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THE RELATIONSHIP BETWEEN VISCERAL ADIPOSITY, HEMATOLOGICAL PARAMETERS, AND GLUCOSE PARAMETERS IN NON-DIABETIC ADULT MALES.

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ABSTRACT

BACKGROUND: Visceral obesity, which is defined as extra adipose tissue accumulation around organs present in the abdominal cavity is also called central/abdominal obesity and glucose parameters with visceral adiposity among obese/non-obese healthy adult males aged between 30-50 years. **METHODS**: A total of 118 adult males took part in this case-control study by the random selection method, Visceral obesity was obtained by the BIA method, and body composition was assessed by WHR (waist to hip ratio), and BMI (body mass index). Hematological parameters including complete cell count, hemoglobin & derivatives, glycemic parameters serum HbA1c and Random blood sugar were done. This study used independent t-tests, chi-square testing, Pearson correlation, and regression analysis, with an alpha threshold of 0.05 being considered significant. **RESULTS**: Comparative hematological parameters between obese and non-obese show great diversity, mean hemoglobin in the obese group is 14.37 ± 1.0, and in the non-obese is 15.1 ± 1.48 along with significant variation in hemoglobin derivatives and RBC count In Leukocyte count mean neutrophils which are 59.08 ± 8.11 shows the significant difference when compared with an opposite group which is 55.52 ± 7.77. Hb is negatively correlated with visceral obesity in the control group (P = 0.03, r = -0.19) HCT (P = .006, r = - 0.2511), RBC, MCV, MCH, and MCHC were also negatively correlated. Among White blood cells neutrophils and lymphocytes were positively correlated with VA (P = 0.54, r = 0.05), Platelet (P =0.61, r =-0.046), and erythrocyte sedimentation rate (P = 0.0165, r =0.2204). A significant positive correlation was also shown between Hba1c with visceral obesity, (P = .0001, r = 0.51). In regression analysis, the similarly negative coefficients -.048, -.223, -.118, and -18812.9 for Hb, HCT, MCH, and PLT show that these blood measurements fall as visceral fat levels rise and increased visceral fat positively predicted increased HbA1c with coefficient 0.61. **CONCLUSION:** Increased visceral fat regardless of age can lead to disturbed blood parameters and impairment in glucose metabolism in non-diabetic apparently healthy adults. The study's limitations, however, are the limited sample size and potential confounding variables that were not considered.

Keywords: Visceral Obesity, Glucose Metabolism, Hematology, Body Composition, Bioimpedance Analysis.

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INTRODUCTION

Visceral obesity, which is defined as extra adipose tissue accumulation around organs present in the abdominal cavity is also called central/abdominal obesity[1]. Visceral fat generally consists of mesenteric and omental fat, retroperitoneal fat deposits, and fat present on the ventral surface of the kidneys [2]. It has unique characteristics involved in normal as well as pathological processes in the human body. Visceral adipose tissue possesses different types of cells among them adipocytes plays critical function in protein production, and hormones such as adiponectin,[3] leptin, interleukin 6, and tumor necrosis factor, however, leptin and adiponectin are of great importance[4]. Interestingly increased visceral fat accumulation beyond normal limits is inversely proportional to biomolecules secreted by VAT for instance the circulating levels of plasma adiponectin are inversely correlated with the proportion of VAT and increased VAT is associated with multiple medical pathologies such as metabolic syndrome,[5] various carcinomas such as breast, colorectal, and prostate cancers and cardiovascular disease[6] Increased visceral fat deposition is responsible for inducing insulin resistance, and insulin resistance plays a major in the development of diabetes[7,8]. Visceral adiposity is also linked with altered lipid metabolism and impaired blood pressure[9]. Deregulation in the production of adipokines and cytokines from adipose tissue mainly give rise in the development of obesity linked disorders[10]. Moreover it is greatly associated with frequent hospital visits and extended hospital stays due to increased morbidity, high chance to get infections, complications, and elevated mortality rate in hospital, Nonalcoholic fatty liver disease[NAFLD], is also linked to central obesity [11]. Studies show, that visceral obesity is more prevalent in males than females across the world additionally in both genders fat percentage gradually increases with increasing age[12] It is also reported that obese Asians possess more VAT with a low/normal BMI as compared to western people so the VAT evaluation among Asians is of much more importance[13]. Pakistan among Asia has exhibited an "Asian Indian phenotype" in its population, it is a unique term that represents a high-risk ethnic group of Indian origin people that possess relatively lower prevalence of generalized obesity but have greater tendency for abdominal /central obesity[14] . Pakistan is one among the ten countries, which consist of approximately half of the 693 million people with obesity in the world[15]. Asian people when compared with population of other ethnic origin reported more visceral adipose tissue deposition but at lower BMI values in multiple studies. This could be the reason for Asians being more prone to Type 2 DM at comparatively lower BMI value than whites[16]. Insulin resistance is among the major health consequence of abdominal obesity and According to IDF by the year 2025 Pakistan is expected to reach 5th position among the countries which have prevalent diabetic population with every 4th individual will be effected with this multi systemic disease[17,18]. In Pakistan, the role of body composition in adult populations has been thoroughly researched. but no or very less research is carried out specifically on visceral adiposity and its association with blood parameters and glucose metabolism. To our knowledge, no study has investigated these two connected variables associated with VFA in a non-diabetic group.

