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Profile of Diabetic Patients Subjected for Spirometry

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Abstract

Introduction: When the air passes through the nasal cavity and pharynx, it gets warmed and takes up water vapour. And it passes down the trachea and through bronchioles, respiratory bronchioles and alveolar ducts to the alveoli. Between the trachea and the alveolar sacs, the airway divides 23 times forming 23 generations. The first 16 generations form the conducting zone of the airways made up of bronchi, bronchioles and terminal bronchioles. The remaining 7 generations from the transitional and respiratory zones where gas exchange occur and are made up of respiratory bronchioles, alveolar ducts and alveoli.

Methodology: Information will be collected through a pre tested and structured proforma for each patient Qualifying patients will be undergoing detailed history, clinical examination, routine investigations like FBS, PPBS, HBA1c, fundus evaluation and spirometric evaluation using a easy one flow spirometer Glycemic control is taken as HBA1C below 7.5 and HBA1C more than 7.5 is considered as uncontrolled sugars.

Results: Group 1 had 15 patients with uncontrolled sugars i.e HbA1c of more than 7.5 Group 2 had 26 patients with HbA1c of more than 7.5, out of 70 patients of type 2 DM, 41 (58.6%) patients had HBA1C of more than 7.5.

Conclusion: Diabetes mellitus leads to increased hepatic glucose output. First, liver glycogen stores are mobilised then hepatic gluconeogenesis is used to produce glucose. Insulin deficiency also impairs non-hepatic tissue utilization of glucose, particularly in adipose tissue and skeletal muscle, insulin stimulates glucose uptake.

Keywords: Diabetes; HBA1C; Spirometry.

Introduction

During 129 - 200 AD (mien did volumetric experiment on human ventilation. In 1680 G. A. Borelli measured the inspiratory volume of lung. Jurin. J-measured 650 mltidal volume and 3610ml of maximal expiration in 1718. Bernouli. D described the method of measuring expired volume in 1749. Kentish.E used simple 'Pulmometer' to study ventilatory volume in disease in 1813. John Hutchinson named subdivisions of the lung volumes and described the methods for measuring them in 1852.Later in 1854 Wintrich developed modified spirorneter and concluded that 3 parameters determine vital capacity: body height, weight and age. In 1860 N.Grehant

calculated the volume of gas (FRC). In 1882 Zuntz gave the formulation of pulmonary dead space. In 1890 Christian Bohr explained the equation for calculating respiratory dead space in terms of gases in alveolar and expired air. In the year 1894 Loewy determined dead space to be 140 ml.[1,2]

Lavoisier and Seguin measured lung volume during late 18th century. At the beginning of 19th century Humphrey Day described indirect measurement of lung volumes. Haldane and Priestly explained alveolar sampling technique in 1905. Knipping H.W introduced a standardized method of spirometry in 1929. Wright B.M. and McKerrow C.B introduced the peak flow meter in 1959. Campbell et al presented light weight peak flow meter in 1974.[3]

Both the lungs are protected within the thoracic cage. The bifurcation of trachea corresponds with the lower border of manubrium sternum that is at the angle of Louis.

When the air passes through the nasal cavity and pharynx, it gets warmed and takes up water vapour. And it passes down the trachea and through bronchioles, respiratory bronchioles and alveolar ducts to the alveoli.

Between the trachea and the alveolar sacs, the airway divides 23 times forming 23 generations. The first 16 generations form the conducting zone of the airways made up of bronchi, bronchioles and terminal bronchioles. The remaining 7 generations from the transitional and respiratory zones where gas exchange occur and are made up of respiratory bronchioles, alveolar ducts and alveoli. These multiple divisions greatly increase the total cross sectional area of the airways, from 2.5 cm^2 in the trachea to 11,800 cm^2 in the alveoli.[4]

These are surrounded by pulmonary capillaries and in most areas the structures between the air and capillary blood across which O_2 and CO_2 diffuse are very thin. There are approximately about 300 million alveoli in humans, and the total area of the alveolar walls in contact with capillaries in both lungs is about 70 m^2.

The alveoli are lined by 2 types of epithelial cells. Type 1 cells are the primary lining cells and are flat cells with large cytoplasmic extension. Type II cells also called as granular pneumocytes are comparatively thicker and contain numerous lamellar inclusion bodies and secrete surfactant.[5,6]

Methodology

Source of Data

Patients visiting medicine OPD and patients admitted in IPD in medical college hospital taken for study considering the inclusion and exclusion criteria.

