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Obesity with cardiopulmonary wellbeing and oxidative stress in asymptomatic individuals with/without family background of T2DM

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Abstract

Introduction: Obesity and type 2 diabetes mellitus have emerged as new drivers of CVD risk as a result of our contemporary overconsumption of processed and energy-dense food products with low nutritional value, paired with our sedentary lifestyle. Increased oxidative stress is a well-known contributor to the onset and progression of diabetes, as well as its consequences.

Material and Methods: This is a prospective and observational study conducted at Department of Biochemistry. Students and apparently healthy cases in the age of 18-30 years who accompanied DM patients in were included for the study (n = 50). Individuals who are taking any medications for a health issue that prevents them from conducting sub-maximal exercise, as well as those who engage in regular physical activity, yoga, or other biofeedback, were excluded from the research. For the control group (n = 50), age and gender matched seemingly healthy adults with no family history of diabetes were recruited.

Results: The cardiovascular parameters of controls and cases individuals are shown and Heart rate ($p < 0.001$), blood pressure (SBP $p < 0.001$, DBP $p < 0.001$, MAP $p < 0.001$)and rate pressure product (p 0.001) all showed statistically significant differences, but PP (< 0.135) did not. The body fat distribution, cardio respiratory fitness as determined by the Cooper 12-minute run test, and blood glucose readings of controls and patients. Body fat percentage ($p<0.001$), visceral fat ($p<0.001$), Cooper 12-minute run test ($p<0.001$), and FBS ($p<0.001$) were all significantly different. The differences in oxidative stress parameters across groups. Between controls and cases participants, there was a significant difference in TAOS ($p<0.000$) and MDA ($p<0.000$).

Conclusion: In our study, we found that both groups had similar age, height, and waist-hip ratio (WHR), but FDRDM had a considerably higher body mass index (BMI). Fasting percentage body fat and blood glucose levels were significantly higher, and the 12-minute walk distance was much shorter in FDRDM. Visceral fat levels were marginally elevated, but not statistically significant alteration during the early stages of illness significant differences in oxidative markers were observed among the subjects.

Keywords: Obesity, type 2 diabetes mellitus, oxidative stress

Introduction

Diabetes mellitus (DM) is a chronic condition that affects people all over the world. Diabetes is becoming more common all around the world at an alarming rate all throughout the world. [1].Diabetes mellitus (DM) is a metabolic condition defined by persistent hyperglycemia and a bnormal carbohydrate, lipid, and protein metabolism as a result of absolute or relative insulin insufficiency or both. Diabetes mellitus, particularly type 2 diabetes, is a major general medical problem that has reached epidemic proportions due to the disease's rapid spread over

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the globe. Type I and Type II diabetes are becoming more common. The number of diabetics globally is currently estimated to be approximately 347 million. Type II diabetes account for more than 90% of the population [2].

The loss of cell mass in Type 2 diabetes mellitus is also linked to glucose toxicity, which is mitigated by IL-1-induced apoptosis. Numerous investigations have emphasized the importance of the fiery component in the pathophysiology of Diabetes mellitus. Adipose tissue, which produces pro-inflammatory cytokines such as tumour necrosis factor alpha (TNF-), interleukin-6 (IL-6) and C Reactive Protein (CRP), plays a critical role. Hyperglycemia creates reactive oxygen species (ROS), which harm cells in a variety of ways. In diabetes mellitus, cell damage eventually leads to secondary problems [3].

Increased oxidative stress is a wellknown contributor to the onset and progression of diabetes, as well as its consequences [4]. The major reason for incorporating oxidative stress in diabetic s was hyperglycemia. The autoxidation of glucose and advanced glycation end products cause s an increase in oxidative stress. A variety of compounds known as advanced glycation end pr oducts are believed to have a significant role in diabetes and its effects [5]. There is a well-established link between diabetes mellitus and the development of macro and microvascular illness[6].

