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College of Applied Medical Sciences

Relationship of some Physiological and Biochemical Parameters with Occurrence Atherosclerosis in Obese and non-Obese Individuals in Karbala Governorate

A Thesis

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بِسْمِ ٱللَّهِ ٱلرَّحْمَٰنِ ٱلرَّحِيمِ

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١٢ \ ١٠ \ 2023

Dedication

Thanks and praise to God Almighty first for the blessing of patience and the ability to accomplish the work, for God is praise for these blessings.

To the example of dedication and sincerity..... my beloved father.

To whom I offered my happiness and comfort over her happiness... My virtuous mother.

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The completion of my work, I hope it will be to your satisfaction.

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List of Abbreviations

Abbreviations	Full Form
ACAT-1	Acyl CoA:cholesterol acyltransferase-1
Acrp30, AdipoQ	Adiponectin
ACS	Acute coronary syndromes
AHA	American Heart Association
Apo	Apolipoproteins
AS	Atherosclerosis
ASCVD	Atherosclerosis cardiovascular diseases
BBB	Blood brain barrier
BMI	Body mass index
BP	Blood pressure
CABG	Coronary Artery Bypass Graft
CAD	Coronary artery diseases
CCTA	Cardiac computed tomography angioplasty
CE	Cholesterol ester
CHD	Coronary heart diseases
CM	Chylomicrons
CAs	Coronary arteries
CRP	C-reactive protein

CT	Computerized tomography
CVD	Cardiovascular diseases
DM	Diabetes mellitus
ECG	Electrocardiogram
ECs	Endothelial cells
ELISA	Enzyme-Linked Immunosorbent Assay
FAs	Fatty acids
HDL	High density lipoprotein
HIN	Hypertension
HMW	High molecular weight
HRP	Avidin-Horseradish Peroxidase
hs-CRP	High sensitive C-reactive protein
hs-cTn	High sensitive cardiac Troponin
ICAM-1	Intercellular adhesion molecule-1
IMT	Intima-media thickness
IVUS	Intravascular ultrasound
LDL	Low density lipoprotein
LEP	Leptin
LMW	Lower molecular weight
MCP-1	Monocyte chemoattractant protein-1
M-CSF	Macrophage colony-stimulating factor
MDA	Malondialdehyde
MENA	Middle East and North Africa

MI	Myocardial infarction
MMP	Matrix metalloproteinases
MMW	Middle molecular weight
MnSOD	Manganese superoxide dismutase
NDPH	Nicotinamide adenine dinucleotide phosphate
OCT	Optical coherence tomography
OD	Optical density
OS	Oxidative stress
Ox-LDLs	Oxidized low-density lipoproteins
PAI-1	Plasminogen activator inhibitor type-1
PCI	Percutaneous coronary intervention
PPAR-γ	Peroxisome proliferator-activated receptor-γ
PVAT	Perivascular adipose tissue
RNS	Reactive nitrogen species
ROS	Reactive oxygen species
S.error	Standard error
Sd	Standard deviation
SMC	Smooth muscle cell
SPSS	Statistical package for the social sciences
SR-A	Scavenger receptor-A
T2DM	Type 2 diabetes mellitus
TAFI	Thrombin activatable fibrinolysis inhibitor
TAGs	Triacylglycerol

T-AOC, TAO	Total antioxidant capacity, Total antioxidant
TC	Total cholesterol
TGs	Triglycerides
TnC	Troponin C
TnI	Troponin I
TnT	Troponin T
t-PA	Tissue-type plasminogen activator
u-PA	Urokinase-type plasminogen activator
VCAM- 1	Vascular cell adhesion molecule-1
VEGF	Vascular endothelial growth factor
VLDL	Very low density lipoprotein
VSMC	Vascular smooth muscle cell
WHO	World Health Organization

Summary

Atherosclerosis is a chronic, complex, inflammatory disease that affects medium to large size arteries (coronary, carotid, peripheral arteries). This condition begins when oxidized low-density lipoproteins build up in the artery intima. This resulted in the production of proinflammatory oxidized lipids by the overlapping endothelial cells. This leads to hardening and narrowing of blood vessels.

Aim of study: To find association between obesity with occurrence of atherosclerosis.

The study included (100) males with an average age of (40-65), and they were divided as follows: (60) patients groups suffering from atherosclerosis were divided into two groups, depending on the body mass index (BMI):

- The obese group (BMI = 25 ≥ 30) included 30 male subjects.
- The normal weight group, BMI = 18.5-24.9, included 30 male subjects.

(40) control groups were divided into two groups, depending on the BMI

- The obese group (BMI = 25 ≥ 30) included 20 male subjects.
- The normal weight group, BMI = 18.5-24.9, included 20 male subjects.

This study was conducted from November 2022 to May 2023. The samples were taken from the Karbala Center for Cardiac diseases and surgery, where (5ml) blood samples were taken from patients and control, and the biochemical and physiological tests were conducted on them (adiponectin, leptin, plasminogen activator inhibitor-1 (PAI-1), malondialdehyde (MDA), total antioxidant capacity (T-AIC), troponin I, C-reactive protein (CRP), lipid profile).

The result of this study found a highly significant decrease ($p \leq 0.001$) in concentration of adiponectin in (atherosclerosis obese, atherosclerosis normal, control obese) as compared to control normal. While highly significant increase ($p \leq 0.001$) in concentration of leptin in (atherosclerosis obese, atherosclerosis normal, control obese) as compared to control normal. A highly significant increase

($p \leq 0.001$) in concentration of PAI-1 in (atherosclerosis obese, atherosclerosis normal, control obese) as compared to control normal. While highly significant increase ($p \leq 0.001$) in concentration of MDA in (atherosclerosis obese, atherosclerosis normal, control obese) as compared to control normal. A highly significant decrease ($p \leq 0.001$) in concentration of T-AOC in (atherosclerosis obese, atherosclerosis normal, control obese) as compared to control normal. A highly significant increase ($p \leq 0.001$) in concentration of troponin I in atherosclerosis groups as compared to control groups. Found a highly significant increase ($p \leq 0.004$) in the concentration of CRP in (atherosclerosis obese, atherosclerosis normal, and control obese) as compared to control normal. A significant increase ($p \leq 0.001$), ($p \leq 0.005$), ($p \leq 0.021$), ($p \leq 0.001$) in the concentration of total cholesterol (TC), triglyceride (TG), low density lipoprotein (LDL), very low density lipoprotein (VLDL), respectively in atherosclerosis obese as compared to (atherosclerosis normal, control obese, control normal). Also a highly significant decrease ($p \leq 0.001$) in the concentration of high density lipoprotein (HDL) in (atherosclerosis obese, atherosclerosis normal, control obese) as compared to control normal.

This study clarified several relationships among parameters in the atherosclerosis obese group. There was a significant negative correlation between adiponectin and MDA. It observes significant positive correlation between BMI and both TG and VLDL. In addition to the positive correlation between TC and LDL. It shows positive correlation between TG and both VLDL, MDA.

This study observed several relationships among parameters in the atherosclerosis normal group. There was a significant negative correlation between BMI and T-AOC. It shows a negative correlation between TC and HDL. It shows positive correlation between TG and VLDL.

This study observed several relationships among parameters in obese group. There was a significant positive correlation between BMI and PAI-1. In addition,

negative correlation between BMI and HDL, positive correlation between TC and LDL, positive correlation between TG and VLDL.

Conclusion: there is association between (adiponectin, leptin, PAI-1) in the development of atherosclerosis in obese patients. Increased MDA concentration indicates that the rate of oxidative stress is high. Decrease in concentration of T-AOC in all obese groups. Leptin ,which controls metabolism, was found to be increasing, which may be a sign that the metabolic issue in obese individuals in this study.

Chapter One

Introduction

Chapter One

1.1 Introduction

German physician Félix Marchand first used the term "atherosclerosis" (AS) in 1904; it is derived from the Greek words "athere" and "sclerosis," which indicate gruel and hard, respectively Atherosclerosis is the primary contributor to diseases of the coronary arteries, carotid artery, and the peripheral blood vessels (Kenny *et al*, 2019). Cholesterol deposition and chronic inflammation are two crucial factors in the pathogenesis of atherosclerosis, including three main stages: the generation of fatty streaks, the induction of atheroma, and atherosclerotic plaques (Malek mohamed *et al.*, 2021). This condition begins when oxidized low-density lipoproteins (Ox-LDLs) build up in the artery intima (Khatana *et al.*, 2020). This resulted in the production of proinflammatory oxidized lipids by the overlapping endothelial cells (ECs) (Björkegren & Lusis, 2022). Within the intima of the artery, monocytes develop into proinflammatory the macrophages locally enhance the inflammatory reaction, eventually, macrophages ingest lipoproteins to produce foam cells rich in lipids, which cause early atherosclerosis lesions to appear (Chen *et al.*, 2022).

Obesity is the unnatural accumulation of fat in the human body, which increases the risk for diabetes, heart disease, hypertension, dyslipidemia, and high cholesterol (Yartaşı *et al.*, 2022). By causing arterial inflammation and oxidative stress, proinflammatory adipocytokines and free fatty acids generated by malfunctioning fatty tissue can systematically accelerate atherosclerosis (Kim *et al.*, 2020).

Adiponectin is a "adipokine" that is virtually mostly formed by adipose tissue (Da Silva Rosa *et al.*, 2021). Adiponectin levels decrease in illnesses such as

coronary artery disease (CAD), diabetes mellitus (DM), and high blood pressure (D'Marco *et al.,* 2020). Adiponectin levels are low and have limited capacity to control inflammatory reactions in obese individuals (Lempesis *et al.,* 2020).

An important adipokine is leptin secreted by adipocytes and recognized for its proinflammatory effect (Landecho *et al.,* 2019). The obese individuals found high levels of leptin in circulation (Izquierdo *et al.,* 2019). One of the main risk factors for atherosclerosis is hyperleptinemia, which is a feature of obesity (Raman & Khanal, 2021).

Plasminogen activator inhibitor type-1 (PAI-1) is the major biological inhibitor of the activation of plasminogen in the bloodstream (Song *et al*., 2017). It has been found that atherosclerotic and coronary artery disease (CAD) are connected to elevated PAI-1 levels that induce the breakdown of fibrin and eventually affects the risk of thrombus (Jung *et al.,* 2018). The primary elements of metabolic syndrome are influenced by PAI-1, and it is believed that higher PAI-1 values have a positive association with obesity (Mira *et al.,* 2020).

Cardiac troponins constitute critical components of the heart contractile mechanism that damaged cardio myocytes leak into the circulatory system (Aakre & Omland, 2019). Heart troponin release with intense exercise is connected to coronary atherosclerosis or indications of plaque susceptibility (Paana *et al.,* 2019).

Reactive species of oxygen and nitrogen are produced as a result of numerous internal and external events, and antioxidant defenses prevent their harmful effects (Liguori *et al*., 2018). The imbalance between the generation of oxidants and antioxidant defenses that might harm a biological system is referred to as "oxidative stress" (OS) (Wang *et al*., 2022). Increased oxidative stress, production of cytokines and chemokines, and activation of proinflammatory signaling pathways are all factors in the development of atherosclerosis (Kattoor *et al.,* 2017).

Malondialdehyde (MDA) is a particular aldehyde that has been used as a biomarker for OS and is considered to be a frequent end product of the peroxidation of lipids (Wang *et al.,* 2017). One of the principal causes of reactive oxygen species (ROS) is the oxidation of lipids (Domínguez *et al.,* 2019). The amount of lipid peroxidation in addition to the severity of OS are both reflected in MDA plasma levels (Cho *et al.,* 2022).

Total antioxidant capacity (T-AOC), an antioxidant is a substance that despite acting at a smaller amount than the substrate being protected, can stop or slow down oxidation of a substrate (Pisoschi *et al.,* 2021). ROS and free radicals are both threats that are mitigated by tissue antioxidants, certain enzyme- and non-enzyme-based antioxidants offer this protective effect (Amin *et al.,* 2020). Antioxidants such as transferrin, vitamins C, and E, are destroyed free radicals by disrupting the chain reactions of lipid peroxidation (Ganjifrockwala *et al.,* 2017).

Aims of the study:

- To find an association between obesity and occurrence of Atherosclerosis.

1. Evaluation level of biomarkers study (Adiponectin, Leptin, PAI-1) in healthy and atherosclerosis people with normal and increased body mass index (BMI), then comparison between them.
2. To find association between levels of biomarkers study (Adiponectin, Leptin, PAI-1), and lipid profile in both healthy and patient groups.
3. Study association between levels of biomarkers study (Adiponectin, Leptin, PAI-1) with level of inflammation indicators C-reactive protein (CRP) in healthy and patient groups.
4. Study the association between these biomarkers with oxidation stress and antioxidant level.

5. Study the association between these biomarkers with Troponin I level in healthy and atherosclerosis people with normal and increased body mass index (BMI), then comparison between them.

Chapter Two

Literatures Review

Chapter Two

2.1. Atherosclerosis

The most widespread pathological condition of peripherals and coronary artery disorders, as well as of cerebellar problems, is atherosclerosis (AS), a chronic inflammatory condition of the arterial marked by the growth of distinctive lesions called atheromatous plaques (Ferrari *et al.*, 2021). The pituitary, pancreas, liver, heart, renal fatty tissue, and the adrenals, in addition to the sex glands are all affected by atherosclerosis, a long-term inflammatory disease that is lipid-driven (Melaku & Dabi, 2021). Fasting glucose levels, high blood pressure, and low-density lipoprotein cholesterol (LDL-c) are the three primary cardio metabolic risk factors that mediate between 40 and 50 percent of the connection between obesity and cardiovascular illnesses (Christen *et al.*, 2019). Lipid retention and oxidative changes inside the artery intima start the atherosclerotic process, which then leads to persistent inflammatory cascades that eventually lead to thrombosis and plaque formation (Ahmad *et al.*, 2020). Russell Ross initially suggested the idea that atherosclerosis is a condition caused by inflammation in 1999, based on his findings that monocytes that circulate enter a developing fatty streak (Kong *et al.*, 2022).

2.1.1. Pathogenesis

The wall of coronary arteries (CAs) consists of three layers: the innermost layer (tunica intima), the middle layer (tunica media), and the outermost layer (tunica adventitia or externa); all encapsulated in perivascular adipose tissue (PVAT) (Zorc-Pleskovič *et al.*, 2018). Atherosclerosis affects the structure and function of all three layers of the coronary artery wall (Tellides & Pober, 2015). Oxidized fatty acids produced from LDL promote several phases of atherosclerotic plaque growth and

development by producing inflammatory cytokines, AS contains a variety of different kinds of cells and cellular mediators (Alfarisi *et al.*, 2020).

1.Development of fatty streaks: LDLs enter the middle layer, undergo accumulation as blood levels rise, and oxidize to proinflammatory cells, this accumulation lead to inflammatory mediators activate and secrete vascular smooth muscle cell (VSMC), the last, attracting neutrophils, lymphocytes, mast cells, and monocytes, which then develop into foam cells through the creation of macrophages (Reddy, 2020).