METHODOLOGY

Recruitment of study participants:

Sample size was calculated by using two mean formula, that was 152 with attrition rate of 10%, a total of 162 healthy adult males were randomly selected initially, working in various

private banks of Qasim Abad district, from July 2020 to September 2020. The selected participants were healthy men aged 30-50 years with no known diseases and were not on any kind of medication, initially fulfilling the selection criteria. Of these, 09 participants were excluded as they had consumed water and meals during the last hour, and 15 participants were excluded due to recent illness, co-morbidities, and taking supplements. Finally, 148 eligible participants were invited for the study, among whom 23 subjects withdrew from the study prior to blood sampling. However, 125 participants filled the consent form for study. They filled in the questionnaires and provided blood samples, resulting in a response rate of 86.4%. After obtaining the blood analysis results, seven participants were not included in analysis due to HbA1c in diabetic ranges and abnormally high ESR values. In total, 118 adult men out of the 146 invited were included in analysis.

Fat analysis: visceral fat and total body fat were calculated by using Omron fat monitor **BF508.** BIA measurements were obtained by using a tetra polar (4 electrodes) technique; participants who consumed food or water in the last hour before fat analysis were excluded. Results were recorded as total body fat, visceral fat percentage, BMI, and weight. Visceral Fat % more than /9% was considered high as per criteria and body fat more than % was considered high.

Laboratory investigation

Complete blood count

Complete blood was performed to get the picture of blood components, minimum 2 cubic centimeter (CC) of blood was drawn, and test was performed by using automated blood analyzer sysmax 505.

Serum hba1c

Serum Hba1c record was obtained by kit method, by utilizing commercially available kit of COBAS.

Erythrocyte sedimentation rate (ESR)

Erythrocyte sedimentation rate was done by Westergren method [19],2 ml of venous blood was collecting in a tube having 0.5ml of sodium citrate, then blood was transferred into Westergren-Katz tube up to the mark of 2mm.The tube was kept at normal room temperature, straight in a standing position for 1 hour, results were recorded after one hour.

Random blood sugar

Random blood glucose levels were obtained by using glucometer ACCU-CHECK Performa.

STATISTICAL ANALYSIS.

Data was analyzed by using SPSS software version26. Descriptive analysis was done, rate and frequency were determined for visceral obesity and total body fat, whereas average, standard deviation, and standard error of mean (SEM) was derived for continuous variables. An independent T test was applied to observe the level of difference between means of hematological parameters of both groups. Pearson Chi Square Test was carried out to observe association of anemia and visceral obesity, alpha <0.5 is considered significant. Correlation was applied to observe the relationship between variables. Simple linear regression was conducted to examine the relationship between the visceral fat and hemoglobin, HCT, MCH, platelets and Hba1c, alpha <0.05 considered significance.

