International Journal of Research in Medical Sciences
Kotwal S et al. Int J Res Med Sci. 2022 Sep;10(9):1904-1907
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20222081

Original Research Article

Obesity and glycated hemoglobin: is there a relation with oxygen saturation in type 2 diabetes mellitus?

Nusrat Chouhan[1], Monica Manhas[1], Suman Kotwal[2]*, Mohsina Majid[1], Mrityunjay[1]

[1]Department of Physiology, GMC, Jammu, Jammu and Kashmir, India
[2]Department of Endocrinology, SSH, GMC, Jammu, Jammu and Kashmir, India

Received: 25 July 2022
Revised: 06 August 2022
Accepted: 08 August 2022

***Correspondence:**
Dr. Suman Kotwal,
E-mail: sumankk1230@rediffmail.com

ABSTRACT

Background: Diabetics and obese persons are highly susceptible to cardiovascular diseases (CVD). Obesity causes hypoxemia and glycated hemoglobin (HbA1c) also is known to lower the oxygen-carrying capacity and related systemic vascular vasodilatory adaptations and responses. Aim was to assess the effect of obesity and glycosylated hemoglobin on oxygen saturation in patients of type 2 diabetes mellitus (T2DM).
Methods: HbA1c level and oxygen saturation (SpO_2) were measured in 100 adult, obese (Body mass index>30) T2DM patients.
Results: Mean HbA1C and SpO_2 values were 8.69±2.41% and 95.24±3.23% respectively. P value for correlation between SpO_2 and BMI was 0.3. On the other hand, p-value for correlation between SpO_2 and HbA1c was 0.679
Conclusions: There was no significant effect of obesity and HbA1c on oxygen saturation in T2DM patients.

Keywords: HbA1c, Oxygen saturation, Obesity, T2DM

INTRODUCTION

Obesity, defined as a BMI ≥30 kg/m^2, is accompanied by profound changes in physiological function. Obese patients are at high risk of comorbid disorders like diabetes, CVD, and cancer.[1-3] Obesity also causes hypoxemia and it does so in three ways: (i) through obesity-hypoventilation syndrome, (ii) through co-morbid conditions such as congestive heart failure, and (iii) reduction in the functional residual capacity (FRC).[4] Obesity augments the size of individual fat cells without increasing blood flow. Tissue is therefore relatively hypo-perfused and likely to be poorly oxygenated.[5]

Hemoglobin is the principal carrier of oxygen in the body. HbA1c is produced by a ketoamine reaction between glucose and the N-terminal valine of both β-chains of the hemoglobin molecule. The major form of HbA1c is hemoglobin A1c (HbA1c).[6,7] Glycosylation of hemoglobin also lowers the oxygen-carrying capacity, thereby promoting hypoxia and its related systemic vascular vasodilatory adaptations.

Analysis of HbA1c in blood provides evidence about an individual's average blood glucose levels during the previous two to three months, which is the predicted half-life of red blood cells (RBCs). The HbA1c is now recommended as a standard of care (SOC) for testing and monitoring the type 2 diabetes.[8]

T2DM constitutes the majority of diabetes burden, comprising some 85% of cases.[9] Most patients with this form of diabetes are obese, and obesity itself causes some degree of insulin resistance. Such patients are at increased risk of developing macrovascular and microvascular complications.[10] Insulin resistance and hyperglycemia, acting via oxidative stress, inflammation, and advanced glycation end products, can induce microvascular abnormality.[11]

Aim of the present work was to verify the relationship of obesity and HbA1c with the resting level of SpO_2 in T2DM population. Since diabetes in associated with obesity, such information would be useful for clinicians to gauge whether decreased SpO_2 readings can be explained by increased weight.

METHOD

The study was conducted at Department of Physiology, GMC, Jammu, over a period of three months (March to May 2022). Total 100 subjects above 18 years of age diagnosed with T2DM and having BMI >30 was included. Pregnant females, patients with history of lung disease and/or heart disease were excluded from the study. Also, patients on medications which interfere with oxygen saturation were not included in the study. The research protocol was approved by the institutional ethics committee for human research vide IEC/GMCJ/2022/1053 dated 18/04/2022.

A written informed 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 cm using a wall mounted scale. BMI was calculated as weight (kilogram) divided by square of height in meter. 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.

Biochemical analysis

Fasting plasm glucose (FBG) was measured by hexokinase method in a fully automated analyzer (Siemens-dimension Rx L max).

Glycated hemoglobin (HbA1c%): 1 mL venous blood was withdrawn and measurement was done by HPLC technique in biochemistry lab.