2.Fibro atheroma formation: when macrophages are activated, they produce a number of cytokines, undergo a transformation into foam cells, and then succumb to necrosis, eventually, activated macrophages release additional inflammatory stimuli and promote the formation of advanced atheroma (Mozos *et al.*, 2017). Atheroma often have a lipid core, a necrotic core formed by apoptotic macrophages, and an extensive fibrous cover that is growing because of smooth muscle cells' (SMC) production of collagen, elastin, fibronectin, and extracellular matrix (Charla *et al.*, 2020).

3.Atherosclerotic rupture: over time, plaque builds up and narrows the artery, increasing the risk of ulceration, intraplaque bleeding, and rupture, this reduces the amount of oxygenated blood that reaches the tissues and organs (Tam *et al.*, 2021).

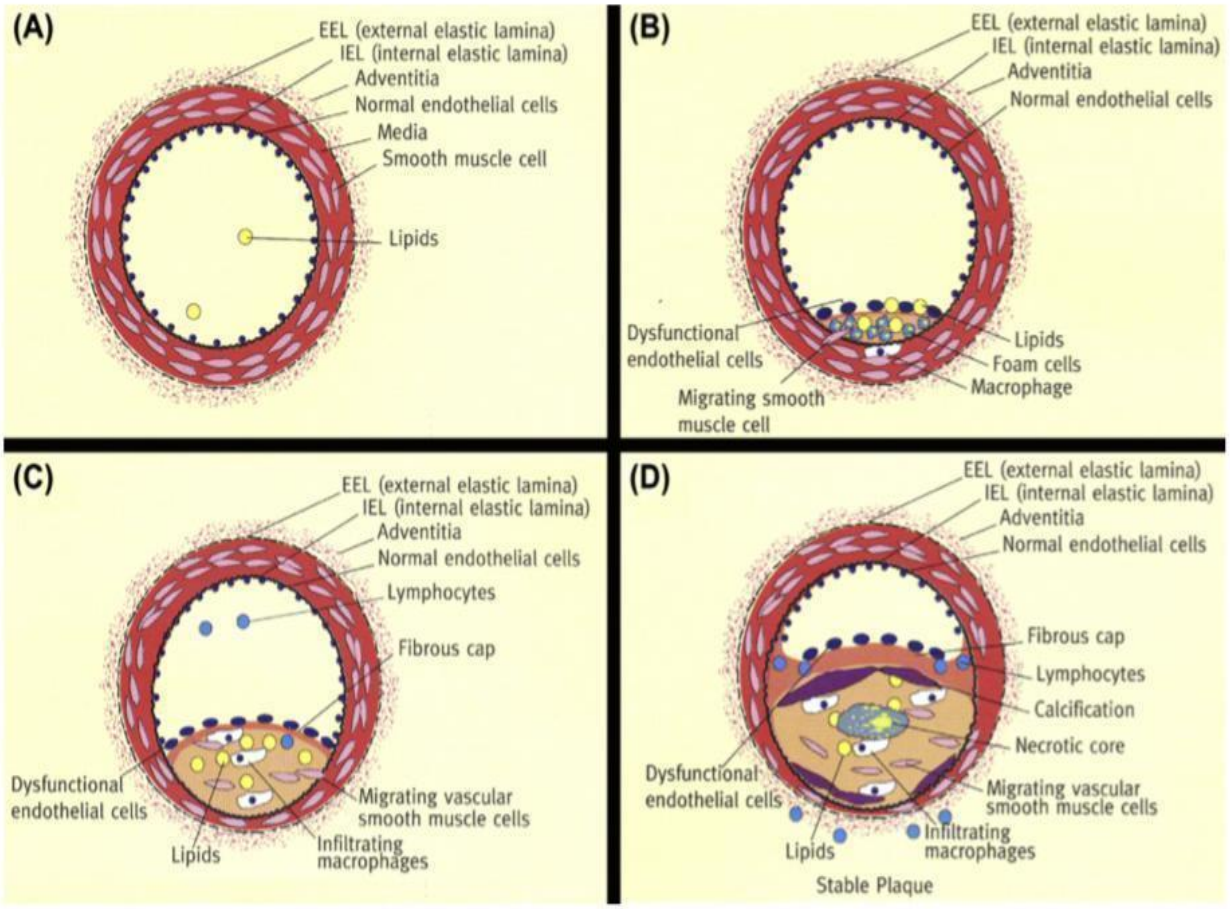

Figure 1: Progression of atherosclerotic plaques (Seidman *et al.*, 2014).

The figure (1) shows the pathology of atherosclerosis. This process is brought on by the buildup of cholesterol, primarily in the form of LDL, in the intimate layer of the arteries, this accumulation results in extensive narrowing of arteries, which significantly lowers blood flow and leads the development of tissue hypoxia (Wolf & Ley, 2019). A proinflammatory monocytes seems to proliferate in the beginning atherosclerotic plaques, where they can develop into macrophages that can absorb excessive lipids to form foam-like cells (Milutinović *et al.*, 2020). In addition, another significant pathogenic factor that contributes to the emergence of atherosclerosis is inflammation, endothelial cells (ECs) in blood vessels respond to LDL buildup by expressing raised concentrations of chemokines and cytokines that increase inflammation, those mediators, such as IL-8 and P-selectin, draw

leukocytes to the focal point of atherosclerotic lesions (Chistiakov *et al.*, 2018). In the damaged intimal layer of the aorta, a number of cell groups, include dendritic cells, different kinds of T and B lymphocytes, VSMC, ECs, and multiple kinds of macrophages, have a role in the growth of the inflammatory response (Mohmmad-Rezaei *et al.*, 2021). In addition to inflammation, the latter contributes to the aggregation of cholesterol by the production of foam cells, playing one of the key roles in the degenerative process in atherosclerosis (Blagov *et al.*, 2023).

2.1.2. Epidemiology

Based on the World Health Organization (WHO), cardiovascular disease (CVD) is the leading cause of death, 17.5 million people die annually from CVDs, approximately 7.4 million people die from coronary heart disease (Sharifi-Rad et al., 2020). According to the World Health Organization's third report, by 2025, it is predicted that cardiovascular mortality will reach global levels (Kalaf et al., 2016). Atherosclerotic CVD, previously the primary source of passing away in the United States of American, is predicted to be the most common cause of death in all parts of the world by the year 2020 (Ateyah et al., 2021). Cardiovascular disease is the leading cause of both death and premature death in China, the cause of 40% of deaths in the Chinese population, China and India have the highest burdens of CVD (Zhao et al., 2019). One of the major cardiovascular illnesses and the most common cause of deaths in developing as well as industrialized countries is coronary artery disease (CAD) (Malakar et al., 2019). The Middle East and North Africa (MENA) area has many countries with a high prevalence of CAD, which suggests that cardiovascular morbidity and death will continue to be a major problem (Laher, 2020). In the Middle East, increased metabolic dyslipidemia is more prevalent than in other locations (Traina et al., 2017). Oman and Kuwait had the largest percentage of deaths attributable to cardiovascular disease (49% and 46%, respectively), while SaudiyArabia, the United Arab Emirates, Bahrain, and Qatar also had a sizable

percentage (42%, 38%, 32%, and 23%, respectively) (Kalaf et al., 2016). In Iraq, the mortality rate from cardiovascular disease was projected to be 33% in 2014 (Mehsen et al., 2020).

2.1.3. Symptom

Early control of risk factors is essential in the management of atherosclerosis because AS does not exhibit evident symptoms in its early stages (Chen *et al.*, 2022). The symptoms of AS vary because it is a systemic disease that often affects all of the body arteries (Héraud, 2017). Angina (chest pain) and myocardial infarction (MI) can result from the main coronary artery atherosclerosis that reduces the blood flow to the heart (Shenton *et al.*, 2021). Atherosclerotic causes the arteries feeding the lower limbs to thin, which frequently causes intermittent claudication, which is characterized by pain or cramping when walking (Harwood *et al.*, 2020).

2.1.4. Causes

- Genes: genes associated with lipid metabolism and linked with inflammation are thought to play a role in atherogenesis (La Sala *et al.*, 2019). Numerous genes have been shown to be important for the individual's susceptibility to atherosclerosis. mutations and variations in genes that raise the chance of atherosclerosis (Poznyak *et al.*, 2022).
- Dyslipidemia: circulating low-density lipoprotein (LDL) is the primary form of lipids that deposit in atherosclerotic plaques, and it plays key role in the growth of atherosclerotic lesions by causing lipid buildup in the artery walls (Summerhill *et al.*, 2019).
- Atheroma: the most frequent reason for acute coronary syndromes (ACS) and sudden fatalities is atheromatous plaques (Hegazy & Hegazy, 2022).

2.1.5. Risk factors

- Aging: growing older is a major risk factor for atherosclerotic cardiovascular disease since it is linked to a reduction in the function of mitochondria and a

rise in IL-6 levels in the blood vessels, both of which likely promote AS independently of chronic hyperlipidemia (Tyrrell & Goldstein, 2021).

- Gender: women often experience less CVD than men do (Gao *et al.,* 2019). A lack of the hormone testosterone affects men who have atherosclerosis (Auda *et al.,* 2021).
- Smoking: is an individual risk factor for atherosclerosis, nicotine inhibits the activity of endothelial nitric oxide synthase (eNOS), which in turn reduces the production and bioavailability of nitric oxide (NO), and increases the oxidative stress that results (Digiacomo *et al.,* 2019).
- Diet and physical activity: atherosclerosis can be prevented, delayed, and treated through diet and exercise (Al-Sharea *et al.*, 2019).
- Obesity: a significant predictor for atherosclerotic cardiovascular disease, a person's probability of developing AS increases by 10% for every point their body mass index (BMI) rises above normal weight (Henning, 2021).
- Other diseases: because diabetic macro vascular disease causes an imbalance in vascular homeostasis brought on by the malfunctioning of ECs and SMC, which eventually results in atherosclerotic thrombosis, insulin resistance increases the prevalence of atherosclerosis in individuals with T2DM (Wei *et al.,* 2022).

2.1.6. Complications of atherosclerosis

As a result of medium- and large-sized arteries being affected, the lumen eventually becomes compromised, causing ischemic syndromes that affect the heart, brain, and extremities (Shah, 2019). Fatty streaks in the artery walls gradually transform into atheroma and distinctive plaques, rapid rupture of these atheromatous plaques induces local thrombosis, the clinical consequences of these plaques depend on their location and the extent and speed of artery blockage (Herrington *et al.,* 2016). Plaques that cause a coronary channel to suddenly become blocked, seriously

endangering cardiac and, if left untreated, myocardial infarction (MI) and irreversible heart attack (Janjusevic *et al.*, 2022). In essence, plaque atherosclerosis accumulation leads to chronic complete occlusions (Artery *et al.*, 2022). Acute cardiovascular events, including several occurrences of sudden cardiac death, acute myocardial infarction, and unstable angina (Partida *et al.*, 2018).

2.1.7. Diagnosis

Complete family and personal medical histories, as well as basic (first-line) diagnostics such as a resting electrocardiogram (ECG), potentially ambulatory monitoring, resting echocardiogram, and a chest X-ray, may all be used to diagnosis AS (Artery *et al.*, 2022).

- Electrocardiogram (ECG): acute coronary syndrome (ACS) mortality and disability can be reduced by early diagnosis with an ECG, which is an initial-line diagnostic method for individuals with chest discomfort (Cho *et al.*, 2019).
- Intravascular ultrasound (IVUS) or optical coherence tomography (OCT) have been employed to determine the existence, size, degree, and features of coronary atherosclerosis, and they have long helped explain the causes of MI (Nakanishi *et al.*, 2019).
- Cardiac computed tomography: assist a main diagnostic test in the evaluation of suspected coronary artery disease and is an effective way to find the patient's susceptible plaque (Oikonomou *et al.*, 2019).
- Computerized tomography (CT) scan: any thickening and constriction of the body's major arteries can be seen with this form of X-ray (Roth *et al.*, 2020)

2.1.8. Treatment

Treatment for AS may need changes in lifestyle, such as more exercise, dietary modifications, body weight management, and quitting smoke (Nording *et al.*,

2020). However, if you have serious symptoms or an obstruction, medicines or surgical procedures can be necessary (Zhang *et al.*, 2019).

2.1.8.1. Medication

- Cholesterol medications: the most significant therapeutic drugs for treating AS are statins (HMG-CoA reductase inhibitors), That is primarily prevent and manage atherosclerosis because of their ability to decrease cholesterol and their anti-inflammatory actions (Michos *et al.*, 2019).
- Antiplatelet medications like aspirin are recommended to stop platelets from clumping in the small arteries, forming blood clots, and causing further blockages (Lordan *et al.*, 2021).
- Fibrinolytic therapy: aims to dissolve the thrombus by activating plasminogen, resulting in the formation of plasmin, which cleaves the fibrin cross links within the thrombus (Vogel *et al.*, 2019).
- Antihypertensive drugs: such as beta-blocker, leading to reduction in cardiac output and vasodilatation, and angiotensin converting enzyme inhibitors, leading to vasodilation and large artery destiffening (Laurent , 2017).

2.1.8.2. Surgical

- Angioplasty: has since taken over as an especially popular treatment for vascular problems involving the heart and the peripheral arteries, endarterectomy surgery is required to remove plaque from the balloons (Barton *et al.*, 2014).
- Coronary Artery Bypass Graft (CABG) surgery: is still the preferred course of treatment for individuals with multiple vessel coronary artery disease (CAD), particularly those who have diabetes or ischemic heart failure (Pölzl *et al.*, 2022).

- The most common procedure for vascularizing the coronary arteries is percutaneous coronary intervention (PCI) with the placement of a coronary stent (Jung *et al.,* 2018).
- Angioplasties with stenting are extremely effective, minimally invasive endovascular procedures that widen stenosis arteries (Burle *et al.,* 2022).

2.1.9. Prevention

Making healthy lifestyle adjustments, including giving up smoking, eating well, maintaining a healthy weight, and monitoring and maintaining normal cholesterol and blood sugar levels, can maintain the arteries in good condition (Koller *et al.,* 2022). Exercise can improve health even if you don't lose weight because it lessens the harmful physiological effects of obesity and its comorbidities (Petridou *et al.,* 2019). To preserve vascular function, aerobic exercise encourages the release of nitric oxide, lowers blood pressure, and induces antioxidant enzymes (Huang *et al.,* 2021).

2.2. Obesity

Obesity is a significant worldwide health problem linked to higher morbidity and mortality rates (De Lorenzo *et al.,* 2019). According to the WHO, obesity is defined as "abnormal excessive fat accumulation that presents risk to health" (Blüher, 2020). The buildup of too much body fat results in a variety of metabolic disorders and diseases, such as resistance to insulin and atherogenic dyslipidemia (Klein *et al.,* 2022). Figure (2) shows the role of dysfunctional adipose tissue in the development of obesity associated health risks. Obesity poses a serious threat to public health since it adversely affects almost all bodily physiological processes and raises the chance of acquiring a number of disease conditions, including DM and CVD (Chooi *et al.,* 2019). Obesity is primarily brought on by a chronic energy imbalance between calories taken and calories burned (Lin & Li, 2021). Therefore, obesity-related unbalanced adipocyte production may aid in an increase of metabolic

and cardiovascular problems (Shibata *et al.,* 2017). Body mass index (BMI) was used to categorize people into four categories: underweight (18.5kg/m-2), normal weight (18.5–24.9% kg/m-2), overweight (25.0–29.9% kg/m-2), and obese (> 30 kg/m-2) (Ciroma *et al.,* 2017). Obesity is not just a problem for adults, since there were over 1.9 billion overweight adults in 2016 and over 650 million obese individuals, in 2016, 340 million kids and teenagers aged 5 to 19 and over 40 million young children under 5 were considered overweight or obese (Bentham *et al.*, 2017). Sedentary behavior is a recurring problem in the Arab World, aggravating the region's continuous obesity and metabolic disease crises, which affects about 60% of the population (Sharara *et al.*, 2018). In addition to being considered as chronic inflammatory disease states with activation of both innate and adaptive immunity, obesity and atherosclerosis are now also seen as lipid storage diseases including triglycerides in fat tissue and cholesteryl esters in atheroma (Mandviwala *et al.,* 2016).

