SJIF Impact Factor: 6.711

WORLD JOURNAL OF ADVANCE HEALTHCARE RESEARCH

ISSN: 2457-0400
Volume: 8.
Issue: 8
Page N. 9-18
Year: 2024

Original Article www.wjahr.com

CARDIOVASCULAR STUDY OF OXIDATIVE STRESS AND ECG WAVE PATTERN IN OBESE AND NON OBESE ADULT FEMALE IN EKPOMA, NIGERIA

[1*]Aigbiremolen A. A., [1]Edebiri O. E., [2]Akhile A. O., [3]Odigie O. M. and [4]Aloamaka C. P.

[1]Department of Human Physiology, Faculty of Basic Medical Sciences, College of Medicine, Ambrose Alli University, Ekpoma, Edo State, Nigeria.
[2]Department of Chemical Pathology, Faculty of Medical Laboratory Sciences, College of Medicine, Ambrose Alli University, Ekpoma, Edo State, Nigeria.
[3]Department of Human Physiology, Faculty of Basic Medical Sciences, Edo State University, Uzairue, Edo State, Nigeria.
[4]Department of Human Physiology, Faculty of Basic Medical Sciences, College of Health Sciences, Delta State University, Abraka, Delta State, Nigeria.

Article Received date: 23 May 2024 **Article Revised date:** 13 June 2024 **Article Accepted date:** 03 July 2024

***Corresponding Author: Aigbiremolen A. A.**
Department of Human Physiology, Faculty of Basic Medical Sciences, College of Medicine, Ambrose Alli University, Ekpoma, Edo State, Nigeria.

ABSTRACT
The study assessed oxidative stress biomarkers enzyme activities, side by side a recorded surface ECG on non obese and obese amongst female adult to determine changes in ECG patterns. Twenty (20) consenting adult female subjects were recruited from Ekpoma metropolitans, Edo State. Five milliliters (5 ml) of venous blood was drawn from consenting participants and placed in a lithium heparin sample bottles. Blood samples was spun in a bucket centrifuge at 2500 RPM (rounds per minute) for 10 minutes after which plasma was collected and stored frozen in plain sample bottles and was analyzed for oxidative stress biomarkers concentrations. Oxidative stress biomarker (catalase) activities in Obese and non obese adult females were significantly different $p<0.05$ and greater in obese. The mean value catalase of Obese was (84.99±1.235) and Non obese (34.82 ± 1.590). Superoxide Dismutase(u/l) 0.4240± 0.008327and 0.2010± 0.01810 significantly $p<0.05$ greater in obese. Glutathione peroxidase(u/ml) 7.509± 0.3769 and 3.389± 0.1967 significantly greater $p<0.05$ in obese. The overall findings shows that oxidative stress is prominent in obese as evident in the higher values of catalase, Superoxide Dismutase biomarker and Glutathione peroxidase enzyme activities compared to non obese.

INTRODUCTION
Obesity is a disease process in which excess body fat has accumulated to an extent that health may be adversely affected. According to WHO classification of body mass index(BMI) a person whose BMI is more than or equal to 30 Kg/m2 is obese and when BMI is between 18.5 to 24.99 then the person is considered normal (WHO, 2004). Obesity is the first wave of a defined cluster of non-communicable diseases called 'New World Syndrome's creating anenormous socio-economic and public health burden (Pednekar, 2008). It has a strong impact on cardiovascular changes which is manifested in electrocardiogram (ECG). Currently it is a serious public health problem with established cardiovascular co-morbidities and a major cause of sudden death in developed as well as developing countries (Prentice, 2006).

According to the National Family Health Survey-4 (NFHS-4) in 2015-16 conducted by Ministry of Health and Family Welfare(MOHFW) in India, the percentage of men and women aged 15–49 years who are obese are19% and 21% respectively. In a large prospective study 'Framingham Heart study there is evidence for inclusion of obesity as a major modifiable cardiovascular risk factor by American Heart Association and also sudden cardiac death has been reported 40 times higher in obese men and women (Eckel & Krauss, 1998).

Wang et al., and Seyfeli et al., have showed that obesity as apotential risk factor for atrial fibrillation (AF) and P–wave dispersion is highly specific in screening healthy obese individuals for the risk of cardiovascular diseases. It has been observed for more than last fifty years that obesity induces changes in the normal ECG pattern in young healthy adults (Frank et al., 2011). P- Wave depression (pd) is a measure of heterogeneity of atrial refractoriness and prolongation of Pd shows the intraatrial and interatrial non–uniform

conduction. Studies have shown that Pd prolongation is an independent risk factor for development of atrialfibrillation (Dilaveris et al., 2012).

Few studies have been conducted on effect of obesity on the duration and dispersion of P–wave and it is non-invasive and cost effective tool for early detection of patients who are at risk of cardiac arrhythmias and it is also important for developing country like India.

Epidemiological, clinical, and animal studies have reported the role of oxidative stress in the pathogenesis of obesity and its associated risk factors (Savini, et al., 2013). Oxidative stress could trigger obesity by stimulating the deposition of white adipose tissue (WAT) and altering food intake; both cell culture and animal studies have demonstrated that oxidative stress can cause an increase in preadipocyte proliferation, adipocyte differentiation, and the size of mature adipocytes (Furukawa et al., 2004).

Reactive oxygen species (ROS) have been found to be involved in the control of body weight by exerting different effects on hypothalamic neurons, which control satiety and hunger behavior. Obesity can also induce systemic oxidative stress through multiple biochemical mechanisms, such as superoxide generation from NADPH oxidases (NOX), oxidative phosphorylation, glyceraldehyde auto-oxidation, protein kinase C (PKC) activation, and polyol and hexosamine pathways (Savini, et al., 2013). Other factors that also contribute oxidative stress to obesity include hyperleptinemia, tissue dysfunction, low antioxidant defense, chronic inflammation, and postprandial ROS generation(Patel et al., 2007).

