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ESTIMATION OF CYSTATIN-C(CYS-C),GALECTIN-3(GAL-3), MYONECTIN LEVELS AND NUMBER OF BIOCHEMICAL PARAMETERS IN ANGINA PECTORIS PATIENTS

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May 2022

We certify that we have read this thesis and that in our opinion it is fully adequate, in scope and in quality, as a thesis for the degree of Master of Science

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ABSTRACT

ESTIMATION OF CYSTATIN-C(CYS-C),GALECTIN-3(GAL-3), MYONECTIN LEVELS AND NUMBER OF BIOCHEMICAL PARAMETERS IN ANGINA PECTORIS PATIENTS

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Master of Science in Chemistry

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May 2022

Recent studies have related Angina is chest pain due to transient myocardial ischaemia, which usually occurs with physical activity or emotional stress, and is relieved by rest or sublingual nitroglycerin. Angina is common, affecting 3.8% of people in New Zealand. About half of patients with ischaemic heart disease initially present with symptoms consistent with a pattern of stable angina. The results of this study included the first of patients with angina pectoris by performing the analysis of troponin and the second of the healthy, and it was divided into four tables, each table compared between the control group and patients, and the first table includes four groups: CYC_C, GAL_3,Myonectin, INS and The second table also includes four groups Cholesterol, Triglycerid, HDL, LDL, VLDL and the third table includes three groups GOT, GPT, ALP and the fourth and last table is between Age (year) and Weight (kg).

2022, 48 pages

Keywords: Laminin, Prolipın levels, Number of biochemical, Inengina Pectoris

ÖZET

ANGİNA PECTORİS HASTALARINDA SİSTATİN-C(CYS-C),GALECTIN-3(GAL-3), MYONECTİN DÜZEYLERİ VE BİYOKİMYASAL PARAMETRE SAYILARININ TAHMİNİ

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Son araştırmalar, Angina'yı, genellikle fiziksel aktivite veya duygusal stres ile ortaya çıkan ve dinlenme veya dilaltı nitrogliserin ile hafifleyen geçici miyokard iskemisine bağlı göğüs ağrısı ile ilişkilendirmiştir. Angina yaygındır ve Yeni Zelanda'daki insanların %3,8'ini etkiler. İskemik kalp hastalığı olan hastaların yaklaşık yarısı başlangıçta stabil angina paterni ile uyumlu semptomlarla başvurur. Bu çalışmanın sonuçları, troponin analizi yapılarak anjina pektorisli hastalardan birincisini ve sağlıklıların ikincisini içermiş ve her biri kontrol grubu ve hastalar arasında karşılaştırılan dört tabloya bölünmüş ve birinci tablo dört tabloyu içermektedir. gruplar: CYC_C, GAL_3,Myonectin, INS ve İkinci tablo ayrıca dört grup Kolesterol, Trigliserid, HDL, LDL, VLDL içerir ve üçüncü tablo GOT, GPT, ALP üç grubu içerir ve dördüncü ve son tablo Yaş (yıl) arasındadır. ve Ağırlık (kg).

2022, 48 sayfa

Anahtar Kelimeler: Laminin, Prolipın seviyeleri, Biyokimyasal sayısı, İnengina Pektoris

PREFACE AND ACKNOWLEDGEMENTS

I would like to thank my thesis advisor, Prof. Dr. Volkan EYÜPOĞLU and Dr. Aseel Mokdad Hatam ABDULWAHED, for their patience, guidance and understanding. Dedication I dedicate this research to every student of knowledge who seeks to gain knowledge and provide his scientific and cultural knowledge to the one who taught me that the world is a struggle and its weapon is science and knowledge to the one who did not spare me anything.....to From striving for my comfort and success to the greatest and dearest man in the universe: my dear father. To the one who supported me in her prayers and supplications, to the one who stayed up late nights, illuminating my path. To those who share my joys and sorrows, to the source of kindness and tenderness, to the most beautiful smile in my life, to the most wonderful woman in existence: my dear mother. To those who were given a gift from predestination as brothers, so they knew the meaning of brotherhood, my beloved brothers: Saafa and Dayaa. To my dear sisters: Nisreen and Tabarak, my sister and my brother Dunya's wife, I offer a special dedication to the family's buds: Tho Alfikar, Azal, Sama, Rose and Fatima. To all my classmates: And to my dear wife, who has stood by me throughout my studies and helped me my dear wife (Narmen) and I thank her for all the assistance and to Everyone who helped me in accomplishing my message to all of them, I dedicate this research.

Bahaa Jalil Ibrahim WAISI

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CONTENTS

LIST OF SYMBOLS

µL	Microliter
g	Gram
L	Liter
mg/dL	Milligrams per decilitre
mL	Milliliter
mmol/L	Millimoles per liter
nm	Nanometer

LIST OF ABBREVIATIONS

CABG	Coronary artery bypass grafting
CAS	Coronary artery spasm
CHARM	Candesartan in heart failure assessment of reduction of mortality and morbidity
CYS-C	Cystatin-C
ECG	Electrocardiogram
ECM	Extracellular matrix
GAL-3	Galectin-3
IHD	Ischaemic heart disease
MI	Myocardial infarction
MVA	Microvascular angina
PCI	Percutaneous coronary intervention
VSA	Vasospastic angina

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1. INTRODUCTION

Rest or sublingual nitroglycerin treat angina, which is caused by brief myocardial ischaemia and often occurs in response to physical or mental stress (Panchmatia 2009, Aguiar-Souto *et al.* 2005). In New Zealand, 3.8% of the population suffers from angina. Approximately 50% of individuals with ischemic heart disease present with symptoms compatible with a stable angina pattern (Marzilli *et al.* 2006) Heterotrimeric proteins, laminins include an a-chain, which is present in five of the genetic variations, a ß-chain (four), and a y-chain (three). The chain composition is used to name the laminin molecules. As a result, the a5, 31, and y1 chains seen in laminin-511 (Aumailley et al. 2005). More than a dozen different chain combinations have been discovered in living organisms. Intersecting trimeric proteins create a cross-like shape, which may interact to other cell membrane and extracellular matrix proteins (Fisher *et al.* 1995). To form sheets, the three shorter arms of laminin bind especially well to those of other laminin molecules. Using the long arm's ability to connect to cells, the membrane is held in place. As a structural protein and a signaling molecule, laminin a4 is a key component of the extracellular matrix (ECM) laminin-8 and-9. Because laminin is so plentiful.

Seek care immediately. The clinical syndrome of angina pectoris occurs in a number of cases in which the oxygenated blood is not sufficient to nourish the heart muscle, meaning an imbalance occurs between the oxygenated blood supplied to the heart muscle and the oxygen required by this muscle.

1.1 Objectives of Study

The aim of this study is to evaluate the importance of Cystatin-C(CYS-C),Galectin-3(GAL-3), Myonectin levels and number of biochemical parameters inEngina Pectoris patients.

2. LITERATURE REVIEW

2.1 Area of Study

A "variant form of angina pectoris" was coined in 1959 by Prinzmetal and colleagues to describe a novel kind of angina pectoris that varied significantly from the classical variety. (Prinzmetal *et al.* 1959). This syndrome had the following characteristics: (Prinzmetal *et al.* 1959). At rest, the attacks occurred, and London was the target. ST segment elevation on an ECG was shown to be a common feature among those who had attacks. Atrial with CCBs may be sufficient for the assessment of potential spasm since the attack occurred at rest and was notcardiac catheterization laboratory. London. When a patient had an incident of variant angina in the early morning, we found that anepicardial coronary artery spasm or coronary spasm might be seen. About the same when Endo and his colleagues released their findings to the world (Kaski *et al.* 2019). coronary spasms have been associated to a wide range of cardiovascular diseases, including exertion angina, unstable angina, acute myocardial infarction, and sudden death (sudden death) (Yasue and Maseri 1978). As a result, angina pectoris produced by coronaryspasm is now referred to as "coronary spastic angina" or "vasospastic angina" because of the vast range of myocardial ischemia syndromes generated by coronaryspasm. A study published in 1983 by Yasue et al. Prior to this article, we provided a comprehensive analysis of coronary artery spasm (CAS). In this article, we amend that study and give updates on our knowledge of the condition (Yasue et al. 2008).

As the top cause of mortality and lost life years in adults worldwide, IHD is particularly prevalent in younger women (less than 55 years of age) (Prinzmetal *et al.* 1959). Angina pectoris is the medical term for chest discomfort induced by coronary artery disease (derived from the Latin word "angere" to suffocate). Prevalence estimates range from 3 percent to 4 percent among adults in the United Kingdom. Invasive coronary angiograms are conducted more than 250,000 times a year, with more than 20,000 new instances of angina each year. Over 110000 inpatient episodes occur each year, resulting in significant morbidity, which is a significant use of healthcare resources. In 1768,

William Heberden noticed myocardial ischaemia, and in 1809, Allen Burns (Lecturer in Anatomy, University of Glasgow) formulated the idea that this may explain angina. Histology and clinical case studies by John Hunter, John Fothergill, Edward Jenner, and Caleb Hiller Parry after Heberden's work all pointed to c nm,oronary artery disease (CAD) as a likely cause (Kligfieldt *et al.* 1982).

Myocardial oxygen depletion (ischemia) is a common cause of angina, which often develops during physical exertion or physiological stress (box 1).

The first invasive coronary angiogram was performed six decades ago; despite this, many clinicians still believe that obstructive epicardial CAD must be detected on coronary angiography in order to accurately diagnose angina.

Finding obstructive CAD enables for evidence-based medical treatment and the possibility of cardiac revascularization in patients (MR). There are several factors that contribute to the underlying pathophysiology, including structural atherosclerosis, as well as abnormalities in the epicardial arteries and microcirculation (Kaski *et al.* 2018).

