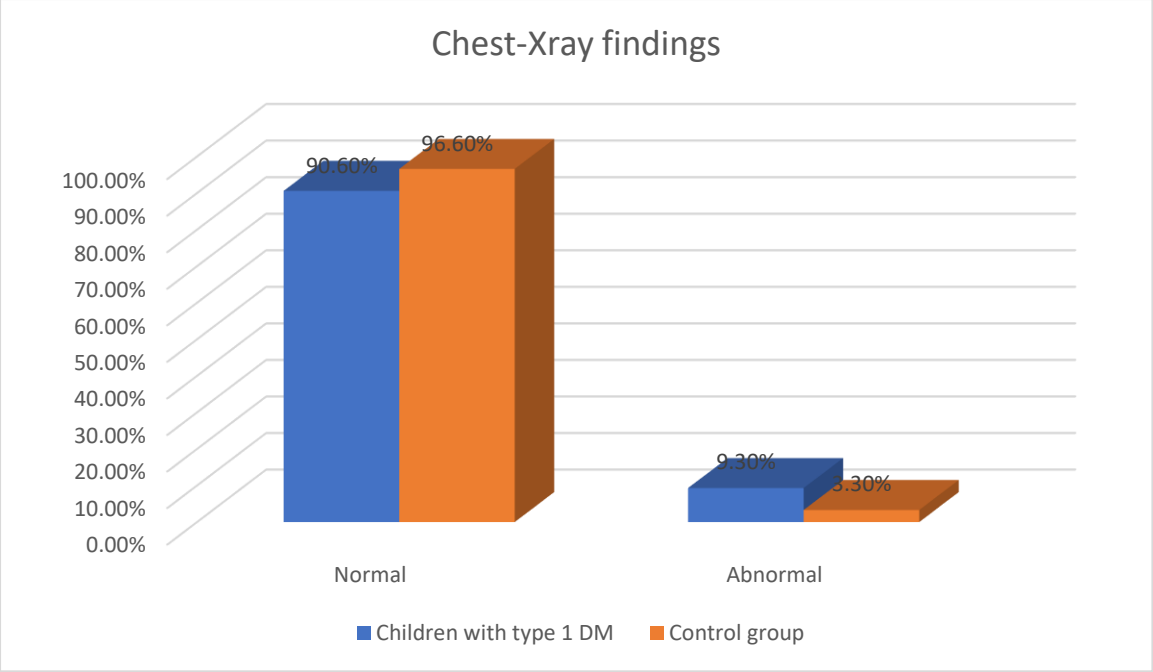

Figure 9: Chest X ray findings wise distribution of Children with Type 1 Diabetes mellitus group and control group

Table 5: FVC wise distribution of Children with Type 1 Diabetes mellitus group and control group

Sl no	FVC(L)	Children with Type 1 Diabetes mellitus	Control
1	Mean	2.03	2.2230
2	Median	2.00	2.0000
3	Std. Deviation	.474	.85702
4	Range	2	3.00

This table represents the mean and standard deviation **of** FVC among Children with Type 1 Diabetes mellitus group and control group and found that the mean and standard deviation is 2.03(0.474) among cases and 2.23(0.85) among control group

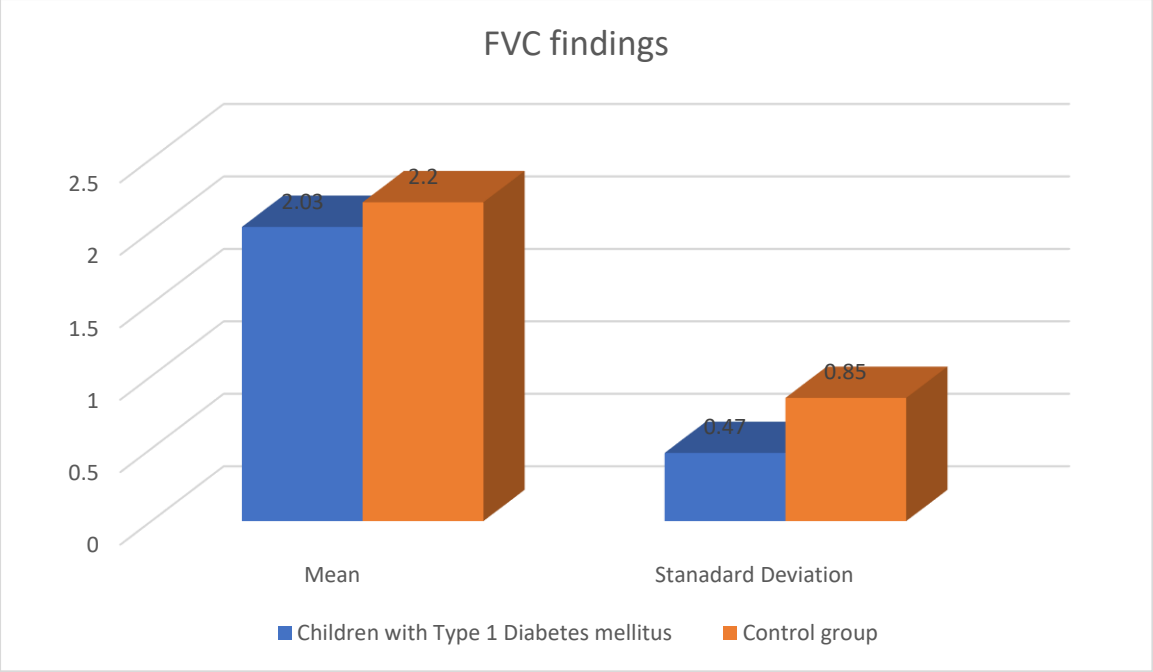

Figure 10: **FVC wise distribution of Children with Type 1 Diabetes mellitus group and control group**

Table 6: FEV1 wise distribution of Children with Type 1 Diabetes mellitus group and control group

Sl no	FEV1(L)	Children with Type 1 Diabetes mellitus	Control
1	Mean	1.72	2.1027
2	Median	2.00	2.0000
3	Std. Deviation	.523	.71559
4	Range	2	3.00

This table represents the mean and standard deviation of FEV1 among Children with Type 1 Diabetes mellitus group and control group and found that the mean and standard deviation is 1.72(0.523) among cases and 2.10 (0.71) among control group

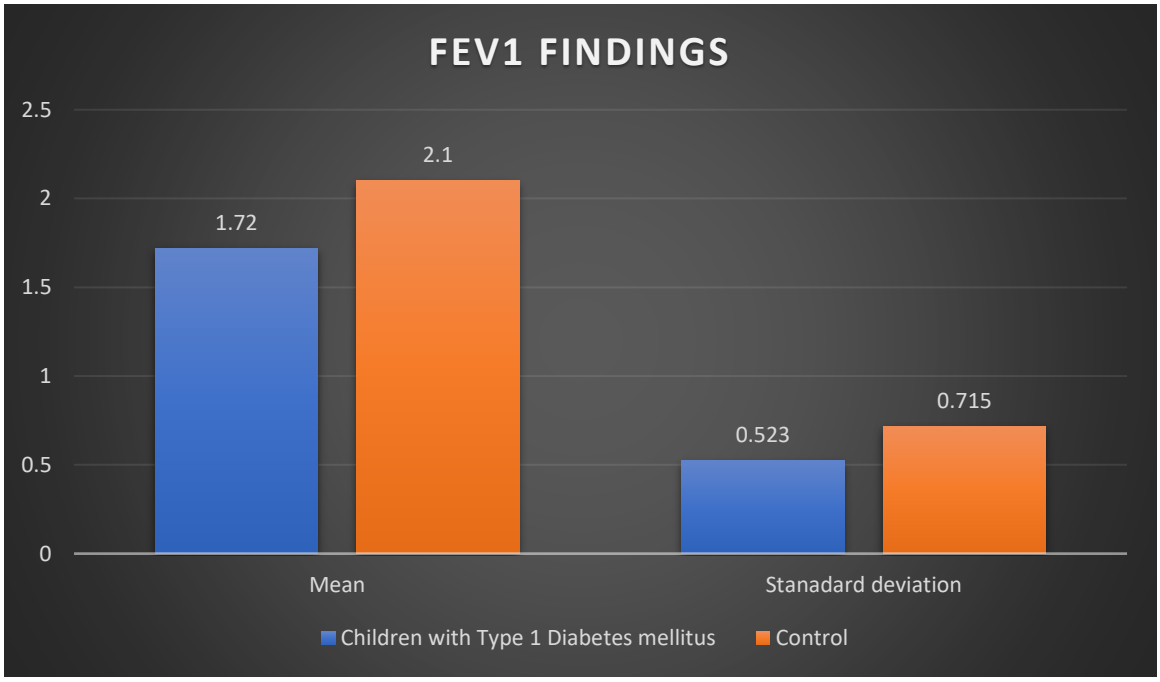

Figure 11: **FEV1 wise distribution of Children with Type 1 Diabetes mellitus group and control group**

Table 7: FEV1/FVC wise distribution of Children with Type 1 Diabetes mellitus group and control group

Sl no	FEV1/FVC(%)	Children with Type 1 Diabetes mellitus	Control
1	Mean	80.41	91.30
2	Median	83.00	93.50
3	Std. Deviation	16.710	6.834
4	Range	99	26

This table represents the mean and standard deviation of FEV1/FVC among Children with Type 1 Diabetes mellitus group and control group and found that the mean and standard deviation is 80.41 (16.71) among cases and 91.30 (6.8) among control group