Oxidative Stress

Oxidative stress reflects an imbalance between the systemic manifestation of reactive oxygen species (ROS) and a biological system's ability to readily detoxify the reactive intermediates or to repair the resulting damage. Oxidative stress from oxidative metabolism causes base damage, as well as strand breaks in DNA.. Any perturbation in the balance in the level of antioxidants and the reactive species results in a physiological condition called "oxidative stress." Superoxide dismutase (SOD) is an enzyme found in all living cells.

Superoxide Dismutase

Superoxide dismutase helps break down potentially harmful oxygen molecules in cell which prevent damage to tissues. The lack in extracellular superoxide dismutase (SOD3, ecSOD) contributes to the development of hypertension. Diminished SOD3 activity has been linked to lung diseases such as Acute Respiratory Distress Syndrome (ARDS) or Chronic obstructive pulmonary disease (COPD). As a shot, superoxide dismutase is used for treating pain and swelling (inflammation) caused by osteoarthritis, sports injuries, and rheumatoid arthritis; a kidney condition called interstitial cystitis; gout; poisoning caused by a weed-killer called paraquat; cancer; and lung problems in newborns. Notably, epidemiological studies have convincingly shown that higher circulating concentrations of the antioxidant enzymes GPx, SOD, and catalase are associated with a significant reduction in the risk of coronary heart disease. This suggests that pharmacological strategies that upregulate these enzymes may exert a key protective role against atherosclerosis and cardiovascular disease. The different units of measurement for Glutathione Peroxidase, Superoxide Dismutase, Catalase are (U/mL, U/gHb, nmol/mg, or µmol/L) used to express the concentrations(Yog-Gyun et al., 19. The reference value Superoxide Dismutase antioxidant enzyme activity in man is between 0.12-0.33u/l (H. Younus H, 2018).

Glutathione Peroxidase

Glutathione Peroxidase (GPx) is a cytosolic enzyme that catalyzes the reduction of hydrogen peroxide to water and oxygen as well as catalyzing the reduction of peroxide radicals to alcohols and oxygen. Glutathione reductase is responsible for maintaining the supply of reduced glutathione; one of the most abundant reducing thiols in the majority of cells. In its reduced form, glutathione plays key roles in the cellular control of reactive oxygen species. Heterozygous deficiency of GPx-1 leads to endothelial dysfunction, possibly associated with increased oxidant stress, and to significant structural vascular and cardiac abnormalities. The different units of measurement for Glutathione Peroxidase, Superoxide Dismutase, Catalase are (U/mL, U/gHb, nmol/mg, or µmol/L) used to express the concentrations. The reference value antioxidant enzyme activity in man is between 2.65-4.80u/ml (Lavi et al., 2008). The current knowledge of the molecular determinants influencing the expression and function of GPx-1, with an emphasis on the role of GPx-1 in modulating cellular oxidant stress and redox-mediated signaling responses. Importantly, by regulating cellular hydroperoxides (and RNS), GPx-1 may protect against oxidative stress, but, in excess, GPx-1 may also have deleterious effects due to a lack of essential cellular oxidants (McClung et al., 2004; Handy et al., 2009) that result in a reductive stress characterized by a lack of oxidants and/or excess reducing equivalents(Ajasekaran et al., 2007). Although reductive stress may appear to be a new concept, it has been known for some time that lack of cellular oxidants can diminish cell growth responses. Newer evidence points to additional cellular and physiological effects caused by lack of cellular oxidants and accumulation of excess reducing equivalents, including changes in protein disulfide bond formation, diminished mitochondrial function, and decreased cellular metabolism.

Catalase

A catalase is one of the crucial antioxidant enzymes that mitigates oxidative stress to a considerable extent by destroying cellular hydrogen peroxide to produce water and oxygen. Deficiency or malfunction of catalase is

postulated to be related to the pathogenesis of many age-associated degenerative diseases like diabetes mellitus, hypertension, anemia, vitiligo, Alzheimer's disease, Parkinson's disease, bipolar disorder, cancer, and schizophrenia(Ankita et al.,2019).

ECG Abnormalities in Obesity

A number of ECG abnormalities may be associated with obesity of various causes. A left shift of the P, QRS and T axes, morphological deviation of the P wave, low QRS amplitude, flattening of T waves (mainly in inferolateral leads) and potentially prolonged QT and QTc intervals are present at a significantly higher rate in obese than in non-obese individuals. The prolongation of the QT and QTc intervals is caused by the heightened sympathetic activity characteristic of obesity that increases the reduced heart rate variability; all these elements have the potential to cause arrhythmia (Matthew, et al., 2008). Various arrhythmias occur more commonly in obese individuals, especially in those with co-existing sleep apnoea or left ventricular hypertrophy. Many ECG abnormalities are reversible, since they may change proportionately with reduced body weight, as was confirmed in a study demonstrating a reduction of the mild left shift of the P and QRS axes after weight loss. In obesity, ECG may change in many ways due to an elevation of the diaphragmatic level, left ventricular hypertrophy caused by increased cardiac output, the presence of epicardial and subcutaneous adipose tissue - functioning as electric insulation layers - as well as sleep apnoea/hypoventilation syndrome (Fraley, et al., 2005).Complete bundle branch blocks caused by elevated pressure are easy to recognise, but the diagnosis of, for instance, an incomplete right bundle branch block is often wrong. The assessment of right ventricular pressure elevation is even more difficult. Left ventricular hypertrophy is a significant predictor of cardiovascular morbidity and mortality(Fraley, et al., 2005).

