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CONTRIBUTION OF BMI ON OXYGEN SATURATION IN DIABETIC PATIENTS

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INTRODUCTION

Diabetes mellitus is a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both.[1] The World Health Organization states that 347 million people worldwide were suffering from diabetes in 2008, which equates to 9.5% of the adult population. The incidence of diabetes is rapidly increasing with estimations suggesting that this number will almost double by 2030.[2]

Diabetes is strongly associated with both microvascular and macrovascular complications, including retinopathy, nephropathy, and neuropathy (microvascular) and ischemic heart disease, peripheral vascular disease, and cerebrovascular disease (macrovascular), resulting in tissue and organ hypoxia and damage in approximately one third to one-half of people with diabetes.[3]

Hemoglobin is the principal carrier of oxygen in the body. The American Diabetes Association has recommended glycated hemoglobin (HbA1c) as a possible substitute for fasting blood glucose for the diagnosis of diabetes.[3][4] The major form of glycated hemoglobin is hemoglobin A1c (HbA1c)[5] Glycosylation of hemoglobin is proposed to be vasoactive is via the formation of reactive oxygen species. Glycosylation of hemoglobin also lowers oxygen-carrying capacity, thereby promoting hypoxia and its related systemic vascular vasodilatory adaptations and responses.[4]

Obesity, defined as a body mass index (BMI) $\geq$ 30 kg/m^2.[6] Obesity causes or exacerbates many health problems, both independently and in association with other diseases. In particular, it is associated with the development of type 2 diabetes mellitus.[7] Large, high-quality longitudinal or prospective studies have confirmed that obesity is a significant risk factor for and contributor to increased morbidity and mortality, primarily from CVD and diabetes.[6]

Obesity can cause hypoxemia by decreasing lung volumes to where there is closure of lung units during normal breathing.[8] Cardiac output, circulating blood volume, and resting oxygen consumption are all increased in obese persons; however, total blood flow is subnormal in relation to body weight. Obesity augments the size of individual fat cells without increasing blood flow. Fat tissue is therefore relatively hypo perfused.[9][10]

Therefore, the present study was conducted to evaluate the effect of obesity and glycated hemoglobin on oxygen saturation in type II diabetic patients.

MATERIALS AND METHODOLOGY

The present study was conducted at a private clinic in Vijaya health care. In present study a total of 100 subjects, age above 18 years with BMI greater than 30 and a known history of Type II diabetes are included. Pregnant females, patients with history of lung disease and/or heart disease are excluded. Also, patients on medications that can interfere with oxygen saturation are not included in the study. Informed written consent was taken from all the participants.

Anthropometry and clinical examination

Weight was measured using a digital scale with sensitivity of 0.1 kg. Height was measured to the nearest 0.1 cm using wall mounted scale. Body mass index (BMI) was calculated as weight (kilogram) divided by squared height (meter2). Waist to hip ratio (WHR) was calculated as ratio of waist circumference (WC), measured at the level of umbilicus after expiration, to hip circumference (HC), measured as maximal horizontal circumference at the level of the buttocks. Blood pressure was measured using mercury sphygmomanometer auscultatory method.

Biochemical analysis

Fasting blood sample was withdrawn from ante-cubital vein under aseptic precautions and collected in fluoride, EDTA and plain vacutainers. For HbA1c 1 mL whole blood was kept in EDTA as aliquot at 4–8 ^{0}C and its concentration was assayed using high performance liquid chromatography.

Pulse Oximeter

SpO2 was monitored in the sitting position with a pulse-oximeter. The fundamental physical property that allows the pulse oximeter to measure the oxygen saturation of hemoglobin is that blood changes color as hemoglobin absorbs varying amounts of light depending on its saturation with oxygen. Oxyhemoglobin does not absorb much red light, but as the hemoglobin oxygen saturation drops, more and more red light is absorbed, and the blood becomes darker. At the near infrared range of light however, oxyhemoglobin absorbs more light than reduced hemoglobin.[11] The probe was applied to the index finger of left hand. Mean of the two readings taken 10 min apart was recorded.

Routine biochemical investigation such as fasting and random blood glucose was done.

Statistical analysis

All statistical tests were performed using SPSS version 20. For comparisons of different variables, student's t-test was used. The statistical analysis was carried out using Pearson coefficient of correlation for assessment of relationship between variables. Bivariate regression analysis was carried out to assess the effect of WC on SpO2. A p value <0.05 was considered statistically significant (two-tailed).

Table 1: Distribution of anthropometric parameters of study subjects.