Sample Size: 70 diabetic subjects meeting the criteria for the present study

Inclusion Criteria

All patients presenting to OPD and patients from IPD who fulfil the inclusion criteria for the study

1. Previously diagnosed type 2 Diabetic patients
2. Type 2 Diabetes mellitus with age group of 30 – 60 years.
3. Who gives written informed consent.

Exclusion Criteria

1. Bronchial Asthma.
2. COPD
3. History of Pulmonary Tuberculosis.
4. History of cardiovascular disease
5. Smokers
6. ILD
7. Those not willing for the study

Information will be collected through a pre tested and structured proforma for each patient

Qualifying patients will be undergoing detailed history, clinical examination, routine investigations like FBS, PPBS, HBA1c, fundus evaluation and spirometric evaluation using a easy one flow spirometer

Glycemic control is taken as HBA1C below 7.5 and HBA1C more than 7.5 is considered as uncontrolled sugars

Diabetic patients of different durations are selected Using criteria laid down

Group A: Type 2 diabetes mellitus of 5-10 year duration

Group B: Type 2 diabetes mellitus of 11-15 year duration

Results

Table 1: Comparison of two study groups with status of HBA1c.

Status of HBA1c	Group 1	%	Group 2	%	Total	%
<7.50	20	57.1	9	25.7	29	41.4
>=7.50	15	42.9	26	74.3	41	58.6
Total	35	100.0	35	100.0	70	100.0

Chi-square = 7.1244 P = 0.0080*

Group 1 had 15 patients with uncontrolled sugars i.e HbA1c of more than 7.5

Group 2 had 26 patients with HbA1c of more than 7.5, out of 70 patients of type 2 DM, 41(58.6%) patients had HBA1C of more than 7.5 (Table 1)

Table 2: Comparison of two study groups with FBS scores by t test.

Group	Mean	SD	SE	t-value	p-value
Group 1	164.49	21.71	3.67	-0.8727	0.3859
Group 2	169.63	27.28	4.61		

Group 1 had mean FBS of 164.1 and group 2 had mean FBS of 169.2 (Table 2)

Table 3: Comparison of two study groups with PPBS scores by t test.

Group	Mean	SD	SE	t-value	p-value
Group 1	204.09	43.14	7.29	-0.0947	0.9248
Group 2	204.94	31.70	5.36		

In our study the mean PPBS value in both group was 204 (Table 3)

Discussion

In this study, Group 1 had mean FBS of 164.1 and group 2 had mean FBS of 169.2. In our study the mean PPBS value in both group was 204

Our study is in agreement with Walter.E.Rohert et al who studied the relationship of FBS to Pulmonary function and found a decrease in FVC with increasing quartile of blood glucose with a P value of 0.04 which is significant. Relationship between FBS and FEVI was analyzed and found a decrease in FEV1 with increasing quartile of blood glucose with a P value of 0.03 which was significant.

Framingham heart study wherein association of glycemic status and lung functions was studied and results were a larger decrease in FVC than FEVI resulted in high FEV1/FVC in diabetic subjects suggestive of restrictive pattern.

A polyuric state similar to diabetes mellitus was described over 3500 years ago and the name diabetes' came from the Greek word for a Syphon'. The sweet taste of diabetic urine was recognized in 1st Millennium. Later in the year 1776 Mathew Dobson stated that diabetes was a systemic condition and not a disease of the kidney and diabetic urine and serum tasted sweet John Rollo added Me1litus (honeyed) in 18th century. Chevreul in 1815 identified excess sugar in diabetes as glucose. In 1840 Claude Bernard showed that glucose was normally present in the body and is stored in the liver as glycogen and secreted into the blood stream during fasting. In 1864 Ma Eduard Von Jaeger- defined Diabetic retinopathy. In 1869 Marchal de Calvi identified the association of neuropathy with diabetes mellitus. Paul langerhans explained the production of an internal secretion, which regulated glucose metabolism.[7]

In 1880 Etienne Lancereaux sub dived on clinical grounds into, diabetic maiqre (leen subject) and diabetic obese gras (obese). The same year Stephan Mackenzie and Edward Nettle described specific lesions (micro aneurysm and new vessels). In 1885 Frederick Pavy explained symptoms of neuropathy with diabetes. In 1889 Oskar Minkowski and Josef Von Mering demonstrated role of pancreas in diabetes by producing diabetes in a dog by pancreatectomy.

In 1893 GustaveLaguesse suggested pancreatic islets produced internal secretion that regulated glucose metabolism During 1900, 1920 George Zuelzer and Nicolas pauluco attempted to isolate insulin.