Arrhythmias in Obesity

The risk of arrhythmias and sudden death is higher in obese individuals even without cardiac dysfunction. The Framingham Study has shown that the mortality rate is increased 6- to 12-fold in seriously obese men . QT prolongation was observed in 30% of the patients with impaired glucose tolerance in the NHANES III (Third National Health and Nutrition Examination Survey) (Medvegy, et al., 2011). The high blood glucose level increases vasomotor tone and ventricular instability, since it reduces the accessibility of nitrogen monoxide. In extremely obese patients, fatal arrhythmia and sudden cardiac death caused by the common dilated cardiomyopathy are not rare. Serious hypoglycaemic episodes requiring intervention occur with a frequency of 62-170 episodes/100 patient-years with the treatment of type 1 diabetes mellitus(Medvegy, et al., 2011), while in type 2 diabetes mellitus, the frequency of serious hypoglycaemic episodes may be nearly 73/100 patient-years. Enhanced sympathoadrenergic activity induced by hypoglycaemia results in tachycardia, feeling of stress and vasoconstriction. As a sign of this, the systolic blood pressure increases while the diastolic value tends to decrease. Previous reports have shown a relationship between hypoglycaemia and atrial fibrillation. However, the changes in repolarisation (prolongation of the QT interval) during hypoglycaemia have greater clinical significance. The significance of the prolongation of the QT interval during hypoglycaemia lies in the fact that it is an independent risk factor of sudden death and Torsade de pointes. The 'late potential' test is less frequently used, although a positive result indicates 'late' depolarisation induced by ischaemia or myocardial disorder and thereby susceptibility to arrhythmia (e.g. it yields positive results in 100% of extreme obesity cases) (Robinson, et al., 2003).

P wave

The sinoatrial node lies high in the wall of the right atrium and initiates atrial depolarisation, producing the P wave on the electrocardiogram. Although the atria are anatomically two distinct chambers, electrically they act almost as one. They have relatively little muscle and generate a single, small P wave. P wave amplitude rarely exceeds two and a half small squares (0.25 mV). The duration of the P wave should not exceed three small squares (0.12 s). Normal ECG values for waves and intervals are as follows: RR interval: 0.6-1.2 seconds. P wave: 80 milliseconds. PR interval: 120-200 milliseconds. Abnormal R–R intervals differ from sinus rhythm in their length and they represent disturbances of both technical and physiological origins and are present in almost all Holter ECG recordings. Physiological artifacts occur especially in patients suffering from different cardiovascular diseases.

The measurement is normally 0.12-0.20 seconds, or 3-5 small squares in duration. The second measurement is normally the width of the QRS which normally less than 3 small squares, or less than 0.12 seconds in duration. R-R interval means beat-to-beat intervals and beats-per-min (BPM). low RR means Bradypnea which is an abnormally slow breathing rate. The normal breathing rate for an adult is typically between 12 and 20 breaths per minute. The RR interval and heart rate (HR) are hyperbolically related.

Heart Rate Variability

Beat-to-beat variability in RR intervals is referred to as heart rate variability. It is determined by the interplay of the sympathetic and parasympathetic input. In normal subjects, age and heart rate are the major determinants of heart rate variability. The variability is reduced in patients with congestive heart failure, left ventricular dysfunction, coronary artery disease, and diabetic neuropathy(Ewing et al., 1985). In these patients the sympathovagal balance shifts toward sympathetic predominance. Therefore more blunted heart variability occurs concomitantly with an elevated heart rate (Pomeranz et al., 1985). Conversely, increased heart rate

variability by exercise training is attributed to an increase in parasympathetic tone(Pagani et al.,1986).

Q Wave duration

Small Q waves are present in the left precordial leads in more than 75 percent of normal subjects. They are seen most frequently in lead V6, less frequently in leads V5 and V4, and rarely in V3. Q waves in these leads are present more often in young subjects than in subjects older than 40 years. Q waves are likely to be present in more leads when the transitional zone is located on the right side of the precordium. The duration of the Q waves is 0.03 second or less. The amplitude usually is less than 0.2 mV, although it may reach 0.3 mV or even 0.4 mV. The deeper Q waves are seen more often in young adults. An amplitude of 0.4 mV or more may be encountered in teenagers. In the posterior leads V7-V9, Q wave duration of ≥0.03 second was seen in 20 percent of normal male subjects(Tetsuo *et al.,* 2004).

Q Waves amplitude

Q waves are normally seen in the inferior and left lateral precordial leads in pediatric patients. The duration of these Q waves is almost always less than 20 ms. The amplitude can be rather large (up to 14 mm), especially in infants. Q waves are usually absent in leads I and aVL in infants, and their presence is often suggestive of cardiac pathology. A deep (≥3 mm) and broad (≥30 ms) Q wave in leads I and aVL, especially when accompanied by absence of Q waves in the inferior leads, may suggest the diagnosis of anomalous origin of the left coronary artery from the pulmonary artery. Q waves in the right precordium are always pathologic and are commonly associated with right ventricular hypertrophy. Deep Q waves in the left lateral precordial leads are often seen with left ventricular hypertrophy of many etiologies. In the assessment of children and adolescents for familial hypertrophic cardiomyopathy, Q waves ≥3 mm in depth or ≥40 ms in duration in ≥2 leads other than lead V1, V2, or III have low sensitivity, but high specificity in the affected genetically tested children (Mithilesh et al., 2006).

ST segment

The ST segment is the interval between the end of the QRS complex (J point, or ST junction) and the beginning of the T wave. In the limb leads, the ST segment is isoelectric in about 75 percent of normal adults. ST segment elevation or depression up to 0.1 mV generally is considered within normal limits.

The ST segment is that portion of the ECG cycle from the end of the QRS complex to the beginning of the T wave. It represents the earliest phase of ventricular repolarization. Bęćkowski et al., 2021). The normal ST segment is usually *isoelectric* (i.e., flat on the baseline, neither positive nor negative), but it may be slightly elevated or depressed normally (usually by less than 1 mm). Pathologic conditions, such as myocardial infarction (MI), that produce characteristic abnormal deviations of the ST segment, are a major focus of clinical ECG diagnosis(Spina et al., 2018).