Individuals who have signs and symptoms of ischaemia but do not have obstructive coronary artery disease are now referred to as having "Chronic Coronary Syndromes" according to the ESC6 guidelines (INOCA) (Montalescot *et al.* 2013, Ford *et al.* 2018).

About half of all patients with angina who do not have obstructive epicardial CAD get elective coronary angiography (Patel and colleagues 2010)

Heart problems in the INOCA population are almost always caused by either MVA or VSA, which is why INOCA chronic coronary syndrome is so difficult to diagnose. We must emphasize that several pathways may cause myocardial ischaemia in distinct coronary compartments. This fact, however, might help us better stratify therapy and better comprehend those who do not respond well to it (eg, angina after

revascularisation). These indices are all numerically equivalent, implying that they may all be used as a guide for treatment plan (Lee *et al.* 2019).

Patients with angina who also have an underlying form of obstructive coronary artery disease (CAD) might benefit from symptom-guided myocardial revascularization, which is often accomplished by percutaneous coronary intervention (PCI) (in many patients). Functional coronary diseases may overlap and have a role in angina even in individuals with epicardial coronary artery disease (CAD), according to recent investigations. In individuals with obstructive CAD, dynamic changes in the 'tone' of the lesion or artery, as well as their predisposition to vasoconstriction, are critical and may produce rest angina. KG. Gould KL. A dynamic stenosis of the coronary artery. A study published in 1980 by Gould et al.

At FFR of 0.76, which is very low in patients with hyperaemia caused by adenosine, ischaemia occurred in Asrress et al during invasive physiological examination of exercise-induced ischaemia (Asrress *et al.* 2017).

According to these observations, subendocardial ischaemia may be caused by mechanisms other than those listed above (myocardial supply:demand factors). That angina is not the same as ischemia or flow-limiting coronary disease has been shown by this study (eg, abnormal FFR or NHPR). It's important to keep the patient's condition in mind while thinking about coronary architecture and physiology. What are the risks associated with angina, such as heart attacks, strokes, and heart failure, if you have angina? The large VA cohort study by Spertus and colleagues recruited outpatients with stable ischemic coronary artery disease (>4400). 1 Those with severe angina, based on their responses.

2.2 Questionnaire Survey of General

The Seattle Angina Questionnaire found that people with mild or minimal angina had a higher one-year mortality risk than those with moderate or severe angina. More than

1,000 persons with stable ischemic heart disease were selected to participate in the Heart and Soul Study, which monitored patients for 8.9 years. These individuals' angina was categorized as nonexistent, occurring once a month, once a day, or once a week. The study's endpoints were inpatient admissions for angina or heart failure, revascularization, myocardial infarction, or death. Hospitalization, revascularization, or death from any cause owing to angina was more common in patients who had angina on a daily or weekly basis than those who only had the symptoms monthly or did not have angina at all. According to Scirica, long-term angina is related with an increased risk of death and recurrent myocardial infarction (Scirica *et al.* 2009).

Jespersen *et al.* (2004) showed that individuals with no signs of angina had a higher chance of avoiding a significant adverse cardiac event than those who did (including patients with 1-, 2-, 3-vessel coronary artery disease, diffuse, nonobstructive coronary disease, and even in patients whose epicardial coronary arteries appeared normal, suggesting that this later group may have had microvascular disease).

For further information, see (Jespersen and colleagues 2012). To put it another way, individuals with angina had a poorer prognosis than those who did not have angina, regardless of their coronary structure. Aside from forecasting.

2.3 The Worst Possible Cardiac Condition

Serious cardiac episodes may cause angina, a common side effect. A research by Hlatky et al. indicated that angina patients had a worse quality of life over the course of a decade than those who did not have the condition. For instance (Hlatky *et al.* 2004). In other investigations, angina was linked to a worse quality of life (Weintraub *et al.* 2008). Angina-free and angina-inducing patients with heart failure have lately been examined for their clinical outcomes. In the Candesartan in Heart Failure Assessment of Reduction of Mortality and Morbidity (CHARM) study, individuals with angina pectoris had greater mortality and morbidity than angina-free patients in the NYHA functional class. A larger risk of major cardiovascular events did not raise the risk of all-cause death in individuals with angina (myocardial infarction, unstable angina, the

requirement for percutaneous coronary intervention [PCI], or coronary artery bypass grafting [CABG].). in the work of Montalescot and associates

The long-term results of patients with ischemic cardiomyopathy with angina pectoris or without angina pectoris were compared. However, the mortality, myocardial infarction, and death or hospitalization rates were not different between patients with and without angina after multivariate adjustment for patients with angina. As of (Kaski and coworkers 2018).

The presence of angina in patients with ischemic cardiomyopathy has a significant impact on their quality of life, even though angina does not seem to alter all-cause mortality, according to these studies. There is a worse prognosis for people with angina who have ischemic cardiomyopathy. According to the study, angina may sometimes go into remission. Angina patients who have been on medicine for a decade or more have had their symptoms completely disappear. A sedentary way of life, ischemic segment hibernation, the recruitment of new collaterals, or even chronic ischemia conditioning might be to blame (purposefully limiting oxygen demand). Remissions and exacerbations of angina are possible, but they're not the same thing.

2.4 Secondary Causes

Secondary and treatable angina exacerbations include poorly controlled hypertension, anemia, tachycardia, hyperthyroidism, infections with fevers and chills, hypoglycemia, and heavy meals, all of which raise oxygen demand and increase the risk of angina. As a general rule, angina has been associated to lower outcomes. Short-term occlusion and reperfusions of the coronary arteries (short enough to mimic angina but not long enough to cause tissue necrosis) were first described in experimental studies in the 1980s, when it was discovered that these preconditioning procedures protected the heart from damage when the coronary arteries were later occluded for longer periods. Myocardial infarctions were decreased considerably by using this ischemia preconditioning. Every species investigated so far has experienced ischemic preconditioning. It was shown that angina history had an impact on enzyme-based myocardial infarction in the TIMI 4

research. ST-segment elevation myocardial infarction (STEMI) patients with previous angina had less total creatine kinase-MB units than those without. Individuals who had a history of angina were less likely to die in the hospital and suffer from acute congestive heart failure and/or shock as a consequence. Angina patients' coronary artery collaterals did not play a role in this improvement in outcomes. 9 Preinfarct angina that occurs within 24 hours of a STEMI had a protective impact in TIMI 9, but we restricted that protective effect in TIMI 10. 10 Studies currently suggest that preinfarct angina improves the size of myocardial infarctions, the function of the left ventricle and other factors. It's possible that this preinfarct angina is clinically identical to ischemia preconditioning.

In spite of this, ischemia preconditioning in the context of STEMI is difficult, if not impossible, to use as a therapeutic. For example, remote ischemia conditioning, in which a distant organ, such as a hand or foot, is forced to experience an ischemic state for a short amount of time and subsequently re-perfused, may be employed therapeutically in the context of STEMI.

Many clinical investigations currently suggest this treatment is helpful in decreasing the extent of the infarct in patients with STEMI who are treated with short, repetitive, blood pressure-induced brachial artery occlusions initiated in the ambulance (Kloner and colleagues 2016). To protect the heart, either a protective humoral substance from preconditioning or neural reflexes that are induced by preconditioning may be discharged into the circulatory system.

2.5 Abstract

As many as one in four individuals will die from coronary artery disease, or CAD (CAD). Angina pectoris, a condition in which a person has discomfort in the chest, neck, or left arm, is one of the numerous indications and symptoms of CAD. The gold standard for detecting and evaluating coronary artery disease is a coronary angiography (CAD). Therapy is focused at decreasing the risk of death from heart disease, which includes factors including diabetes, high blood pressure, and high cholesterol. For the

treatment of cardiovascular disease, antiplatelet medicines and nitrates are often recommended. coronary artery disease (CAD), also known as coronary heart disease (CHD), coronary artery atherosclerosis (CAA), or stable ischemic heart disease, is caused by an inadequate flow of blood to the myocardium (SIHD). In many cases, coronary artery disease is to blame. The leading cause of mortality in the world is coronary artery disease (CAD). For further details, please check (Go et al. 2014). Angina pectoris is one of the numerous possible clinical manifestations of coronary heart disease, which is characterized by chest, neck, or left arm discomfort.

Chronic stable angina pectoris (CSAP) is a common sign of coronary artery disease (CAD). At least 15.5 million persons in the United States have chronic coronary artery disease (CAD), and angina affects more than 7 million. Angina is the initial symptom experienced by almost half of those suffering from coronary artery disease (CAD). Patients with persistent angina have a roughly twofold increased risk of serious cardiovascular events (CVEs). (Fihn and colleagues 2014). With physical activity, discomfort in the chest known as chronic stable angina occurs, which is eased by rest or nitroglycerin. At repose, chest discomfort is frequently a sign of a more serious problem, such as angina or an acute coronary syndrome (ACS).

The pain of angina is not universally perceived by patients, but rather as a sense of tightness, pressure or discomfort. If you are a woman or an older person, you may have uncommon symptoms such as nausea, vomiting or discomfort in the middle of the esophagus. It is common for ischemia-induced anginal discomfort to persist a few minutes. As the discomfort spreads, it might be felt in the neck and jaw, as well as in the esophagus and arms. For the most part, angina isn't characterized by pain above the mandible, lower than the epigastrium, or on the left side of the chest. Exertion or mental stress may cause angina, which can be eased by relaxation. Within 30 seconds to several minutes, sublingual nitroglycerin generally resolves angina. The gold standard for identifying coronary artery disease in people with SIHD is coronary angiography (Jespersen and colleagues 2012). X-rays and a contrast agent may show how the coronary arteries function.

