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Patient awareness about causes &associated risk factors of coronary artery disease in coronary care unit of Omdurman military hospital from May to July 2017

A thesis submitted in partial full filament for the requirement of degree of MSc in critical care nursing.

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Septemper 2017

بسم الله الرحمن الرحيم

Dedication

To my parent who cared me after birth until child hood and providing more attention and teaching me how to communicate with others........

To my husband mohanad who support me in all important stages in my life ..

To my supervision Dr:mohammed jeber aldar

To all my brothers and sisters...

To any person who help me in this research .

To all my friends....

Abstract

The aim of this research is to assess the knowledge of Patient with coronary artery disease   about causes ,associated risk factors and prevention measure because the number of patient with CAD had increase recently in the Sudan for all aged group ,while coronary artery disease is still the number one killer of people in the united states and has epidemic spread all over the word for both developed and developing country ,there for it is important to evaluate the knowledge of patient with coronary artery disease about causes ,associated risk factors and prevention measure through changes I n their life style and behaviors that may improve and reduce complication .therefore this descriptive cross sectional hospital base study was conducted among convenience sample of 35 patient with CAD (20 male,15 female) and data was collected by asking them using close ended questionnaire in duration of   eight week to assess their knowledge about causes ,associated risk factors and prevention measure in CCU of Omdurman military hospital ,then data were analyzed for result by using SPSS software program .the result revealed that more than half were male that account 57.1% ,and their age above 50 years old, more than half 54.3% their education was secondary level.

The result show that The knowledge about the primary cause of coronary artery disease is poor only 32% of study population were know that atherosclerosis is the primary cause for coronary artery disease .

All participants were assessed their knowledge and awareness about risk factors such as(smoking, blood pressure ,hypertension, diabetes, obesity)and prevention methods such as diet, physical exercise ,control blood pressure and diabetic mellitus and reducing weight. Result was revealed that only less than half (40%) had a good knowledge   about risk factors for CAD.

The result revealed that The common risk factors between the study populations is HTN common for one select, family history and smoking for two selects (options). Hyperlipidemia & Increasing weight & DM for three select.

The knowledge about the prevention of coronary artery disease is good Majority of study population 92.9% was knowledgeable about the prevention methods.

This study concluded that that   study population had a fair knowledge about the awareness of coronary artery disease development, risk factors, complication, prevention methods and they had poor knowledge regarding the primary causes, so the study recommended comprehensive education about causes &risk factors. therefore the study recommended that Establish of health educational program to increase the awareness about coronary artery disease especially in the community of health persons to minimize the risk for developing coronary artery disease education about life style changes Increase control on all modifiable risk factors for those whom at risk for developing coronary artery disease.

الخلاصة

الهدف من هذا البحث هو معرفة مرضي أمراض الشرايين التاجيه بالعوامل المسببه والمؤثره للمرض والاجراءت الوقائيه لان عدد المرضي في إزدياد مستمر كل يوم في السودان في كل الفئات العمريه كما أن أمراض القلب التاجيه تعتبر المسبب الأول للوفيات في الولايات المتحده الأمريكيه ولديها إإنتشار وبائي عالمي في كل انحاء العالم في كلا الدول الناميه والمتقدمه لهذا السبب من الضروري تقييم معرفة المرضي بالعوامل المسببه للمرض والإجراءت الوقائيه عن طريق تغيير نمط الحياة والسلوك الذي يؤدي الي تحسيين ومنع حدوث المضاعفات .لذا اجريت هذه الدراسه علي 35 مريض عينه مناسبه (20 من الذكور,15 من الإناث)وجمعت البيانات بواسطة الإستبيان عن طريق طرح الاسئلة للاجابه عليها في فترة ثمانيه اسابيع داخل وحدة العنايه التاجيه في مستشفي السلاح الطبي ام درمان ثم حللت البيانات للحصول علي النتائج عن طريق برنامج التحليل الإحصائي للعلوم الاجتماعيه زبينت النتائج ان معظم المرضي الذين شاركو في الدراسه كانو من الذكور %57.1 واعمارهم فوق الخمسين ومعظمهم درجة تعليمهم متوسطه .