QRS Angle of Deviation

There is some disagreement on the exact degrees that define each type, but some general cutoffs can be used for the QRS axis(Meek et al., 2002). The QRS axis moves leftward throughout childhood and adolescence and into adulthood. At birth, the normal QRS axis lies between +30 degrees and +190 degrees. Between the ages of 8 to 16, the axis moves leftward with normal lying between 0 degrees to +120 degrees. The normal adult QRS axis is between -30 degrees and +90 degrees, which is directed downward and to the left. This adult range is sometimes extended from -30 degrees to +100 degrees (Lévy, 1991; Surawicz et al., 2009).

Positive T-waves

Usually, though, the amplitude in V2–V3 is around 6 mm and 3 mm in men and women, respectively. T-waves that are higher than 10 mm and 8 mm, in men and women, respectively, should be considered abnormal.

Inverted T wave

Inverted T wave is considered abnormal if inversion is deeper than 1.0 mm. Inverted T waves found in leads other than the V1 to V4 leads is associated with increased cardiac deaths. Inverted T waves associated with cardiac signs and symptoms (chest pain and cardiac murmur) are highly suggestive of myocardial is chaemia Hiss et al., 1960). Other ECG changes associate with myocardial is chaemia are: ST segment depression with an upright T wave; ST segment depression with biphasic T wave or inverted T wave with negative QRS complex; T wave symmetrically inverted with a pointed apex, while the ST segment is either bowed upwards or horizontally depressed, or not deviated; and ST segment depression progressing to abnormal T wave during ischaemia free intervals((Marcus, 2005). However, ST segment depression is not suggestive of ischaemic location of the heart. ST segment depression in eight or more leads, associated with ST segment elevation in aVR and V1 are associated with left main coronary artery disease or three-vessel disease (blockage of all three major branches of coronary arteries). ST segment depression most prominent from V1 to V3 is suggestive of posterior infarction. Furthermore, tall or wide QRS complex with an upright T wave is further suggestive of the posterior infarction(Papadakis et al., 2009).

MATERIALS AND METHODS

Materials used

- Syringes
- Lithium heparin sample bottles
- plain tubes
- Cotton wool
- ECG machine (HeartScreen 112C-1/INNOMED ZRT)
- ECG gel
- Methylated spirit

- Cotton wool

SAMPLE SIZE

Sample size is the number of subject or participants recruited and to which the study findings will be generalized. The sample size was calculated using the Taro Yamani's formula;

$$n = \frac{n}{1 + N(d)^2}$$

Where:

n= sample size

N=population size

d= level of precision (0.05 at 95% confidence level).

The sample for this study is 20 respondents who are non obese pre-pubertal and adult female obese from Ekpoma town, Edo State.

SUBJECTS

Twenty (20) consenting adult female subjects were recruited from Ekpoma metropolitans, Edo State. These subjects consist of 10 non obese female with blood pressure below 120/80 mm/Hg without previous history of obesity and 10 (ten) obese female adult with previous history of obesity.

INFORMED CONSENT

Written informed consent was obtained from subjects prior to commencement of the study.

BLOOD SAMPLING

Five milliliters (5 ml) of venous blood was drawn from consenting participants and placed in a lithium heparin sample bottles. Blood samples was spun in a bucket centrifuge at 2500 RPM (rounds per minute) for 10 minutes after which plasma was collected and stored frozen in plain sample bottles and was analyzed for oxidative stress biomarkers concentrations.

EXPERIMENTAL PROTOCOLS

After the subjects where identified and recruited into the study, they were taken to the lab where their ECG patterns where recorded. After which blood samples was collected by venipuncture and taken to the chemistry laboratory for analysis.

INCLUSION CRITERIA

Non obese and obese female adult, within the age rage of 20 to 40years. Adult female recruited for this study were obese with body mass index greater than 30kg/m^2 and non obese female adult with normal body mass index.

EXCLUSION CRITERIA

Non obese and obese adult female who were on drugs and with a known history of hyperlipidemia, Diabetes and other comorbidity.

STATISTICAL ANALYSIS

Data analysis was done with Graph pad Prism 8.0. The results were expressed as means ± SEM and analysed using Student t-test. P < 0.05 was considered statistically significant.

RESULTS

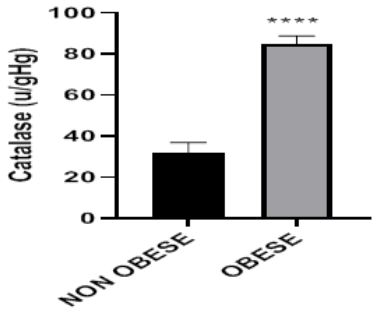

Figure 1: Compares the level of Catalase (u/gHg) in Non Obese and obese of adult female, relatively in Oxidative stress biomarkers.

N=20 (n=10); Mean ± SEM; *significant at p<0.05 compared with control, ^significant at p<0.05 compared with non obese

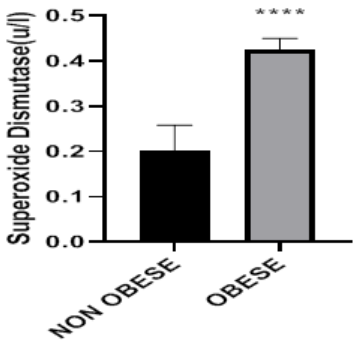

Figure 2: Compares the level of superoxide dismutase in Non Obese and obese of adult female, relatively in Oxidative stress biomarkers.

N=20 (n=10); Mean ± SEM; *significant at p<0.05 compared with control, ^significant at p<0.05 compared with non obese.