RESULTS:

Baseline characteristics of participants are provided in **table 01**, among 118 participants 60 (50.8%) are obese and 58 (49.2%) as non-obese, the mean age of the obese group (38.18 years, SD=6.6) is slightly higher than the mean age of the non-obese group (35.29 years, SD=5.5). In comparison to the non-obese group (4.98%, SD=2.2), the obese group's mean visceral fat percentage is significantly higher (14.52%, SD=3.1). Like this, the mean percentage of total body fat in the obese group (33.12%, SD=4.3) is also significantly higher than the non-obese group (18.35%, SD=5.9).

Table 01. Characteristics of study participants (n=118)

Visceral obesity	Frequency n (%)
Non-obese	58 (49.2%)
Obese	60(50.8%)
Age	means (SD)
Non-obese	35.29(5.5)
Obese	38.18(6.6)
Visceral fat	**means (SD)**
Non-obese	4.98(2.2)
Obese	14.52(3.1)
Total body Fat	**means (SD)**
Non-obese	18.35(5.9)
Obese	33.12(4.3)

Fig.1 Average visceral fat % in obese and non-obese group (n=118).

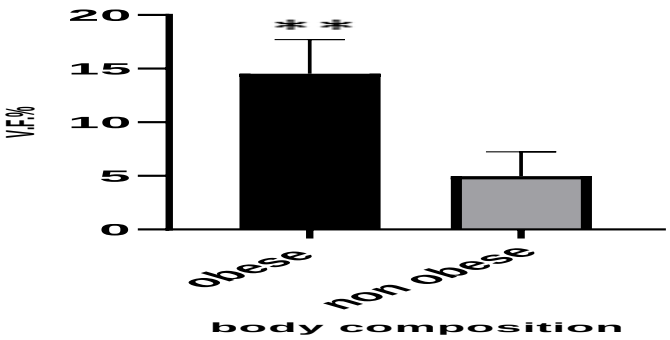

Table 02: Comparison of hematological and glucose parameters of obese and non-obese groups.

Blood parameters	Obese	Non-obese	p-Value
Hb	14.37±1.0	15.1±1.48	0.0022***
HCT (%)	45.56±5.29	48.31±4.21	0.0023*
RBCs(million/mm)	5.67±0.68	5.71±0.53	0.7542
MCV (µm3)	81.17±9.66	84.71±9.65	0.0487*
MCH (pg)	25.57±3.57	27.15±2.92	0.0101*
MCHC (%)	31.14±1.81	31.57±2.38	0.2801
WBCs (per mm3)	8.38±1.61	8.70±2.87	0.46
Neutrophils (%)	59.08±8.11	55.52±7.77	0.0166
Lymphocytes (%)	31.65±7.52	32.18±7.18	0.6948
Monocytes (%)	7.48±1.26	8.11±2.24	0.0624
Eosinophils (%)	2.748±1.86	3.24±1.6	0.129
Basophils (%)	0.496±0.27	0.548±0.39	0.4089
Platelets	262015±63339	274362±66489	0.3037
HbA1c	5.67±0.61	4.88±0.43	0.0001***
RBS	125.3±10.4	124.2±11.00	0.5828

Hb=hemoglobin, HCT=hematocrit, RBC=red blood cell, MCV=mean corpuscular volume, MCH=mean corpuscular hemoglobin, MCHC=mean corpuscular hemoglobin concentration, WBC=white blood, cell, RBS= random blood sugar, *Results are statistically significant.