Figure 2: Role of dysfunctional adipose tissue in the development of obesity associated health risks (Manna & Jain, 2015)

2.3. Biomarkers

2.3.1. Adiponectin

Adiponectin is type of adipokines are released by adipose tissue, which is a tissue that is found throughout the body and is considered an important endocrine system (organ), adipokines are essential for the control of immunological and energy responses (López-Ortega *et al.,* 2022). Adiponectin is a factor exclusive to adipocytes that was originally identified in 1995 (Maeda *et al.*, 2020). Adiponectin has a molecular weight of 28 kDa and 244 amino acids, and is involved in a number of physiological processes, such as lipid metabolism, energy control, immunological response, and inflammation (Khoramipour *et al.,* 2021). Multiple names for adiponectin include (Acrp30, AdipoQ), it exerts its physiologic effects through three widely dispersed receptors called AdipoR1, AdipoR2, and T-cadherin (Parida *et al.*, 2019). Adiponectin's physiological actions are triggered by related receptors in conditions like obesity, inflammation, and CVD (Ishtiaq *et al.*, 2019). Adiponectin is a multimeric protein that can occur as trimmers (lower molecular weight, LMW), hexamers (middle molecular weight, MMW), and high molecular weight (HMW) multimers (Solarewicz *et al.*, 2019). HMW adiponectin may be particularly significant since it possesses insulin-sensitizing and vasoprotective effects via its interactions with AdipoR1 and AdipoR2 receptors (Achari & Jain, 2017). In figure (3) show the source, function, effect of adiponectin. Adiponectin is inversely connected with fat mass, makes up to 0.05% of the total proteins in the plasma, and is low in obese individuals (Zhang *et al.*, 2020). Decreased adiponectin levels have been linked to metabolic syndrome, cardiovascular disease (CVD), and hypertension, it has a wide range of biological effects, including anti-diabetic, anti-atherogenic, anti-inflammatory properties (Meshkini *et al.,* 2018). In skeletal muscle, adiponectin deficiency promotes fatty acid oxidation (Wang & Scherer, 2016). In contrast to several other "adipokines" that rise with obesity, such as tumor

necrosis factor- (TNF-), leptin, and resistin (Mallardo *et al.*, 2021). It has been demonstrated that adiponectin levels are inversely correlated with carotid artery intima-media thickness (IMT), a marker of early atherosclerosis (Shibata *et al.*, 2017). Additionally, adiponectin changes the pro-inflammatory M1 macrophage phenotype to the anti-inflammatory M2 (Sharma *et al.*, 2021). It is well known that men's serum levels of adiponectin are lower than women's (Lindberg *et al.*, 2017). Adiponectin promotes fatty acid oxidation while suppressing hepatic gluconeogenesis and lipolysis (Shabalala *et al.*, 2020). Dyslipidemia and low adiponectin levels are correlated (Ghoshal *et al.*, 2021). High levels of adiponectin have been linked to these physiological processes because they increase in HDL-C, which is known to be good for the cardiovascular system (Ciroma *et al.*, 2017).

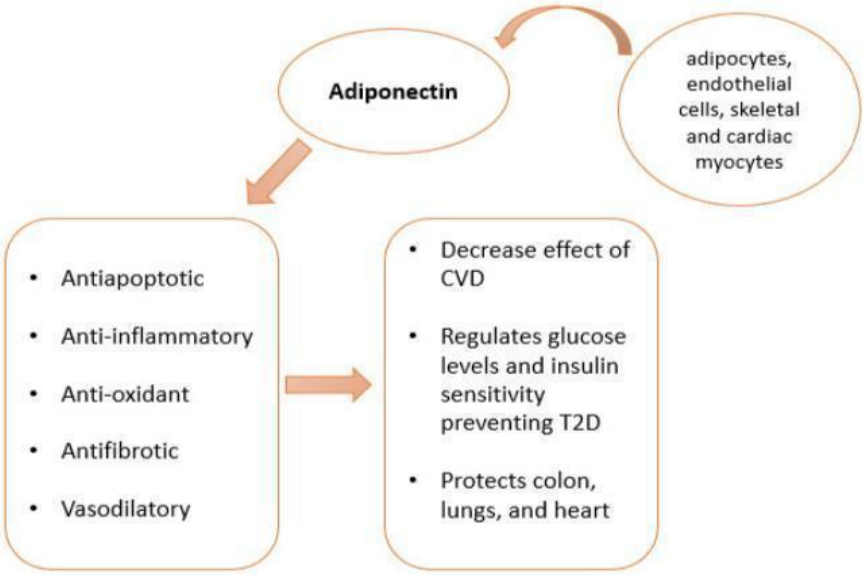

Figure 3: Adiponectin source, function, effect (Aljafary & Al-Suhaimi, 2022)

2.3.2. Leptin

The obese gene encodes leptin, a single-chain 16 kDa protein that is mostly released by adipocytes but can also be generated by vascular smooth muscle cells (VSMC) and cardiomyocytes (D'Marco *et al.*, 2020). In the 1990s, leptin was the

first adipokine to be discovered, it is known to reduce food intake by reducing hunger and to regulate energy balance, including blood sugar and lipid metabolism (Szekeres *et al.,* 2023). Produced mostly by adipose tissue in relation to the volume of fat reserves, with a key role in the management of lipid reserves (Picó *et al.*, 2022). Leptin is a well-known proinflammatory adipokine that is thought to contribute together to a "low-grade inflammatory state" observed in overweight and obese people (Recinella *et al.,* 2020). Fasting Leptin levels between obese and lean individuals showed an inverse relationship with cerebral gray matter volume (Battineni *et al.,* 2021). The altered leptin transport over the blood brain barrier (BBB) into the hypothalamus caused by hypertriglyceridemia may be key factors in the start and progression of obesity (Gong *et al.,* 2018). Leptin levels and BMI are positively correlated, and it's thought that hyperleptinemia, which is linked to adipose tissue in obese people without the predicted hunger reduction, may signify a leptin resistance state (Goh *et al.,* 2022). An increase in food intake and a decrease in energy expenditure are caused by a loss of leptin signaling, which can result from mutations in leptin or its receptors (Gruzdeva *et al.*, 2019). Leptin levels may rise as a result of inappropriate or excessive fat storage in the context of obesity, resulting in a condition known as "leptin resistance" when leptin signaling is reduced, and Leptin loses its ability to suppress feeding during this physiological resistance, increasing energy expenditure (Izquierdo *et al.,* 2019). Resistance to leptin's catabolic effects has been explained by a number of mechanisms, including issues with leptin transport across the blood-brain barrier, changes in development programming, and/or variations in leptin expression of receptors (Martínez-Sánchez, 2020). Leptin is a proatherogenic substance that plays a special role in the pathogenesis of CVD (Mućka *et al.*, 2022). Human obesity-related hyperleptinemia or leptin resistance affects the endothelium cardiovascular structure and functioning, inflammation, and sympathetic nerve activity and may result in cardiovascular

disease (Kang *et al.*, 2020). Figure (4) show the effect of hyperleptinemia on vascular macrophage.

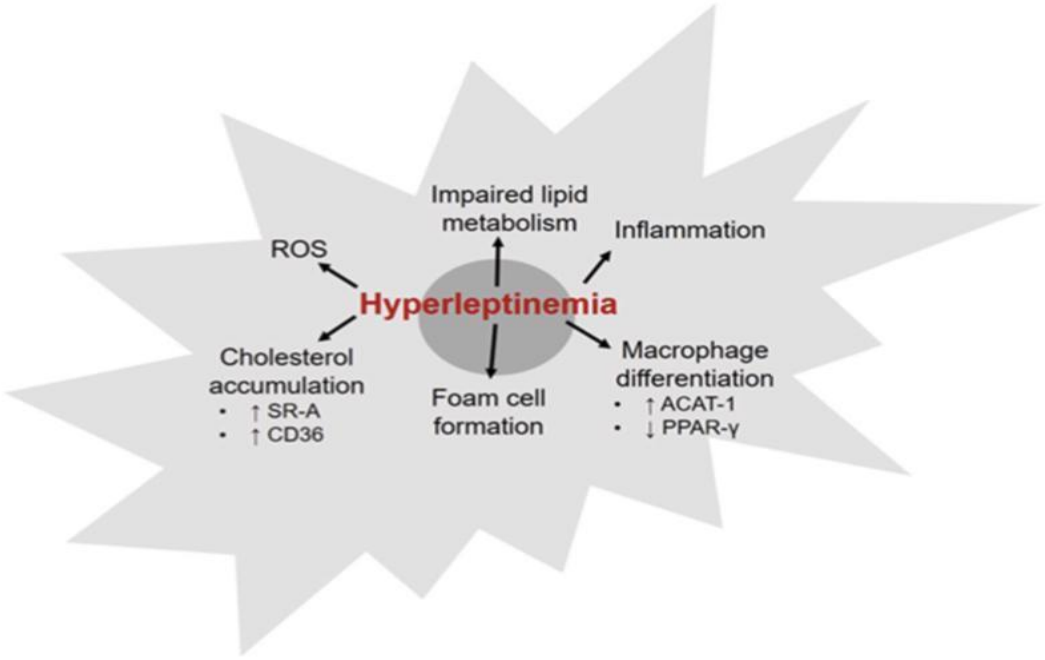

Figure 4: Hyperleptinemia-induced changes on vascular macrophages that contribute to atherosclerosis. ROS: reactive oxygen species; PPAR-γ: peroxisome proliferator-activated receptor-γ; SR-A: scavenger receptor-A; ACAT-1: acyl CoA: cholesterol acyltransferase-1 (Raman & Khanal, 2021)

2.3.3. Plasminogen activator inhibitor type-1 (PAI-1)

PAI-1his a single-chain glycoprotein with a 45-kDa mass and 379 or 381 amino acids (Sillen & Declerck, 2021). PAI-1 is primarily produced by Platelet precursor cells, megakaryocytes, although it is also produced by endothelial, adipocyte, hepatocyte, and cardiomyocyte cells (Morrow *et al.*, 2021). Aging, obesity, diabetes, heart disease, and cerebrovascular illness are only a few of the conditions for which adipose-derived PAI-1 has been identified as a key mediator (Bruno *et al.*, 2022). Obesity and metabolic syndrome, including increased plasma levels of the PAI-1 (Yarmolinsky *et al.*, 2016). The fibrinolytic system consists of a

balance between rates of plasminogen activation and fibrin degradation, both of which are finely regulated by spatiotemporal mechanisms, three distinct inhibitors of the fibrinolytic system that differently regulate these two steps are plasminogen activator inhibitor type-1 (PAI-1), α2-antiplasmin, and thrombin activatable fibrinolysis inhibitor (TAFI) (Urano *et al.,* 2019). The primary protease of the fibrinolytic system, plasmin is essential for cell migration and remodeling of tissues because it can break down extracellular matrix proteins by activating metalloproteases, plasmin is produced from plasminogen by the endogenous enzymes tissue-type plasminogen activator (tPA (Simon & Simon, 2013). Increased plasma levels of PAI-1 have been proven to have significant effects on the onset and progression of cardiovascular illnesses (Ploplis, 2011). In people, PAI-1 over-expression is associated with atherosclerosis, especially in those who have the metabolic syndrome, which is marked by overweight or obese people, dyslipidemia, and high blood pressure (Khoukaz *et al.,* 2020). Hepatic lipid metabolism is also significantly regulated by PAI-1 (Levine *et al.,* 2021). Fibrous deposit in plaques can be eliminated by plasminogen activators, and fibrinolytic imbalances contribute to the course of AS (Ormazabal *et al.,* 2018).

2.4. Oxidative Stress (OS)

OS refers to oxidative stress, which is the word for an imbalance between antioxidants pro-oxidant compounds in cells, OS may cause cellular damage interfering with the state of proteins, DNA, and lipids and is associated with the formation of many diseases (Yaribeygi *et al.,* 2019). Oxidative stress is considered to be crucial in several clinical illnesses (Sifuentes-Franco *et al.,* 2017). Reactive Oxygen Species (ROS), which are naturally produced by a variety of metabolic processes and are produced in minute quantities during normal metabolism, are very reactive and take electrons from any substance that stands in their way, ROS can

harm cells when they are present in higher concentrations (Walton, 2017). Among the radicals that make up ROS include hydroxyl, superoxide, hydroperoxyl, and peroxyl, non-radical ROS examples include hydrogen peroxide and hypochlorous acid, while reactive nitrogen species (RNS) examples include nitrogen dioxide, nitric oxide, nitrous oxide, and peroxynitrite, which are produced in minute amount during normal physiological conditions (Ganjifrockwala et al., 2017). Endothelial cells deposit cholesterol-rich low density lipoprotein (LDL) in the membrane's inner layer of the vascular wall, where it is oxidized and transformed into oxidized LDL under conditions of elevated oxidative stress (Y. Wang *et al.,* 2021). OS refers to a series of actions that frequently initiates and follows molecular/cellular pathogenic events that cause a number of human illnesses, such as cardiovascular disease, atherosclerosis, and cancer(Alfei *et al.*, 2020).

2.4.1. Malondialdehyde (MDA)

Malondialdehyde (MDA), a small, reactive chemical complex containing 2 groups of aldehyde at the carbon 3 and carbon 1 positions, is present in all eukaryotes (Morales & Munné-Bosch, 2019). MDA is a toxic aldehyde that can covalently bond to other biomolecules including DNA, lipids, or proteins and is frequently employed as an indicator of stress caused by oxidation in biological materials (Busch & Binder, 2017). MDA is a constant end product of a chain events known as peroxidation of lipids, which produces a constant supply of radicals called free radicals that start more peroxidation (Cui *et al.,* 2018). MDA plasma levels not only indicate the degree of lipid peroxidation but additionally the severity of OS) (Maurya *et al.,* 2021). Lipid peroxidation is a condition where OS causes an excess of ROS or RNS, which can then combine with other biomolecules like lipids to produce various chemicals like MDA (Isola *et al.,* 2019).