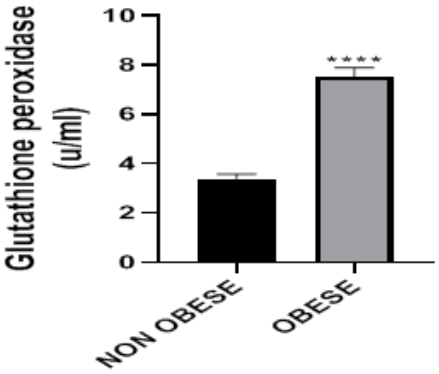

Figure 3: Compares the level of Glutathione peroxidase (u/ml) in Non Obese and obese of adult female, relatively in Oxidative stress biomarkers.

N=20 (n=10); Mean ± SEM; *significant at p<0.05 compared with control, ^significant at p<0.05 compared with non obese.

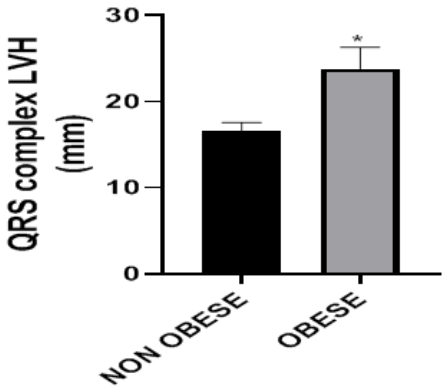

Figure 4: Compares the ECG QRS Complex Left Ventricular Hypertrophy (LVH) pattern in Non Obese and obese of adult female individuals.
N=20 (n=10); Mean ± SEM; *significant at p<0.05 compared with control, ^significant at p<0.05 compared with non obese

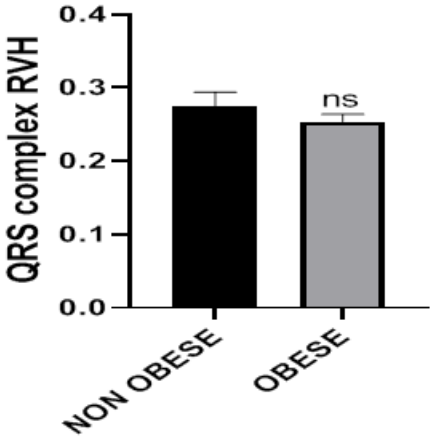

Figure 5: Compares the ECG of QRS Complex Right Ventricular Hypertrophy (RVH) pattern in Non Obese and obese of adult female individuals.
N=20 (n=10); Mean ± SEM; insignificant at p<0.05 compared with control (non obese)

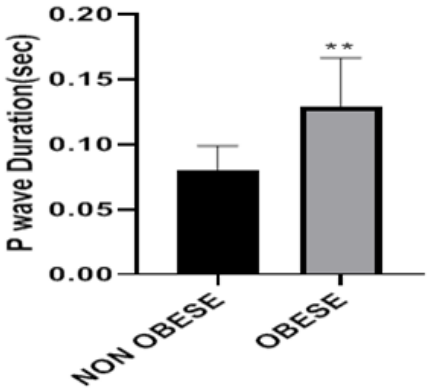

Figure 6: Compares the ECG (P wave duration-sec) pattern in Non Obese and obese of adult female individuals.
N=20 (n=10); Mean ± SEM; *significant at p<0.05 compared with control, ^significant at p<0.05 compared with non obese.

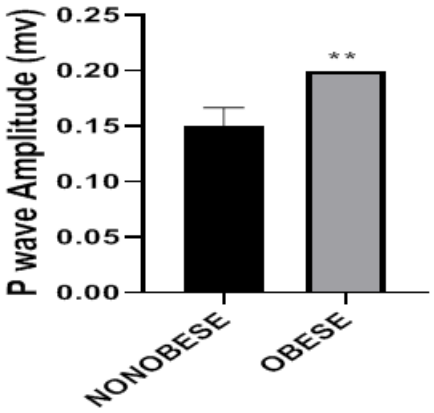

Figure 7: Compares the ECG (P wave Amplitude-mv) pattern in Non Obese and obese of adult female individuals.
N=20 (n=10); Mean ± SEM; *significant at p<0.05 compared with control, ^significant at p<0.05 compared with non obese

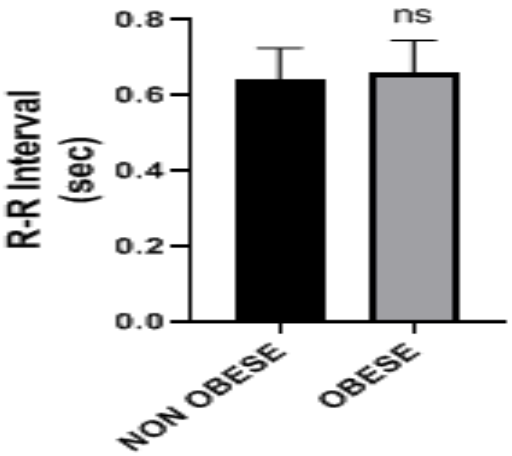

Figure 8: Compares the ECG (R-Rinterval) pattern in Non Obese and obese of adult female individuals.
N=20 (n=10); Mean ± SEM; not significant at p<0.05 compared with control (non obese).

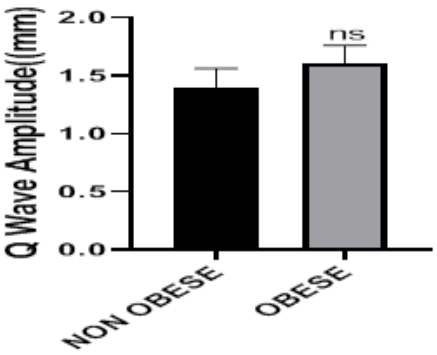

Figure 9: Compares the mean ECG (ST Segment) pattern in Non Obese and obese of adult female.
N=20 (n=10); Mean ± SEM; not significant at p<0.05 compared with control (non obese).