Table 02 shows comparison of hematological and glucose parameters among both groups, the obese group's hemoglobin (Hb) level was substantially lower than that of the non-obese group (15.1 1.48 g/dL). With a p-value of 0.0023, the hematocrit (HCT) level was similarly substantially lower in the obese group (45.56 5.29%) than in the non-obese group (48.31 4.21%). With a p-value of 0.0487, the mean corpuscular volume (MCV) in the obese group was substantially smaller than that in the non-obesity group (84.71 9.65 m3) (81.17 9.66 m3). The mean corpuscular hemoglobin (MCH), which had a p-value of 0.0101, was also substantially lower in the obese group (25.57 3.57 pg) than in the non-obese group (27.15 2.92 pg). With a p-value of 0.0166, the proportion of neutrophils was significantly greater in the obese group (59.08 8.11%) than in the non-obese group (55.52 7.77%). With a p-value of 0.0001, the obese group's HbA1c level was considerably higher (5.67 0.61%) than the non-obesity group's (4.88 0.43%). Red blood cells, mean corpuscular hemoglobin concentration, white blood cells, lymphocytes, monocytes, eosinophils, basophils, and platelets were among the other blood parameters that did not differ significantly between the two groups.

Table 03: Frequency of pre-diabetic & normal HbA1c range in obese &non obese subjects

Body composition	Normal HbA1c	Pre-diabetic	
	< 5.7%	(HbA1c5.7%to6.4)	
obese (n=60)	42(70%)	18(30%)	
Non-obese (n=58)	56(96.55%)	02(3.44%)	

The distribution of body composition and HbA1c levels are displayed in table **03**. Based on body composition, there are two groups: obese (n=60) and non-obese (n=58). Two categories of HbA1c values have been established: normal (5.7%) and pre-diabetic (5.7% to 6.4%), 30% of the obese group had pre-diabetic levels (5.7% to 6.4%), while 70% had normal HbA1c levels (5.7%). Only 3.44% of the non-obese group had pre-diabetic levels of HbA1c, while 96.55% of them had normal levels.

Table 4: Association of Anemia with Obesity

Body composition	**Anemic**	**Non-Anemic**	**X2(df)**	**LR**	**O. R**	**P-value**
	Hb < 13mg/dl	13mg/dl or more				
obese (n=60)	7(11.7%)	53(88.3%)	0.30(1)	0.301	7.48	0.5
Non-obese **(n=58)**	5(8.6%)	53(91.4%)				

L. R= Likelihood ratio, O. R=odd ratio.

Association of Anemia was found between obese and non-obese individuals and presented in table no.3, 7 (11.7%) participants in obese group shown anemic status that is HbA1c less than 13mg/dl and 5(8.6%) participants in non-obese group, however the difference is not statistically significant, p value .0.05, alternative hypothesis is rejected.

Table 5: Pearson's correlation of visceral fat with blood & glucose parameters in obese and non-obese participants.

Parameter	OBESE (60) R P-VALUE	NON-OBESE (58) R	P-VALUE
Hb	-0.19	.03*	0.4	.40
HCT (%)	-0.25	.006**	.06	.96
RBCs (million/mm3)	-0.00	.99	.21	.10
MCV (µm3)	-0.20	.02*	.16	.23
MCH (pg)	-0.19	.03*	-.01	.90
MCHC (%)	-0.08	.37	.07	.59
WBCs (per mm3)	-0.08	.35	-.27	.03*
Neutrophils (%)	0.3	.01*	-.18	.17
Lymphocytes (%)	0.09	.29	.15	.24
Monocytes (%)	-0.12	.16	.17	.59
Eosinophils (%)	-0.09	.28	.13	.30
Basophils (%)	-0.12	.17	-.06	.63
Platelets	-0.04	.61	-.39	.002*
ESR	0.22	.01*	-.10	.45
HbA1c	0.51	.0001***	0.12	.35
RBS	0.3	.01*	-.04	.72

n= total number of participants, R=correlation coefficient*Results are statistically significant.