2.4.2. Total antioxidant capacity (T-AOC)

An antioxidant is a substance that may prevent oxidation of a substrate while operating at lower levels than the substrate being protected (Pisoschi *et al.*, 2021). Enzymes neutralize free radicals, proteins like transferrin may bind to metals that promote their formation, vitamins E and serve as free radical scavengers, and vitamin C, which is a water soluble molecule, usually scavenges hydroxyl radicals,vitamin E, lipid soluble vitamin, breaks down the chain reactions of lipid peroxidation (Ganjifrockwala *et al.,* 2017). Important physiological processes involving glutathione have consequences that are relevant to a variety of diseases and pathologies, including the preservation of redox balance, decreased oxidative stress, improvement metabolism detoxification, and control of of the immune system's activity (Minich & Brown, 2019). It is recognized as the most important antioxidant for protecting the cell membrane from oxidative damage brought on by free radicals (Maciejczyk *et al.*, 2021).

An antioxidant is a molecule that is capable of "neutralizing" the oxidation of ROS before they react with cellular biomolecules and change their structure or function (Salvayre *et al.,* 2016). Antioxidant include the enzymes superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) and lipid-soluble antioxidants, such as glutathione, ascorbate (vitamin C), α- tocopherol (vitamin E), and β-carotene, and also endogenous antioxidant, for example, albumin, bilirubin (Gutiérrez-Salinas *et al.*, 2013).

Antioxidant defense has two levels:

- Primary defense mechanism: this defense mechanism inhibits oxidative damage directly by scavenging free radicals before they can damage intracellular biomolecules, endogenous enzymes play an important role in this step (Goszcz *et al.*, 2015).

- Secondary defense mechanism (chain-breaking defense): Vitamin C, and vitamin E, also nuclear enzymes which participate in DNA repair can be considered as a secondary defense system against oxidative damage caused by oxygen free radicals (Siekmeier *et al.,* 2007).

2.5. Physiological parameters

2.5.1. Troponin I

Troponin is a protein, which are a member of the contractile apparatus in skeletal and cardiac muscle, control and promote the connection between actin and myosin filaments, as part of the sliding filament process of muscle contraction (Garg *et al.,* 2017). Troponin releases into the circulation when myocytes are injured (Sorodoc *et al.*, 2022). Troponin T (TnT), Troponin I (TnI), and Troponin C (TnC) are the three troponin isoforms (subunits) that make up the troponin complex, which is the most significant regulation of the contraction/relaxation of striated muscle tissues (Jin, 2016)**.** The processes of cTnI and cTnT leakage in the early stages of many pathological illnesses may be linked to a rise in the permeability of cell membranes and/or intracellular fragmentation of cTnI and cTnT molecules into smaller pieces that may easily flow through intact cell membrane (Chaulin, 2021). After strenuous activity, cardiac troponin is connected to measures of plaque susceptibility or coronary atherosclerosis (Paana *et al.,* 2019). Although cTnT and cTnI are expressed in heart tissue in approximately equal amounts in patients with myocardial necrosis, such as in acute myocardial infarction (MI), cTnI frequently reaches peak levels ten times higher than cTnT (Janssen *et al.,* 2022). In obese people, troponin I serves as an indicator of myocardial damage (El Saiedi *et al.,* 2018).

2.5.2. C-Reactive Protein (CRP)

is a type of protein, mainly produced by liver organ, it can be elevated in plasma patients with acute inflammation (Fu *et al.*, 2020). Structurally, CRP is a 206 amino acid cyclic pentameric protein with five identical subunits that are not covalently bonded, excessive body weight that has been associated with elevated CRP levels (Stanimirovic *et al.*, 2022). Since fat cells secrete a variety of inflammatory molecules called adipocytes, including leptin, adiponectin, and others, obese people constantly have high levels of inflammation (Kim & Yeun, 2022). Serum-CRP is generally 0.5 mg/dl in healthy individuals and is a not specific biologic measure of systemic inflammation that may be raised in chronic as well as acute inflammation (Pourhassan *et al.*, 2022). In response to infection or tissue injury, CRP levels increase significantly, ranging from 5–10 mg/L in mild instances to 320–550 mg/L in the greatest severe cases (Melnikov *et al.*, 2023). Because of its capacity to bind to change LDL and its effect on the functionality of ECs, the stability of plaque, and thrombosis, CRP is believed to have a part in atherogenesis (Schenkein *et al.*, 2020). Subclinical atherosclerosis, intima media thickness (IMT), the presence of plaque, and total plaque area are all associated with CRP (Eltoft *et al.*, 2017).

2.6. Dyslipidemia

An increased serum total cholesterol (TC), TGs, LDL-C, and decreased HDL-C levels are symptoms of the metabolic condition dyslipidemia, which progresses to an atherogenic metabolic disorder and causes cardiovascular problems (Ghoshal *et al.*, 2021). Low Levels of HDL-C, an accumulation of small dense LDL particles, and high triglyceride levels in both the fasting and postprandial phases are the hallmarks of atherogenic dyslipidemia (Björnson *et al.*, 2017). Apolipoproteins, which are structural components that serve as

ligands for cell-surface receptors and cofactors for a number of enzymes including lipoprotein lipase (LPL), are present in abnormal amounts of lipids (Ding *et al.*,2022). Apo lipoproteins may alter the generation, transformation, or catabolism of lipoprotein particles, these modifications may be responsible for obesity's enhanced basal lipolysis and the subsequent release of fatty acids into the bloodstream, which has a proatherogenic character (Ormazabal *et al.*, 2018). The amount of fatty streaks was inversely correlated with HDL cholesterol levels and favorably correlated with postmortem LDL cholesterol and VLDL cholesterol (VLDL-C) concentrations (Hari *et al.*, 2020).

2.6.1. Total Cholesterol (TC)

A large number of cells can generate cholesterol, and in humans, the liver contributes for around half of the overall production (Hong *et al.*, 2020). Cholesterol is an essential part of eukaryotic membranes in cells, involved in regulating membrane fluidity, organization, and other physicochemical elements (Chakraborty *et al.*, 2020). Additionally, a range of sterol transportation proteins connect cholesterol to aid in trafficking and control its subcellular distribution, cholesterol has the potential to interact with an array of transmembrane proteins, protecting or modifying their conformations (Luo *et al.*, 2020). Convert cholesterol to cholesteryl esters that are either released as a vital component of plasma lipoproteins like LDLs, VLDLs, and HDLs or retained as a cholesterol storage in the cytosolic lipid droplets (Subczynski *et al.*, 2017). Atherosclerosis and CAD are both greatly increased by hypercholesterolemia (Adam *et al.*, 2020).

2.6.2. Triglycerides (TGs)

Three molecules of fatty acids (unsaturated, saturated, or both) are joined to a single glycerol molecule by ester bonds to form TGs, which are natural fats (Laufs *et al.*, 2020). In the human body, TGs serve as a major cellular energy source for the transfer of dietary lipids, their typical serum concentration range is between 40 and 150 mg/dL (Hasanah *et al.*, 2019). Chylomicrons originating from the intestinal tract carry exogenous (dietary) TG, whereas VLDL derived from the liver carries endogenous TG (Lewis *et al.*, 2015). Fatty acids (FAs) rise from the energy stored in TGs in lipid droplets created by lipogenesis in adipose tissue through the process of lipolysis (Blomquist *et al.*, 2018). The very low density lipoprotein (VLDL) and its remnants produced during the metabolism of TG are chylomicrons (CM), a major component of triglyceride-rich lipoproteins (TRLs), which also contain TG (Peng *et al.*, 2017). Atherosclerotic coronary artery disease (ASCVD) has been associated with TGs as a prevalent risk factor (Chapman *et al.*, 2011).

2.6.3. Low-density lipoprotein (LDL) cholesterol

LDL's physiological function is to supply cells in extrahepatic tissues with cholesterol, which they then attach to and integrate using their plasma membrane receptors (Maaninka, 2018). It is widely acknowledged that oxidized low-density lipoprotein (oxLDL) is a patho-atherogenic lipoprotein. Atherosclerosis, which results in CAD, is more likely to develop when there is higher serum concentration of oxLDL (Chunta *et al.*, 2020). Low-density lipoproteins (LDLs), sometimes known as "bad LDL," are oxidized LDL (Kolonin, 2021). High blood LDL-C concentrations are an indicator of risk

for CVD, however different sizes of LDL particles may have distinct effects on how the disease develops (Froyen, 2021).

2.6.4. Very Low Density Lipoprotein (VLDL)

VLDL is released from the liver into the bloodstream, by endothelial lipoprotein lipase in the blood vessels of muscle and fat tissue causes the release of fatty acids that are free from these triacylglycerol (TAGs) found in the VLDL core (Chapman *et al.*, 2011). Triglyceride (TG) makes up 50–70% of the particle mass carried by VLDL, with cholesterol ester (CE) making up the remaining 10–25% and fatty acids making up less than 10%, Apolipoproteins (Apo) B–100, C–I, C–II, C–III, and E make up the outer layer proteins of VLDL (Huang & Lee, 2022). A diet high in fat and excessive calorie intake increase the release of VLDL (Lambert & Parks, 2012). Additionally, vascular stiffness and carotid intima-media thickness are linked to VLDL (Gentile *et al.*, 2020).

2.6.5. High-density lipoprotein (HDL) cholesterol

The smallest and densest lipoprotein [Lipoproteins are complex particles with a central core of free cholesterol, phospholipids, and a number of apolipoproteins (Apo) that are essential to lipoprotein creation and function (Sidorkiewicz, 2023)] in plasma are called HDL,both ApoA- II and ApoA-I, the two main HDL apolipoproteins, are necessary for typical HDL biosynthesis (Srivastava *et al.*, 2018). HDL, a lipid, transfers extra cholesterol across peripheral tissues to the liver for storage and breakdown (Beverly & Budoff, 2020). Consequently, it is referred to as "good cholesterol" (Xiang & Kingwell, 2019). There is a distinct inverse correlation between serum high-density lipoprotein cholesterol levels and risk for CHD (Kosmas *et al.*, 2018).

Low HDL-C is the main indicator of dyslipidemia in obese people (Zhang *et al.*, 2019)**.**

Chapter Three

Materials and Methods

Chapter Three

Materials and Methods

3.1. Materials

3.1.1. Chemicals and Kits

Table 3-1: Kits and Chemicals that have been utilized in this study.

No	Kits	Companies	Origin
1	CRP kit	Abbott	USA
2	Cholesterol kit	DIRUI	China
3	Ethanol	Teeba	Iraq
4	HDL kit	DIRUI	China
5	Human Adiponectin ELISA Kit	BT LAB	China
6	Human Leptin ELISA Kit	BT LAB	China
7	Human MAD ELISA Kit	BT LAB	China
8	Human PAI- 1 ELISA Kit	BT LAB	China
9	Human T-AOC ELISA Kit	BT LAB	China
10	Human Troponin I ELISA Kit	BT LAB	China
11	LDL kit	DIRUI	China
12	Triglyceride kit	DIRUI	China

3.1.2. Tools and Devices.

Table 3-2: Tools and devices of this study.

NO	Devices and Tools	Companies	Origins
1	Centrifuge	BIOBASE ROTOFIX 32A	Germany
2	Deep freeze	ALS	Italy
3	Disposable syringe 5ml	EASYMED	China
4	ELIZA apparatus	Bioteck	U.S.A
5	Eppendorf tube	Carl ROTH	Switzerland
6	Gel tube	Vacuum blood collection tubes	Iraq
7	Gloves	Mumu plus +	Malaysia
8	Mask	Disposable 3-layer Mask	China
9	Micropipettes	Micropipettes	Germany
10	Refrigerator	LG	South Korea
11	Tourniquet	Voltaren	China

3.2. Methods

3.2.1. Study Design

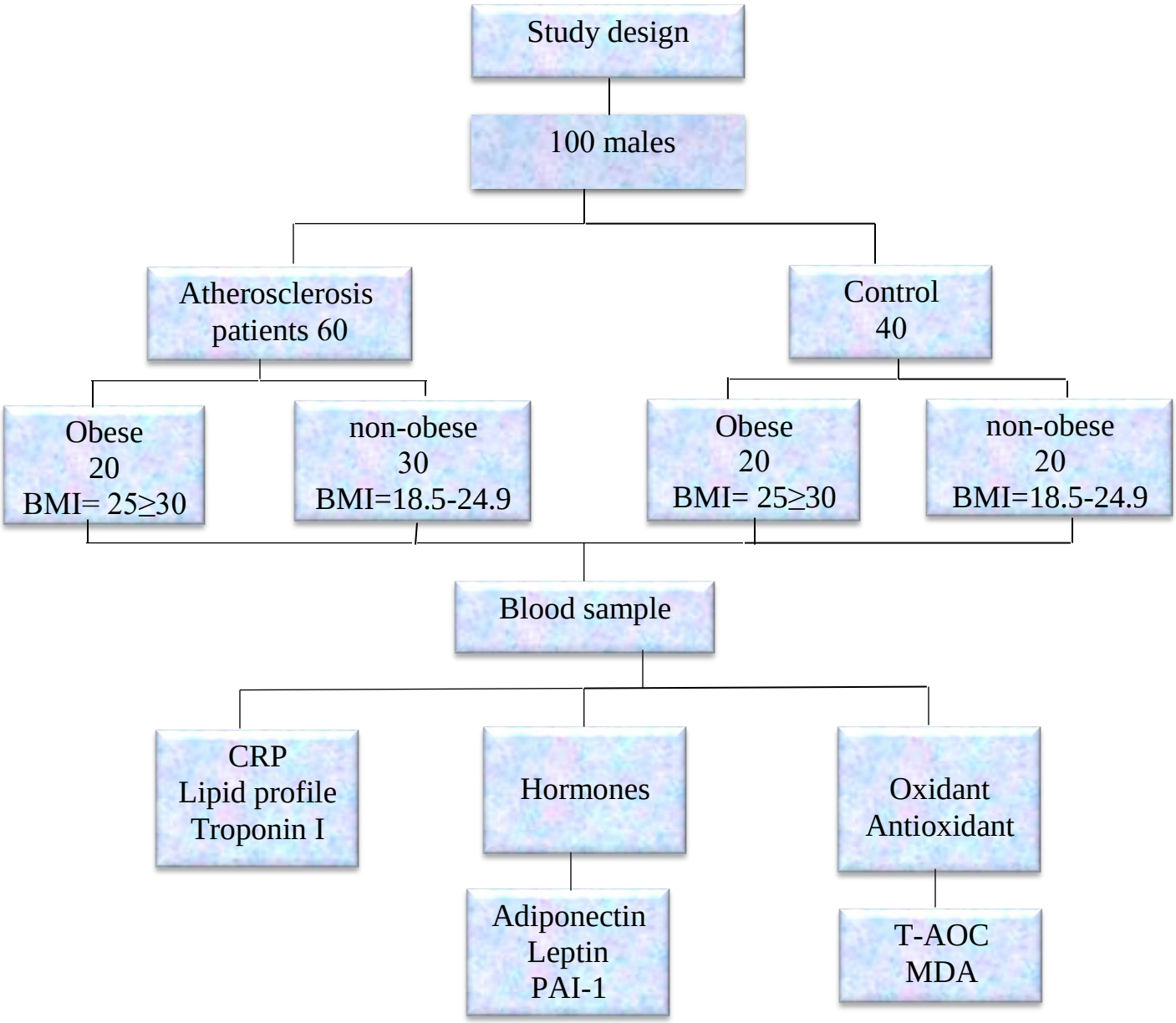

Figure (3-1): Study Design

3.2.2. Patients

A case-control studt was used in the design of the current investigation. 100 participants in the present study were divided as follows: 60 atherosclerosis individuals, 30 obese patients, 30 normal weight patients, and 40 healthy people, 20 of who were obese and 20 of who were of normal weight, had serum samples drawn. In Iraq, Karbala the work was conducted during November 2022 to May 2023. Participants from the Karbala Center for Cardiac Disease and Surgery constituted the majority of the group. The questionnaire form, which inquired about each participant's age, weight, height, and other details, was used to capture their data.