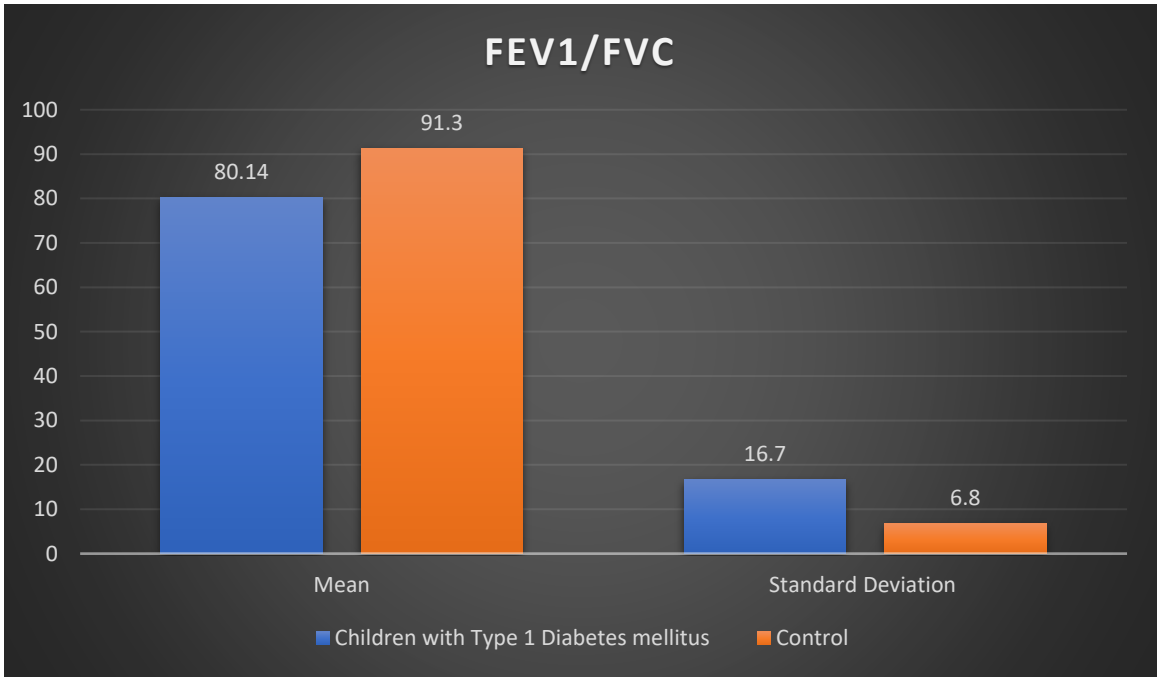

Figure 12: **FEV1/FVC wise distribution of Children with Type 1 Diabetes mellitus group and control group**

Table 8: RBS wise distribution of Children with Type 1 Diabetes mellitus group and control group

Sl no	RBS (mg/dl)	Cases	Control
1	Mean	170.70	78.35
2	Median	171.00	78.50
3	Std. Deviation	63.465	11.475
4	Range	271	42

This table represents the mean and standard deviation **of** RBS among Children with Type 1 Diabetes mellitus group and control group and found that the mean and standard deviation is 170.70 (63.4) among cases and 78.35 (11.47) among control group

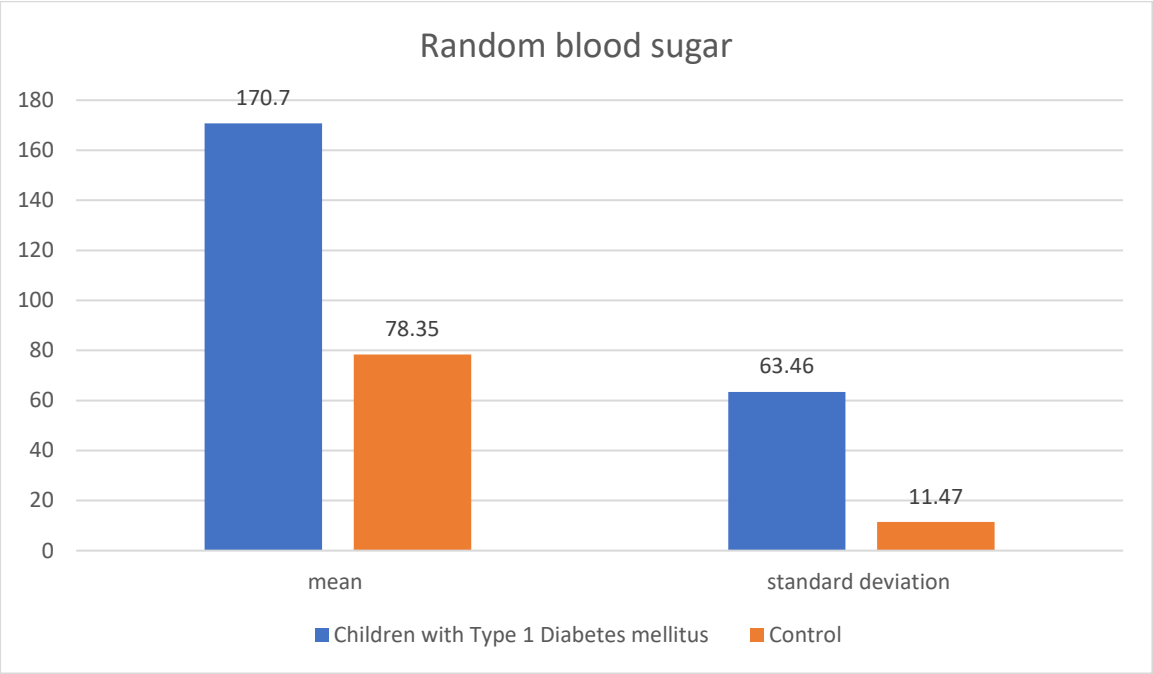

Figure 13: **RBS wise distribution of Children with Type 1 Diabetes mellitus group and control group**

Table 10: PEFR wise distribution **of Children with Type 1 Diabetes mellitus group and control group**

Sl no	PEFR (L/min)	**Children with Type 1 Diabetes mellitus group**	Control
1	Mean	4.8120	3.84
2	Median	5.0000	4.00
3	Std. Deviation	.70018	.515
4	Range	2.70	2

This table represents the mean and standard deviation of **PEFR** level among Children with Type 1 Diabetes mellitus group and control group and found that the mean and standard deviation is 4.8 (0.700) among cases and 3.84 (0.515) among control group

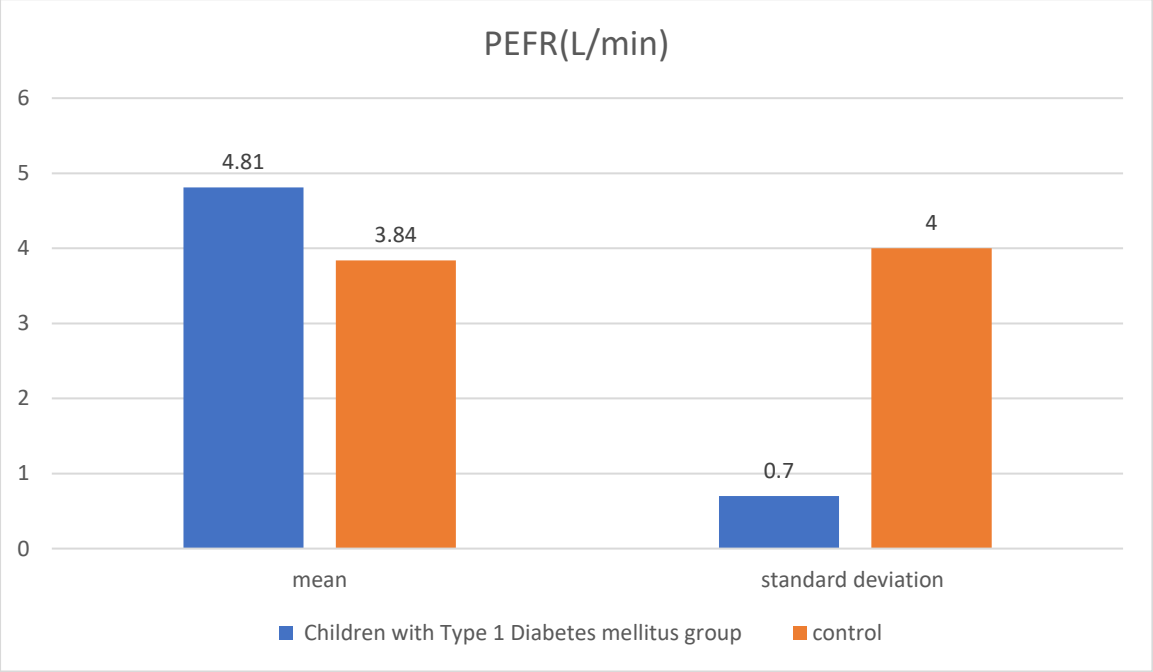

Figure 14: PEFR wise distribution **of Children with Type 1 Diabetes mellitus group and control group**

Table 11: DLCO wise distribution **of Children with Type 1 Diabetes mellitus group and control group**

Sl no	DLCO(ml/min/mmHg)	Children with Type 1DM(n=25)	Control (n=30)
1	Mean	9.96	11.10
2	Median	10	11
3	Std. Deviation	1.24	0.89
4	Range	5	4.0

This table represents the mean and standard deviation of **DLCO** level among Children with Type 1 Diabetes mellitus group and control group and found that the mean and standard deviation is 9.96 (1.24) among cases and 11.10 (0.89) among control group