A correlation coefficient between HB, HCT, MCV and MCH suggests a moderate to weak negative correlation between visceral obesity and hemoglobin/HCT, in obese participants but the p-value less than 0.05 tells that there is a statistically significant negative correlation between the two variables. The p-value suggests that the probability of obtaining such a correlation coefficient by chance is less than 0.05 (assuming a significance level of 0.05), which is considered statistically significant when comparing with the other group. This means that as visceral obesity increases, HCT tends to decrease. Visceral obesity and neutrophils have a 0.3 correlation, which indicates a somewhat good link between the two factors. However, the significance level (p-value) of 0.01 suggests that the relationship is statistically significant because it is extremely unlikely that such a correlation would be observed by chance. As a result, we can say that there is proof that visceral adiposity and neutrophils are significantly positively correlated. Visceral obesity and ESR both have p-values of 0.01, which is less than 0.05 and hence statistically significant, as are visceral obesity and random blood sugar. Visceral obesity and HbA1c had p-values that are much smaller, 0.0001, indicating a very high level of statistical significance. According to these findings, visceral obesity and HbA1c show a statistically significant positive link, while visceral obesity and random blood sugar show a statistically significant positive correlation. Visceral obesity and ESR also show a statistically significant positive link, however this one is less strong than the other two.

Table 06. Regression analysis of visceral fat as a predictor variable for Hb, HCT, MCH, PLT AND HbA1c.

Dependent Variable	Independent variable	Coefficient	S.E	T value	95% CI		P value
					Lower bound	Upper bound	
Hb	Visceral fat	-.048	.022	-2.14	-.093	-.004	0.03*
HCT	Visceral fat	-.223	.080	-2.76	-.382	-.063	0.04*
MCH	Visceral fat	-.118	.55	-2.15	-.228	-.009	0.03*
PLT	Visceral fat	-18812.9	8698.2	-2.16	-360.8	-158.0	0.03*
HBA1C	Visceral fat	0.61	0.06	9.71	0.48	0.73	0.01*

The findings of a regression analysis are reported in table 06, in which visceral fat served as the independent variable and Hb, HCT, MCH, PLT, and HBA1C served as the dependent variables are shown in table 06. The coefficients in the table show, while maintaining all other variables constant, the change in the dependent variable that results from a one-unit change in the independent variable. According to the findings, visceral fat significantly predicts levels of Hb, HCT, MCH, PLT, and HBA1C. The negative coefficients for Hb, HCT, MCH, and PLT show that these blood measurements fall as visceral fat increases. As visceral fat increases, however, HBA1C also rises, as shown by the positive HBA1C coefficient. The coefficient estimate's level of statistical significance is shown by the p-value. At the 95% confidence level, a p-value of less than.05 implies that the coefficient is statistically significant.

DISCUSSION

In total118 participants were included, among them 60(50. 84%) participants were found obese (having visceral fat more than 9% and 58(49.15%) were categorized as non-obese possess visceral fat less than 9%. Comparative hematological parameters between obese and non-obese shows great variation, mean HB in obese group is on lower side as shown in table 1, this notion is also supported by the earlier researches [20].Red blood cell count, MCV &MCH above mentioned blood parameters shows significant differences when compared with controls ,these results are further supported by other studies.in case of WBC count mean neutrophils shows significant difference , this phenomenon is also reported by the study [21].Increased leukocyte count and specifically neutrophils could be associated with the state of mild chronic inflammation possibly caused by the obesity, which is further confirmed by Purdy et al[22. 23] Decreased mean platelet count was observed in obese participants possibly due to platelet aggregation, which is potentially associated with increased leptin levels in obesity and platelets has shown high number of leptin receptors on their surface which ultimately leads to aggregation, this phenomenon is referred as hyperleptinemia, so as reported earlier [24]. Mean Erythrocyte sedimentation rate (ESR) of obese group is observed higher than controls, this finding is supported by the recent research carried out by Alendo et al [25]. Current results shown increased mean RBS in obese population, this is in consistent with the study by Anuradha [26] suggest that with increased body fat and BMI, there will be elevated RBG levels. Similar results were observed in Jammu and Kashmir, [27]showing increased Triglycerides and RBGs have positive relation to obesity and cardiovascular disorders. On the contrary a research carried out at Africa [28] shows no relation of obesity and RBG in men.HbA1c is observed higher in obese which coincide with another study by Bower et al. [29]showing that total fat of the body and trunk fat, is closely linked to increased HbA1c.Another research showing relationship between BMI with glycemic control concludes that obesity is related to poor glycemic control which agrees to the findings of this study [30]. These results are in consistent with the hypothesis presented earlier that certain proinflammatory cytokines secreted by VAT could contribute to impaired glucose metabolism and insulin resistance [31] Goel et al [32] suggest that viscerally obese men shows markedly elevated risk of getting diabetes when compared to lean people. In present study the Mean hemoglobin in obese individuals is on the lower side although not considered as anemic but showed relatively low average when compared with controls ,it could be possibly due to low levels of body iron stores, Iron deficiency (ID) either with anemia or without iron deficiency anemia is prevalent in obese population and certain précised mechanisms are observed to be take part in its pathogenesis predominantly a condition of low grade subclinical inflammation.[33]because obesity often leads a person to anemia which is known as Inflammation associated anemia (AI also named as anemia of chronic disease) in which rarely Hb <8mg/dl, mainly occurs as a result in the regulation of iron metabolism primarily due to Hepcidin. Hepcidin is a small, cysteine-rich peptide hormone produced by hepatocytes in liver,[34] it is a key regulator of body iron which plays a crucial role in the