3.2.2.1. Collection Data

The study's participants were all affected by the condition atherosclerosis. Their BMI was calculated based on their height and weight.

3.2.2.2. Collection samples

Each participant had their venous blood collected using a disposable syringe for a total of (5ml) was divided into two gel tubes and left at room temperature for about 30 minutes to coagulate. The first gel tube containing the serum was utilized to automatically calculate the amount of lipids, CRP, after the gel tubes were centrifuged at 4000 x g for five minutes to extract the serum. The second gel tube that was transferred to an eppendorf tube and kept at a temperature of (-30 C°) to measurement of Adiponectin, leptin, plasminogen activator inhibitor-1, malondialdehyde (MDA), total antioxidant capacity (TAO) and Troponin I.

3.2.3. Determination of Body Mass Index (BMI)

The BMI was calculated using the formula shown below:

BMI = Weight (kg) / Height (meters)^2 (Cnop *et al.*, 2003).

According to BMI, patients' and control groups' weight status was categorized in the table (3-3).

Table 3-3: Based on their BMI, weight status was classified.

Weight Status	(BMI)(kg/m^2)
Normal weight	18.5 to 24.9
Obese	25 to more than 30

3.2.4. Hormones

3.2.4.1. Determination of Human Adiponectin Level

- **Principle:**

This ELISA kit uses the Sandwich-ELISA. The micro ELISA plate has been pre-coated with an antibody specific to Human ADP/Acrp30. Samples are added to wells and combined with the specific antibody. Then a biotinylated detection antibody specific for Human ADP/Acrp30 and Avidin-Horseradish Peroxidase (HRP) conjugate are added successively to each microplate well and incubated.The substrate solution is added. Only those wells that contain Human ADP/Acrp30, biotinylated detection antibody and Avidin-HRP conjugate will appear blue in color. The enzyme-substrate reaction is terminated by the addition of a stop solution and the color turns yellow. The optical density (OD) is measured spectrophotometrically at a wavelength of 450 nm (Biosciences, 2019).

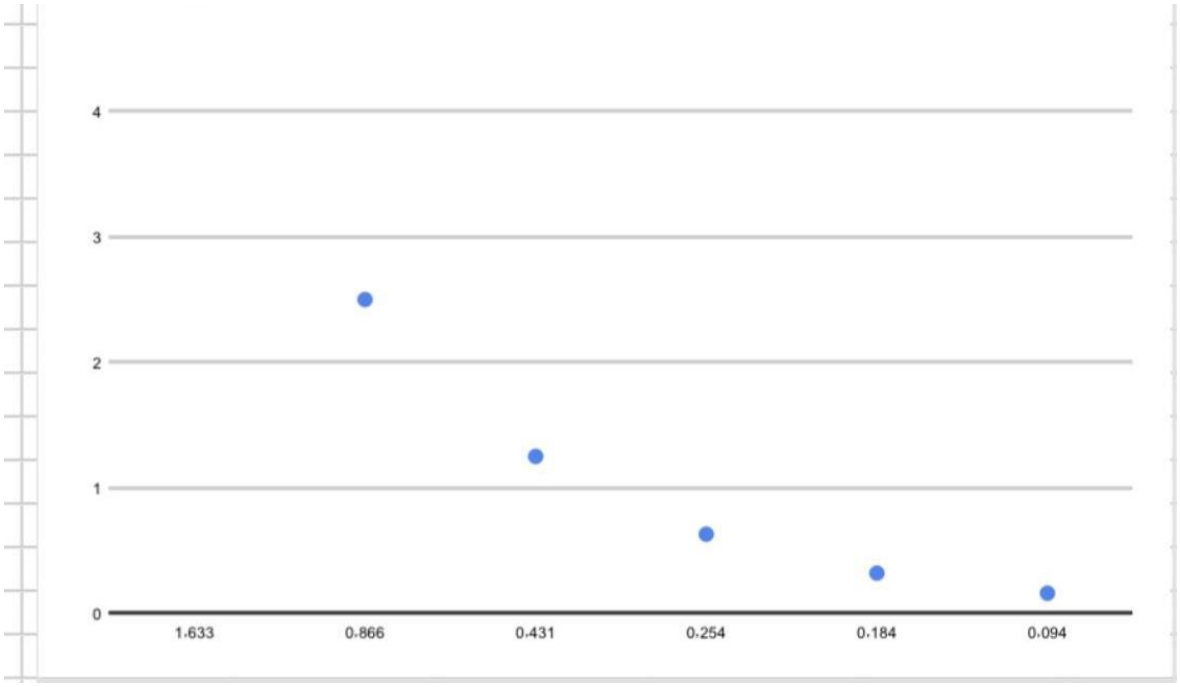

Figure (3-2): Standard curve for Adiponectin

3.2.4.2. Determination of Human Leptin Level

- **Principle:**

This ELISA kit uses the Sandwich-ELISA principle. The micro ELISA plate has been pre-coated with an antibody specific to Human LEP. Samples and biotinylated detection antibodies specific for Human LEP are added to the wells. Human LEP would combine with the specific antibody. Then Avidin-Horseradish Peroxidase (HRP) conjugate are added successively to each well and incubated. The substrate solution is added to each well. Only those wells that contain Human LEP, biotinylated detection antibody and Avidin-HRP conjugate will appear blue in color. The enzyme-substrate reaction is terminated by the addition of a stop solution and the color turns yellow. The optical density (OD) is measured spectrophotometrically at a wavelength of 450 nm (Vadacca *et al.*, 2013).

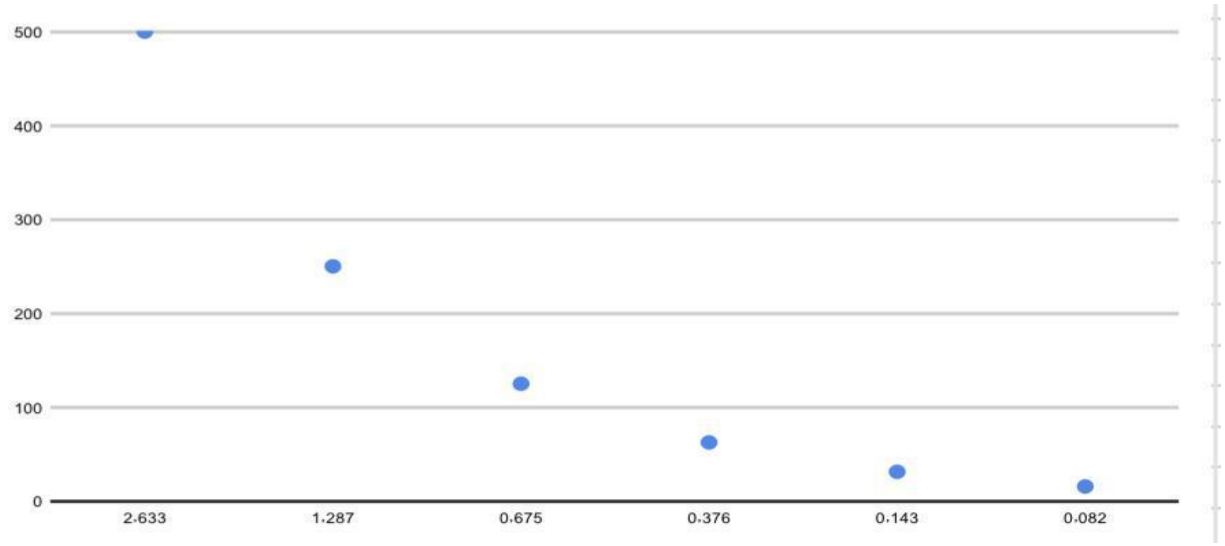

Figure (3-3): Standard curve for Leptin

3.2.4.3. Determination of Human Plasminogen activator inhibitor-1 Level

- **Principle**

This ELISA kit uses the Sandwich-ELISA principle. The micro ELISA plate has been pre-coated with an antibody specific to Human PAI1. Samples are added to the wells and combined with the specific antibody. Then a biotinylated detection antibody specific for Human PAI-1 and Avidin-Horseradish Peroxidase (HRP) conjugate are added successively to each microplate well and incubated. The substrate solution is added to each well. Only those wells that contain Human PAI1, biotinylated detection antibody and Avidin-HRP conjugate will appear blue in color. The enzyme-substrate reaction is terminated by the addition of a stop solution and the color turns yellow. The optical density (OD) is measured spectrophotometrically at a wavelength of 450 nm (Elabscience, 2022).

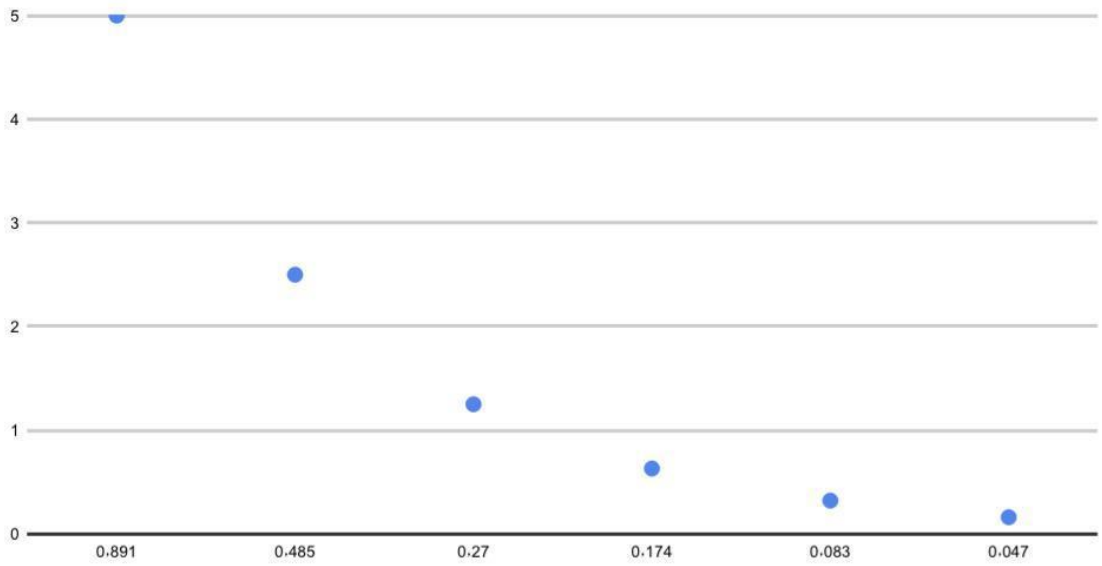

Figure (3-4): Standard curve for plasminogen activator inhibitor-1

3.2.5. Oxidant / antioxidant

3.2.5.1. Determination of Human Malondialdehyde (MDA) Level

- **Principle:**

This kit is an Enzyme-Linked Immunosorbent Assay (ELISA). The plate has been pre-coated with Human MDA antibody. MDA present in the sample is added and binds to antibodies coated on the wells. And then biotinylated Human MDA Antibody is added and binds to MDA in the sample. Then Streptavidin-HRP is added and binds to the Biotinylated MDA antibody. After incubation unbound Streptavidin-HRP is washed away during a washing step. Substrate solution is then added and color develops in proportion to the amount of Human MDA. The reaction is terminated by addition of acidic stop solution and absorbance is measured at 450 nm (Malondialdehyde, n.d.)

3.2.5.2. Determination of Human Total Antioxidant Capacity (T-AOC) Level

- **Principle:**

This kit is an Enzyme-Linked Immunosorbent Assay (ELISA). The plate has been pre-coated with human T-AOC antibody. T-AOC present in the sample is added and binds to antibodies coated on the wells. And then biotinylated human T-AOC Antibody is added and binds to T-AOC in the sample. Then Streptavidin-HRP is added and binds to the Biotinylated T-AOC antibody. After incubation, unbound Streptavidin-HRP is washed away during a washing step. Substrate solution is then added and color develops in proportion to the amount of human T-AOC. The reaction is terminated by addition of acidic stop solution and absorbance is measured at 450 nm (ThermoFisher, 2018).

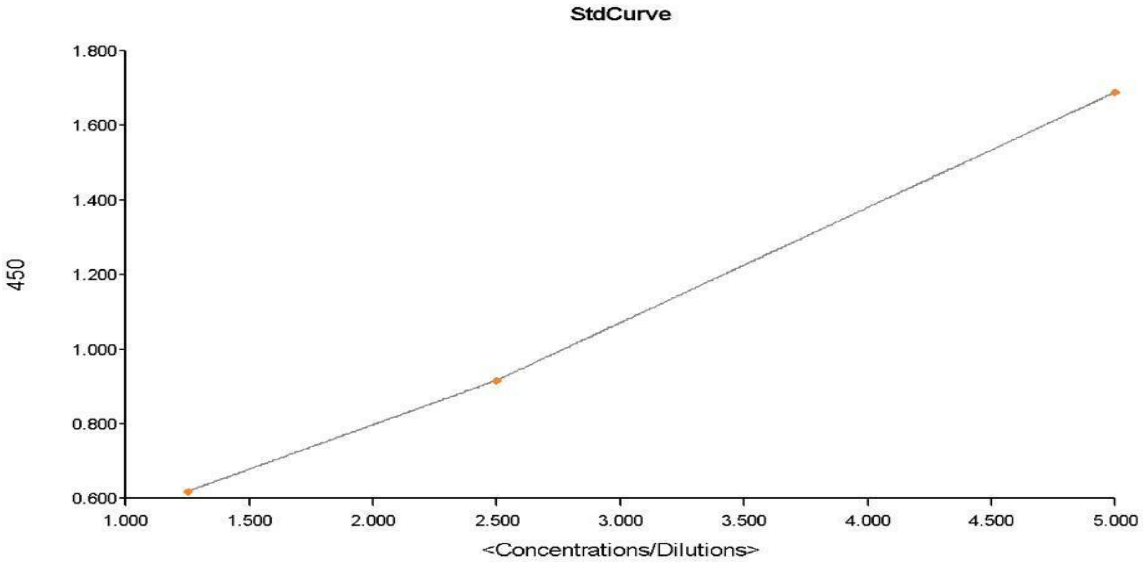

Figure (3-5): Standard curve for total antioxidant capacity

3.2.6. Human Troponin-I ELISA Kit

- **Principle:**

This kit is an Enzyme-Linked Immunosorbent Assay (ELISA). The plate has been pre-coated with Human Tn-1 antibody. Tn-1 present in the sample is added and binds to antibodies coated on the wells. And then biotinylated Human Tn-1 Antibody is added and binds to Tn-1 in the sample. Then Streptavidin-HRP is added and binds to the Biotinylated Tn-1 antibody. After incubation, unbound Streptavidin-HRP is washed away during a washing step. Substrate solution is then added and color develops in proportion to the amount of Human Tn-1. The reaction is terminated by addition of acidic stop solution and absorbance is measured at 450 nm (Chaulin et al., 2022)

3.2.7. Estimate of CRP Level

- **Principle**:

For accurate and reliable measurement of CRP levels in serum and plasma, a latex immunoassay known as MULTIGENT CRP Vario has been developed. CRP in a sample and anti-CRP antibody that has been adsorbed on latex particles interact, it results in a process known as agglutination. The rate of agglutination, as measured by a change in absorbance at 572 nm, is directly correlated with the concentration of CRP in the sample (Swastini et al., 2019).