Obese people exhibit oxidative stress indicators include higher reactive oxygen species (ROS) [7] and weakened antioxidant defense, which is linked to decreased antioxidant enzymes [8]. Systemic inflammation, endothelial cell proliferation and death, and enhanced vasoconstriction are all linked to oxidative stress, making it a significant contributor to endothelial dysfunction. Oxidative stress is a phrase used to describe cellular damage induced by an imbalance between pro-oxidants like reactive oxygen species (ROS) and antioxidants like reactive nitrogen species (RNS). ROS are oxidizing agents that are produced during cellular metabolism when oxygen is chemically reduced to form unstable free radicals with an unpaired electron [9]. ROS are required for a variety of physiological tasks, including gene expression, cellular development, infection defence, and endothelial function modulation[9-11].

To our knowledge, so far there is no proper evidence about the relationship between excessive visceral fat and cardio-respiratory fitness, oxidative stress in first-degree relatives of type 2 diabetes mellitus (cases). As a result, this study looked at the relationship between visceral fat and cardiopulmonary fitness, oxidative stress in healthy people with and without a family history of Type 2 diabetes.

Materials &Methods

Study participants were given written and informed consent after receiving approval from the institutional ethics committee. Experiments were conducted at, and, India, at the Department of Biochemistry research lab. The sample size N=100 which was calculated based on the expectation of a 0.56 correlation between visceral fat composition and oxidative stress and inflammation.

Students and apparently healthy cases in the age of 18-30 years who accompanied DM patients in were included for the study (n = 50). Individuals who are taking any medications for a health issue that prevents them from conducting sub-maximal exercise, as well as those who engage in regular physical activity, yoga or other biofeedback, were excluded from the research. For the control group (n = 50), age and gender matched seemingly healthy adults with no family history of diabetes were recruited.

Procedure: One day prior to recording, participants were asked to get enough sleep and abstain from caffeinated beverages, exercise, alcohol, and nicotine for 24 hours. After overnight fasting, participants were requested to report to the Department of Biochemistry in the morning. As soon as they reported to the lab, a fasting venous blood sample (5 mL) was taken in sodium fluoride tubes from the median cubital vein for biochemical examination of lipid profile, inflammatory markers and oxidative stress indicators. They were then shown about the research facility and told about the operations that will be carried out the next day.

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Anthropometric and basal characteristics (Height, Weight, BMI etc. were recorded after the participants had emptied their bladders. Blood was obtained through vein puncture, allowed to clot, Semi auto analyzer (ERBA CHEM 5x) was used to measure fasting blood glucose and calorimetry was used to study oxidative stress markers (ALERE AM 2100). Observations were noted and compared with students test using SPSS software version 17.

Results

Table 1: Shows the baseline and anthropometric parameters of controls and cases subjects

Parameter	Mean ± SD		p value
	Controls Without family history of type 2 diabetes	Cases Withfamily history of type 2 diabetes	
Age (years)	20.26 ± 1.89	20.90 ± 1.80	0.446
Height (cms)	165.04 ± 8.01	162.00 ± 10.11	0.089
Weight (kg)	52.12 ± 9.68	67.10 ± 11.05	0.001
BMI (kg/m^2)	20.83 ± 2.35	25.56 ± 3.76	0.001
WHR	0.81 ± 0.07	0.81 ± 0.02	0.006

There was no significant difference in age (p 0.446) or height (p 0.089) of the study subjects, as indicated in Table 1 and Fig 1-5. Between controls and cases, there was a significant difference in weight (p< 0.001), BMI (p <0.001), and WHR (p <0.006) in table 1.

Table 2: The cardiovascular parameters of controls and cases

Parameter	Mean ± SD		p value
	Controls Without family history of type 2 diabetes	Cases Withfamily history of type 2 diabetes	
HR (bpm)	76.72± 3.24	87.26 ± 3.40	0.001
SBP (mmHg)	112.48 ± 4.26	122.20 ± 4.12	0.001
DBP (mmHg)	74.20 ± 3.40	82.45 ± 3.36	0.001
PP (mmHg)	38.28 ±3.85	37.76 ± 5.87	0.142
MAP(mmHg)	85.96 ± 3.30	97.37 ± 2.36	0.001
RPP	9046.32 ± 472.86	10905.88 ± 665.20	0.001

Individuals are shown and Heart rate (p <0.001), blood pressure (SBP p< 0.001, DBP p <0.001, MAP p <0.001), and rate pressure product (p 0.001) all showed statistically significant differences, but PP (< 0.135) did not in table 2.