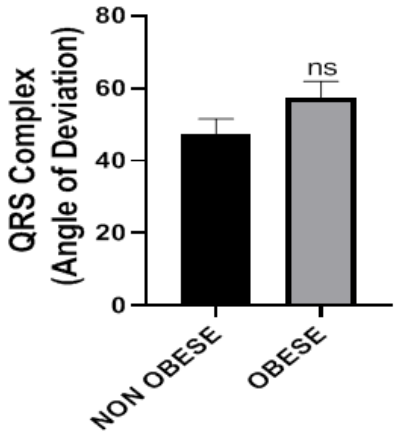

Figure 10: Compares the mean ECG (Angle of Deviation) pattern in Non Obese and obese of adult female.
N=20 (n=10); Mean ± SEM; not significant at p<0.05 compared with control (non obese).

DISCUSSION

Oxidative stress reflects an imbalance between the systemic manifestation of reactive oxygen species (ROS) and a biological system's ability to readily detoxify the reactive intermediates or to repair the resulting damage. Disturbances in the normal redox state of cells can cause toxic effects through the production of peroxides and free radicals that damage all components of the cell, including proteins, lipids, and DNA. Oxidative stress from oxidative metabolism causes base damage, as well as strand breaks in DNA. Reactive species produced in the cell during normal cellular metabolism can chemically react with cellular biomolecules such as nucleic acids, proteins, and lipids, thereby causing their oxidative modifications leading to alterations in their compositions and potential damage to their cellular activities. Fortunately, cells have evolved several antioxidant defense mechanisms (as metabolites, vitamins, and enzymes) to neutralize or mitigate the harmful effect of reactive species and/or their byproducts. Any perturbation in the balance in the level of antioxidants and the reactive species results in a physiological condition called "oxidative stress." A catalase is one of the crucial antioxidant enzymes that mitigates oxidative stress to a considerable extent by destroying cellular hydrogen peroxide to produce water and oxygen. Deficiency or malfunction of catalase is postulated to be related to the pathogenesis of many age-associated degenerative diseases like diabetes mellitus, hypertension, anemia, vitiligo, Alzheimer's disease, Parkinson's disease, bipolar disorder, cancer, and schizophrenia. Therefore, efforts are being undertaken in many laboratories to explore its use as a potential drug for the treatment of such diseases (Ankita Nandi et al., 2019). The different units of measurement for Glutathione Peroxidase, Superoxide Dismutase, Catalase are (U/mL, U/gHb, nmol/mg, or µmol/L) used to express the concentrations.

The reference value antioxidant enzyme activity is between 27.5-73.6u/gHb. From this study, oxidative stress biomarker (catalase) as shown in **Figure 1** of Obese(84.99±1.235) was significantly greater than non obese(34.82 ± 1.590) adult females p<0.05. However the mean values in obese was relatively higher than normal while the non obese was consistent with normal values for antioxidant enzyme activities in human of earlier documented reports.

The reference value Superoxide Dismutase antioxidant enzyme activity is between 0.12-0.33u/l. From this study, oxidative stress biomarker (Superoxide Dismutase) as shown in **Figure 2** of Obese(0.4240± 0.008327) was significantly greater than non obese(0.2010± 0.01810) adult females p<0.05. However the mean values in obese was relatively higher than normal while the non obese was consistent with normal values for antioxidant enzyme activities in human of earlier documented reports.

The reference value antioxidant Glutathione peroxidase enzyme activity is between 2.65-4.80u/ml. From this study, oxidative stress biomarker Glutathione peroxidase(u/ml) as shown in **Figure 3** of Obese(7.509± 0.3769) was significantly greater than non obese (3.389± 0.1967) adult females p<0.05. However the mean values in obese was relatively higher than normal while the non obese was consistent with normal values for antioxidant enzyme activities in human of earlier documented reports.

The QRS complex represents the electrical impulse as it spreads through the ventricles and indicates ventricular depolarization. The normal duration (interval) of the QRS complex is between 0.08 and 0.10 seconds. That is, 80 and 100 milliseconds. When the duration is between 0.10 and 0.12 seconds, it is intermediate or slightly prolonged. QRS duration of greater than 0.12 seconds is considered abnormal. Right ventricular hypertrophy (RVH) is an abnormal enlargement or pathologic increase in muscle mass of the right ventricle in response to pressure overload, most commonly due to severe lung disease. Right bundle branch block (RBBB) is an electrocardiogram finding that occurs when the physiologic electrical conduction system of the heart, specifically in the His-Purkinje system, is altered or interrupted resulting in a widened QRS and electrocardiographic vector changes. QRS complex: amplitude greater than 0.5 mV in at least one standard lead, and greater than 1.0 mV in at least one precordial lead. Upper limit of normal amplitude is 2.5 - 3.0 mV. Increased QRS voltage is often taken to infer the presence of **left ventricular hypertrophy**. However, high left ventricular voltage (HLVV) may be a normal finding in patients less than 40-45 years of age, particularly slim or athletic individuals. There are multiple "voltage criteria" for left ventricular hypertrophy. *RVH **less than**1mm indicates that there is*

*no RVH and LVH **less than** 35mm also indicates that there is no RVH*

From the ECG patterns in this study QRS complex LVH as shown in **Figure 4** of Obese (23.80±2.476mm) and non obese(16.50 ± 1.067 mm) adult females was significantly higher in obese compare to non obese, p<0.05. However, the mean values of both were consistent generally acceptable normal range values. Also from the ECG patterns in this study QRS complex RVH as shown in **Figure 5** of Obese (0.2540 ±0.009798 mm) and non obese(0.2740 ± 0.01973 mm) adult females was significantly higher in obese compare to non obese, p<0.05. However, the mean values of both were consistent generally acceptable normal range values. The normal P-wave duration is 80 milliseconds. PR interval: 120-200 milliseconds. PR segment: 50-120 milliseconds. The duration of the P wave does not usually exceed three small squares (0.12 s). An abnormal P-wave axis (aPWA) obtained in the routine 12-lead electrocardiogram (ECG) is thought to be a marker of left atrial fibrosis and delayed conduction. Consistent with this, aPWA has been linked to the development of atrial fibrillation, stroke and total mortality.