3.2.8. Estimate of Lipid Profile

3.2.8.1. Total Cholesterol (TC)

- **Principle:**

Enzymatically, the 3-OH group of cholesterol is oxidized and hydrolyzed in a sequence of linked processes in blood or plasma to evaluate cholesterol levels. H2O2, one of the process's byproducts, is quantified in a peroxidase-catalyzed reaction that generates color. 500 nm is used to quantify absorbance. According to

the level of cholesterol, the color intensity increases. The following is the reaction order:

cholesteryl ester hydrolase

Cholesteryl ester + H2O ——————————>cholesterol + fatty acid

cholesterol oxidase

Cholesterol + O2 ——————————————> cholest-4-en-3-one + H2O2

peroxidase

2H2O2+ 4-aminophenazone + phenol ——————> 4-(p-benzoquinone-monoimino)-phenazone + 4 H2O

The concentration of cholesterol is connected with the color's intensity (Hopkins, n.d.).

3.2.8.2. Triglycerides (TG)

- **Principle:**

Through a sequence of linked processes in which triglycerides are digested to create glycerol, triglycerides are quantified enzymatically in serum or plasma. Then, using glycerol oxidase, glycerol is subjected to oxidation, while one of the resultant products, H2O2, is measured as with cholesterol. At 500 nm, absorbance is measured. The following is the order of the reactions:

lipase

Triglycerides + 3H2O ——————————> glycerol + fatty acids

glycerokinase

Glycerol + ATP ——————————————> glycerol-3-phosphate + ADP

glycerophosphate

glycerophosphate oxidase

Glycerol-3-phosphate + O2 ——————————> dihydroxyacetone phosphate + H2O2

peroxidase

H2O2+4-aminophenazone+4-chlorophenol ———> 4-(p-benzoquinone-monoimino)-

phenazone + 2H2O + HCl

(Hopkins, n.d.).

3.2.8.3. Low Density Lipoprotein (LDL)

- **Principle**:

Without using off-line preparation or centrifugation, the test offers a homogenous approach for figuring out how much LDL cholesterol is in plasma or serum, using the liquid selective detergent method.

The procedure, which uses 2 reagents, depends on the characteristics of one detergent. Just the non-LDL particles are dissolved by this detergent (Reagent 1). VLDL, CM, and HDL. The generated cholesterol is consumed by the enzymes cholesterol oxidase and cholesterol esterase in a non-color generating reaction. Following the second detergent (Reagent 2) dissolves the leftover LDL particles, a chromogenic coupler enables color formation. When a coupler is present, the total amount of LDL cholesterol of the sample is depicted by the color created by the reaction of the enzyme with LDL cholesterol. The BIOBASE auto chemical analyzer used a technique that included testing cholesterol (TC), TG, HDL-C, and LDL automatically (Hopkins, n.d.).

3.2.8.4. High Density Lipoprotein (HDL)

- **Principle**:

(1) ApoB containing lipoproteins + α-cyclodextrin + Mg+2 + dextran SO4 ———> soluble non-reactive complexes with apoB-containing lipoproteins

PEG-cholesteryl esterase

(2) HDL-cholesteryl esters ——————————> HDL-unesterified cholesterol + fatty acid

PEG-cholesterol oxidase

(3) Unesterified chol + O2 ——————————————> cholestenone + H2O2

(4) H2O2 + 5-aminophenazone + N-ethyl-N-(3-methylphenyl)-N'_succinyl ethylene diamine + H2O + H+ peroxidase————> qunoneimine dye + H2O

Absorbance is measured at 600 nm (Hopkins, n.d.).

3.3. Ethical management of studies

The research adhered to the protocols for handling biological substances established by the Department of Clinical Laboratories at the University of Kerbala's College of Applied Medical Sciences. The samples used in this study were taken from patients arriving at the Karbala Center for Cardiac Diseases and Surgery / Karbala Health Directorate after receiving the required consent from the hospital administration and patients.

3.4. Statistical analysis

The computer application SPSS, version 12, ANOVA one way was used for statistical analysis of the data. The data are shown as mean standard deviation (Sd). The results were deemed to have statistical significance since they were calculated differences between groups using the P value (i.e., the least significant difference) that was discovered for the comparison between the groups (Delacre *et al.*, 2019).

Chapter Four

Results and Discussion

Chapter Four

4.1. Biomarkers

4.1.1. Adiponectin

Table 4-1 :The level of (Adiponectin) in Atherosclerosis in obese, non-obese patients compared to obese, non-obese control

Groups	N	Mean of Adiponectin (mg/dl)	Std. Deviation
Atherosclerosis obese	30	*0.820 c*	0.133
Atherosclerosis normal	30	1.426 b	0.191
Control obese	20	0.874 c	0.301
Control normal	20	4.071 a	0.684

P value = 0.001 * LSD= 0.384 * means different letters refers to significant differences

The result of table (4-1) demonstrated very high significant decrease in the concentration of adiponectin ($p \leq 0.001$) in atherosclerosis obese, atherosclerosis normal, control obese group (0.820 ± 0.133), (1.426 ± 0.191), (0.874 ± 0.301) respectively as compared to control normal group (4.071 ± 0.684). This decrease may be related to many reasons such as the manner by which adipose tissue expands (increases in size, hypertrophy, and/or in number of cells, hyperplasia) could regulate synthesis and secretion of adiponectin, demonstrated an inverse relationship between mean adipocyte diameter and adiponectin secretion, also AdipoR1 and AdipoR2 expression is significantly decreased in obesity (Nigro *et al.*, 2014).

The results in the table (4-1) have been addressed and supported by another study, it is well established that levels of the hormone adiponectin decrease as BMI

rises, and that obese people have a lower level of this hormone than lean people (Von Frankenberg *et al.*, 2017). In obese people, the ability of adiponectin levels to regulate inflammatory responses is limited (Lempesis *et al.*, 2020). Decreasing serum levels of adiponectin are linked to metabolic conditions with chronic inflammation, such as obesity, and atherosclerosis (Choi *et al.*, 2020).

In obese people, low levels of adiponectin can possibly lead to endothelial dysfunction and a proatherogenic effect (Amin *et al.*, 2020). Adiponectin levels have been shown to be related to intima-media thickness (IMT), a measure of early AS (Shibata *et al.*, 2017). Additionally, research showed that adiponectin overexpression reduces the development of atherosclerotic plaque (Okamoto *et al.*, 2008). This corresponds to the result in the table (4-1), a control who is non obese has a high concentration of adiponectin. By preventing macrophages from transforming into foam cells and monocytes from adhering to endothelial cells, adiponectin has antiatherogenic actions (Mihalopoulos *et al.*, 2020). It additionally inhibits smooth muscle cell proliferation, increases nitrogen oxide synthesis, and promotes blood vessel development (Csongrádi *et al.*, 2017).

4.1.2. Leptin

Table 4-2: The level of (Leptin) in Atherosclerosis in obese, non-obese patients compared to obese, non-obese control

Groups	N	Mean of Leptin (ng/ dl)	Std. Deviation
Atherosclerosis obese	30	*657.842 a*	32.049
Atherosclerosis normal	30	545.324 c	92.184
Control obese	20	599.802 b	75.523
Control normal	20	120.954 d	39.047

P value = 0.001 * LSD= 25.981 * means different letters refers to significant differences

The result of table (4-2) demonstrated very high significant increase in the concentration of leptin ($p \leq 0.001$) (atherosclerosis obese, atherosclerosis normal, control obese) (657.824 ± 32.049), (545.324 ± 92.184), (599.802 ± 75.523) respectively as compared to control normal (120.954 ± 39.047). This increase can be related to obesity-associated enlargement of adipocytes in humans' results in accelerated secretion of leptin and therefore higher serum leptin levels (Poetsch *et al.*, 2020). Another study thought that hyperleptinemia, which is linked to adipose tissue in obese people without the predicted hunger reduction, may signify a leptin resistance state (Goh *et al.*, 2022).

Numerous studies have been investigated and supported the result in the table (4-2) such as the obese individuals found high levels of leptin in circulation (Izquierdo *et al.*, 2019). Early investigations into the levels and expression of leptin in human organs indicated that serum leptin levels rise together with increases in body fat mass, supporting the idea that adipose tissue is a major source of this hormone (Rawal *et al.*, 2020). Leptin is primarily produced by adipose tissue in

response to the amount of fat stores, and it plays a crucial part in the control of lipid reserves (Picó *et al.*, 2022). It is believed that leptin has a role in the "low-grade inflammatory state" seen in overweight and obese individuals (Recinella *et al.*, 2020)**.**

Higher oxidative stress, atherogenesis, thrombosis, dysfunction of endothelial cells, and inflammation have all been linked to hyperleptinemia (Szekeres *et al.*, 2023). Elevated leptin levels are associated with atherosclerosis and are thought to be a possible indication for problems associated with obesity (Csongrádi *et al.*, 2017). The first crucial stage in the development of atheroma has been identified as the binding of leptin with its receptor (Raman & Khanal, 2021). According to a recent study, leptin induces macrophages to take cholesterol, which causes the formation of atheromatous lesions (Amin *et al.*, 2019). Hyperleptinemia, which is a characteristic of obesity, is one of the primary risk factors for atherosclerosis (Raman & Khanal, 2021).

4.1.3. Plasminogen activator inhibitor-1 (PAI-1)

Table 4-3: The level of (PAI-1) in Atherosclerosis in obese, non-obese patients compared to obese, non-obese control

Groups	N	Mean of PAI-1 ng/dl	Std. Deviation
Atherosclerosis obese	30	*8.207 a*	0.886
Atherosclerosis normal	30	7.296 b	0.972
Control obese	20	4.171 c	0.807
Control normal	20	1.180 d	0.497

P value = 0.001 * LSD= 0.201 * means different letters refers to significant differences

The result of table (4-3) demonstrated very high significant increase in the concentration of PAI-1 ($p \leq 0.001$) (atherosclerosis obese, atherosclerosis normal, control obese) (8.207 ± 0.886), (7.296 ± 0.972), (4.171 ± 0.807) respectively as compared to control normal (1.180 ± 0.497), this increase may be related to many reason such as obesity has linked endothelial dysfunction and inflammation as major contributors to the development of atherosclerotic plaques, which is a chronic, systemic disease, endothelial dysfunction has the potential to alter the fibrinolytic system, which is crucial for the formation of atherosclerotic plaques (Poredos *et al.*, 2021). Atherosclerosis tend to exhibit increased PAI-1 expression levels, PAI-1, which is the primary inhibitor of tissue plasminogen activator (tPA) and urokinase (uPA), is an inhibitor of fibrinolysis and plays a crucial role in AS (Chen *et al.*, 2017).

Increased levels of a serine protease inhibitor (PAI-1), being obese and the metabolic syndrome (Yarmolinsky *et al.*, 2016). BMI and plasma PAI-1 levels are significantly linked (Levine *et al.*, 2021). Obesity, hypertension, and increased TGs, are characterized by elevated PAI-1 levels. (Morrow *et al.*, 2021). In addition, studies show BMI and PAI-1 are significantly related in obese and overweight persons (Ahirwar *et al.*, 2014). Individuals with obesity presented elevated PAI-1; however, we also found that subjects with metabolic alterations, both obese and lean, showed increased PAI-1 (Basurto *et al.*, 2019).

PAI-1 is reportedly involved in the etiology of AS, according to a recent large meta-analysis (Liu *et al.*, 2018). Clinical studies have demonstrated elevated PAI-1 levels in individuals with early-stage and established atherosclerotic disease (Córdova-Pérez *et al.*, 2015).

4.2. Oxidant / Antioxidant

4.2.1. Malondialdehyde (MDA)

Table 4-4: The level of (MDA) in Atherosclerosis in obese, non-obese patients compared to obese, non-obese control

Groups	N	Mean of MDA (umol/l)	Std. Deviation
Atherosclerosis obese	30	*21.594 a*	7.615
Atherosclerosis normal	30	16.745 b	5.481
Control obese	20	12.906 c	2.326
Control normal	20	4.636 d	2.642

P value = 0.001 * LSD= 3.251 * means different letters refers to significant differences

Based on the results in the table (4-4) found highly significant increase ($p \leq 0.001$) in the concentration of (MDA) in (atherosclerosis obese, atherosclerosis normal, control obese) (21.594 ± 7.615), (16.745 ± 5.481), (12.906 ± 2.326) respectively as compared to the control normal (4.636 ± 2.642), this increase may be related to Endothelial cells deposit cholesterol-rich low density lipoprotein (LDL) in the membrane's inner layer of the vascular wall, where it is oxidized and transformed into oxidized LDL under conditions of elevated oxidative stress (Y. Wang *et al.,* 2021). The initial phase of atherosclerosis is demonstrated by the oxidation of lipids in the form of Ox-LDL, while MDA, a marker of elevated oxidative stress that indicates the level of lipid peroxidation (Rafieian-Kopaei *et al.,* 2014).

The result was supported by other studies MDA levels in obese individuals were significantly higher than those in normal (Adnan *et al.,* 2019). The level of lipid peroxidation and the severity of OS are both reflected in MDA plasma levels (Cho

et al., 2022). MDA is one of the significant end products of lipid peroxidation, which is one of the most often used biomarkers to measure oxidant status since it is connected to the severity of lipid peroxidation (Uçkan *et al.*, 2022). The lipid hypothesis regarding atherosclerosis suggests that peroxidation of lipids and the oxidation of LDL cause the onset and persistent progression of atherosclerosis (Gaggini *et al.*, 2023).

4.2.2. Total antioxidant capacity (T-AOC)

Table 4-5: The level of (T-AOC) in Atherosclerosis in obese, non-obese patients compared to obese, non-obese control

Groups	N	Mean of T-AOC (mmol/l)	Std. Deviation
Atherosclerosis obese	30	*2.887 c*	0.529
Atherosclerosis normal	30	3.969 b	0.871
Control obese	20	2.764 c	0.552
Control normal	20	6.785 a	0.715

P value = 0.001 * LSD= 0.908 * means different letters refers to significant differences

In table (4-5) Total antioxidant capacity (TAOC) showed highly significant decrease ($p \leq 0.001$) in (atherosclerosis obese, atherosclerosis normal, control obese) (2.887 ± 0.529), (3.969 ± 0.871), (2.764 ± 0.552) respectively as compared to the control normal (6.785 ± 0.715), this decrease may be related to many reason such as the level of expression and action of antioxidant enzymes like catalase, decreased in the adipose tissue of obese people, although the fact that adipose tissue has compared high levels of antioxidant defense enzymes for controlling high ROS generation (Okuno *et al.*, 2008). the main center of ROS production, particularly in obese individuals, is in the mitochondria of white adipose tissue, where NDPH

(nicotinamide adenine dinucleotide phosphate) oxidase is overexpressed and antioxidative enzyme expression is diminished (Catalán *et al.*, 2018).