Table 3: Show the body fat distribution, cardio respiratory fitness

Parameter	Mean ± SD		p value
	Controls Without family history of type 2 diabetes	Cases Withfamily history of type 2 diabetes	
FBS (mg/dl)	82.20 ± 4.39	86.62 ± 4.46	0.001
Visceral fat (%)	6.34 ± 0.52	8.52 ± 2.70	0.001
12 min run(meters)	2762.14 ± 81.46	2309.48 ± 234.21	0.001
Body fat (%)	23.15 ± 1.64	27.89 ± 2.76	0.001

In table 3, as determined by the Cooper 12 minute run test, and blood glucose readings of controls and patients. Body fat percentage (p<0.001), visceral fat (p<0.001), Cooper 12-minute run test (p<0.001), and FBS (p<0.001) were all significantly different.

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Table 4: The differences in oxidative stress parameters across groups

Parameter	Mean ± SD		p value
	Controls Without family history of type 2 diabetes	Cases Withfamily history of type 2 diabetes	
TAOS (mM)	1.34 ± 0.32	0.57± 0.36	0.001
MDA (mM)	6.02 ± 0.95	11.78 ± 9.32	0.001

The differences in oxidative stress parameters across groups. Between controls and cases participants, there was a significant difference in TAOS (p<0.000) and MDA (p<0.000).

Discussion

The prevention and treatment of diabetes mellitus is a serious public health issue that we are currently confronted with. Although there is no cure for diabetes, problems can be avoided or postponed by following a balanced diet, exercising regularly, taking oral medicines, and using insulin as necessary to regulate blood glucose levels. For a decrease in these consequences, adequate therapy aimed at early management of hyperglycemia is critical. The Diabetes Control and Complications Trial (DCCT) and the UK Prospective Diabetes Study both confirmed this (UKPDS) [15]. The findings of recent studies show that early intervention can improve diabetic outcomes [12].

In our research, we anticipated that having a family history of type 2 diabetes mellitus will lead to diabetes development in the future. According to the research protocol, we enrolled a case group (with a family history of type 2 diabetes) and a control group (without a family history of type 2 diabetes). Cardiovascular system parameters (HR, SBP, DBP, PP, MAP, RPP), FBS, visceral fat, and indicators of oxidative stress (TAOS, MDA).

In our study, we found that there was no statistically significant difference between the control group (without family history of type 2 diabetes) and the case group (with family history of type 2 diabetes) in the mean and standard deviation of age control group (20.62 ± 1.98 years) and case group (20.90 ± 1.80 years) (p=0.463). On the other hand, there was no statistically significant difference in height between 18-21 years in the control and case groups (166.87 ± 8.06 cms) and (165.69 ± 10.41 cms) respectively (p=0.334) and there was no statistically significant difference in height between 22-25 years in the control and case groups (167.75 ± 8.28 cms) and (161.67 ± 9.32 cms) respectively (p=0.66).(See Table 1) In a similar research, Jago R. *et al*., found no difference in age groups between the case and control groups [13]. In a similar research, Reas DL, *et al*., found no difference in BMI between the case and control groups [14].

In this study, there was no statistically significant difference in mean and standard deviation of heart rate in 18-21 years between the control group (80.34 ± 3.34 bpm) and the case group (88.45 ± 3.92 bpm) (p=0.57), as well as change in HR in 22-25 years between the control group (77.83 ± 2.86 bpm) and the case group (86.81 ± 2.94 bpm) (p=0.62) in the control group (77.83 ± (Table 2). Another study by Koizumi K *et al*.,found a significant increase in heart rate with a family history of type 2 diabetes [15]. There is a difference in heart rate between the case and control groups, similar to a research done by Nielsen *et al*.,[16] In contrast, a research published by Eckberg DL *et al*.,found no difference in heart rate between the case and control groups [17].