Diabetes mellitus leads to increased hepatic glucose output. First, liver glycogen stores are mobilised then hepatic gluconeogenesis is used to produce glucose. Insulin deficiency also impairs non-hepatic tissue utilization of glucose, particularly in adipose tissue and skeletal muscle, insulin stimulates glucose uptake. Reduced glucose uptake by peripheral tissues in turn leads to a reduced rate of glucose metabolism. In addition, the level of hepatic glucokinase is regulated by insulin. Therefore, a reduced rate of glucose phosphorylation in hepatocytes leads to increased delivery to the blood. The combination of increased hepatic glucose production and reduced peripheral tissues metabolism leads to elevated plasma glucose levels. When the capacity of the kidneys to absorb glucose is surpassed, glycosuria ensues. Glucose is an osmotic diuretic and an increase in renal loss of glucose is accompanied by loss of water and electrolytes, termed polyuria. The result of the loss of water leads to the activation of the thirst mechanism (polydipsia). The negative caloric balance, resulting from the glycosuria and tissue catabolism as well as failure of hypothalamic

regulation leads to an increase in appetite and food intake (polyphagia).[8]

Although chronic hyperglycemia is an important etiologic factor leading to complications of DM, the mechanism by which it leads to such diverse cellular and organ dysfunction is unknown. At least four prominent theories, which are not mutually exclusive, have been proposed to explain how hyperglycemia might lead to the chronic complications of DM. An emerging hypothesis is that hyperglycemia leads to epigenetic changes in the affected cells.

One theory is that increased intracellular glucose leads to the Formation of advanced glycosylation end products (AGEs), which bind to a cell surface receptor, via the non enzymatic glycosylation of intra- and extracellular proteins. Non enzymatic glycosylation results from the interaction of glucose with amino groups on proteins. Ages have been shown to cross-link proteins (e.g., collagen. extracellular matrix proteins), accelerate atherosclerosis, promote glomerular dysfunction, reduce nitric oxide synthesis, induce endothelial dysfunction, and alter extracellular matrix composition and structure. The serum level of AGEs correlates with the level of glycemia, and these products accumulate as the glomerular filtration rate (GFR) declines.[9]

A second theory is based on the observation that hyperglycemia increases glucose metabolism via the sorbitol pathway. Intracellular glucose is predominantly metabolized by phosphorylation and subsequent glycolysis. But when increased, some glucose is converted to sorbitol by the enzyme aldose reductase. Increased sorbitol concentration alters redox potential and increases cellular osmolality, generates reactive oxygen species, and likely leads to other types of cellular dysfunction. However, testing of this theory in humans, using aldose reductase inhibitors, has not demonstrated significant beneficial effects on clinical endpoints of retinopathy, neuropathy, or nephropathy.

A third hypothesis proposes that hyperglycemia increases the formation of diacyl glycerol leading to activation of protein kinase C (PKC). Among other actions, PKC alters the transcription of genes for fibronectin, type IV collagen, contractile proteins, and extracellular matrix proteins in endothelial cells and neurons. Inhibitors of PKC are being studied in clinical trials.[10]

A fourth theory proposes that hyperglycemia increases the flux through the hexosamine pathway, which generates fructose-6-phosphate a substrate for O-linkedglycosylation. The hexosamine pathway may alter function glylcosylation of proteins such as caduthelialnoneoxide synthase or by changes in gene expression of transforming growth factor beta or plasminogen activator inhibitor-I (PAI-I).[5]

Growth factors appear to play an important role in some DM-related complications, and their production is increased by most of these proposed pathways. Vascular endothelial growth factor A (VEGF-A) is increased locally in diabetic proliferative retinopathy and decreases after laser photocoagulation. TGF beta is increased in diabetic nephropathy and stimulates basement membrane production of collagen and fibronectin by mesangial cells. Other growth factors such as platelet- derived growth factor, epidermal growth factor, insulin-like growth factor I, growth hormone, basic fibroblast growth factor, and even insulin, have been suggested to play a role in DM-related complications. A possible unifying mechanism is that hyperglycemia leads to increased production of reactive oxygen species or superoxide in the mitochondria; these compounds may activate all four of the pathways described above.[8] Although hyperglycemia serves as the initial trigger for complications of diabetes, it is still unknown whether the same pathophysiologic processes are operative in all complications or whether some pathways predominate in certain organs.

Conclusion:

In this study, Group 1 had mean FBS of 164.1 and group 2 had mean FBS of 169.2. In our study the mean PPBS value in both group was 204

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