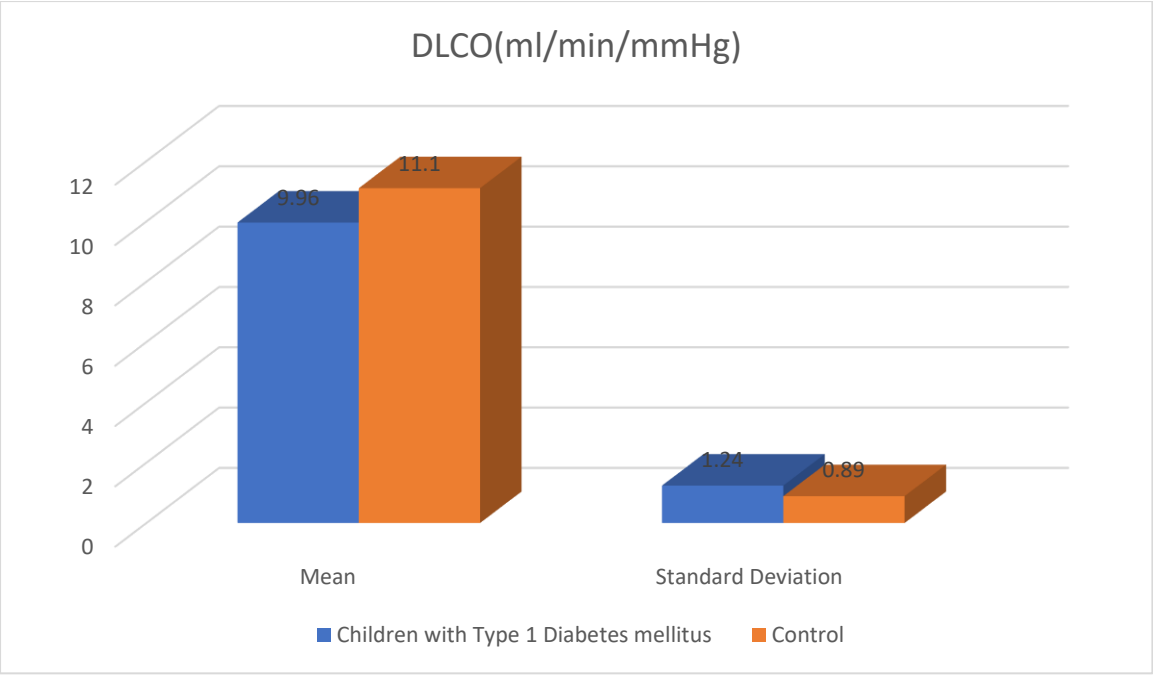

Figure 15: DLCO wise distribution **of Children with Type 1 Diabetes mellitus group and control group**

Table 12: HBA1c level wise distribution of Children with Type 1 Diabetes mellitus group and control group

Sl no	HBA1c(%)	Cases	Control
1	Mean	11.19	5.57
2	Median	12	5.50
3	Std. Deviation	3.03	0.643
4	Range	13	3

This table represents the mean and standard deviation of HBA1c level among Children with Type 1 Diabetes mellitus group and control group and found that the mean and standard deviation is 11.19 (3.03) among cases and 5.5 (0.643) among control group

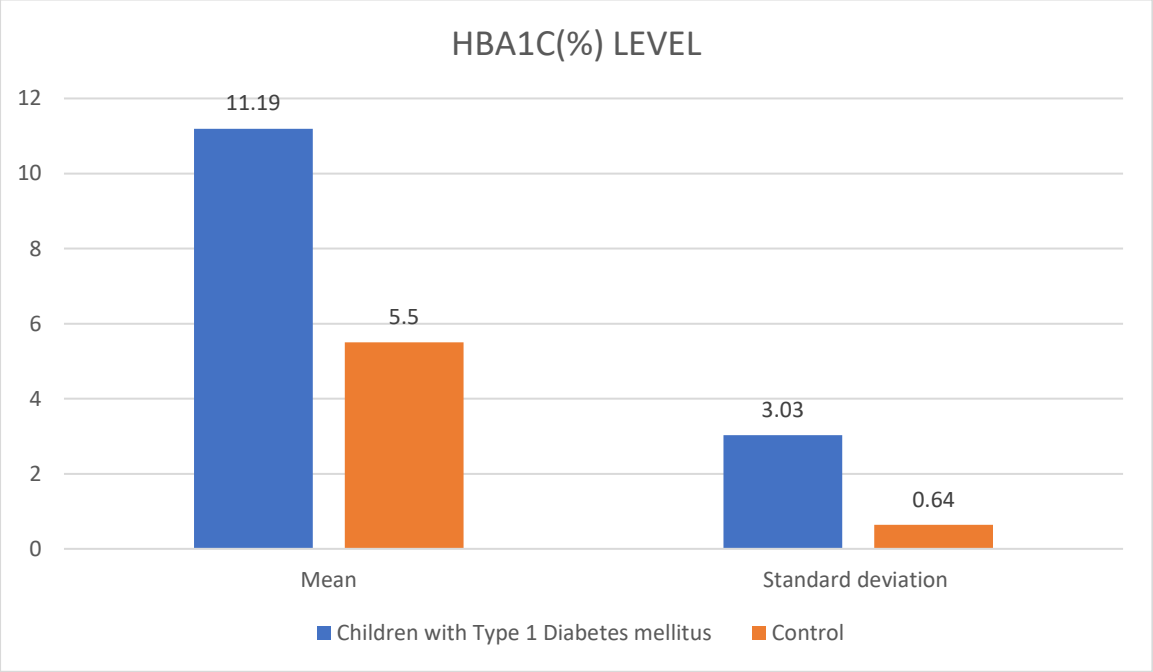

Figure 16: **HBA1c level wise distribution of Children with Type 1 Diabetes mellitus group and control group**

Table 13: Association between Gender wise distribution of Children with Type 1 Diabetes mellitus group and control group

Sl no		Children with type 1 DM	Control group	P value
Gender	Male	18 (56.25%)	19 (63.3%)	P value-0.29
	Female	14 (43.7%)	11 (36.6%)	X^2-0.3,
Age(Years)	<10 years	20 (62.5%)	15 (50%)	P value-0.16
	>10 years	12 (37.5%)	15(50%)	X^2-0.98
Maternal history of DM	Yes	4 (12.5%)	0	P value-0.03
	No	28 (87.5%)	30 (100%)	X^2-4.009
Family history of DM	Yes	4 (12.5%)	0	P value-0.03
	No	28 (87.5%)	30 (100%)	X^2-4.009

This table represents the gender and age variables do not lead to meaningful relationships with Type 1 Diabetes Mellitus (T1DM) because their p-values exceed 0.05 at 0.29 and 0.16. The distributions between T1DM and control participants along with their gender composition and age groups exhibit no statistically significant differences because observed variances would likely result from chance occurrences. However, a maternal history of diabetes mellitus (DM) and a family history of DM show significant associations with T1DM, with both having p-

values of 0.03 and chi-square values of 4.009. The diagnostic results indicate that T1DM subjects carry genetic or hereditary elements associated with the development of diabetes

mellitus while the control group displays no such symptoms. The results confirm that genetics strongly influences Type 1 Diabetes Mellitus but age and sex demographics seem to have minimal impact on disease development.

Table 14: Spirometry findings in children with Type 1 diabetes mellitus

Sl no	**Spirometry findings(in comparison to control group)**	Mean	Standard Deviation	P value
1	FVC	2.03(0.474)	2.2(.85702)	**<0.001**
2	FEV1	1.72 (0.523)	2.10 (.71559)	0.08
3	FEV1/FVC	80.14 (16.710)	91.30 (6.834)	**<0.005**
4	PEFR	4.8 (0.700)	3.84(.515)	0.1002
5	DLCO	9.96(1.24)	11.10(0.89)	**<0.03**

This table presents the mean and standard deviation of various spirometry findings among children with Type 1 diabetes mellitus in comparison to control group and found that FVC and FEV1/FVC ,DLCO are statistically significant (p value <.001) and FEV1, PEFR were not significant

Table 15: Association between Spirometry findings and HBA1c level

This table shows the association between HBA1c and various Spirometry findings and found that only FEV1/FVC is found to be significant but other are not significant.

Sl no	Spirometry findings (	HBA1c		P value
		<9.5	>9.5	
1	FVC	2.02 (0.366)	1.96(0.501)	1.01
2	FEV1	1.59(0.300)	1.65(0.366)	0.54
3	FEV1/FVC	79.63(7.065)	80.76(19.8)	**0.003**
4	PEFR	3.65(0.522)	3.84(0.44)	0.48
5	DLCO	9.80(0.912)	10.05(1.4)	0.18

Table 16: Time of diagnosis

Sl no	Time of diagnosis	Frequency	Percentages
1	< 5 years	10	31.5%
2	>5 years	22	68.7%
3	Total	32	100%

This table presents the time of diagnosis and found that 31.5 % (n-10) had diagnosis within 5 years and majority 68.7% (n-22) had time of diagnosis >5 years periods and it is shown in pie diagram