Hainsworth R *et al*.,back up our findings concerning an increase in heart rate in the case group. Exaggerated cardiovascular reactivity to stress is known to be a risk factor for cardiovascular diseases, whereas reduced reactivity is an indicator of fitness, according to the researchers [18].

In the Case Group, the mean percentage of body fat increased from 26.91 to 27.91±2.25, compared to 24.10 ± 1.66 in the control group (p=0.21), same body fat in 22-25 years. The case group had a higher mean percentage of 24.49 ± 2.62 than the control group.24.32 ± 1.66 = 24.32 ± 1.66 = 24.32 ± 1.66 = It shows that in the case group, body fat increased by 1.39 percent. Similar findings were also found by Ainsworth BE *et al*.,in a study population [19]. The majority of fat people despite being insulin resistant, individuals do not develop

hyperglycemia. In a similar research, Alahmadi MA *et al*.,found a difference in body fat between the case and control groups [20].

In the Case Group, the FBS between the ages of 18 and 21 years showed an increase in mean levels of 88.03± 4.95 mg/dl, compared to 84.29 ± 4.37 mg/dl in the control group (p=0.53), and FBS in the Case Group aged 22-25 years showed an increase in Mean levels of 87.29 ± 4.37 mg/dl, compared to 83.91 ± 4.74 mg/dl in the control group (p=74). In our study, the mean rise in blood glucose was 3.74 mg/dl in the case group versus 3.74 mg/dl in the control group. According to King P *et al*.,in a case control study, there was an increase in blood glucose levels [21]. In a research published by Koyama Y *et al*.,there was a difference in FBG between the case and control groups [22].

In our study, mean and standard deviation of 12 minute run test in control group (2767.0 ± 65.39 metres) and case group (2313.7 ± 179.32 meters) were statistically significant differences between both groups (p=0.007), and mean and standard deviation of 12 minute run test in control group (2734.2 ± 119.83 meters) and case group (2313.7 ± 179.32 meters) were statistically significant differences between both groups (p=0.007). According to Chatterjee S *et al*.,the case group had less run metres than the control group [23]. In a similar research, Fox EL et colleagues found no difference between the case and control groups [24].

In our study, mean and standard deviation of TAOS in 18- 21 years in control group (1.45 ± 0.34 mM) and case group (0.71 ± 0.46 mM) were statistically not significant differences between both groups (p=0.061), whereas TAOS in 22- 25 years in control group (1.37 ± 0.47 mM) and case group (0.45 ± 0.14 mM) were statistically significant differences between both groups (p=0.061). In the Case Group, MDA increased from 18 to 21 years old.11.05 ± 9.26 mM, compared to 6.14 ± 0.89 mM in the control group (p0.0001). MDA levels in the Case Group increased by 13.05 ± 9.34 mM above the control group's 5.74 ± 1.19 mM in 22- 25 years. Kashyap MK *et al*.,found similar results in the case and control groups [25]. According to Banarjee S *et al.'s* study, there is a difference in TAOS and MDA between the case and control groups [26]. In contrast, according to a study published by Kunwar A *et al*.,there is no difference in TAOS or MDA between the case and control groups [27]. In a research published by Yoshikawa T *et al*.,there was no difference in TAOS or MDA between the case and control groups [28].

Conclusion

Numerous studies show that dietary changes, such as vitamin D supplementation, can help to reduce oxidative stress; nevertheless, the relationship between obesity and physical activity has yet to be confirmed. The effects of these therapies are likely explained by a combination of metabolic, inflammatory, and cardiovascular processes. Best significantly, more mechanistic research is required to find the most effective intervention(s) for certain advantages. It appears that weight loss has a major impact on the reduction of oxidative stress. Obesity, inflammation, vascular function and diabetes are all linked to oxidative stress [29,30]. To reduce oxidative stress, appropriate lifestyle changes (e.g., exercise training, nutritional treatments) might be made. The development of specific treatment options for oxidative stress and illness can benefit from a better knowledge of the processes involved.

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