The result supported by another study, one of the causes of obesity-related diseases such atherosclerosis, hypertension, and obesity is oxidative damage to essential cellular components, the human body has created defenses that protect biomolecules from OS's harmful effects (Di Meo *et al.*, 2022).

A number of mechanisms support the action of antioxidants, including the inhibition of oxidation of LDL, the reduction of produced free radicals, the limitation of secretion of cytokines, prevention of the development of atherosclerotic plaque and aggregation of platelets, a reduction of dysfunctional endothelial cells and vascular dilation, the modulation of adhesion molecule expression, and the inhibition of foam cell formation (Malek mohammad *et al.*, 2019).

4.3. Physiological parameters

4.3.1. Troponin I

Table 4-6: The level of (Troponin I) in Atherosclerosis in obese, non-obese patients compared to obese, non-obese control

Groups	N	Mean of Troponin I (pg/ ml)	Std. Deviation
Atherosclerosis obese	30	*6901.810 b*	1497.823
Atherosclerosis normal	30	16916.490 a	6778.663
Control obese	20	6.040 c	1.616
Control normal	20	0.740 c	0.482

P value = 0.001 * LSD= 7031.647 * means different letters refers to significant differences

The present data showed highly significant increase ($p \leq 0.001$) in the concentration of Troponin I in atherosclerosis obese, atherosclerosis normal (6901.810 ± 1497.823), (16916.490 ± 6778.663) respectively as compared to control

obese and control normal (6.040 ± 1.616), (0.740 ± 0.482) respectively, this increase may be caused by Troponin release into the circulation when myocytes are injured (Sorodoc *et al.,* 2022). A further investigation demonstrated a direct correlation between elevated troponin levels and the severity of coronary stenosis (Liberale *et al.,* 2017). There is a correlation between low levels of high-sensitivity cardiac troponin (hs-cTn) and a low risk of atherosclerotic cardiovascular disease (Sandoval *et al.,* 2020).

It is well established that body mass index (BMI) and troponins are independently associated (Huang *et al.,* 2021).

Heart troponin release with exercise is connected to coronary atherosclerosis or indications of plaque susceptibility (Paana *et al.,* 2019). Because one of the primary risk factors for developing cardiac angina is coronary atherosclerosis, this condition may induce discomfort or retrosternal pain (Sazonova *et al.,* 2019).

4.3.2. C-reactive protein (CRP)

Table 4-7: The level of (CRP) in Atherosclerosis in obese, non-obese patients compared to obese, non-obese control

Groups	N	Mean of CRP (mg/ dl)	Std. Deviation
Atherosclerosis obese	30	* 23.183 a*	5.386
Atherosclerosis normal	30	13.833 ab	2.599
Control obese	20	8.260 ab	1.021
Control normal	20	1.360 b	0.865

P value = 0.004* LSD= 19.784 * means different letters refers to significant differences

In table (4-7) result found highly significant increase ($p \leq 0.004$) in the concentration of C-reactive protein in (atherosclerosis obese, atherosclerosis normal, and control obese) (23.183 ± 5.286), (13.833 ± 2.599), (8.260 ± 1.021) respectively as compared to control normal (1.360 ± 0.865). This increase related to fat cells

secrete a variety of inflammatory molecules called adipocytes, including leptin, adiponectin, and others, obese people constantly have high levels of inflammation (Kim & Yeun, 2022). Because of CRP's capacity to bind to change LDL and its effect on the functionality of endothelial cells, the stability of plaque, and thrombosis, CRP is believed to have a part in atherogenesis (Schenkein *et al.*, 2020).

The result agrees with other studies, obesity is the main factor that contributes to high CRP in people with the metabolic syndrome, these people are more likely than non-obese people to have CRP levels (Aronson *et al.*, 2004). Obesity is related to elevated CRP levels (Pope & Choy, 2021). Excessive body weight has been associated with elevated CRP levels (Stanimirovic *et al.*, 2022).

4.4. Lipid profile

Table 4-8: The level of Lipid Profile in Atherosclerosis in obese, non-obese patients compared to obese, non-obese control

Groups	N	TC (mg/dl)	Std. Deviation	P value
Atherosclerosis obese	30	*196.733 a*	23.675	0.001 *
Atherosclerosis normal	30	153.366 b	18.985	
Control obese	20	141.166 b	17.005	
Control normal	20	136.933 b	16.623	
Groups	**N**	**TG (mg/dl)**	**Std. Deviation**	**P value**
Atherosclerosis obese	30	*202.466 a*	31.545	0.005 *
Atherosclerosis normal	30	134.733 b	20.658	
Control obese	20	133.833 b	17.288	
Control normal	20	102.100 b	15.346	
Groups	**N**	**LDL (mg/dl)**	**Std. Deviation**	**P value**
Atherosclerosis obese	30	*107.133 a*	27.593	0.021 *
Atherosclerosis normal	30	94.800 ab	19.643	
Control obese	20	82.133 b	34.333	
Control normal	20	75.866 b	32.732	
Groups	**N**	**VLDL (mg/dl)**	**Std. Deviation**	**P value**
Atherosclerosis obese	30	*44.400 a*	21.390	0.001 *
Atherosclerosis normal	30	36.026 ab	18.690	
Control obese	20	25.563 bc	16.357	
Control normal	20	19.523 c	13.094	
Groups	**N**	**HDL (mg/dl)**	**Std. Deviation**	**P value**
Atherosclerosis obese	30	*32.386 b*	8.708	0.002 *
Atherosclerosis normal	30	34.866 b	6.157	
Control obese	20	34.933 b	11.719	
Control normal	20	43.133 a	8.683	

* means different letters refers to significant differences

The result in the table (4-8) showed highly significant increase ($p \leq 0.001$) in the concentration of TC in atherosclerosis obese (196.733 ± 23.675) as compared to (atherosclerosis normal, control obese, control normal) (153.366 ± 18.985), (141.166 ± 17.005), (136.933 ± 16.623) respectively.

The result in the table showed highly significant increase ($p \leq 0.005$) in the concentration of TG in atherosclerosis obese (202.466 ± 31.545) as compared to (atherosclerosis normal, control obese, control normal) (134.733 ± 20.658), (133.833 ± 17.288), (102.100 ± 15.346) respectively.

The result in the table showed significant increase ($p \leq 0.021$) in the concentration of LDL in atherosclerosis obese (107.133 ± 27.593 as compared to (atherosclerosis normal, control obese, control normal) (94.800 ± 19.643), (82.133 ± 34.333), (75.866 ± 32.732) respectively.

The result in the table showed highly significant increase ($p \leq 0.001$) in the concentration of VLDL in atherosclerosis obese (44.400 ± 21.390) as compared to (atherosclerosis normal, control obese, control normal) (36.026 ± 18.680), (25.563 ± 16.357), (19.523 ± 13.094) respectively.

The result in the table showed highly significant degrease ($p \leq 0.001$) in the concentration of HDL in (atherosclerosis obese, atherosclerosis normal, control obese) (32.386 ± 8.708), (34.866 ± 6.157), (34.933 ± 11.719), respectively as compared to control normal group (43.133 ± 8.683).

An increased serum total cholesterol (TC), TGs, LDL-C, and decreased HDL-C levels are symptoms of the metabolic condition dyslipidemia, which progresses to an atherogenic metabolic disorder and causes cardiovascular problems (Ghoshal *et al.*, 2021). Obesity and atherosclerosis are now seen as lipid storage diseases including triglycerides in fat tissue and cholesterol ester in atheroma (Mandviwala *et al.*, 2016). A causative role for triglycerides (TGs) in the occurrence of AS is highly supported by observational and genetic epidemiology data (Sandesara *et al.*,

2019). It has been demonstrated that triglyceride buildup in macrophages, which is associated with macrophage oxidative stress, increases mitochondrial production of reactive oxygen compounds and stimulates the development of foam cells (Peng *et al.*, 2017). TGs have been identified as a common risk factor for atherosclerotic coronary artery disease (Chapman et al., 2011).

Low levels of HDL-C, an accumulation of small dense LDL particles, are the hallmarks of atherogenic dyslipidemia (Björnson *et al.*, 2017). Atherosclerosis is brought on by the buildup of cholesterol, primarily in the form of LDL, in the intimate layer of the arteries, this accumulation results in extensive narrowing of arteries (Wolf & Ley, 2019). Low HDL-C is the main indicator of dyslipidemia in obese people (Zhang *et al.*, 2019)**.** The origin of obesity and related dyslipidemia is multifactorial, not all obese individuals develop dyslipidemia, and not all dyslipidemia patients are obese (Kanwar & Kabra, 2016).

4.5. Correlation

Table 4-9: Correlation between markers in study groups

Correlation between markers	**R2**	
Correlation between Adiponectin and MDA in Atherosclerosis obese group	r	-0.371
	p	0.043
Correlation between BMI and TG in Atherosclerosis obese group	r	0.767
	p	0.001
Correlation between BMI and VLDL in Atherosclerosis obese group	r	0.758
	p	0.001

Correlation between TG and MDA in Atherosclerosis obese group	r	0.556
	p	0.001
Correlation between BMI and T-AOC in Atherosclerosis normal group	r	-0.393
	p	0.032
Correlation between TC and HDL in Atherosclerosis normal group	r	-0.638
	p	0.001
Correlation between TG and VLDL in Atherosclerosis normal group	r	0.708
	p	0.003
Correlation between BMI and PAI-1 in control obese group	r	0.539
	p	0.038
Correlation between BMI and HDL in control obese group	r	-0.569
	p	0.027
Correlation between TG and VLDL in control obese group	r	0.650
	p	0.001

Table (4-9) showed there was a significant negative correlation between adiponectin and MDA (-0.371) in atherosclerosis obese group. This finding agrees with (Dolab et al., 2020) that found MDA has negative correlation with adiponectin. Also in skeletal muscle, adiponectin deficiency promotes fatty acid oxidation (Wang & Scherer, 2016). Adiponectin promotes fatty acid oxidation while suppressing hepatic gluconeogenesis and lipolysis (Shabalala et al., 2020). Also showed a significant positive correlation between BMI, TG, and VLDL (0.767,0.758) in

atherosclerosis obese group. All lipid profile parameters S.TC, S.TG, S. VLDL and S.LDL showed significant increase in obese except S.HDL whose level showed significant decrease with BMI (Kanwar & Kabra, 2016). There was a significant positive correlation between TG and MDA (0.556) in atherosclerosis obese group. This agrement with (Yang et al., 2008) they found hypertriglyceridemia was associated with oxidative modification of LDL, thus leading to excess production of lipid peroxidation products which may cause elevation of oxidative stress.

Table (4-9) showed there was a significant negative correlation between BMI and T-AOC (-0.393) in atherosclerosis normal group. This agrees with (Hosseini et al., 2017) that found concentrations of circulating antioxidants are inversely related to body mass index. Also showed a significant negative correlation between TC and HDL (-0.638) in atherosclerosis normal gruop. An increased serum total cholesterol (TC), TGs, LDL-C, and decreased HDL-C levels are symptoms of the metabolic condition dyslipidemia (Ghoshal et al., 2021). Also showed a significant positive correlation between TG and VLDL in atherosclerosis normal and control obese. Triglyceride (TG) makes up 50–70% of the particle mass carried by VLDL (Huang & Lee, 2022).

Table (4-9) showed there was a significant positive correlation between PAI-1 and BMI (0.539). This results in agreement with (Barnard et al., 2016) they found obesity provides more evidence for the influence of adipose tissue depots on plasma PAI-1 levels . BMI, as a general marker of body fat, was found to be strongly associated with PAI-1 (Fang et al., 2018). There was a significant negative correlation between BMI and HDL (- 0.569) in control obese. Low HDL-C is the main indicator of dyslipidemia in obese people (Zhang et al., 2019).

Conclusions and Recommendations

Conclusion

1. There is association between adipokine derived adipose tissue (adiponectin, leptin, PAI-1) in the development of atherosclerosis in obese patients.
2. Increased concentration of adiponectin protects individuals from atherosclerosis, because it has an antiatherogenic effect.
3. The hormone Leptin, which controls metabolism, was found to be increasing, which may be a sign that the metabolic issue in obese individuals in this study.
4. Increased levels of PAI-1 have associations between obesity and atherosclerosis.
5. The end result of lipid peroxidation, increased MDA concentration, indicates that the rate of oxidative stress is high.
6. Decrease in concentration of T-AOC in all obese groups, which means increased body mass index leads to decreased antioxidant.
7. A person with increased body mass index is more susceptible to development of atherosclerosis.

Recommendations

1. Studying the relationship of the parameters in their occurrence of atherosclerosis in obese and non-obese females.
2. Comparing study parameters between male and female atherosclerosis patients.
3. Study the parameters with diabetic atherosclerosis, and find correlation between them.
4. We recommend to use (adiponectin, leptin, PAI-1) as predicting markers in obese individuals

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Appendix

Appendix 1:

Questionnaire for participants

Name: **Height:** **Weight:**
Age: **Phone Number:** **Address:**

Serial	Content	
1	Work	
2	Simple date	
3	Family history	
4	Other diseases	
5	Any treatment	
6	BMI	
7	Smoker	
8	Hypertension	

Serial	Content	
1	Adiponectin	
2	Leptin	
3	PAI-1	
4	CRP	
5	Troponin I	
6	MAD	
7	T-AOC	
8	Total cholesterol	
9	LDL	
10	Triglyceride	
11	HDL	
12	VLDL	

Appendix 2:

Procedure to determination of human Adiponectin level

1. The sample, the blank, and the diluted standard should go in separate wells. Add 100 ml of each dilute into the appropriate wells. The plate with the sealant from the package. Incubate for 90 minutes at 37 °C.

2. Washing, pour the solution from each well. 100 µL of Biotinylated detection Ab working solution should be added to each well right away. Fill the plate with new sealant. For one hour, incubate at 37 °C.

3. After pouring the mixture from each well, 350 µL of wash buffer should be added to each well. After soaking for 1-2 minutes, draw out or decant the solution from each well, then pat it dry with fresh absorbent paper.

4.100µL of the working solution for HRP conjugate should be placed in each well.Apply a new layer of sealer on the plate. Incubate for 30 min at 37°C.

5. Each well's solution should be emptied before five more times of the three-step wash process are completed.

6. Add 90 µL of the substrate reagent to each well. Refresh the sealer on the plate. Incubate for about 15 minutes at 37 °C.

7. Fifty µL of Stop Solution should be put into each well. As with the substrate solution, the stop solution should be introduced in the same way.

8. Concurrently determine each well's optical density (OD value) using a reader for micro plates set to 450 nm in wavelength (Biosciences, E. 2019).

Appendix 3:

Procedure to determine human Leptin level.