SpO_2: Pulse-oximeter (Omron) was applied to the index finger of left hand after ensuring that nail polish was not applied. Mean of the two readings taken 10 min apart was recorded.

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 waist circumference on the SpO_2. A p value<0.05 was considered the statistically significant.

RESULTS

A total of 100 individuals participated in the study. The mean age of all participants was 50.99±10.5 years (range 27-78 years).

Table 1: Anthropometric parameters of study subjects.

Anthropometric Parameters	N	Percentage (%)
BMI (kg/m^2)		
<18.5 (Underweight)	1	1
18.5-24.99 (Normal BMI)	36	36
25-29.99 (Overweight)	38	38
≥30 (Obese)	25	25
Mean ± SD	27.03±4.63	
Range	18.05-37.13	
Height (cm)		
Mean ± SD	158.34±9.3	
Range	140-176	
Weight (kg)		
Mean ± SD	67.36±11.68	
Range	39-98	
Waist circumference (cm)		
Mean ± SD	93.59±11.2	
Range	70-117	
Hip circumference (cm)		
Mean ± SD	96.43±9.54	
Range	71-121	
WHR		
Mean ± SD	0.96±0.06	
Range	0.82-1.22	

Mean duration since diagnosis of T2DM of the subjects was 6.89±5.83 years.

Table 2: Biochemical parameters and SpO_2% of the study subjects.

Parameters	Mean ± SD	Range
FBG (mg/dL)	182.59±63.22	86-365
Random blood sugar (mg/dL)	253.5±89.14	96.8-470
HbA1C (%)	8.6±2.41	5-15
SpO_2 (%)	95.24±3.23	80-99

Subjects were divided into underweight, normal weight, overweight and obese on the basis of their BMI (Table 1 and 3).

No significant correlation was seen between body mass index (BMI) and SpO_2 measured by pulse oximeter.

Table 3: Association between SpO_2 and BMI.

SpO_2 (%)	<18.5 kg/m² (Underweight), (n=1)	18.5-24.99 kg/m² (Normal BMI), (n=36)	25-29.99 kg/m² (Overweight) (n=38)	≥30 kg/m² (Obese) (n=25)	Total	P value
Mean ± SD	97±0	94.53±3.34	95.53±2.73	95.76±3.73	95.24±3.23	0.397

DISCUSSION

Excess adiposity is associated with a chronic state of vascular inflammation, with increased levels of tumor necrosis factor- alpha (TNFα). Deposits of fat around arterioles may be involved in local TNFα signaling, resulting in impaired perfusion and insulin resistance.[11]

Individuals with diabetes and obesity might be at higher risk for infection-related morbidity caused by altered defense mechanisms, including the effects of hyperglycemia and impaired tissue perfusion on injury and wound healing.[12-14] Studies conducted in rodent models have shown that hypoxia is involved in the pathogenesis of obesity-induced insulin resistance.[15]

Hypoxia in adipose tissue is an early event in the course of obesity and leads to dysregulated adipokine production, inflammation and the metabolic syndrome. This contributes to the progression of diabetes and its complications.[16]

However, in present study no significant correlation has been observed between BMI and SpO_2. Contrary to our findings, Garg et al concluded that obesity strongly contributes to decrease in oxygen saturation.[6] Similarly negative correlation between SpO_2 and BMI was seen by Littleton and Kabon respectively.[4,5]

The ordered secondary structure of hemoglobin, as examined by FTIR-fourier transform infrared spectroscopy, was significantly altered in diabetics with persistent hyperglycemia.[17]

No significant correlation between HbA1c and SpO_2 has been observed in present study. Study by Garg et al found obesity to be a strong independent contributor to reduction in oxygen carrying capacity in ambulatory T2DM subjects but there was no effect of glycated Hb on SpO_2 in the same population.[6]

Glycosylation of hemoglobin alters its structure and function which tend 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.[19] In the present study mean glycosylated hemoglobin was 8.6±2.41%. This is quite similar to the value reported by Hammad et al in the obesity cohort but differs from HbA1c 8.2±2.5% reported by Hassan in obese diabetics.

The inconsistent relation between BMI and HbA1c on one hand and BMI and SpO_2 signifies those further studies need to be conducted to ascertain the role of obesity and HbA1c on oxygen saturation in diabetic patients.

Limitation

Some of the values obtained using the pulse oximeter could have been compared with direct arterial blood sample measurements to verify results at random.

A bigger study sample with more representation from obese diabetics will provide a more useful result.