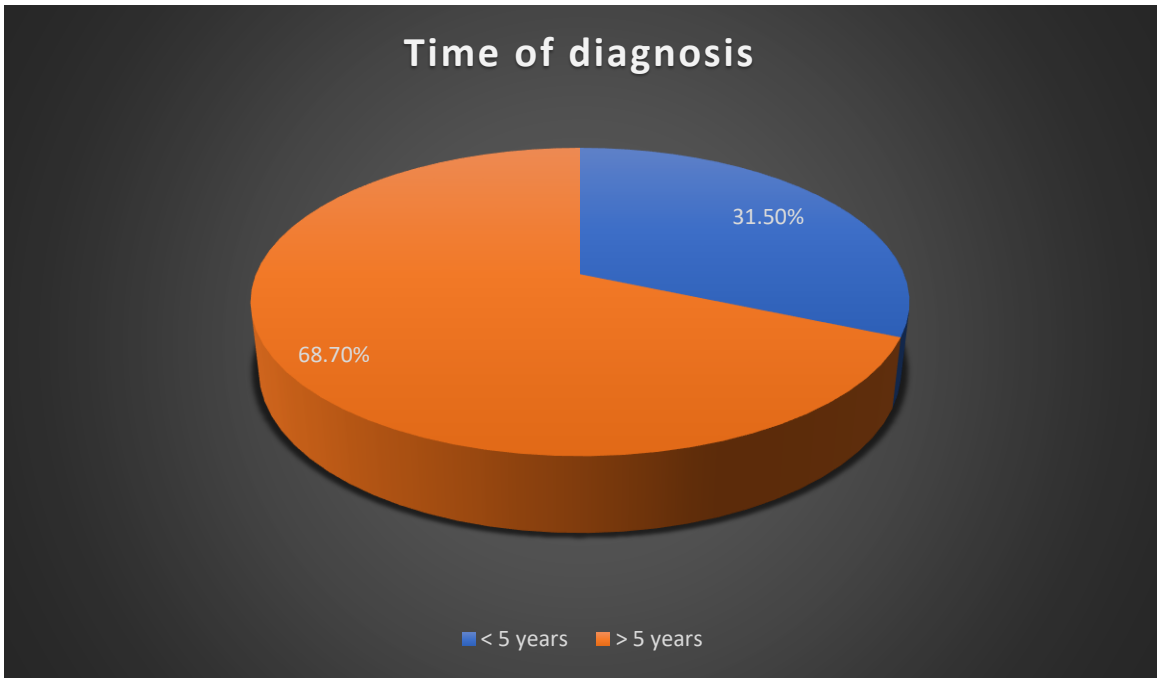

Figure 17: Time of diagnosis

Table 17 : Association between spirometry findings and the time of diagnosis

Sl no	Spirometry parameters	< 5 years	>5 years	P value
1	**FVC**	2.5 (0.90)	2.18 (0.72)	0.38
2	**FEV1**	2.30 (0.91)	2.03 (0.63)	0.15
3	**FEV1/FVC**	92.57 (3.4)	90.63 (8.10)	0.01
4	**PEFR**	4.91 (0.80)	4.65 (0.64)	0.381
5	**DLCO**	11.52 (0.980)	10.9 (0.802)	0.423

This table presents the association between time of diagnosis and the spirometry findings and found that only FEV1/FVC is significant.

Table 18: Distribution of BMI among study participants

Sl no	**BMI**	Cases Mean (SD)	Control Mean (SD)	P value
1	**Underweight**	**3 (13.6%)**	**1 (5%)**	
2	**Normal**	**18 (81.8%)**	**15 (75)**	**0.22**
3	**Overweight**	**1 (4.5%)**	**4 (20%)**	

X2=2.9,df-2

Table 19

Sl no		Cases Mean (SD)	Control Mean (SD)	P value
1	BMI	16.63 (3.16)	18.19 (3.11)	0.93

This table presents the BMI among cases and control ,majority were having normal BMI among case and control and is 81.8% and 75 % respectively

Table 20: Association of BMI with respect to spirometry findings

Sl no	**Underweight**	**Normal**	**Overweight**	P value
FVC	**2.4 (0.66)**	**2.01 (0.38)**	**1.9**	**0.75**
FEV1	**2.03 (0.75)**	**1.67 (0.19)**	**1.4**	**0.43**
FEV1/FVC	**81.8 (7.4)**	**78.38 (20.6)**	**73.6**	**0.51**
PEFR	**4.5(0.51)**	**3.8 (0.40)**	**3.4**	**0.28**
DLCO	**9.4 (0.69)**	**10.1**	**8.4**	**0.43**

This table presents the association between **BMI** among spirometry findings and found that it is not significant

Discussion

Pulmonary function tests (PFTs) are non-invasive assessments used to evaluate lung volumes, airflow, and gas exchange efficiency. In children with Type 1 Diabetes Mellitus (T1DM), studies have shown that lung function may be impaired due to chronic hyperglycemia-induced structural and functional changes in the lungs. These include reduced lung elasticity, microvascular damage, and increased airway resistance.

Medical tests including like Forced Vital Capacity (FVC) together with Forced Expiratory Volume in 1 second (FEV_1) and Diffusing Capacity for Carbon Monoxide (DLCO) show diminished results in children with T1DM when compared to healthy participants.

Chronic blood sugar level management presents an essential role for protecting children with T1DM as poor diabetes control leads to worsened lung function. The research team examined pulmonary function measurements between Type 1 Diabetes Mellitus (T1DM) patients and healthy children groups. The study researched how pulmonary function test outcomes relate to glycemic control levels. Children who have Type 1 Diabetes Mellitus demonstrate reduced values of spirometry measures including Forced Vital Capacity (FVC) and Forced Expiratory Volume in 1 second (FEV_1) as well as FEV_1/FVC ratio along with Peak Expiratory Flow Rate (PEFR) when compared to healthy groups. Researchers have already shown that pulmonary function deteriorates in children who have T1DM through their previous studies.

The pulmonary function parameters of children with Type 1 Diabetes Mellitus (T1DM) were assessed in our study and contrasted with those of healthy controls. We also looked at the connection between glycemic management and alterations in the pulmonary function test (PFT). In comparison to healthy controls, our results show that children with type 1 diabetes have significantly lower spirometry parameters, such as forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), FEV_1/FVC ratio, and peak expiratory flow rate (PEFR). These findings are consistent with earlier research showing impaired lung function in kids with type 1 diabetes.

A similar study by **Prasanth et al. in** 2015 established an association between T1DM and diminished FVC, FEV_1, and FEV_1/FVC ratio results in children. The research showed that poor glycemic control led children to experience major reductions in FEV_1 and FVC while fair

glycemic control demonstrated no such impact. This finding suggests inadequate glycemic control intensifies pulmonary impairment. [33]

Children with T1DM displayed reduced volumes of air in their lungs and lower oxygen and carbon dioxide transfer compared to healthy subjects according to **Cazzato et al. (2004).** The research data demonstrates that T1DM produces unfavorable effects on the lungs in children. [9]

Our study data shows that the 62.5% T1DM group participants under 10 years old compared to 50% in the control group could explain their different pulmonary test results. The literature shows pulmonary function abnormalities develop during the early stages of T1DM no matter

how old or experienced patients are with the condition[38]. The research by **Cazzato et al. (2004)** demonstrated that children with newly detected T1DM presented with pulmonary function abnormalities from the beginning. [9]

PFT measurements showed important correlations with diabetic control levels according to investigators in our study. Children with poor blood sugar management demonstrated greater FEV_1 and FVC reduction compared to children who had effective glycemic control. Research has confirmed that lack of effective blood sugar control leads to reduced lung capacity among children who have Type 1 Diabetes Mellitus. Children with poor glycemic control demonstrated lower FEV_1 and FVC values in comparison to children with fair glycemic control according to **Prasanth et al. (2015).** [33]

The understanding of lung damage mechanisms in children with T1DM remains incomplete but scientists believe that respiratory protein glycosylation promotes tissue changes with subsequent stiffness reduction because of non-enzymatic reactions. The prolonged presence of elevated blood sugar levels may trigger complications in the tiny blood vessels of the lungs therefore worsening respiratory function. Medical evidence shows why controlling blood sugar levels remains essential for protecting the lung function of children who have Type 1 Diabetes Mellitus.

The investigation revealed that 4 out of 32 children with Type 1 Diabetes Mellitus (T1DM) had diabetic mothers which represented 12.5% of our study group but no control group participant had diabetic parents. The evidence indicates maternal diabetes could play a role in producing Type 1 Diabetes Mellitus in offspring.

Available studies about maternal diabetes history and its connection to Type 1 Diabetes Mellitus (T1DM) in children remain scarce in the Indian setting but wider research demonstrates that family medical history increases T1DM risk significantly. A research study conducted in Belgrade by **S Sipetic et al** showed that people with T1DM family background in their past had four times higher chances of developing T1DM (Odds Ratio = 4.04; 95% CI, 2.31-7.07) [39.]

The research in Kashmir showed 26.4% of youth diabetes patients carried diabetes in their family history according to the study[40.] The research did not specify whether patients obtained their diabetes from their mothers or fathers. The research into infantile-onset diabetes mellitus in Southern India excluded any analysis of maternal diabetes history. [41]

Our research showed spirometry test values displayed important variations between youth with Type 1 Diabetes Mellitus (T1DM) and regular child participants. Specific results indicated T1DM participants demonstrated lower Forced Vital Capacity and Diffusing Capacity of the Lungs for Carbon Monoxide compared to controls with statistical significance reaching <0.001 and <0.03. Data showed lower FEV_1 and PEFR levels existed in the T1DM subjects while the results refused statistical significance due to p-values of 0.08 and 0.1002. The T1DM group showed reduced FEV_1/FVC ratio values that statistical analysis demonstrated as significant below $p<0.005$ suggesting the presence of restrictive lung pattern.

The results match data that has been previously reported in Indian studies. A research by **Prashanth et al. (2022)** measured pulmonary function in 96 children with T1DM against pulmonary function in 102 healthy controls. The assessment of T1DM patient spirometry showed substantial decreases in FVC, FEV_1, FEV_1/FVC, PEFR and MMFR measurements ($p<0.001$). Children with inadequate glycemic control experienced statistically significant reductions in FEV_1 and FVC values compared to those maintaining fair glycemic control thus indicating poor blood glucose regulation might worsen their pulmonary dysfunctions. [33]

A study conducted by **Gangani et al. (2023)** examined pulmonary function tests within Type 2 Diabetes Mellitus (T2DM) patients and discovered that their FVC and FEV_1 mean values were lower than controls at the 0.0001 and 0.0015 p-value levels. The results of this T2DM-specific research demonstrate that diabetes mellitus in all its forms may harm lung function. [42]

A author by Van Gent R et al studied airway obstruction alongside lung volumes and pulmonary carbon monoxide diffusion capacity and airway resistance through body plethysmography measurements in this study group of 27 T1DM children. Research data showed that children with T1DM had higher total airway resistance than reference values at ($P < 0.001$). The measurements of other lung function tests maintained their similar values without showing signs of change. [34]

The researcher by Chhabra SK et al provides valuable prediction methods for spirometry results in Northern Indian children between 6 and 17 years of age but fails to specify its focus on T1DM patients exclusively. All spirometry parameters were strongly influenced by both height and age according to the research findings. The Author underlined how mistakes will occur when interpreting spirometry data in Indian children by relying on prediction equations created for different populations. The proven need exists to employ regional reference values due to their importance for performing accurate pulmonary function assessments.[43]

The respiratory function test results from our study confirm that FEV1 and FVC measurements between diabetic patients with HbA1c <9.5 and >9.5 are significant and equivalent according to Davis et al. (2004)44 and other relevant studies. The FEV1/FVC ratio proved significant ($p=0.003$) when examining subjects with uncontrolled glycemia and demonstrated findings which matched those reported in Klein et al. (2010) and Meo et al. (2019). Research data shows that diabetes-related microvascular changes do not always substantially impact the diffusion capacity measurement. The data from your study upholds previous research showing that diabetes causes minor impairment of lung function that primarily appears as restrictive defects across different studies.[45]

Strengths:

1.Research delivers essential evidence about pulmonary dysfunction in children with Type 1 Diabetes Mellitus (T1DM) despite its rare recognition as a disease consequence.