From the ECG patterns in this study, the P wave duration-sec as shown in Figure **6** of Obese and non obese adult females was significantly higher in obese compare to non obese, p<0.05. P wave Duration (sec) of Obese was (0.1289 ± 0.01252) and Non obese (0.08000±0.005963) respectively. The Obese P wave Duration (sec) was abnormally higher (0.1289 ± 0.01252-0.12) with 0.0089sec while that of non obese was less than 0.12sec with 0.04sec duration. Also, the ECG (P wave amplitude) pattern in **Figure 7** Obese and non obese of adult female was significantly higher in obese compare to non obese, p<0.05. P wave Amplitude (mV) of Obese was 0.2000±0.0000 and Non obese (0.1500±0.01667) respectively. Although the mean value of (P wave amplitude) in this study was 0.2 mv, there was a significant statistical difference between that of non obese. The RR interval, the time elapsed between two successive R waves of the QRS signal on the electrocardiogram (and its reciprocal, the HR), is a function of intrinsic properties of the sinus node as well as autonomic influence. Abnormal R–R intervals differ from sinus rhythm in their length and they represent disturbances of both technical and physiological origins and are present in almost all Holter ECG recordings. Physiological artifacts occur especially in patients suffering from different cardiovascular diseases. Normal ECG values for RR interval: 0.6-1.2 seconds. From the ECG patterns in this study, the mean RR intervals as shown in **Figure 8** of Obese and non obese adult females were not significantly different in obese compare to non obese, p<0.05. The mean RR interval (sec) of Obese was (0.66 ± 0.02667) and Non obese (0.64±0.02667) respectively were not significantly different. However the mean values of this present obese and non obese were consistent with earlier reports of normal RR intervals. The **ST segment** is the flat, isoelectric section of the ECG between the end of the S wave (the J point) and the beginning of the T wave. The ST Segment represents the interval between ventricular depolarization and repolarization. The most important cause of ST segment abnormality (elevation or depression) is **myocardial ischaemia or infarction. Causes of ST segment elevation are:** Acute myocardial infarction, Coronary vasospasm (Printzmetal's angina), Pericarditis, Benign early, repolarization, Left bundle branch block, Left ventricular hypertrophy, Ventricular aneurysm, Brugada, syndrome, Ventricular paced rhythm, Raised intracranial pressure, Takotsubo Cardiomyopathy etc. An ST elevation is considered significant if the vertical distance inside the ECG trace and the baseline at a point 0.04 seconds after the J-point is at least 0.1 mV (usually representing 1 mm or 1 small square) in a limb lead or 0.2 mV (2 mm or 2 small squares) in a precordial lead. From the ECG patterns in this study, the mean **ST segment** (mm) as shown in **Figure 9** of Obese and non obese adult females were not significantly different in obese compare to non obese, p<0.05. The mean **ST segment (mm)** of Obese was (ST segment 1.600±0.2211mm) and Non obese (1.400±0.1633mm) respectively were not significantly different. However the mean values of this present obese and non obese were consistent with normal **ST segment (mm)** acceptable values of earlier reports

CONCLUSION

The overall findings shows that oxidative stress was not prominent in obese as evident in the higher values of catalase, Superoxide Dismutase biomarker and Glutathione peroxidase enzyme activities compared to non obese. Deficiency or malfunction of catalase is postulated to be related to the pathogenesis of many age-associated degenerative diseases like diabetes mellitus, hypertension which was not the case. Glutathione peroxidase-1 (GPx-1) is an intracellular antioxidant enzyme that enzymatically reduces hydrogen peroxide to water to limit its harmful effects. Hence cardiovascular disease may not be much concern in this study when oxidative stress is considered. Excess Glutathione peroxidase-1 (GPx-1) is thought to have deleterious effects due to a lack of essential cellular oxidants that result in a reductive stress characterized by a lack of oxidants and/or excess reducing equivalents. Reductive stress may appear to be a new concept, it has been known for some time that lack of cellular oxidants can diminish cell growth responses. Newer evidence points to additional cellular and physiological effects caused by lack of cellular oxidants and accumulation of excess reducing equivalents, including changes in protein disulfide bond formation, diminished mitochondrial function, and decreased cellular metabolism. Also ECG wave patterns of the P wave duration-sec and P wave amplitude were significantly higher in obese compared to non obese. An abnormal P-wave axis (aPWA) obtained in the routine 12-lead electrocardiogram (ECG) is thought to be a marker of left atrial fibrosis and delayed

conduction. Consistent with this, aPWA has been linked to the development of atrial fibrillation, stroke and total mortality. Deliberate efforts should be made at addressing associated cardiovascular and oxidative stress compromise in obesity.