1. Prepare all of the reagents, samples, and standard solutions as instructed. Bring each reagent to the room's temperature before using it.
2. Add 50µl of standard to the standard well. Don't add it to the standard well because a standard solution already contains biotinylated antibodies.
3. The sample and standard wells should each contain 50 µL of streptavidin-HRP after 40 µL of sample and 10 µL of anti-LEP antibody have been added to the sampling wells. Apply sealer on plate after mixing it thoroughly. Incubate for 60 minutes at temperatures of 37 °C.
4. After removing the sealant, wash the plate five times with a washing buffer. For each wash, immerse the wells for 30 to 60 seconds in 300 µL for the washing buffer. Thoroughly draw out or decant, then wash five times automatically using the washing buffer.
5. Before administering 50 µL of the solution of the substrate B to each well, 50 µL of substrate solution A should be administered. At a temperature of 37°C and in complete darkness, the plate must be incubated for 10 minutes.
6. The color blue will rapidly change to yellow when 50 µL of this Stopping Solution is added to each well.
7. Measure OD of each well using a reader for micro plates set to 450 nm within ten minutes after adding the stop solution (Vadacca et al., 2013)

Appendix 4:

Procedure to determine Plasminogen activator inhibitor-1

1. Place the sample, diluted standard, and blank in separate wells. 100 µL for each dilute of the standard, blank, and sample should be placed in the appropriate wells. It is recommended that all standards and samples be examined in duplicate.To estimate a dilution of samples, it is advisable to use preparatory tests or technical guidance. Seal the plate using the sealer that is provided in the kit. Incubate for 90 minutes at 37 °C. Please be aware that, in order to prevent foaming, solutions should only be added to the bottom of the micro ELISA plate.

2. Decant the liquid from each well; do not wash. The working Biotinylated Detection Ab solution should then be added to each well in 100 µL increments. Refresh the plate with a new coat of sealant. Place at 37 °C for one hour.

3. After decanting is the solution from every well, add 350 µL of wash buffer to each well. The solution should be soaked, aspirated, or decanted from each well after one minute, then dried with new, absorbent paper. During the washing, three times. As said, you can use a microplate washer to complete this step and other wash procedures. Use the tested strips once they have been washed. Never leave a well empty, please.

4. 100 µL of the working solution for HRP conjugate should be added to each well. Fresh sealant should be applied to the plate. 30 minutes of incubation at 37 °C.

5. Decant the solution from each of the wells after that, then repeat step 3's wash instructions a further five times.

6. Each well should receive 90 µL increments of the substrate reagent. Fresh sealant should be applied to the plate. Incubate for roughly 15 minutes at 37 °C. Keep the light out of the plate.

Note: According to the actual color change, the reaction time could be increased or decreased by a maximum of thirty minutes. The Microplate readers should be warmed for around 15 minutes prior to taking an OD reading.

7. The Stop Solution should be 50 μL in each well. Remember that the solution for stopping needs to be introduced in the same way in the substrate solution.

8. Find The OD value of each well individually with a reader for micro plates set to 450 nm (Elabscience, 2022).

Appendix 5:

Procedure to determine human Malondialdehyde level

1. The reagents, samples, and standard solutions have all been produced according to the instructions. Each and every one of the reagents is warmed to room temperature before use. The test is run in a temperature-neutral environment.
2. Find out how many test strips are needed. The strips are inserted into the frames before usage. Between 2 and 8 oC must be maintained for the strips.
3. Add 50 µl of standard to the standard well. Note: Avoid adding it to the standard well since the standard solution already contains the biotinylated antibody.
4. The sample should be diluted to 40 µl in the sample wells, followed by the addition of 50 µl of streptavidin-HRP in the standard wells and 10 µl of anti-MDA antibody (Not the blank control well). Mix well, then seal the plate with a sealer. Incubate for 60 minutes at a temperature of 37 °C.
5. The sealant is removed, and the plate is then washed using a wash buffer five times. For every wash, the wells are immersed in 300 µL of a buffer for washing for between thirty and sixty seconds. Complete aspiration or decantation is followed by five automatic washing cycles with a buffer. Place paper towels or another absorbent material nearby to dab the plate.
6. Before administering 50 µl of the substrate solution B to each well, 50 µl of substrate solution A should be administered. A 37°C incubation period should be followed by ten minutes of darkness.
7. Every well will turn yellow when 50 µl of Stop Solution is added, which causes the blue color to change.

8. Measure the OD for each well using a microplate reader that has been set to 450 nm within ten minutes of applying the stop solution (Malondialdehyde, n.d.)

Appendix 6:

Procedure to determine Total Antioxidant Capacity level

1. Preparing all the reagents standard solutions, and sample in accordance with the directions. Put every reagent to room temperature prior to use. The procedure is carried out at room temperature.

2. Determine the number of test strips required. Place the strips into the frames to use. Keep the strips somewhere from 2 and 8 °C.

3. Add 50 μL of standard to the standard well. Remember: Do not add the biotinylated antibody to the standard well since it is present in the standard solution already.

4. For the standard wells, 50 μL of streptavidin-HRP should be added after 40 μL of sample (not a blank control well), 10 μL of anti-T-AOC antibody, and. Mix everything well. Apply a sealant to the plate. Incubation at 37 °C for 60 minutes.

5. Remove the sealer off the plate, and then apply a buffer to wash it five times to clean it. Wells should be soaked during 30 to 120 seconds in a minimum of 0.35 cc of buffer for washing for each wash. For automatic washing, aspirate every well, then five times pour excessive amounts of wash buffer into each well. To blot the dish, cover it with a paper towel or another material that is absorbent.

6. The substrate solution A should be diluted by 50 μL before the substrate solution B is diluted by 50 μL in each well. 10 minutes of dark incubation at 37 °C is required for the plate.

7. The blue color of each well will change to yellow immediately when 50 μL of Stopping Solution is added.

8. Use a microscope reader with the wavelength set to 450 nm to gauge each well's density of light (OD value) after ten minutes following the addition of the stop solution (ThermoFisher, 2018).

Appendix 7:

Procedure to determine Human Troponin-I level

1. Prepare each reagent, standard solution, and sample in accordance with the provided instructions. Each reagent must be room temperature before use. A room-temperature setting is used for the test.

2. The number of test strips required should be calculated. In order to use the strips, place them in the frames. The temperature should be between two and eight degrees Celsius for the strips.

3. The well needs to receive 50 µL of standard. Note: Avoid including it to the standard well since the standard solution usually has a biotinylated antibody in it.

4. For the standard wells, 40 µL of sample should be put first, then 10 µL of anti-Tn-1 antibody, and 50 µL of streptavidin-HRP (not a blank control well). Combine well. The plate should be sealed. For 60 minutes, incubate at 37 °C.

5. Wash the plate five times with a wash buffer once the sealant has been removed. Soak wells in a 300 µL wash buffer for 30 seconds to 1 minute before each wash. Each well should be aspirated or decanted before being washed five times automatically with a wash buffer. Paper towels or another absorbent material should be used to dry the plate.

6. Each well needs to have substrate solution A supplied to it before substrate solution B. A plate with a fresh sealer on it should be incubated for 10 minutes without any light at 37°C.

7. The blue color of each well will change to yellow immediately as 50 µL of Stopping Solution gets added.

8. Use a reader for micro plates set to 450 nm to ascertain each well's the optical density (OD value) after 10 minutes of applying the stop solution (Chaulin et al., 2022).

الخلاصة

تصلب الشرايين هو مرض التهابي مزمن و معقد يؤثر على الشرايين المتوسطة والكبيرة الحجم (التاجية، الشرايين السباتية، والشرايين الطرفية). تبدأ هذه الحالة عندما تتراكم البروتينات الدهنية منخفضة الكثافة المؤكسدة في الطبقة الداخلية للشرايين. يؤدي ذلك إلى إنتاج الدهون المؤكسدة المسببة للالتهابات بواسطة الخلايا البطانية المتداخلة. وهذا يؤدي إلى تصلب وتضييق الأوعية الدموية.

هدف الدراسة: إيجاد علاقة بين السمنة وحدوث تصلب الشرايين

شملت التجربة الحالية على (100) شخص من الذكور بمتوسط أعمار (40-65) وتم تقسيمهم بالشكل التالي

(60) مريض يعانون من تصلب الشرايين قسمت بالاعتماد على قيمة مؤشر الجسم إلى مجموعتين :

- مجموعة البدناء BMI= 25≤30وشملت 30 فرداً من الذكور.
- مجموعة الوزن الطبيعي BMI=18.5-24.9 وشملت 30 فرداً من الذكور

(40) فرداً أصحاء قسمت بالاعتماد على قيمة مؤشر الجسم إلى مجموعتين :

- مجموعة البدناء BMI= 25≤30وشملت 20 فرداً من الذكور.
- مجموعة الوزن الطبيعي BMI=18.5-24.9وشملت 20 فرداً من الذكور

أجريت هذه الدراسة للمدة من تشرين الثاني 2022 إلى مايو 2023 . وتم أخذ العينات من مركز كربلاء لأمراض القلب والقسطرة حيث أخذ (٥ مل) عينات الدم من المرضى والأصحاء وأجريت عليها الاختبارات الكيميائية والفسلجية اللازمة (الاديبونكتين، اللبتين، منشط مثبط البلازمينوجين-1، مالوندايالديهايد، القدرة الكلية لمضادات الاكسدة ، تروبونين I، بروتين سي التفاعلي، الدهون)

أظهرت نتائج هذه الدراسة انخفاضاً عالي المعنوية ($p\leq0.001$) في تركيز الاديبونيكتين في (مجاميع مرضى تصلب الشرايين البدناء وغير البدناء ،الأصحاء البدناء) مقارنة مع الأصحاء ذوي الوزن الطبيعي .

أظهرت نتائج هذه الدراسة أرتفاعاً عالي المعنوية ($p\leq0.001$) في تركيز اللبتين في (مجاميع مرضى تصلب الشرايين البدناء وغير البدناء ،الأصحاء البدناء) مقارنة مع الأصحاء ذوي الوزن الطبيعي . أظهرت نتائج هذه الدراسة أرتفاعاً عالي المعنوية ($p\leq0.001$) في تركيز منشط مثبط البلازمينوجين-١ في (مجاميع مرضى تصلب الشرايين البدناء وغير البدناء ،الأصحاء البدناء) مقارنة مع الأصحاء ذوي الوزن الطبيعي. أظهرت نتائج هذه الدراسة أرتفاعاً عالي المعنوية ($p\leq0.001$) في تركيز المالوندايالديهاد في (مجاميع مرضى تصلب الشرايين البدناء وغير البدناء ،الأصحاء البدناء) مقارنة مع الأصحاء ذوي الوزن الطبيعي. أظهرت نتائج هذه الدراسة أنخفاضاً عالي المعنوية ($p\leq0.001$) في تركيز القدرة الكلية لمضادات الاكسدة في (مجاميع مرضى تصلب الشرايين البدناء وغير البدناء ،الأصحاء البدناء) مقارنة مع الأصحاء ذوي الوزن الطبيعي. أظهرت

الخلاصة

نتائج هذه الدراسة أرتفاعاً عالي المعنوية ($p\leq0.001$) في تركيز التروبونين I في (مجاميع مرضى تصلب الشرايين البدناء وغير البدناء مقارنة مع الأصحاء البدناء و الأصحاء ذوي الوزن الطبيعي. أظهرت نتائج هذه الدراسة أرتفاعاً عالي المعنوية ($p\leq0.004$) في تركيز بروتين سي التفاعلي في (مجاميع مرضى تصلب الشرايين البدناء وغير البدناء ،الأصحاء البدناء) مقارنة مع الأصحاء ذوي الوزن الطبيعي . لوحظ زيادة معنوية ($p\leq0.001$،($p\leq0.005$) ،($p\leq0.021$ $p\leq0.001$) في تركيز الكولسترول الكلي، الدهون الثلاثية، البروتين الدهني منخفض الكثافة، البروتين الدهني منخفض الكثافة جداً، على التوالي في مرضى تصلب الشرايين البدناء بالمقارنة إلى (مرضى تصلب الشرايين غير البدناء، الأصحاء البدناء وغير البدناء). كما حدث انخفاض معنوي كبير ($p\leq0.001$) في تركيز البروتين الدهني عالي الكثافة في (مجاميع مرضى تصلب الشرايين البدناء وغير البدناء ،الأصحاء البدناء) مقارنة مع الأصحاء ذوي الوزن الطبيعي .

أوضحت هذه الدراسة عدة علاقات بين المعلمات في مجموعة مرضى تصلب الشرايين البدناء كان هناك ارتباط سلبي كبير بين الاديبونيكتين والمالونديالدهيد. ويلاحظ وجود علاقة إيجابية كبيرة بين مؤشر كتلة الجسم وكل من الدهون الثلاثية والبروتين الدهني منخفض الكثافة للغاية. بالإضافة إلى وجود علاقة إيجابية بين الكولسترول الكلي والبروتين الدهني منخفض الكثافة. ويظهر وجود علاقة إيجابية بين الدهون الثلاثية وكل من البروتين الدهني منخفض الكثافة جدا، المالونديالدهيد.

لاحظت هذه الدراسة عدة علاقات بين المتغيرات في المجموعة مرضى تصلب الشرايين غير البدناء. كان هناك ارتباط سلبي كبير بين مؤشر كتلة الجسم والقدرة الكلية لمضادات الأكسدة. يظهر وجود علاقة سلبية بين الكوليسترول الكلي والبروتين الدهني عالي الكثافة. ويظهر وجود علاقة إيجابية بين الدهون الثلاثية والبروتين الدهني منخفض الكثافة للغاية.

لاحظت هذه الدراسة عدة علاقات بين المتغيرات في مجموعة الأصحاء البدناء. كان هناك ارتباط إيجابي كبير بين مؤشر كتلة الجسم ومثبط منشط البلازمينوجين -1. بالإضافة إلى ذلك، هناك ارتباط سلبي بين مؤشر كتلة الجسم والبروتين الدهني عالي الكثافة، و ارتباط إيجابي بين الكوليسترول الكلي والبروتين الدهني منخفض الكثافة، و ارتباط إيجابي بين الدهون الثلاثية والبروتين الدهني منخفض الكثافة جدًا.

الاستنتاج: هناك علاقة بين (الاديبونيكتين، اللبتين، مثبط منشط البلازمينوجين-1) في تطور تصلب الشرايين لدى مرضى تصلب الشرايين البدناء. تشير زيادة تركيز المالونديالدهيد إلى أن معدل الإجهاد التأكسدي مرتفع. انخفاض تركيز القدرة الكلية لمضادات الأكسدة في جميع مجموعات السمنة. وقد وجد أن هرمون الليبتين الذي يتحكم في عملية التمثيل الغذائي في ازدياد، مما قد يكون علامة على أن المشكلة الأيضية لدى الأشخاص الذين يعانون من السمنة في هذه الدراسة

جامعة كربلاء

كلية العلوم الطبية التطبيقية

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رسالة مقدمة

الى مجلس كلية العلوم الطبية التطبيقية - جامعة كربلاء

وهي جزء من متطلبات نيل شهادة الماجستير في التحليلات المرضية

كتبت بواسطة

رفل حليم نوماس

جامعة الفرات الاوسط التقنية

كلية التقنيات الصحية والطبية/تحليلات مرضية/٢٠١٧

بإشراف :

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