Hlatky *et al.* (2004). Heart angiography may help individuals with suspected SIHD who have unbearable ischemia symptoms and don't respond to recommended therapy. Angiography may assist patients with suspected SIHD identify the extent and severity of coronary artery disease (CAD). They must be willing and eligible for coronary revascularization and have completed noninvasive testing, excluding stress tests that indicate a high chance of severe IHD, to be eligible for this procedure. Patients who are unable to undertake stress testing or who have an ambiguous or nondiagnostic stress test may benefit from coronary angiography if the results might lead to a change in treatment. Even in individuals who have had a satisfactory stress test, coronary angiography should be explored if clinical suspicion is strong and it is likely that the findings will lead to changes in treatment (Hlatky *et al.* 2004).

Allen Burns (Lecturer in Anatomy, University of Glasgow) proposed the notion that angina may be caused by myocardial ischaemia in 1809, after William Heberden first discovered it in 1768. After Heberden's publication, other pathologists and clinicians, including John Hunter, John Fothergill, Edward Jenner, and Caleb Hiller Parry, began conducting research (Scirica *et al.* 2009).

Ischaemia (deficiency in myocardial oxygen supply) is the most common cause of angina, and it often arises after physical or physiological stress (box 1). While invasive coronary angiograms have been around for more than 60 years, many doctors still regard the presence of epicardial obstructive cardiomyopathy (CAD) to be an absolute need in the diagnosis of angina (Jespersen *et al.* 2012).

Evidence-based medical therapy and the contemplation of myocardial revascularization are possible when obstructive coronary artery disease is discovered. Epicardial arteries and microcirculation are both affected by atherosclerosis, as are structural atherosclerotic and functional alterations (Hlatky *et al.* 2004).

Individuals with Ischemia without Obstructive Coronary Artery Disease may now be referred to as having "Chronic Coronary Syndromes" (INOCA). The great majority of

individuals with angina who undergo coronary angiography are found to be devoid of epicardial coronary artery disease after the procedure (CAD) (Kloner *et al.* 1995).

Heart problems in the INOCA population are almost always caused by either MVA or VSA, which is why INOCA chronic coronary syndrome is so difficult to diagnose. We emphasize that diverse causes of myocardial infarction may develop in distinct coronary compartments through different processes. As a result, recognizing this fact may assist us better stratify therapy and help us understand patients who do not respond well to treatment (eg, angina after revascularisation).

As a first step, we'll categorize angina based on its pathogenesis. The existing criteria for screening patients with recent development of stable chest discomfort are then examined in terms of their strengths and limitations. Our discussion of coronary artery function testing includes both noninvasive and invasive techniques and their accompanying treatment plans. Our discussion will conclude with prospective options for the future, which might lead to better patient outcomes and the creation of disease-modifying medicines specific to a certain ailment. Epicardial, microcirculatory, and coronary vasospasm dysfunction are all taken into consideration in this study to provide a more current approach to angina diagnosis and treatment.

2.6 Myocardial Infarction (MI)

Based on accepted definitions of anatomical (Weiner 1979) and prognostic (Murabito 1990):

- 34th recurrence.
- Chest discomfort that comes and goes (up to 15 minutes).
- Exertion-induced or -emotion-induced.
- Rest or nitroglycerin relieve the pain.
- A person's personality and level of exposure match the diagnosis.

3. MATERIALS AND METHODS

3.1 Used Specimens

3.1.1 Control group

The current study included healthy normal people as a comparison group (control) and their number was (40 samples) who were from all regions of Diyala Governorate from the auditors to the private laboratories and their ages ranged between (30-85) years

3.1.2 Patients gruep

Blood samples were obtained from patients with angina pectoris from the intensive care unit at Ibn Al-Nafis Hospital affiliated to the Rusafa Health Department - Baghdad. The number was (50 samples) males and their ages ranged between (40-85) years. Tests conducted by experts in the hospital and based on clinical symptoms and information gathered from patients using a questionnaire form designed for this purpose, the specifics of which are given in an appendix, were used to confirm that they had the condition.

3.1.3 Collection of blood

Samples Samples were collected by drawing blood from a vein by means of a plastic wine syringe a volume of (10 mL) and the amount of blood withdrawn was (7 mL), then the blood samples were divided into three sections

The first section: was taken (4 mL) of the withdrawn blood and put it in plastic tubes with a tight cover and free of anticoagulant (Plain tube) in order to obtain the serum. At a speed of (3000) revolutions / minute for (15) minutes, it was used to conduct biochemical tests on it

The second section: Take (1mL) of the blood drawn into plastic tubes with a tight lid and containing Na2 EDTA, and then put it directly into a cooling centrifuge at a speed of (1000) revolutions / min at 4°C for a period of (10) minutes in order to separate and store the plasma In special tubes in deep freezer at 20C°- for the purpose of HPLC homocysteine measurement

The third section: (2mL) of the blood drawn into clean and sterile plastic tubes containing sodium heparin to prevent blood clotting, with light shaking of the sample in the tube to ensure that it mixes with heparin, then the plastic tubes containing the blood are transferred to the laboratory (after Put them in the cool box (taking into account the proper preservation of samples and the prevention of contamination) for the purpose of conducting a molecular examination.

3.2 Estimation of the Level of Analogue (GAL_3) in the Blood Serum

The level of the enzyme (GAL_3) was estimated using the kit (Kit) supplied by the company (Competitive ELISA).

The basic principle: The competitive enzyme immunoassay approach is used using a polyclonal anti-GAL-3 antibody and a GAL-3-HRP conjugate in the Enzyme-Linked Immunosrbent Assay (ELISA) kit. For one hour, the GAL-3-HRP conjugate is incubated with the assay sample and buffer on a pre-coated plate. Incubation is followed by five rounds of decanting and washing of the burc wells. Substrate for HRP enzyme is subsequently added to the wells. A blue-colored complex is formed as a result of the enzyme-substrate reaction. Stop solutions are introduced at this point, and the reaction is then stopped, which results in a bright yellow color. A microplate reader is used to detect color intensity spectrophotometrically at 450nm. Since GAL-3 from samples and GAL-3-HRP conjugate compete for the antibody binding site, the color intensity is inversely proportional to GAL-3 concentration. Because there are only a limited number of binding sites, as more GAL-3 from the sample occupy the binding sites, less GAL-3-HRP conjugate binding sites are available. An O.D. standard curve is drawn to show the

relationship between color intensity (O.D.) and standard concentration (S.C.). From this standard curve, each sample's GAL-3 content is interpolated.

3.3 Determination of Serum Aspartate Aminotransferase (AST):

The colorimetric method was used to measure the activity of the enzyme (AST) in the blood serum, which is a method (Reitman and Frankel 1957), using the kit for analysis supplied by the French company (BIOLABO). As a result of this approach, Amount of enzyme activity (AST) may be determined by measuring the absorbance of (2,4 Dinitrophenylhydrazones) formed by the interaction of oxalates with (2,4 Dinitrophenylhydrazine (DNPH) as in the following Table 3.1.

Table 3.1 Determination of serum aspartate aminotransferase (AST)

pH 7.5 Sodium Phosphate Buffer The L-Aspartate 2- Oxoglutarate in Vial R1 is a (GOT Substrate)	85 mmol/L 200 mmol/L 2 mmol/L
In Vial R3, place HCL 2,4-Dinitrophenylhydrazine (DNPH)	1 mmol/L 1 mol/L
In order to use R4 (Standard Solution) Phosphatidylserine Buffer for Sodium Mercurothiolate (pH = 7.5)	2 mmol/L 0.1 % 100 mmol/L

Preparation of the working reagent:

- The buffer solution (GOT Buffer) is ready for use.
- (2,4-Dinitrophenylhydrazine) is also ready to use.
- 3- Sodium Hydroxide (NaOH) at a concentration of (0.4) molar was prepared by dissolving (16 g) of it in (1 L) of distilled water, and thus the solution is ready for use.

Procedure: The reagent solution (R1) was diluted to (0.5 mL) in a test tube, and the tube was then put in the incubator at (37 oC) for five minutes. Incubation was continued for an additional 60 minutes after the tube was removed from the incubator and 100 L of serum was added. The tube was well mixed before being returned to the incubator. The

reagent solution (R3) (0.5 mL) was poured over it when it was removed from the incubator. At room temperature for (20) minutes, it was completely mixed, then (5 mL) of sodium hydroxide was added and left for (5) minutes, and the Absorbance at a wavelength was read (505 nm). After measuring the absorbance intensity using a spectrophotometer, the absorbance of the enzyme (AST) was calculated, then the values of the absorbance of the enzyme were projected onto the standard curve of the enzyme shown in Figure (1.3) below, from which the enzyme activity was calculated in units (IU/L).

3.4 Serum Total Cholesterol Concentration Measurement

The French business provided an analytical kit that was used to measure the serum total cholesterol levels via the Enzymatic Method (BIOLABO) (Tietz 1999). This method depends on the enzymatic oxidation of free cholesterol and cholesterol ester, as shown in the following Table 3.2.

Table 3.2 Determination of serum total cholesterol concentration

Triton x 100 Phosphate Buffer Chloro-4-Phenol Triton (Buffer)	100 mmol/L 5 mmol/L 2.3 mmol/L 1.5 mmol/L
Vacutainer tube number two (R2) (Enzymes) Oxydase Cholesterol The 4-Amino-Antipyrine PEG 6000 Cholesterol Esterase Peroxydase	≥ 100 IU/L ≥ 170 IU/L ≥ 1200 IU/L 0.25 mmol/L 167 µmol/L
Standard Cholesterol Vial R3	200 mg/dL

Preparation of the working reagent: The reagent was prepared by mixing the contents of (R2) with the contents of (R1) well and kept until use Table 3.3.