REFERENCES

1. Alegría A, Barberá R, Clemente G, Farré R, García M J, Lagarda M J. Selenium and glutathione peroxidase reference values in whole blood and plasma of a reference population living in Valencia, Spain. J Trace Elem Med Biol., 1996; 10(4): 223-8.
2. Ankita Nandi, Liang-Jun Yan, Chandan Kumar Jana, and Nilanjana Das(2019). Role of Catalase in Oxidative Stress- and Age-Associated Degenerative Diseases. Oxid Med Cell Longev. : 9613090.
3. Bęćkowski M, Gierlotka M, Gąsior M, Poloński L, Zdrojewski T, Dąbrowski R, Kowalik I, Karwowski J, Drygas W, Szwed H. Factors Affecting Early Mortality and 1-Year Outcomes in Young Women With ST-Segment-Elevation Myocardial Infarction Aged Less Than or Equal to 45 Years. Curr Probl Cardiol., 2021 Mar; 46(3): 100419.
4. Ewing DJ, Martin CN, Young RJ, Clarke BF. The value of cardiovascular autonomic function tests: 10 years' experience in diabetes. Diabetes Care, 1985; 8: 491-498.
5. Handy DE. Lubos E. Yang Y. Galbraith JD. Kelly N. Zhang YY. Leopold JA. Loscalzo J. Glutathione peroxidase-1 regulates mitochondrial function to modulate redox-dependent cellular responses. J Biol Chem., 2009; 284: 11913–11921.
6. Hiss RG, Averill KH, Lamb LE. Electrocardiographic findings in 67,375 asymptomatic subjects. VIII. Nonspecific T wave changes. Am J Cardiol., 1960; 6: 178–189.
7. Lavi S, Yang E, Prasad A, Mathew V, Barsness GW, Rihal CS, Lerman LO, Lerman A. The interaction between coronary endothelial dysfunction local oxidative stress, and endogenous nitric oxide in humans. Hypertension. 2008; 51: 127–133.
8. Lévy S. Diagnostic approach to cardiac arrhythmias. J Cardiovasc Pharmacol., 1991; 17 Suppl 6: S24-31.
9. Marcus FI. Prevalence of T-wave inversion beyond V1 in young normal individuals and usefulness for the diagnosis of arrhythmogenic right ventricular cardiomyopathy/dysplasia. Am J Cardiol., 2005; 95: 1070–1071.
10. Márta Vitai, László Góth. Reference ranges of normal blood catalase activity and levels in familial hypocatalasemia in Hungary. Clinica Chimica Acta, 1997; 261: 35-42.
11. McClung JP. Roneker CA. Mu W. Lisk DJ. Langlais P. Liu F. Lei XG. Development of insulin resistance and obesity in mice overexpressing cellular glutathione peroxidase. Proc Natl Acad Sci U S A., 2004; 101: 8852–8857.
12. Meek S, Morris F. ABC of clinical electrocardiography.Introduction. I-Leads, rate, rhythm, and cardiac axis. BMJ., 2002 Feb 16; 324(7334): 415-8.
13. Mithilesh K. Das, Bilal Khan, Sony Jacob, Awaneesh Kumar and Jo Mahenthiran((2006).
14. Papadakis M, Basavarajaiah S, Rawlins J, Edwards C, Makan J, Firoozi S, Carby L, Sharma S. Prevalence and significance of T-wave inversions in predominantly Caucasian adolescent athletes. Eur Heart J., 2009; 30: 728–1735.
15. Mithilesh K Das 1, Bilal Khan, Sony Jacob, Awaneesh Kumar, Jo Mahenthiran Significance of a Fragmented QRS Complex Versus a Q Wave in Patients With Coronary Artery Disease. Circulation, 2006; 113: 2495–2501.
16. Pagani M, Lombardi F, Guzzetti S, Rimoldi O, Furlan R, Pizzinelli P, Sandrone G, Malfatto G, Dell'Orto S, Piccaluga E, Turiel M, Baselli G, Cerutti S, Malliani A. Power spectral analysis of heart rate and arterial pressure variabilities as a marker of sympathovagal interaction in man and conscious dog. Circ Res., 1986; 59: 178-193.
17. Pomeranz M, Macaulay RJB, Caudill MA, Kutz I, Adam D, Gordon D, Kilborn KM, Barger AC, Shannon DC, Cohen RJ, Benson M. Assessment of autonomic function in humans by heart rate spectral analysis. Am J Physiol., 1985; 248: H151-H153.
18. Rajasekaran NS. Connell P. Christians ES. Yan LJ. Taylor RP. Orosz A. Zhang XQ. Stevenson TJ. Peshock RM. Leopold JA. Barry WH. Loscalzo J. Odelberg SJ. Benjamin IJ. Human alphaB-crystallin mutation causes oxido-reductive stress and protein aggregation cardiomyopathy in mice. Cell., 2007; 130: 427–439.
19. Serum and Erythrocyte Lipoperoxides and Superoxide Dismutase Levels in Normal Persons and Hypertensive Patients. Korean J Intern Med., 1(1): 113–119.
20. Spina R, Song N, Kathir K, Muller DWM, Baron D. Takotsubo cardiomyopathy following unintentionally large subcutaneous adrenaline injection: a case report. Eur Heart J Case Rep., 2018 Jun; 2(2): yty043.
21. Surawicz B, Childers R, Deal BJ, Gettes LS, Bailey JJ, Gorgels A, Hancock EW, Josephson M, Kligfield P, Kors JA, Macfarlane P, Mason JW, Mirvis DM, Okin P, Pahlm O, Rautaharju PM, van Herpen G, Wagner GS, Wellens H., American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology. American College of Cardiology Foundation. Heart Rhythm Society. AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: part III: intraventricular conduction disturbances: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart

Rhythm Society. Endorsed by the International Society for Computerized Electrocardiology. J Am Coll Cardiol. Mar 17; 53(11): 976-81.

22. Tetsuo Konno, Masami Shimizu, Hidekazu Ino, Masato Yamaguchi, Hidenobu Terai, Katsuharu Uchiyama, Kotaro Oe, Tomohito Mabuchi, Tomoya Kaneda, Hiroshi Mabuchi. Diagnostic value of abnormal Q waves for identification of preclinical carriers of hypertrophic cardiomyopathy based on a molecular genetic diagnosis. European Heart Journal, 2004; 25: 246–251.
23. Yog-Gyun Kim, M.D., Jong-Koo Chae, M.D., Sang-Ho Park, M.D., Choon-Hae Chung, M.D., Soon-Pyo Hong, M.D., and Kun-Kook Cho, M.D. Diagnostic value of abnormal Q waves for identification of preclinical carriers of hypertrophic cardiomyopathy based on a molecular genetic diagnosis, European Heart Journal, 1986; 25: 246–251.