regulation of iron, Hepcidin was observed in higher concentration in overweight and obese individuals, it works mainly by causing subclinical mild inflammation , as a result iron absorption of iron from gut reduced[35]. According to the findings of this linear regression analysis there may be a connection between certain blood measurements and visceral fat. In particular, the negative coefficients for Hb, HCT, MCH, and PLT show that these blood measurements fall as visceral fat levels rise. This shows that elevated visceral fat may be linked to decreased quantities of platelets and red blood cells [36] which can have harmful effects on one's health. As visceral fat increases, however, HBA1C also rises, as shown by the positive HBA1C coefficient. HBA1C is a test that is frequently used to identify and track diabetes. It measures the long-term blood sugar control of a patient. Because visceral fat and HBA1C are positively correlated, having more visceral fat may result in higher blood sugar levels and a higher risk of developing diabetes[37]. The new thing observed in this study is less difference is present in between the mean RBS of both groups, on the contrary the significant differences were observed in mean hba1c of both groups , perhaps it could be due to less hemoglobin found in obese group as compared to lean, as is earlier reported that Haba1c is not a transparent and a clear diagnostic test in people with anemia or having low levels of hemoglobin , however various types of anemia are linked with minimizing HbA1c results ,but iron deficiency has been shown to move HbA1c a bit upward. The precise mechanism through which IDA disturbs HbA1c levels, is still remains unclear,[38] Another study conducted on Korean adults reveals the same phenomenon that hemoglobin concentration affects hba1c in non-anemic adults [39]. Further studies [37] reported that , a decrease in the hemoglobin concentration in non-diabetic people might lead to an enhanced glycation fraction of hemoglobin, however it could be further confirmed by performing other glucose test. However, the participants included in our study were not diabetic and the difference in mean age group is not much, despite, a big number of participants among obese group shows hba1c in pre diabetic category (> than 5.7 acc.to WHO) thus it is clear that obese showing more odds for developing diabetes in future.

CONCLUSION: Visceral fat associated with inflammatory parameters, which significantly leads to impaired glucose metabolism, and deranged blood profile, Estimation of HbA1c levels should not use as an only diagnostic tool for estimation of blood glucose in obese, more précised techniques should be included in diagnostic protocol like Glycated Albumin.

RECOMMENDATIONS:

Large studies are needed with many resources which could address the Hba1c effects especially the mechanism behind elevated Hba1c, glycation process in iron deficient state, and its effects on transparency of HbA1c.

ETHICS APPROVAL: The ERC gave ethical review approval.

CONSENT TO PARTICIPATE: written and verbal consent was taken from subjects and next of kin.

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AUTHORS' CONTRIBUTIONS:

All persons who meet authorship criteria are listed as authors, and all authors certify that they have participated in the work to take public responsibility of this manuscript. All authors read and approved the final manuscript.

CONFLICT OF INTEREST: No competing interest declared

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