Process: Three test tubes were used (Model, Standard Solution, Blank). Each of them contained the solutions installed below.

Table 3.3 Preparation of the working reagent

Solutions	Blank	Standard	Assay
Reagent	1mL	1mL	1mL
Standard	-	10µL	-
Sample	-	-	10µL

After thoroughly mixing the solutions, the absorbance at a wavelength of (500 nm) against the efficient solution was measured after they had been left for (5) minutes at (37 oC) or (10) minutes at room temperature. As the color intensity remained stable for a period of time. hour.

The estimation of the concentration of triglycerides in serum was based on the method (Fossati and Prencipe, 1982), using the analysis kit supplied by the French company (BIOLABO), which is based on this method. Enzymatic analysis of triglycerides to glycerol according to the following Table 3.4.

Table 3.4 Determination of serum triglycerides concentration

Magnesium Chloride Chloro-4-Phenol is piped into Vial R1 (Buffer)	100 mmol/L 9.8 mmol/L 3.5 mmol/L
Vacutainer tube number two (R2) (Enzymes) Lipase \sPeroxydase It is an enzyme that oxidizes glycerol 3 phosphorus Antipyrine for Glycerol Kinase 4-Amino (PAP) Biochemical compound that is made up of three phosphate groups. Na \sl	≥ 1000 IU/L ≥ 1700 IU/L ≥ 3000 IU/L ≥ 660 IU/L 0.5 mmol/L 1.3 mmol/L
Glycerol in Vial R3 (Standard)	200 mg/dL

The working reagent was made by combining the contents of (R2) and (R1) thoroughly and storing it in an airtight container until needed Table 3.5.

Procedure: Three test tubes were used (Model, Standard Solution, Blank). Each of them contained the solutions installed below.

Table 3.5 Preparation of the working reagent

Solutions	Blank	Standard	Sample
Reagent	1mL	1mL	1mL
Standard	-	10µL	-
Sample	-	-	10µL

It was then tested at a wavelength of 500 nm (500 nm/500 nm) against the efficient solution, which had been mixed thoroughly and left for five minutes at (37oC) or 10 minutes at (room temperature). As the color intensity remained stable for an hour.

3.5 Determination of hdl-cholesterol levels in the blood (HDL-C)

The enzymatic approach based on quantitative precipitation of VLDL-C was used to measure HDL-C concentration. As well as (LDL-C) and chylomicrons when phosphotenetic acid (PTA) and magnesium ions are added to a kit provided by the French firm, which provides analytical services (BIOLABO). The filtrate obtained after centrifugal separation contains (HDL-C) only. It was estimated using the enzyme solution of cholesterol Table 3.6 (Badimon *et al.* 1990).

Table 3.6 Detection of Serum HDL-Cholesterol Concentration (HDL-C)

magnesium chloride, precipitant, vial R1 of phosphotungstic acid	13.9 mmol/L 570 mmol/L
It's a Vial R2 (Standard) Cholesterol	100 mg/dL

Procedure: Sedimentation: A test tube was taken and (0.5 mL) of serum was placed in it and (50µL) of the precipitated solution was added to it as shown in the Table 3.7.

Table 3.7 The following reagents are placed in test tubes

Solutions	Sample
Sample	0.5 mL
Precipitant	50µL

The contents of the tube were mixed well and left at room temperature for (10) minutes, after which the solution was separated using a centrifuge (4000 rpm) for (15) minutes. Then the filtrate was separated and the reagent was used to estimate cholesterol.

3.6 Estimation of cholesterol concentration for (HDL-C)

Three test tubes (Form, Standard, Efficient (Blank)) were taken. Each contained the solutions indicated below Table 3.8.

Table 3.8 Estimation of cholesterol concentration for (HDL-C)

Solutions	Blank	Standard	Sample
Reagent	1mL	1mL	1mL
Standard	-	25µL	-
Supernatant	-	-	25µL

A spectrophotometer was used to detect the absorbance at a wavelength of 500 nm, and the concentration of (HDL-C) was determined by comparing the readings to a standard curve.

3.7 Statistical Analysis

Study parameters were analyzed using a Statistical Analysis System- SAS-2018 application to determine the influence of different groups (control vs. patients). Using a T-test, researchers compared the means of two groups in this study.

4. RESULTS AND DISCUSSION

The results of this study included the first of patients with angina pectoris by performing the analysis of troponin and the second of the healthy, and it was divided into four tables, each table compared between the control group and patients, and the first table includes four groups: CYC_C, GAL_3,Myonectin, INS and The second table also includes four groups Cholesterol, Triglycerid, HDL, LDL, VLDL and the third table includes three groups GOT, GPT, ALP and the fourth and last table is between Age (year) and Weight (kg) Table 4.1.

Table 4.1 Comparison of CYS-C, GAL-3, Myonectin, and INS levels between the control and patient groups

Group	Mean ± SE			
	CYS-C	GAL-3	Myonectin	INS
Control	317.29 ± 4.63	6.78 ± 0.27	4.99 ± 0.05	1.99 ± 0.04
Patients	514.96 ± 18.28	12.40 ± 1.01	6.25 ± 0.26	2.42 ±0.20
T-test	41.122 **	2.271 **	0.569 **	0.406 *
P-value	0.0001	0.0001	0.0001	0.0459
* (P≤0.05), ** (P≤0.01).				

The results showed that there were significant differences for each patient according to the information recorded in the form mentioned in Table (1-10), as there was a difference of patients with angina pectoris (317.29) in CYS-C and (6.78) inGAL-3 and (4.99) in Myonectin and (1.99) in INS with angina pectoris, and(514.96) in CYC –C and (12.40)inGAL-3 and (6.25)in Myonectin and(2.42) in INS, which is a high percentage during the collection of samples from the CCU, as shown in the above Table 4.1.

In the early post-infarction period, the concentration dynamics of this biomarker may be altered by a variety of variables, including the development of cardiac dysfunction after an acute myocardial infarction. This was the focus of our study. TECHNIQUES AND MATERIALS: Non-ST-elevation angina (NSTEMI), an ST-height myocardial infarction, and SAP were the diagnosis of this research, which included 50 patients (STEM).

3'-Galectin HTA and triglyceride levels impacted the early post-infarction levels of galectin-3, but by day 30, this biomarker was shown to follow the pattern of myocardial remodeling.

Galectin-3 has long been recognized to have a role in the development of atherosclerosis. Compared to umbilical cord arteries, atherosclerotic arteries had greater amounts of galectin-3 (Arar *et al.* 1998, Nachtigal *et al.* 1998). Animal model studies using experimental atherosclerosis examined the expression of the Galectin-3 gene. Patients with hypercholesterolemia and artificial aortic injury had higher levels of galectin-3 gene expression in their smooth muscle cells. Galectin-3 seems to have a function in the development of atherosclerosis, as seen here. (Arar and colleagues 1998)

As compared to fibrotic atherosclerotic lesions, lesions rich in foam cells showed greater amounts of galectin-3 and galectin-3 positive cells than did atherosclerosis lesions. Atherosclerotic lesions often include a lipid core or patches of fibrosis, hemorrhage, or thrombosis around the galectin-3 positive cells (Nachtigal *et al.* 1998). in the foam cells of atherosclerosis, galectin-3 was highly expressed, but the presence of atherosclerosis was reduced in the absence of galectin-3 expression (Naughty and Nice 2008). In the active phase of atherosclerotic plaques, researchers think that Galectin-3 is involved.

Patients with ACS should have their blood tested to see whether they have ACS. In this investigation, we looked at the galectin-3 levels in patients with AMI and in the SAP control group. No statistically significant differences in galectin concentrations were seen on day one, five, or thirty following PCI. Galectin-3 secretion kinetics in the acute phase of AMI have not been determined, as previously claimed. Acute myocardial infarction has been demonstrated to increase the mRNA levels of galectin-3 in mice from 30 minutes to 1–2 weeks following the onset of the disease (AMI). A significant increase was seen in plasma galectin-3 concentrations 24 hours after MI in patients in the study with the shortest mRNA peak time. To learn more about this subject, check out our sister site (Li *et al.* 2020).

Galectin (Gagno *et al.* 2019), which has been connected to CRP and WBC count, was shown to be much more prevalent among AIM patients than SAP. It is consistent with previous studies (Byrne *et al.* 2004) that indicated that individuals with atypical chest pain had AIM in addition to those with SAP or SAP. ACS patients, on the other hand, had exhibited lower lymphocyte counts than SAP patients, according to (Fang *et al.* 2004). According to Luke et al. As compared to healthy controls, patients with ACS showed significantly higher neutrophil to lymphocyte and monocytic to lymphocyte ratios (NLR and MLR). As a result, it has been shown to be the best indication of ACS (Shumilah *et al.* 2021). The higher the WBC count, the greater the risk of mortality for patients with unstable angina pectoris, acute coronary syndrome, acute myocardial infarction (AMI), percutaneous coronary intervention (PCI), or coronary artery bypass grafting (CABG) (Madjid *et al.* 2013). Considering that past studies have shown a link between blood C-reactive protein levels and the degree of coronary stenosis, patients with STEMI were expected to have higher blood C-reactive protein levels than those with SAP or NSTEMI (Assadpour *et al.* 2012, Seyedin *et al.* 2016).

It is possible to determine the risk of coronary artery disease in individuals with acute chest pain by detecting the levels of hs-CRP (a marker of inflammation) in the blood (Lozano *et al.* 2007).