CONCLUSION

General obesity is a risk indicator for developing diabetes and impaired glucose regulation. This study concludes that oxygen saturation level in diabetic patients is independent of the effects of BMI and HbA1c.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Pi-Sunyer X. CV-behnaz Toluei. Postgr Med. 2010;121(6):21-33.
2. Kopelman PG. Obesity as a medical problem. Nature. 2000;404(6778):635-43.
3. Nuttall FQ. Body mass index: obesity, BMI, and health: a critical review. Nutrition today. 2015;50(3):117.
4. Littleton SW, Tulaimat A. The effects of obesity on lung volumes and oxygenation. Respir Med. 2017;124:15-20.
5. Kabon B, Nagele A, Reddy D, Eagon C, Fleshman JW, Sessler DI, Kurz A. Obesity decreases perioperative tissue oxygenation. J Am Society Anesthesiologists. 2004;100(2):274-80.
6. Garg S, Gupta S, Mobeen S, Venkata S. Diabetes & Metabolic Syndrome: Clinical Research & Reviews Effect of obesity and glycated hemoglobin on oxygen saturation in ambulatory type 2 diabetic individuals: A pilot study. Diabetes Metab Syndr Clin Res Rev. 2016;10-3.
7. Shanthi B, Revathy C, Devi AJ. Effect of iron deficiency on glycation of haemoglobin in nondiabetics. Journal of clinical and diagnostic research: JCDR. 2013;7(1):15.
8. Sherwani SI, Khan HA, Ekhzaimy A, Masood A, Sakharkar MK. Significance of HbA1c test in

diagnosis and prognosis of diabetic patients. Biomark Insights. 2016;11:95-104.
9. Forbes JM, Cooper ME. Mechanisms of diabetic complications. Physiol Rev. 2013;93(1):137-88.
10. Diabetes DOF. Diagnosis and classification of diabetes mellitus. Diabetes Care. 2007;30(1).
11. Levy BI, Schiffrin EL, Mourad J, Agostini D, Vicaut E, Safar ME et al. Impaired Tissue Perfusion A Pathology Common to Hypertension, Obesity and Diabetes Mellitus. Diabetes Care. 2008;968-76.
12. Bertoni AG, Saydah S, Brancati FL. Diabetes and the risk of infection-related mortality in the US. Diabetes Care. 2001;24(6):1044-9.
13. Huttunen R, Syrjänen J. Obesity and the risk and outcome of infection. Int J Obesity. 2013;37(3):333-40.
14. Falagas ME, Kompoti M. Obesity and infection. Lancet infectious diseases. 2006;6(7):438-46.
15. Cifarelli V, Beeman SC, Smith GI, Yoshino J, Morozov D, Beals JW et al. Decreased adipose tissue oxygenation associates with insulin resistance in individuals with obesity. J Clin Investigation. 2020;130(12):6688-99.
16. Catrina SB, Zheng X. Hypoxia and hypoxia-inducible factors in diabetes and its complications. Diabetologia. 2021;64(4):709-16.
17. Ye S, Ruan P, Yong J. The impact of the HbA1c level of type 2 diabetics on the structure of haemoglobin. Sci Rep. 2016;6:33352.
18. Zavorsky GS, Murias JM, Kim DJ, Gow J, Sylvestre JL, Christou N V. Waist-to-hip ratio is associated with pulmonary gas exhange in the morbidly obese. Chest. 2007;131(2):362-7.
19. Pu LJ, Shen Y, Lu L, Zhang RY, Zhang Q, Shen WF. Increased blood glycohemoglobin A1c levels lead to overestimation of arterial oxygen saturation by pulse oximetry in patients with type 2 diabetes. Cardiovasc Diabetol. 2012;11:110.
20. Hammad M A, Syed Sulaiman S A, Aziz N A, Elsayed T M, Mohamed Noor D A. PSY7-Impact of obesity on glycated hemoglobin control among patients with type 2 diabetes mellitus. Value in Health. 2018;21:S436-7.
21. Fawzi H, Hussein AM, Ahmed HJ, Mitham ZS. Study of Glycated Hemoglobin (Hba1c) in Obese Diabetics Patients and Non-Obese Diabetics Patients. Int J Med Res Prof. 2016;2(1):61-4.

Cite this article as: Chouhan N, Manhas M, Kotwal S, Majid M, Mrityunjay. Obesity and glycated hemoglobin: is there a relation with oxygen saturation in type 2 diabetes mellitus? Int J Res Med Sci 2022;10:1904-7.