2.The research builds its findings reliability by evaluating healthy controls in addition to the experimental subjects.

3.The study demonstrates a probable relationship between blood sugar management and lung function deterioration while showing why diabetes management needs to exceed monitoring glycemic levels.

4.The research reveals Type 1 Diabetes Mellitus (T1DM) gives rise to serious lung complications that pediatricians and endocrinologists need to recognize according to this clinical study.

5.This study contributes crucial pulmonary function data which specifically targets the Indian children population thus making it applicable for local healthcare strategies.

6.The research examines both pulmonary function differences between T1DM and healthy children along with establishing a link between glycemic control and lung function deterioration which demonstrates how tight blood glucose management matters.

7.The study provides essential groundwork for forthcoming therapeutic approaches which developers can use to treat lung function deterioration in children with T1DM.

Limitations:

1.The research's general applicability suffers from the small size of the participant sample.

2.The research lacks consideration of potential factors besides diabetes that could affect pulmonary function including environmental elements and nutritional condition along with physical exercise habits.

3.The study lacked continuous follow-up so researchers faced challenges when monitoring T1DM children's lung function deterioration since their adolescence.

4.The analysis failed to include extensive research of advanced pulmonary function tests including body plethysmography

5.Study lacked structured HbA1c follow up to know ,proper association(direct) to lung function worsening

Recommendations:

1.Future studies must conduct their research at multiple research facilities to confirm results in broader racial and ethnic groups.

2.Children with T1DM require ongoing assessment of lung health because **regular observations will help understand the long-term effects of diabetes management strategies**.

3.Future research should examine more lung function factors such as BMI along with insulin treatment methods and disease duration and inflammatory markers to better analyse their effect on lung function results.

4.**All children with diabetes should undergo regular pulmonary function testing through comprehensive care which becomes essential for patients with uncontrolled blood sugar**.

5.Research should develop **structured exercise programs combined with optimized diabetes management to stop or reduce pulmonary function reduction in patients with type 1 diabetes.**

Conclusion:

This study aimed to evaluate pulmonary function parameters in children with Type 1 Diabetes Mellitus (T1DM) and analyze potential impairments associated with the disease. The findings reveal significant alterations in pulmonary function in children with T1DM compared to healthy controls, highlighting the need for regular respiratory monitoring in diabetic children.

The results indicate that key spirometry parameters, including **Forced Vital Capacity (FVC),** and **Diffusion Capacity for Carbon Monoxide (DLCO),** were significantly reduced in children with T1DM. These findings suggest that diabetes-related metabolic changes may **contribute to restrictive lung patterns and decreased lung compliance**.

Additionally, the FEV1/FVC ratio was lower in diabetic children, pointing towards potential airway involvement. However, parameters such as Peak Expiratory Flow Rate (PEFR) did not show a significant difference, **suggesting that obstructive lung disease may not be a primary concern in this population**.

The study explored the relationship between glycemic control and pulmonary function but did not find a strong association between high HbA1c levels and significant pulmonary function impairment. While previous literature suggests potential links, the findings of this study do not establish a definitive correlation.

The study also examined genetic predisposition and environmental factors that could influence pulmonary function in T1DM. A significant association was found between a family history of diabetes and the presence of impaired lung function, reinforcing the role of hereditary factors

in disease progression. These findings suggest that pediatricians should consider lung function assessments as part of routine diabetes care, especially in children with poor metabolic control.

Future research should explore longitudinal trends in pulmonary function decline and investigate potential interventions, such as pulmonary rehabilitation or targeted pharmacological strategies, to mitigate respiratory complications in T1DM patients.Addressing this overlooked aspect of pediatric diabetes care may improve overall patient outcomes and quality of life.

Summary

Pulmonary function tests of children with Type 1 Diabetes Mellitus show significant differences from healthy control subjects and their main abnormal finding is restrictive lung pattern characteristics. The extent of diabetic control and pulmonary disease worsening needs to be studied in detail. The study presents important results about T1DM lung effects yet more comprehensive research with expanded samples and extended longitudinal monitoring is required to achieve comprehensive understanding of T1DM lung effects. Children with T1DM have lower results on spirometry measurements especially in Forced Vital Capacity (FVC) and Diffusing Capacity for Carbon Monoxide (DLCO) which indicates early restrictive lung problems. Research evidence showed that inadequate blood glucose management leads to reduced lung function thus demonstrating how prolonged high blood sugar affects pulmonary complications.

The research presents strong points by applying clinical utility to test results while analyzing an Indian pediatric population which expands regional data compared to other existing studies. The study needs better validation due to its small sample size and lack of prolonged monitoring that requires additional research from multiple clinics.

These results stress why physicians must use routine pulmonary function testing for T1DM patients since good blood glucose management protects their respiratory health from long-term damage. Screening for lung function deterioration at its early stages enables health professionals to provide prompt medical assistance resulting in improved quality of life for children who have diabetes.

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ANNEXURE- I

PROFORMA

SCHEME OF CASE TAKING- PROFORMA

Name:

Ip No:

Gender:

address:

Date of Admission:

Age at Admission

Indication for admission:

Maternal History

Family History:

INVESTIGATIONS

Complete Blood Picture

TC

DC

RBC

HB

PCV

MCV

MCH

MCHC

PLATLET COUNT

RDW

Chest x ray

Test/date								
RBS								
HbA1c								

PFT

	RESULTS	NORMAL VALUES
FVC :		
FEV1:		
FEV1/FVC:		
PEFR:		
DLCO :		

ANNEXURE- II

RESEARCH INFORMED CONSENT FORM

BLDEU (DEEMED TO BE UNIVERSITY) Shri B.M. PATIL Medical College,
Hospital & Research Centre, Vijayapur, Karnataka -586103.

TITLE OF THE PROJECT:

Evaluation of Pulmonary Function Parameters in Children with Type 1 Diabetes Mellitus

GUIDE

DR.M M PATIL

PROFESSOR & HOD

DEPARTMENT OF PEDIATRICS

PG STUDENT

DR. KOSURI MAHESH VARMA

PURPOSE OF RESEARCH:

To study the impairment of lung functions in
T1DM children

PROCEDURE:

I understand that after having obtained a detailed clinical history, thorough clinical examination and relevant investigations, a final work up of the procedure and its outcome is planned.

RISK AND DISCOMFORTS:

I understand that I may experience some pain and discomforts during the examination or during my treatment. This is mainly the result of my condition and the procedures of this study are not expected to exaggerate these feelings which are associated with the usual course of treatment.

BENEFITS:

I understand that my participation in the study will have no direct benefit to me other than the potential benefit of the treatment.

CONFIDENTIALITY:

I understand that the medical information produced by this study will become a part of hospital records and will be subject to the confidentiality. Information of sensitive personal nature will not be part of the medical record but will be stored in the investigations research file. If the data are used for publication in the medical literature or for teaching purpose, no name will be used and other identifiers such as photographs will be used only with special written permission. I understand that I may see the photograph before giving the permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time; Dr. Mahesh K, at the department of pediatrics is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of the study, which might influence my continued participation. A copy of this consent form will be given to me to keep for careful reading.

REFUSAL FOR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice. I also understand that Dr. M.M PATIL may terminate my participation in the study after he/she has explained the reasons for doing so.

INJURY STATEMENT:

I understand that in the unlikely event of injury to my child resulting directly from child's participation in this study, if such injury were reported promptly, the appropriate treatment would be available to the child. But no further compensation would be provided by the hospital. I understand that by my agreements to participate in this study and not waiving any of my legal rights.

I have explained to _____the purpose of the research, the procedures required and the possible risks to the best of my ability

DR.MAHESH KOSURI Date

(Investigator)

Signature

PARENTS / GUARDIAN CONSENT STATEMENT:

We confirm that Dr.KOSURI MAHESH KOSURI is doing a study on .in Shri B. M. Patil Medical College Hospital Vijayapur, Karnataka. Dr…… has explained to us the purpose of research and the study procedure. We are willing to allow our child to get treated in Shri B.M. Patil Medical College Hospital, Vijayapur. We have been explained about the study, benefits and possible discomforts in detail in our native language and we understand the same. We are aware that child will get best treatment and no compensation like financial benefits will be given if our child's condition deteriorates and any untoward event happens, and we will not sue anyone regarding this. Therefore, we agree to give our full consent for child's participation as a subject in this research project.