Results showed that HTA, triglyceride level and day 30 STEMI were related to LA size and percentage of LM stenosis. An independent predictor of galectin-3 concentration was found on day 1 in STEMI and on day 5 in the overall patient group for the E/A ratio. Galectin-3 levels were greater in patients with HTA, particularly those with LV remodeling, even in the earliest stages of illness, according to a study (LVRM). The amount of galectin-3 in the blood has also been linked to LV mass. A biomarker for HTA Yao's early cardiac remodeling may therefore be relevant (Yao *et al.* 2016). The drop in eGFR, the increase in myocardial index, and the categorization of hypertension patients as high or low risk are all linked to the ambulatory ECG biomarker galectin-3 (pusuroglu *et al.* 2016). A growing amount of research suggests that elevated blood pressure may contribute to fibrosis by boosting the synthesis of inflammatory molecules

such galectin-3 Azibani. [source: Azibani] (Azibani *et al.* 2012, Martnez and Azibani 2015).

Cardiovascular disease is often linked to dyslipidemia. Several cardiometabolic diseases, such as dyslipidemia, have been associated to galectin-3 in recent study (Nayor *et al.* 2015). Cardiotoxicity has been shown to cause cardiac remodeling by increasing lipid fractions and free fatty acids and triglycerides (Kanukurti *et al.* 2020).

Along with intramyocellular triglyceride accumulation, lipotoxicity also alters the lipid composition of cells to facilitate the generation and production of activated mediator lipids that affect metabolism and cardiac function in part through the alteration of mitochondrial activity within the heart (Marin *et al.* 2018). Galectin-3 causes damage by binding to advanced lipoxidation end products, which build up in target organs and trigger proinflammatory and prooxidative pathways (Menini *et al.* 2016). Using an animal study, it was shown that galectin-3 inhibition reduced cardiac lipotoxicity and alleviated heart mitochondrial damage (Ansari *et al.* 2018). Galectin-3 was found to have a significant positive correlation with total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and very low-density lipoprotein cholesterol (VLDL-C) in patients with heart failure and preserved ejection fraction, while HDL-C was found to have a significant negative correlation with all of these lipids (Ionin *et al.* 2020).

As a follow-up to our work (Ansari *et al.* 2018), a correlation between galectin-3 levels in patients with an intact ejection fraction and LA dimension and E/A index has been found. metabolic syndrome has been proven to produce structural alterations in the atria, including an increased LA size and the development of interstitial atrial fibrosis, despite its unclear pathophysiological reasons. Fasciitis is generally acknowledged to be an important factor in AF. Galectin-3 blood levels may be beneficial for patients with metabolic syndrome as a fibrosis marker, which may be used to identify the degree of fibrosis and the risk of atrial fibrillation (lonin *et al.* 2020).

AF is more likely to occur in people with dilated mitral annulus when the P-A0 TDI interval (the duration between the P wave on ECG and the peak of the A0 wave determined by tissue Doppler imaging at the septal or lateral mitral annulus) is linked to a dilated mitral annulus in sinus rhythm (Nuzzi *et al.* 2019). As we found in our study, the quantity of galectin-3 in NSTEMI and STEMI patients on day 30 may be predicted independently by TMZ and long-acting nitrates. Use of TMZ in combination with optimum medical treatment may minimize oxidative stress, endothelial abnormalities, inflammation and significant acute cardiovascular events in patients with NSTE-ACS (Bobescu *et al.* 2021). After myocardial ischemiareperfusion damage, this metabolic drug protects myocardial cells from further damage due to its postconditioning, which is in addition to relieving angina and myocardial ischemia symptoms and improving patient outcomes. In the field of biomedical engineering. It has been shown that TMZ may reduce the formation of neutrophil cells after reperfusion in an animal model of ischaemia and re-perfusion (Di *et al.* 2005). Apoptosis and metabolic alterations produced by MI Luo are also prevented by TMZ (Luo *et al.* 2021). CK, LDH, and AST levels fall after a heart attack (Zhang *et al.* 2021).

TMZ may enhance echocardiographic parameters such as LVEF, decreased LV end-systolic volume, and the wall motion score index in people with poor myocardial function. However, nitrate treatment causes an increase in the production of NO and platelet disaggregation by activating the cGMP/cGMP-dependent protein kinase I, which results in the synthesis of endogenous nitric oxide (NO) (Daiber *et al.* 2015, Boden *et al.* 2015). According to research, elevated plasma NO concentration has also been shown to have an immediate positive impact on LV diastolic performance and myocardial hypertrophic remodeling (Zakeri *et al.* 2015). Similar to the anti-inflammatory characteristics of NO, it inhibits neutrophil adhesion and chemotaxis during acute inflammation as well as modifies microvascular permeability (Divakaran *et al.* 2017).

Long-term use of nitrates is related with a change from STEMI to NSTE-ACS and a much reduced release of cardiac enzymes (CK-MB and troponin) compared to nitrate nave individuals (Ambrosio *et al.* 2010). Because these medicines have hemodynamic

effects and may improve collateral flow, they can minimize infarct size or stabilize reperfusion in conjunction with thrombolytic therapy. It has also been shown that nitrates may lower mortality by 35 percent in individuals who fail to return to normal circulation after a heart attack (Meng *et al.* 2020).

In terms of age, male and diabetic percentages, TC, LDL-C, and HCY in various kinds of CAD patients, there was a significant difference between the acute myocardial infarction and stable angina pectoris groups. C-statin C levels in AMI were 317.29 4.63, whereas SAP's were 317.44.43. (514.96 18.28). In comparison to the AMI and UAP groups, the SAP group had considerably higher HDL-C and eGFR readings (P.05). On the other hand, no statistically significant increases (all P>.05) were seen in SBP, DBP, FPG, BUN, or UA (Table 4.1).

In addition to high levels of LDL and uric acid in the blood, the most prevalent risk factors for cardiovascular disease (CAD) are high levels of Cystatin C and homocysteine (HCY). Patients with stable angina pectoris (SAP), unstable angina group (UAP), and acute myocardial infarction (AMI) had significantly different total cholesterol, triglyceride, high-density lipoprotein, low-density lipoprotein, cystatin C, eGFR, and GS values. Patients with AMI had higher levels of Cystatin C, LDL-C, and HCY than those with SAP or UAP. Males and diabetics (HCY) were more common among Q4 patients, as well as those who were more likely to have GS. This study discovered a strong link between the anti-cancer drug Cystatin C and the hormone hormone corticosteroid (GS). Cardiovascular lesions are more likely to occur in patients with high levels of blood Cystatin C, hypertension/diabetes, HCY, age, and gender. A clinical biomarker for cardiovascular disease, cystatin C, seems to be a component in our findings. Cystatin C levels may be utilized to determine the severity of coronary artery lesions because of its significant link to GS.

Initial and long-term effects of isosorbide dinitrate on 12 patients with angina pectoris were studied using a variety of doses of the drug. A wider area of plasma concentration-time curve was seen when isosorbide dinitrate was delivered in dosages of 30, 60, and 120 mg, respectively (p 0.01). This study showed a greater decrease in standing systolic

blood pressure with acute therapy (p 0.001). Systolic blood pressure dropped for eight hours following acute treatment, but only four hours with maintenance medication. This was a dose-related impact Compared to placebo, it required 8 hours of isosorbide dinitrate initial treatment but only 2 hours of sustained therapy before activity duration rose significantly and moderate angina began to develop. During acute administration, Isosorbide dinitrate's impact on exercise tolerance was equivalent to that of a single dose of 60 or 120 mg. Long-term therapy with dosages of 30, 60, or 120 mg four times daily resulted in the same level of improvement in exercise tolerance (15 mg four times daily). After consuming 15 or 30 mg four times a day, most individuals noticed a near-maximum boost in their capacity to exercise. Studies show that isosorbide dinitrate's antianginal and circulatory effects are somewhat resistant to long-term administration.

In obese people, elevated myonectin levels have been linked to a wide range of health issues, including cardiovascular disease, type 2 diabetes, and insulin resistance. It has been established that the amount of myonectin, a myokine synthesized largely by skeletal muscles, is negatively associated with body mass index (BMI) (BMI). For eight weeks, obese and overweight women were monitored for changes in myonectin and insulin resistance as a result of aerobic exercise. Both myonectin and insulin resistance reduced dramatically in the experimental group (P=0.000).

An independent t-test found that whereas myonectin levels increased considerably in the experimental group, insulin resistance was much lower than in the control group (P=0.000) (Table 4.1).

Researchers found that insulin resistance and myonectin levels in obese males decreased dramatically after eight weeks of aerobic exercise. Regular exercise may help regulate or improve insulin resistance risk factors, such as diabetes and cardiovascular disease, which should be encouraged among overweight or obese individuals.

Because of the function that myonectin plays in enhancing fatty acid intake, exercise training may be critical in reducing obesity-associated disorders and the metabolic syndrome, and this impact is connected in part to the action of myonectin. In order to

lower the risk of illnesses linked to obesity and metabolic syndrome, this kind of exercise is highly suggested.

Over the last several years, obesity and overweight have grown dramatically (Arar *et al.* 1998). Diabetes, hypertension, elevated triglycerides in the blood, and cardiovascular disease are all linked to obesity and the central distribution of lipids, which includes abdominal visceral fat. Because of insulin resistance and increased glucose generation by liver cells as a result of visceral fat accumulation around and within abdominal organs and an increase in fatty acid transport, insulin secretion is impeded (Nachtigal et al. 2008). After 8 weeks of aerobic activity, mynoectin levels and insulin sensitivity were substantially different from the control group. In obese people, the quantity of fat in the bloodstream decreases due to the release of myonectin, an entirely novel myokine agent generated by skeletal muscles. A reduction in plasma myonectin levels was linked to obesity, according to (Li *et al.* 2020). These findings back up what I've been saying all along (Assadpour *et al.* 2012)

Compared to the control group, the exercise group had significantly greater levels of myonectin due to a decrease in body mass index (BMI). Due to decreased free fatty acids and higher absorption of acids from tissues, myonectin levels have been demonstrated to decrease with obesity in previous studies (Li *et al.* 2020).