Anthropometric parameters	Frequency	Percentage
Body mass index(kg/m²)		
<18.5 kg/m² {Underweight}	2	2.00%
18.5 -24.99 kg/m² {Normal BMI	33	33.00%
25-29.99 kg/m² {Overweight}	39	39.00%
>=30 kg/m² {Obese}	26	26.00%
Mean ± SD	26.89 ± 3.76	
Median (25th-75th percentile)	26.27(22.96-29.41)	
Range	17.95-36.52	
Height(cm)		
Mean ± SD	157.82 ± 8.45	
Median (25th-75th percentile)	156(152.67-165)	
Range	142-173	
Weight(kg)		
Mean ± SD	68.19 ± 10.92	
Median (25th-75th percentile)	69(59-74)	
Range	38-94	
Waist circumference(cm)		
Mean ± SD	92.37 ± 10.38	
Median (25th-75th percentile)	93(84-100)	
Range	73-113	
Hip circumference(cm)		
Mean ± SD	95.78 ± 10.23	
Median (25th-75th percentile)	94(88-102)	
Range	73-117	
Waist hip ratio		
Mean ± SD	0.91 ± 0.05	
Median (25th-75th percentile)	0.92(0.90-0.96)	
Range	0.85-1.16	

Table 2: Descriptive statistics of biochemical parameters and oxygen saturation of study.

Biochemical parameters and oxygen saturation	Mean ± SD	Median (25th-75th percentile)	Range
Fasting blood sugar(mg/dL)	179.39 ± 60.61	155.52(135.42-206.38)	89-374
Random blood sugar(mg/dL)	251.72 ± 85.37	223.7(178-298)	99-481
Glycosylated hemoglobin HbA1C (%)	8.2 ± 2.39	8.5(6.42-10.65)	4.5-14
Oxygen saturation (%)	94.72 ± 3.81	95(94-97)	83-99

Table 3: Descriptive statistics of duration of disease(years) of study subjects.

Variable	Mean ± SD	Median (25th-75th percentile)	Range
Duration of disease(years)	7.12 ± 4.23	6(3.25-9.65)	1.6-27

Table 4: Correlation of body mass index with and oxygen saturation.

Variables	Oxygen saturation (%)
Body mass index(kg/m^2)	
Correlation coefficient	0.184
p value	0.43

Pearson correlation coefficient.

Table 5: Correlation of glycosylated hemoglobin HbA1C(%) with oxygen saturation.

Variables	Oxygen saturation (%)
Glycosylated hemoglobin HbA1C (%)	
Correlation coefficient	-0.129
p value	0.745

Pearson correlation coefficient.

DISCUSSION

Hypoxia in adipose tissue is an early event in the course of obesity and leads to dysregulated adipokine production, inflammation and the metabolic syndrome.[12] In humans, coronary flow reserve is significantly lower in obese compared to nonobese subjects, and capillary recruitment is reduced in nondiabetic obese individuals compared with lean control subjects.[13]

However, in present study no significant correlation has been observed between BMI and oxygen saturation. Similar results were obtained by Zavorsky and Munro in their respective studies in which no association between obesity and SpO2 was found.[14][15] While the study by Garg et al showed inversely proportional relationship between the two.[3] Similarly negative correlation between SpO2 and BMI was seen by Littleton and Kabon respectively in their studies.[8] This can be attributed to profound effect of obesity that alter lung mechanics, diminish exercise capacity, augment airway resistance resulting in an increased work of breathing, and influence respiratory muscle function, control of breathing, and gas exchange.[16]

Also individuals with diabetes might be at higher risk for moderate or severe infection-related morbidity caused by altered defense mechanisms, including the effects of hyperglycemia, obesity, and/or the effects of neuropathy and impaired tissue perfusion on injury and wound healing.[17]

Insulin resistance and hyperglycemia, acting via oxidative stress, inflammation, and advanced glycation end products, can induce microvascular abnormality. In people with type 2 diabetes, coronary flow reserve is inversely related to hemoglobin A1C and fasting plasma glucose levels, which suggests that chronic hyperglycemia is a key factor.[13]

Glycohemoglobin is produced via a non-enzymatic reaction between the free aldehyde group of glucose or other sugars and the unprotonated form of free amino groups of hemoglobin. Glycosylation alters the structure and function of hemoglobin and tends to shift the oxygen dissociation curve to the left, leading to an increase in hemoglobin-oxygen affinity and a reduction in oxygen delivery to tissues.[18]

In the present study negative correlation was observed between glycated hemoglobin and oxygen saturation. P value was not significant, however. Pu in his study observed that elevated blood HbA1c concentrations lead to an overestimation of SaO2 by SpO2, suggesting that arterial blood gas analysis may be needed for type 2 diabetic patients with poor glycemic control during the treatment of hypoxemia.[18]

However, among diabetic and obese individuals, existing cardiometabolic risk factors should be monitored and treated intensively with diet quality, PA, and pharmacological or other treatments as necessary. Each of these interventions provides benefits in weight loss and maintenance of blood glucose level.

CONCLUSION

The oxygen saturation level is not affected by HbA1c level and body mass index in diabetic patients.

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