(Parents / Guardian) (Witness to signature)

Date Date

ಸಂಶೋಧನೆಯ ಉದ್ದೇಶ:

ಮಧುಮೇಹ ಮಕ್ಕಳಲ್ಲಿ ಶ್ವಾಸಕೋಶದ ಕಾರ್ಯಗಳ ದುರ್ಬಲತೆಯ ಅಧ್ಯಯನ

ವಿಧಾನ:

ವಿವರವಾದ ಕ್ಲಿನಿಕಲ್ ಇತಿಹಾಸ, ಸಂಪೂರ್ಣ ಕ್ಲಿನಿಕಲ್ ಪರೀಕ್ಷೆ ಮತ್ತು ಸಂಬಂಧಿತ ತನಿಖೆಗಳನ್ನು ಪಡೆದ ನಂತರ, ಕಾರ್ಯವಿಧಾನದ ಅಂತಿಮ ಕೆಲಸವನ್ನು ಮತ್ತು ಅದರ ಫಲಿತಾಂಶವನ್ನು ಯೋಜಿಸಲಾಗಿದೆ ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ಅಪಾಯ ಮತ್ತು ಅನಾನುಕೂಲಗಳು:

ಪರೀಕ್ಷೆಯ ಸಮಯದಲ್ಲಿ ಅಥವಾ ನನ್ನ ಚಿಕಿತ್ಸೆಯ ಸಮಯದಲ್ಲಿ ನಾನು ಕೆಲವು ನೋವು ಮತ್ತು ಅಸ್ವಸ್ಥತೆಗಳನ್ನು ಅನುಭವಿಸಬಹುದು ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ. ಇದು ಮುಖ್ಯವಾಗಿ ನನ್ನ ಸ್ಥಿತಿಯ ಫಲಿತಾಂಶವಾಗಿದೆ ಮತ್ತು ಈ ಅಧ್ಯಯನದ ಕಾರ್ಯವಿಧಾನಗಳು ಚಿಕಿತ್ಸೆಯ ಸಾಮಾನ್ಯ ಕೋರ್ಸ್‌ಗೆ ಸಂಬಂಧಿಸಿದ ಈ ಭಾವನೆಗಳನ್ನು ಉತ್ಪ್ರೇಕ್ಷಿಸುವುದಿಲ್ಲ ಎಂದು ನಿರೀಕ್ಷಿಸಲಾಗಿದೆ.

ಪ್ರಯೋಜನಗಳು:

ಚಿಕಿತ್ಸೆಯ ಸಂಭಾವ್ಯ ಪ್ರಯೋಜನವನ್ನು ಹೊರತುಪಡಿಸಿ ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನ ಭಾಗವಹಿಸುವಿಕೆಯು ನನಗೆ ಯಾವುದೇ ನೇರ ಪ್ರಯೋಜನವನ್ನು ಹೊಂದಿಲ್ಲ ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ಗೌಪ್ಯತೆ:

ಈ ಅಧ್ಯಯನದಿಂದ ಉತ್ಪತ್ತಿಯಾಗುವ ವೈದ್ಯಕೀಯ ಮಾಹಿತಿಯು ಆಸ್ಪತ್ರೆಯ ದಾಖಲೆಗಳ ಭಾಗವಾಗುತ್ತದೆ ಮತ್ತು ಗೌಪ್ಯತೆಗೆ ಒಳಪಟ್ಟಿರುತ್ತದೆ ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ. ಸೂಕ್ಷ್ಮ ವೈಯಕ್ತಿಕ ಸ್ವಭಾವದ ಮಾಹಿತಿಯು ವೈದ್ಯಕೀಯ ದಾಖಲೆಯ ಭಾಗವಾಗಿರುವುದಿಲ್ಲ ಆದರೆ ತನಿಖೆಯ ಸಂಶೋಧನಾ ಕಡತದಲ್ಲಿ ಸಂಗ್ರಹಿಸಲಾಗುತ್ತದೆ. ಡೇಟಾವನ್ನು ವೈದ್ಯಕೀಯ ಸಾಹಿತ್ಯದಲ್ಲಿ ಅಥವಾ ಬೋಧನಾ ಉದ್ದೇಶಕ್ಕಾಗಿ ಪ್ರಕಟಣೆಗಾಗಿ ಬಳಸಿದರೆ, ಯಾವುದೇ ಹೆಸರನ್ನು ಬಳಸಲಾಗುವುದಿಲ್ಲ ಮತ್ತು ಛಾಯಾಚಿತ್ರಗಳಂತಹ ಇತರ ಗುರುತಿಸುವಿಕೆಗಳನ್ನು ವಿಶೇಷ ಲಿಖಿತ ಅನುಮತಿಯೊಂದಿಗೆ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ. ಅನುಮತಿ ನೀಡುವ ಮೊದಲು ನಾನು ಫೋಟೋವನ್ನು ನೋಡಬಹುದು ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ಹೆಚ್ಚಿನ ಮಾಹಿತಿಗಾಗಿ ವಿನಂತಿ:

ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದ ಕುರಿತು ಹೆಚ್ಚಿನ ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಬಹುದು ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ; ನನ್ನ ಪ್ರಶ್ನೆಗಳಿಗೆ ಅಥವಾ ಕಳವಳಗಳಿಗೆ ಉತ್ತರಿಸಲು ಮಕ್ಕಳ ವಿಭಾಗದಲ್ಲಿರುವ ಡಾ. ಮಹೇಶ್ ಕೆ. ಅಧ್ಯಯನದ ಅವಧಿಯಲ್ಲಿ ಪತ್ತೆಯಾದ ಯಾವುದೇ ಮಹತ್ವದ ಹೊಸ ಸಂಶೋಧನೆಗಳ ಕುರಿತು ನನಗೆ ತಿಳಿಸಲಾಗುವುದು ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ, ಅದು ನನ್ನ ಮುಂದುವರಿದ ಭಾಗವಹಿಸುವಿಕೆಯ ಮೇಲೆ ಪ್ರಭಾವ ಬೀರಬಹುದು. ಈ ಸಮ್ಮತಿ ನಮೂನೆಯ ಪ್ರತಿಯನ್ನು ಎಚ್ಚರಿಕೆಯಿಂದ ಓದುವುದಕ್ಕಾಗಿ ಇರಿಸಿಕೊಳ್ಳಲು ನನಗೆ ನೀಡಲಾಗುವುದು.

ಭಾಗವಹಿಸುವಿಕೆ ಹಿಂತೆಗೆದುಕೊಳ್ಳುವಿಕೆಗೆ ನಿರಾಕರಣೆ:

ನನ್ನ ಭಾಗವಹಿಸುವಿಕೆಯು ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ ಮತ್ತು ನಾನು ಭಾಗವಹಿಸಲು ನಿರಾಕರಿಸಬಹುದು ಅಥವಾ ಒಪ್ಪಿಗೆಯನ್ನು ಹಿಂಪಡೆಯಬಹುದು ಮತ್ತು ಪೂರ್ವಾಗ್ರಹವಿಲ್ಲದೆ ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವುದನ್ನು ನಿಲ್ಲಿಸಬಹುದು ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ. ಡಾ. ಎಂ.ಎಂ ಪಾಟೀಲ್ ಅವರು ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಕಾರಣಗಳನ್ನು ವಿವರಿಸಿದ ನಂತರ ನನ್ನ ಭಾಗವಹಿಸುವಿಕೆಯನ್ನು ಕೊನೆಗೊಳಿಸಬಹುದು ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ಗಾಯದ ಹೇಳಿಕೆ:

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಮಗುವಿನ ಭಾಗವಹಿಸುವಿಕೆಯಿಂದ ನೇರವಾಗಿ ನನ್ನ ಮಗುವಿಗೆ ಗಾಯವಾಗುವ ಸಾಧ್ಯತೆಯಿಲ್ಲದ ಸಂದರ್ಭದಲ್ಲಿ, ಅಂತಹ ಗಾಯವನ್ನು ತಕ್ಷಣವೇ ವರದಿ ಮಾಡಿದರೆ, ಮಗುವಿಗೆ ಸೂಕ್ತವಾದ ಚಿಕಿತ್ಸೆಯು ಲಭ್ಯವಿರುತ್ತದೆ ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ. ಆದರೆ ಆಸ್ಪತ್ರೆಯಿಂದ ಹೆಚ್ಚಿನ ಪರಿಹಾರ ನೀಡುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನನ್ನ ಒಪ್ಪಂದಗಳ ಮೂಲಕ ಮತ್ತು ನನ್ನ ಯಾವುದೇ ಕಾನೂನು ಹಕ್ಕುಗಳನ್ನು ಬಿಟ್ಟುಕೊಡುವುದಿಲ್ಲ ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ನಾನು

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ಸಂಶೋಧನೆಯ ಉದ್ದೇಶ, ಅಗತ್ಯವಿರುವ ಕಾರ್ಯವಿಧಾನಗಳು ಮತ್ತು ನನ್ನ ಸಾಮರ್ಥ್ಯಕ್ಕೆ ಸಾಧ್ಯವಾದಷ್ಟು ಅಪಾಯ

Dr.kosuri Mahesh Varma (Investigator) Date

ಪೋಷಕರು / ಗಾರ್ಡಿಯನ್ ಸಮ್ಮತಿ ಹೇಳಿಕೆ:

ಶ್ರೀ ಬಿ.ಎಂ. ಪಾಟೀಲ್ ವೈದ್ಯಕೀಯ ಕಾಲೇಜು ಆಸ್ಪತ್ರೆ ವಿಜಯಪುರ, ಕರ್ನಾಟಕದಲ್ಲಿ

_____ ಅಧ್ಯಯನ ಮಾಡುತ್ತಿದೆ ಎಂದು ನಾವು ದೃಢೀಕರಿಸುತ್ತೇವೆ. ಡಾ.....

ಸಂಶೋಧನೆಯ ಉದ್ದೇಶ ಮತ್ತು ಅಧ್ಯಯನ ವಿಧಾನವನ್ನು ನಮಗೆ ವಿವರಿಸಿದ್ದಾರೆ. ನಮ್ಮ ಮಗುವಿಗೆ ಶ್ರೀ ಬಿ.ಎಂ.ನಲ್ಲಿ ಚಿಕಿತ್ಸೆ ನೀಡಲು ನಾವು ಸಿದ್ಧರಿದ್ದೇವೆ. ಪಾಟೀಲ್ ವೈದ್ಯಕೀಯ ಕಾಲೇಜು ಆಸ್ಪತ್ರೆ, ವಿಜಯಪುರ. ನಮ್ಮ ಸ್ಥಳೀಯ ಭಾಷೆಯಲ್ಲಿ ಅಧ್ಯಯನ, ಪ್ರಯೋಜನಗಳು ಮತ್ತು ಸಂಭವನೀಯ ಅಸ್ವಸ್ಥತೆಗಳ ಬಗ್ಗೆ ವಿವರವಾಗಿ ನಮಗೆ ವಿವರಿಸಲಾಗಿದೆ ಮತ್ತು ನಾವು ಅದನ್ನು ಅರ್ಥಮಾಡಿಕೊಳ್ಳುತ್ತೇವೆ. ಮಗುವಿಗೆ ಉತ್ತಮ ಚಿಕಿತ್ಸೆ ದೊರೆಯುತ್ತದೆ ಮತ್ತು ನಮ್ಮ ಮಗುವಿನ ಸ್ಥಿತಿ ಹದಗೆಟ್ಟರೆ ಮತ್ತು ಯಾವುದೇ ಅಹಿತಕರ ಘಟನೆ ಸಂಭವಿಸಿದರೆ ಆರ್ಥಿಕ ಪ್ರಯೋಜನಗಳಂತಹ ಯಾವುದೇ ಪರಿಹಾರವನ್ನು ನೀಡಲಾಗುವುದಿಲ್ಲ ಮತ್ತು ನಾವು ಈ ಬಗ್ಗೆ ಯಾರನ್ನೂ ಮೊಕದ್ದಮೆ ಹೂಡುವುದಿಲ್ಲ ಎಂದು ನಮಗೆ ತಿಳಿದಿದೆ. ಆದ್ದರಿಂದ, ಈ ಸಂಶೋಧನಾ ಯೋಜನೆಯಲ್ಲಿ ವಿಷಯವಾಗಿ ಮಗುವಿನ ಭಾಗವಹಿಸುವಿಕೆಗೆ ನಮ್ಮ ಸಂಪೂರ್ಣ ಒಪ್ಪಿಗೆಯನ್ನು ನೀಡಲು ನಾವು ಒಪ್ಪುತ್ತೇವೆ.

(ಪೋಷಕರು / ಪಾಲಕರು) (ಸಹಿಗೆ ಸಾಕ್ಷಿ)

ದಿನಾಂಕ ದಿನಾಂಕ

ANNEXURE- III

BIODATA OF THE GUIDE

NAME :Dr. M M PATIL

EDUCATION : M B B S – KIMS HUBLI

M D PEDIATRICS – KIMS HUBLI

KMC REGISTRATION NUMBE : 52830

WORK EXPERIENCE

: UG TEACHER EXPERIENCE – 18 YEARS

PG TEACHER EXPERIENCE – 11 YEARS

MEMBERSHIP : IAPL 2007p1100

PRESENTLY WORKING : PROFESSOR & HOD BLDE DU DEPARMENT PEDIATRICS

SHRI B M PATIL MEDICAL COLLEGE AND

HOSPITAL , VIJAYAPURA Karnataka 586103

BIODATA OF THE INVESTIGATOR:

NAME DR. KOSURI MAHESH VARMA

DOB 11-4-1993

QUALIFICATION MBBS ASRAM MEDICAL COLLEGE

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KMC REGISTRATION NUMBER

PRESENTLY WORKING AS

POST GRADUATE STUDENT/JUNIOR RESIDENT,

DEPARTMENT OF PEDIATRICS,

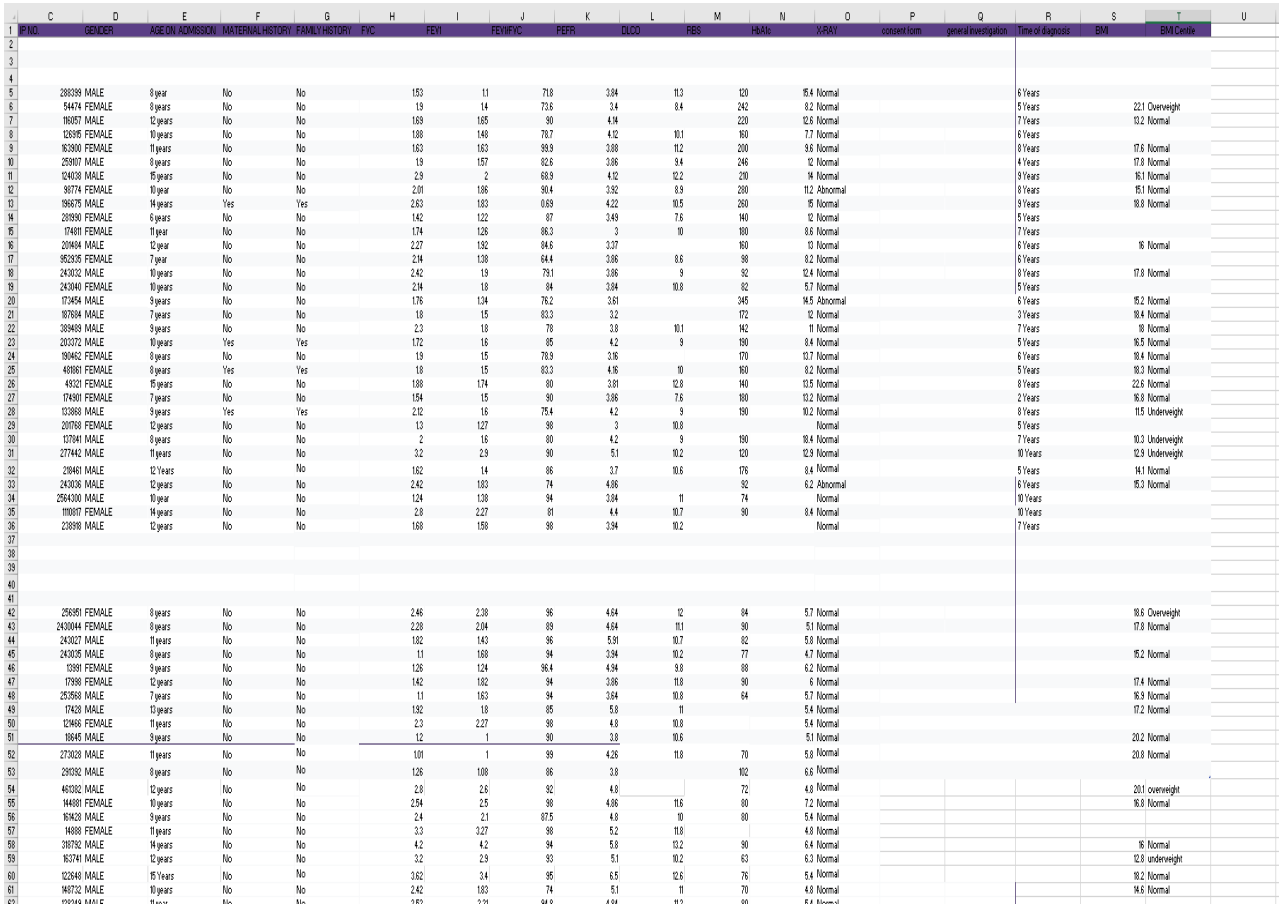
SHRI B M PATIL MEDICAL COLLEGE,

HOSPITAL & RESEARCH CENTRE,

VIJAYAPURA –

ANNEXURE- IV

MASTER CHART



IP NO.	GENDER	AGE ON ADMISSION	MATERNAL HISTORY	FAMILY HISTORY	FVC	FEV1	FEV1/FVC	PEFR	DLCO	FBS	HbA1c	X-RAY	consent form	general investigation	Time of diagnosis	BMI	BMI Centile
288399	MALE	8 year	No	No	1.53	1.1	71.8	3.84	11.3	120	15.4	Normal			6 Years		
54474	FEMALE	8 years	No	No	1.9	1.4	73.6	3.4	8.4	242	9.2	Normal			5 Years		22.1 Overweight
116057	MALE	12 years	No	No	1.69	1.65	90	4.14		220	12.6	Normal			7 Years		13.2 Normal
126195	FEMALE	10 years	No	No	1.88	1.48	78.7	4.12	10.1	160	7.7	Normal			6 Years		
163900	FEMALE	11 years	No	No	1.63	1.63	99.9	3.88	11.2	200	9.6	Normal			8 Years		17.6 Normal
259107	MALE	8 years	No	No	1.9	1.57	82.6	3.86	9.4	246	12	Normal			4 Years		17.8 Normal
124038	MALE	15 years	No	No	2.9	2	68.9	4.12	12.2	210	14	Normal			9 Years		16.1 Normal
98774	FEMALE	10 year	No	No	2.01	1.86	90.4	3.92	8.9	280	11.2	Abnormal			8 Years		15.1 Normal
196675	MALE	14 years	Yes	Yes	2.63	1.83	0.69	4.22	10.5	260	15	Normal			9 Years		18.8 Normal
281590	FEMALE	6 years	No	No	1.42	1.22	87	3.49	7.6	140	12	Normal			5 Years		
174911	FEMALE	11 year	No	No	1.74	1.26	86.3	3	10	180	8.6	Normal			7 Years		
201494	MALE	12 year	No	No	2.27	1.92	94.6	3.37		160	13	Normal			6 Years		16 Normal
952935	FEMALE	7 year	No	No	2.14	1.38	64.4	3.86	8.6	98	9.2	Normal			6 Years		
243032	MALE	10 years	No	No	2.42	1.9	78.1	3.86	9	92	12.4	Normal			8 Years		17.8 Normal
243040	FEMALE	10 years	No	No	2.14	1.8	84	3.84	10.8	82	5.7	Normal			5 Years		
173454	MALE	9 years	No	No	1.76	1.34	76.2	3.61		345	14.5	Abnormal			6 Years		15.2 Normal
187684	MALE	7 years	No	No	1.8	1.5	83.3	3.2		172	12	Normal			3 Years		18.4 Normal
389493	MALE	9 years	No	No	2.3	1.8	78	3.8	10.1	142	11	Normal			7 Years		18 Normal
203372	MALE	10 years	Yes	Yes	1.72	1.6	95	4.2	9	190	9.4	Normal			5 Years		16.5 Normal
190462	FEMALE	8 years	No	No	1.9	1.5	78.9	3.16		170	13.7	Normal			6 Years		18.4 Normal
48161	FEMALE	8 years	Yes	Yes	1.8	1.5	83.3	4.16	10	160	9.2	Normal			5 Years		18.3 Normal
49321	FEMALE	15 years	No	No	1.88	1.74	80	3.81	12.8	140	13.5	Normal			8 Years		22.6 Normal
174901	FEMALE	7 years	No	No	1.54	1.5	90	3.86	7.6	180	13.2	Normal			2 Years		16.8 Normal
133868	MALE	9 years	Yes	Yes	2.12	1.6	75.4	4.2	9	190	10.2	Normal			8 Years		11.5 Underweight
201768	FEMALE	12 years	No	No	1.3	1.27	98	3	10.8			Normal			5 Years		
137041	MALE	8 years	No	No	2	1.6	80	4.2	9	190	18.4	Normal			7 Years		10.3 Underweight
277442	MALE	11 years	No	No	3.2	2.9	90	5.1	10.2	120	12.9	Normal			10 Years		12.9 Underweight
218461	MALE	12 Years	No	No	1.62	1.4	86	3.7	10.6	176	9.4	Normal			5 Years		14.1 Normal
243036	MALE	12 years	No	No	2.42	1.83	74	4.86		92	6.2	Abnormal			6 Years		15.3 Normal
2564300	MALE	10 year	No	No	1.24	1.38	94	3.84	11	74		Normal			10 Years		
110817	FEMALE	14 years	No	No	2.8	2.27	81	4.4	10.7	90	9.4	Normal			10 Years		
238918	MALE	12 years	No	No	1.68	1.58	98	3.94	10.2			Normal			7 Years		
256951	FEMALE	8 years	No	No	2.46	2.38	96	4.64	12	84	5.7	Normal					18.6 Overweight
2430044	FEMALE	8 years	No	No	2.28	2.04	89	4.64	10.1	90	5.1	Normal					17.8 Normal
243027	MALE	11 years	No	No	1.82	1.43	96	5.91	10.7	82	5.8	Normal					
243035	MALE	8 years	No	No	1.1	1.68	94	3.94	10.2	77	4.7	Normal					15.2 Normal
12891	FEMALE	9 years	No	No	1.26	1.24	96.4	4.94	9.8	88	6.2	Normal					
17898	FEMALE	12 years	No	No	1.42	1.82	94	3.86	11.8	90	6	Normal					17.4 Normal
253568	MALE	7 years	No	No	1.1	1.63	94	3.64	10.8	64	5.7	Normal					16.9 Normal
17428	MALE	13 years	No	No	1.92	1.8	85	5.8	11		5.4	Normal					17.2 Normal
124166	FEMALE	11 years	No	No	2.3	2.27	98	4.8	10.8		5.4	Normal					
18645	MALE	9 years	No	No	1.2	1	90	3.8	10.6		5.1	Normal					20.2 Normal
273028	MALE	11 years	No	No	1.01	1	99	4.26	11.8	70	5.8	Normal					20.8 Normal
291092	MALE	8 years	No	No	1.26	1.08	86	3.8		102	6.6	Normal					
461092	MALE	12 years	No	No	2.8	2.6	92	4.8		72	4.8	Normal					20.1 overweight
144881	FEMALE	10 years	No	No	2.54	2.5	98	4.86	11.6	80	7.2	Normal					16.8 Normal
161428	MALE	9 years	No	No	2.4	2.1	87.5	4.8	10	80	5.4	Normal					
14888	FEMALE	11 years	No	No	3.3	3.27	98	5.2	11.8		4.8	Normal					
318792	MALE	14 years	No	No	4.2	4.2	94	5.8	13.2	90	6.4	Normal					16 Normal
163741	MALE	12 years	No	No	3.2	2.9	93	5.1	10.2	63	6.3	Normal					12.8 underweight
123648	MALE	15 Years	No	No	3.62	3.4	95	6.5	12.6	76	5.4	Normal					18.2 Normal
146732	MALE	10 years	No	No	2.42	1.83	74	5.1	11	70	4.8	Normal					14.6 Normal

ANNEXURE- V

BLDE

(DEEMED TO BE UNIVERSITY)

Declared as Deemed to be University u/s 3 of UGC Act, 1956

Accredited with 'A' Grade by NAAC (Cycle-2)

The Constituent College

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 961/2022-23 10/4/2023

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Saturday, 18th March, 2023 at 11.30 a.m. in the CAL Laboratory, Dept. of Pharmacology,** scrutinizes the Synopsis/ Research Projects of Post Graduate Student / Under Graduate Student /Faculty members of this University /Ph.D. Student College from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "EVALUATION OF PULMONARY FUNCTION PARAMETERS IN CHILDREN WITH TYPE 1 DIABETES MELLITUS".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR.KOSURI MAHESH VARMA

NAME OF THE GUIDE: DR.M.M.PATIL, PROFESSOR, DEPT. OF PEDIATRICS.

Dr. Santoshkumar Jeevangi
Chairperson
IEC, BLDE (DU),
VIJAYAPURA

Chairman,
Institutional Ethical Committee,
BLDE (Deemed to be University)
Vijayapura

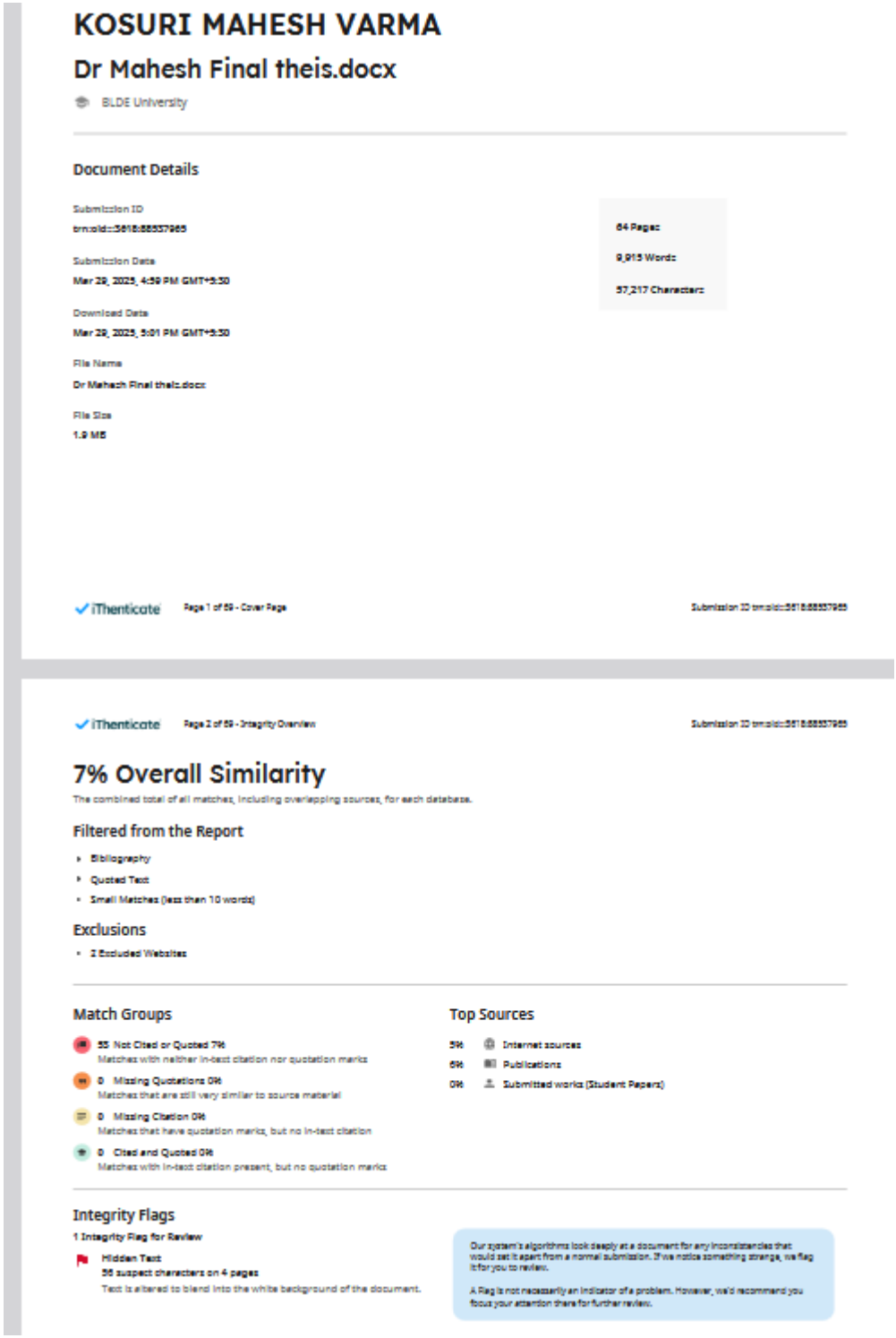
Dr.Akram A. Naikwadi
Member Secretary
IEC, BLDE (DU),
VIJAYAPURA

MEMBER SECRETARY
Institutional Ethics Committee
BLDE (Deemed to be University)
Vijayapura-586103. Karnataka

Following documents were placed before Ethical Committee for Scrutinization.

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

ANNEXURE- VI



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