There are a number of functions that myonectin may help with, including glucose absorption and the breakdown of fats (Li *et al.* 2020).

In line with previous research, endurance exercise significantly increased myonectin levels in obese women. In obese mice, Ford9 (Ford *et al.* 2018) discovered that three weeks of voluntary wheel running exercise increased the expression of the myonectin gene (Kaski *et al.* 2018).

An increase in myonectin levels in the body may help those who are obese or overweight to better absorb carbohydrates and fats, which in turn increases insulin

sensitivity. No measurements of myonectin levels were made in the Paterson study, which contradicts our results (Peterson *et al.* 2014) that treadmill running lowered expression without regard to obesity (Peterson *et al.* 2014).

Aerobic exercise had a significant influence on the improvement of insulin-dependent indices in obese women who were previously insulin resistant and had high blood glucose levels. Exercising for only one hour a day for two days may improve insulin sensitivity and insulin function in obese people, according to a new study (Gagno *et al.* 2019).

Overweight and obese women who engaged in aerobic exercise for eight months had a substantial reduction in their insulin resistance in the central body region, according to a previous study (Le *et al.* 2016, Bryne and Co 2004).

Rhythmic aerobic exercise and core stability training did not have a significant effect on insulin levels or insulin resistance in overweight women after 12 weeks (Faramarzi *et al.* 2016). Instead Using (Luke *et al.* 2019).

By reducing the activation of proinflammatory/stress pathways in overweight/diabetic patients and their detrimental effects on signaling and insulin sensitivity, aerobic exercise seems to promote fat distribution and reduce accumulation (Fang *et al.* 2004). The transport of glucose-4 carrier protein to the plasma membrane is reduced in insulin-resistant people's skeletal muscles, which results in decreased insulin-induced glucose absorption. Adenosine monophosphate kinase (AMPK) activation has been related to exercise-induced alterations in carrier proteins (Shumilah *et al.* 2021).

As the number of plasma membrane glucose transporters rises, this might lead to an increase in the membrane's permeability to glucose under muscular tension (Glut-4). In a study of changes in insulin sensitivity after exercise training, higher glut-4 concentrations were shown to enhance insulin's capacity to control glucose metabolism after exercise (Mann *et al.* 2014). These findings might be utilized to manage and

improve insulin indicators, as proven in their research on contemporary ris (Ginghina *et al.* 2011).

With coronary artery bypass surgery, Firouzabadi and colleagues discovered that patients who participated in physical activity were more satisfied with their lives than those who did not (Firouzabadi *et al.* 2014). Being physically active may help prevent weight gain, which is important since obesity and its accompanying physical and mental diseases (Bădescu et al. 2016) are on the rise in modern society (Bădescu *et al.* 2016). (Sekhavati *et al.* 2015).

In this research, we didn't ask participants about their food history. Studying the effects of training on myonectin and insulin resistance levels during the course of the study and their immediate impacts thereafter is necessary. To get a more accurate picture of the outcomes of this research, it is advised to conduct a study on both genders. In addition, it is recommended that a complete research be conducted to examine various and associated physiological aspects in obese individuals. The molecular and histopathological mechanisms of myonectin and insulin resistance should be studied further.

The results showed that there were significant differences for each patient according to the information recorded in the form mentioned in Table 4.2, Figure 4.1 and Figure 4.2, as there was a difference of patients with angina pectoris (158.45) in Cholesteroi and (137.33) inTriglycerid and (41.91) in HDL and (89.07) in LDL and (27.47) in VLDL with angina pectoris, (195.32) for Cholesteroi and (179.11)inTriglycerid and (36.62)in HDLand(122.88) LDL and (35.82) in VLDL, which is a high percentage during the collection of samples from the CCU, as shown in the above table.

Table 4.2, Figure 4.3 and Figure 4.4 Cholesterol concentration in the blood serum of patients and control group, and when comparing the results that appeared for groups, we find that the disease groups recorded a significant increase compared to the control group and at a significant level ($P < 0.01$), but when comparing between the disease groups, we find that the two groups (HDL)) recorded a significant decrease (41.91 ±

0.52) (36.62 ± 0.57) mmol/L and at a significant level ($p<0.01$ It shows when comparing the results that appeared for the studied samples, we find significant differences in (triglyceride, LDL, VLDL), which recorded a significant increase (137.33 ± 2.65), (179.11 ± 4.60), (89.07 ± 3.37), (122.88 ± 3.82) and (27.47). ± 0.53) and (35.82 ± 0.92), respectively, the results of this study are in agreement with the study carried out by (Ismail 2006), which indicated that people with heart diseases are more likely to accumulate fat in their blood vessels leading to atherosclerosis, as a number of researchers indicated There is a strong relationship between hypercholesterolemia and atherosclerosis (Vogel *et al.* 1998), and it appears from Table 4.2 that those with acute coronary syndrome and heart failure had high cholesterol levels compared to the disease groups, and this corresponds to some extent with the results of both (Khalil *et al.* 1994).

Those with angina pectoris (AP) and myocardial infarction (MI) are more likely to have high cholesterol (Crawford *et al.* 2004). The reasons that lead to high cholesterol in the body are many. They may be due to pathological causes or other pathological conditions associated with coronary heart disease, CAD, CHD, such as diabetes. People with diabetes are more likely to have atherosclerosis, which accelerates the advancement of sclerosis (Mittinen *et al.* 2001), or to have a disorder affecting the liver that prevents it from converting cholesterol into LDL or HDL, as is the case with diabetes (Mittinen *et al.* 2001, Ridges *et al.* 2001). Toxic levels of total cholesterol in the blood plasma are caused by poor eating habits and obesity (Crawford *et al.* 2004).

This study's findings are in line with several others (Mienttinen *et al.* 1988). Another major risk is a low amount of HDL cholesterol. A higher amount of lipoprotein in people with coronary heart disease (CHD) than what is considered normal raises the risk of both IHD and coronary heart disease (CHD) (Chatterjea and Shinde 2005).

We also found substantial increases in the patient groups compared to the control group, but no significant differences across the patient groups. We found no significant differences between the sexes when comparing all of the groups. According to what was mentioned, this research is in agreement. (Al-Obaidi, 2005, Al-Bajari 2006).

It is known that the main component of TG is VLDL, so an increase in TG leads to an increase in VLDL (Crawford *et al.* 2004). The relationship between VLDL and atherogenesis is less certain than that of HDL, but the sub-types of VLDL "intermediate density lipoprotein IDL and LDL", the main product of VLDL metabolism, are important in causing atherosclerosis (Tiernery *et al.* 2006).

People who usually have low levels of HDL have high VLDL levels and this increases the risk of coronary heart disease (CHD), as evidenced by our study. The rise in VLDL may be due to a defect in the work of the enzyme Lipoprotein lipase, which works to convert TG into fatty acids capable of penetrating cell membranes, and in the meantime, Intermediate density Lipoprotein (IDL) is formed, which quickly converts to LDL (Guyton and Hall 2006).

Table 4.2 Comparison of the lipid profile between the control and patient groups

Group	Mean ± SE (mg/dL)				
	Cholesterol	Triglyceride	HDL	LDL	VLDL
Control	158.45 ± 3.54	137.33 ± 2.65	41.91 ± 0.52	89.07 ± 3.37	27.47 ± 0.53
Patients	195.32 ± 3.69	179.11 ± 4.60	36.62 ± 0.57	122.88 ± 3.82	35.82 ± 0.92
T-test	10.33 **	11.204 **	1.565 **	10.371 **	2.240 **
P-value	0.0001	0.0001	0.0001	0.0001	0.0001
** ($P \leq 0.01$).					

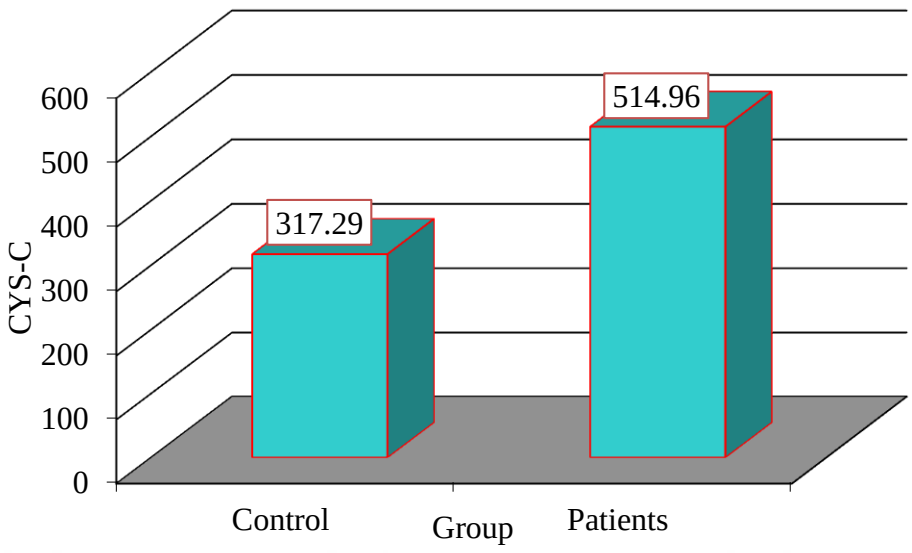

Figure 4.1 Comarisn between control and patients group in CYS-C

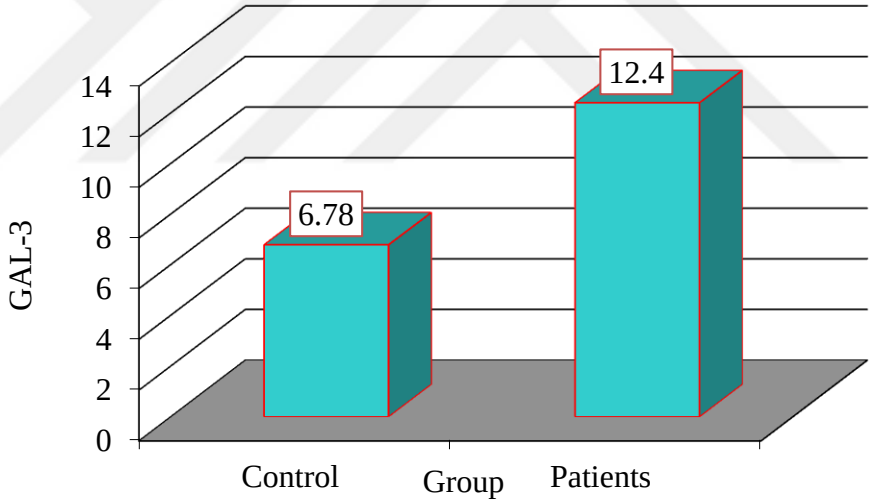

Figure 4.2 Comarisn between control and patients group in GAL-3

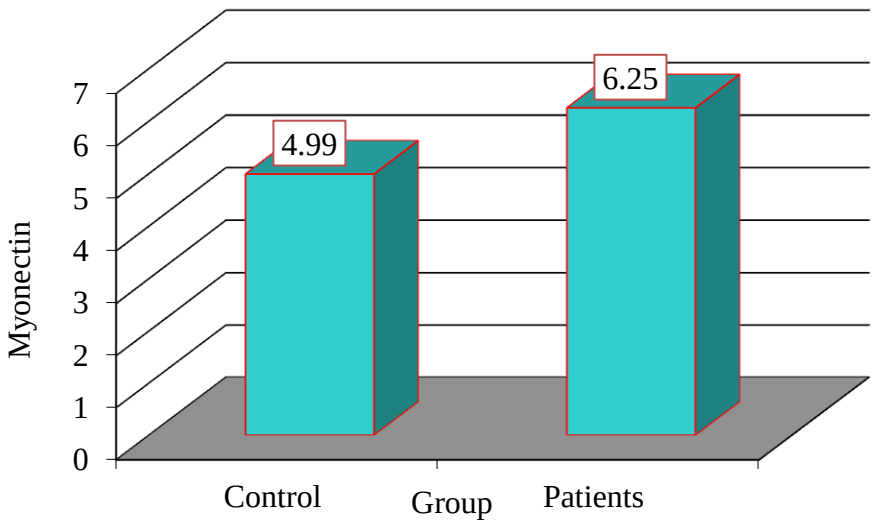

Figure 4.3 Comarisn between control and patients group in myonectin

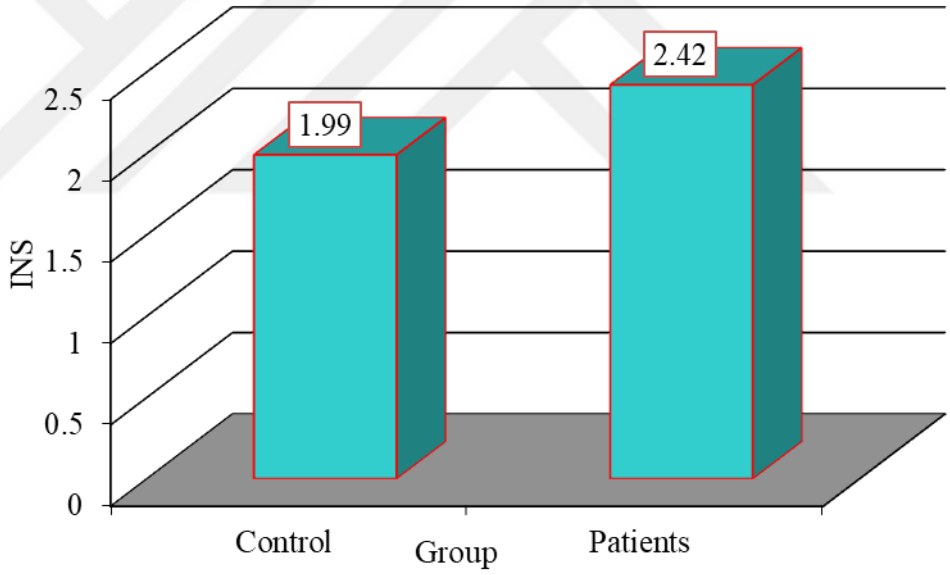

Figure 4.4 Comarisn between control and patients group in INS

The results showed that there were significant differences for each patient according to the information recorded in the form mentioned in Table 4.3, Figure 4.5 and Figure 4.6 as there was a difference of patients with angina pectoris (36.36) in GOT and (32.61) in GPT and (109.32) in ALP with angina pectoris, (48.13) for GOT and (44.33)in GPT (163.99)in ALP, which is a high percentage during the collection of samples from the

CCU, as shown in the above table. Total protein, albumin (Alb),globulin (Glob),and uric acid were assessed in patients with various forms of acute coronary syndrome (ACS) (UA/non-STEMI, AMI, and healthy persons) in this research.

Group AMI had significantly higher GOT concentrations than the control group (UA/NSTEMI) as compared to Table 4.3, Figure 4.47, and Figure 4.48. In comparison to the CTR group, GOT and GOT concentrations increased considerably in all patient groups (p0.01). There is a clear trend across all patient groups (p 0.01), with serum GPT values considerably greater than those in the CTR group (p 0.001). It is clear, however, that there were no differences between the AMI and UA group's blood total protein concentrations when compared to the CTR control group's (p 0.01). Comparing the UA and CTR groups, serum albumin concentrations decreased significantly (p0.01) in the former while remaining almost unchanged in the latter (group AMI) in comparison to both the CTR and UA groups (CTR and UA). Group AMI had considerably greater (p0.01) serum globulin concentrations than group CTR, whereas serum globulin in group UA did not vary significantly from the other groups (CTR and AMI). All patient groups saw a significant increase in blood uric acid concentrations (p 0.01) compared to the CTR control group, however there was no significant difference in serum uric acid concentrations between the AMI and UA groups.

Acute coronary syndromes such as unstable angina (UA) and acute myocardial infarction (AMI) are more dangerous than other kinds of ischemic heart disease because of the fast structural changes that occur in atherosclerotic plaques (IHD). ACS patients and healthy controls were both tested to determine the levels of (GOT, GPT; total protein); albumin; globulin; and uric acid). Materials and Procedures for the Study: The enzymes S.GOT and S.GPT, as well as serum total protein, serum albumin, serum globulin, and serum uric acid, were used to divide 75 healthy volunteers into three groups. In this research, 25 UA and AMI patients and 25 healthy volunteers were compared (control). Results: Our study indicated that individuals with UA and AMI had a significant rise in the levels of uric acid and uric acid in their blood compared to those without these illnesses. When compared to the control group, patients with (UA) had considerably lower serum albumin levels than those with (AMI), however this was not

the case when compared to the control group's levels of serum albumin. AMI patients had higher serum globulin levels than the control group, but these levels did not alter substantially in the blood serum of (UA) patients (p0.01). Low total protein and albumin may contribute to ACS, as can increased levels of GOT, GPT, or globulin.

Table 4.3 Comparison of the Liver enzymes in the control and the sick group

Group	Mean ± SE		
	GOT	GPT	ALP
Control	36.36 ± 0.82	32.61 ± 1.08	109.32 ± 1.69
Patients	48.13 ± 0.82	44.33 ± 0.54	163.99 ± 4.14
T-test	2.323 **	2.281 **	9.616 **
P-value	0.0001	0.0001	0.0001
- ** (P≤0.01).			

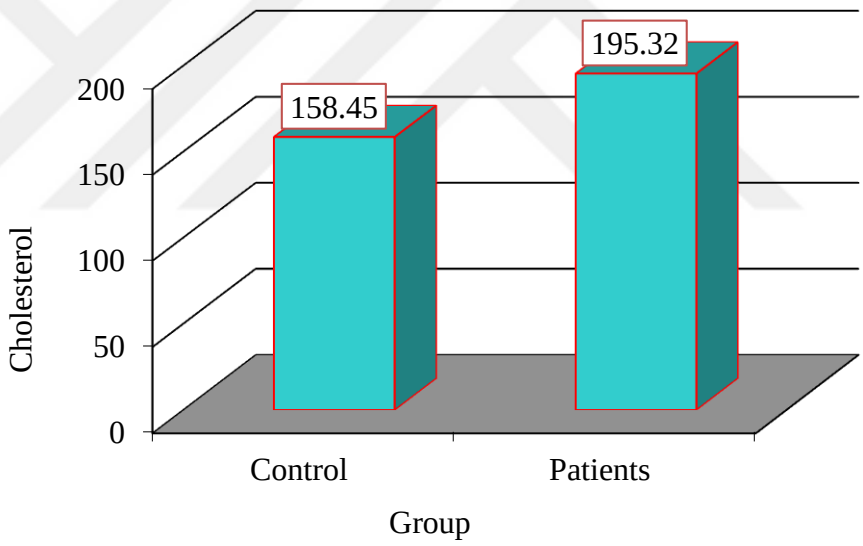

Figure 4.5 Comarisn between control and patients group in Cholesterol

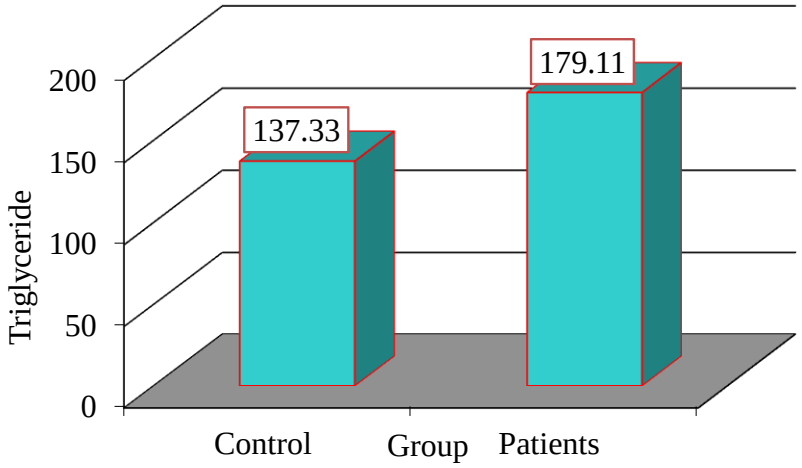

Figure 4.6 Comarisn between control and patients group in Triglicende

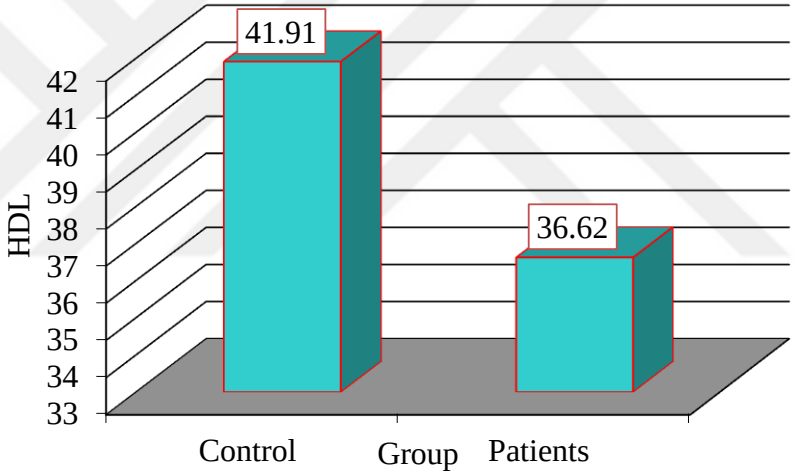

Figure 4.7 Comarisn between control and patients group in HDL

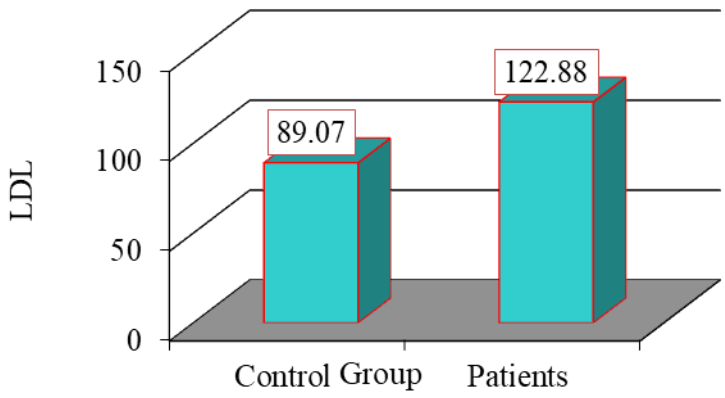

Figure 4.8 Comarisn between control and patients group in LDL

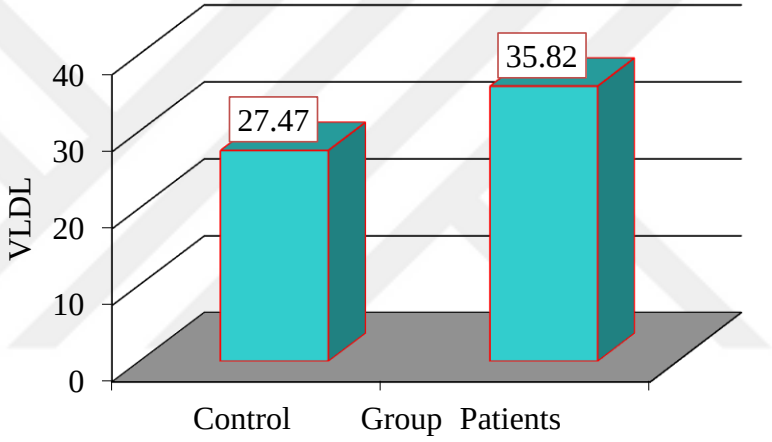

Figure 4.9 Comarisn between control and patients group in VLDL

The results showed that there were significant differences for each patient according to the information recorded in the form mentioned in Table 4.4, Figure 4.10, Figure 4.11 and Figure 4.12 as there was a difference of patients with angina pectoris (53.72) in age and (76.00) For patients with angina pectoris, the healthy weight is (56.20) for age and (83.14) for weight, which is a high percentage during the collection of samples from the CCU, as shown in the above table.

Table 4.4 Age and weight comparisons between the control and sick groups

Group	NO	Mean ± SE	
		Age (year)	Weight (kg)
Control	40	53.72 ±2.73	76.00 ±1.49
Patients	50	56.20 ±1.77	83.14 ±1.18
T-test	---	6.256 NS	3.735 **
P-value	---	0.433	0.0003
** (P≤0.01).			

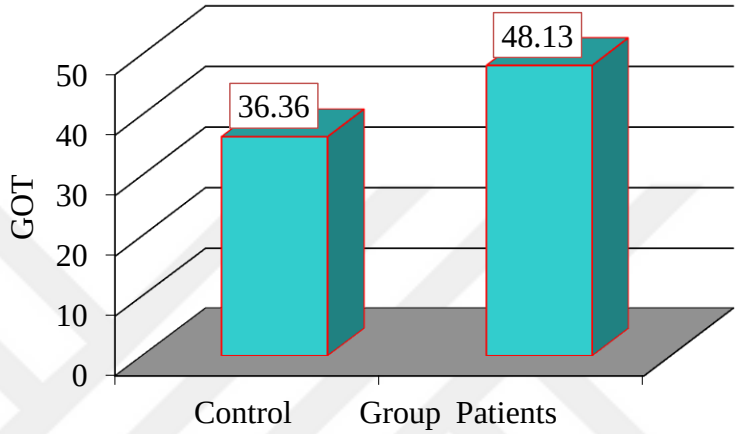

Figure 4.10 Comarisn between control and patients group in GOT

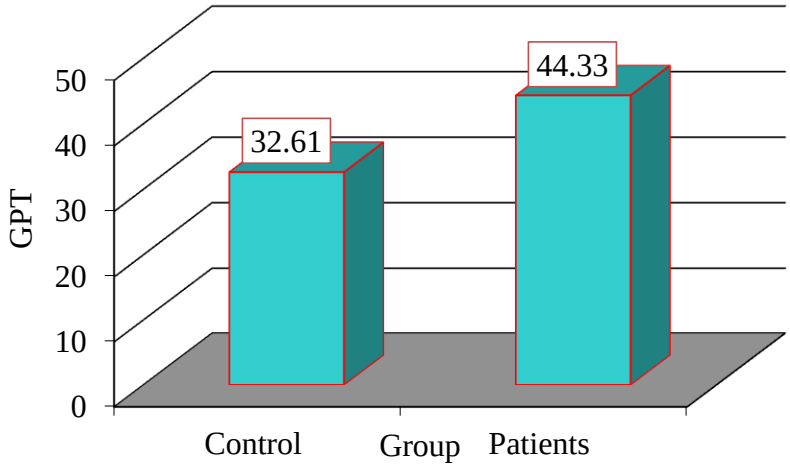

Figure 4.11 Comarisn between control and patients group in GPT

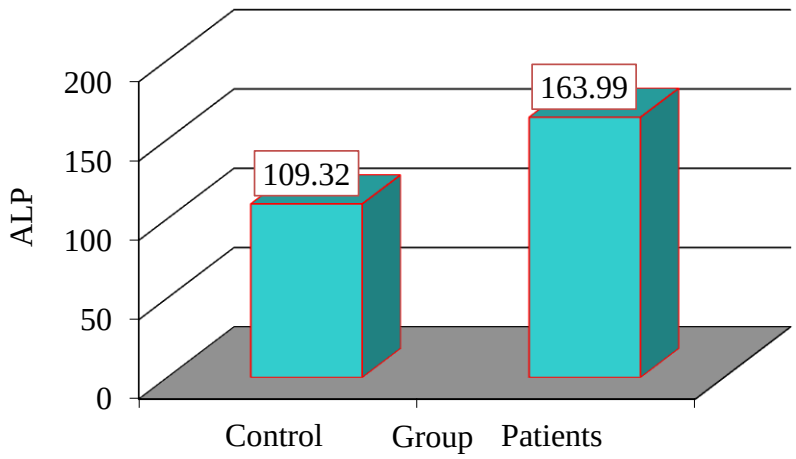

Figure 4.12 Comarisn between control and patients group in ALP

5. CONCLUSIONS AND RECOMMENDATION

Study after study found that Cystatin C levels correlate with cardiovascular events and mortality in people with Acute Myeloid Leukemia (ACS). Higher levels of cystatin C were linked to higher odds ratios or relative risk measures in both the systematic review and the meta-analysis. Only one investigation found statistically significant differences in the percentage of patients with elevated cystatin C levels who had or did not develop outcomes. Our meta-analysis and comprehensive review found a strong link between greater cystatin C levels in ACS patients and an increased risk of cardiovascular events and death. Baseline Predictors of long-term all-cause mortality and MACE in very old patients hospitalized with both AMI and UAP are significant independent predictors of Cys C, and the predictive capacity of Cys C in very old AMI patients rises with increasing concentrations of Cys C. Cys C and Hcy may potentially serve as new indicators for long-term outcomes in individuals with extremely old AMI.

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