الدراسه بينت أن مدي معرفتهم بالسبب الاساسي لمرض الشرايين التاجيه ضعيفه فقط %32 من جملة المشاركيين مدركيين أن تصلب الشرايين هي السبب الرئيسي لامراض القلب التاجيه.تم تقييم كل المشاركيين في عينة الدراسه لتقييم معرفتهم ودرجة وعيهم بالعوامل المسببه للمرض(كالتدخيين .ارتفاع مستوي الكلسترول في الدم ,ارتفاع ضغط الدم,السكري.السمنه)والاجراءت الوقائيه كالتحكم في الطعام وممارسة الرياضه والتحكم في سكر الدم وارتفاغ ضغط الدم والسمنه.اوضحت النتايج أن %40 فقط كان معرفتهم جيده بالعوامل المؤثره %460 معرفتهم متوسطه.

اوضحت الدراسه أن العامل المؤثر المشترك بين معظم المرضي هو إرتفاع ضغط الدم.وان مدي معرفتهم بالاجراءت الوقائيه كانت جيده %92 كانو مدركيين بها.

List of abbreviation

ACE-I	Angiotensin-Converting Enzyme Inhibitors
ACS	acute coronary syndrome
CAD	coronary artery disease
CCU	coronary care unit
CABG	coronary artery bypass graft
CPB	Cardiopulmonary Bypass
CHD	coronary heart disease
CK	creatine kinase
CRP	C-reactive protein
ECG	electrocardiography
HDL	high density lipoproteins
IHD	ischemic heart disease
LDL	low density lipoprotein
MI	myocardial infarction
NSAID	non-steroidal anti-inflammatory drugs
PCI	percutaneous coronary intervention
PTCA	Percutaneous Transluminal Coronary Angioplasty
TMR	transmyocardial laser revascularization
TPA	Tissue plasminogen activator
TLC	therapeutic lifestyle changes

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CHAPTER ONE

1.1 Introduction

Coronary artery disease (CAD) is a common term for the buildup of plague in the heart's arteries that could lead to heart attack, with coronary artery disease, plague first grows in the coronary arteries until blood flow to the heart's muscle is limited this lead to ischemia it may be chronic, caused by narrowing of the coronary artery and limitation of the blood supply to part of the muscle. Or it can be acute, resulting from a sudden rupture of the plague and formation of a thrombus or blood clot [1].

Coronary artery disease (CAD), also known as ischemic heart disease (IHD),is a group of diseases that includes: stable angina, unstable angina, myocardial infarction, and sudden cardiac death. It is within the group of cardiovascular diseases of which it is the most common type.

A common symptom is chest pain or discomfort which may travel into the shoulder, arm, back, neck, or jaw. Occasionally it may feel like heartburn. Usually symptoms occur with exercise or emotional stress, last less than a few minutes, and get better with rest. Shortness of breath may also occur and sometimes no symptoms are present. The first sign is occasionally a heart attack. Other complications include heart failure or an irregular heartbeat.

Risk factors include: high blood pressure, smoking, diabetes, lack of exercise, obesity, high blood cholesterol, poor diet, and excessive alcohol, among others .Other risks include depression. The underlying mechanism involves atherosclerosis of the arteries of the heart. A number of tests may help with diagnoses including: electrocardiogram, cardiac stress testing,

coronary computed tomographic angiography, and coronary angiogram, among others.

Prevention is by eating a healthy diet, regular exercise, maintaining a healthy weight and not smoking. Sometimes medication for diabetes, high cholesterol, or high blood pressure are also used' There is limited evidence for screening people who are at low risk and do not have symptoms.

Treatment involves the same measures as prevention. Additional medications such as antiplatelets including aspirin, beta blockers, or nitroglycerin may be recommended Procedures such as percutaneous coronary intervention (PCI) or coronary artery bypass surgery (CABG) may be used in severe disease. In those with stable CAD it is unclear if PCI or CABG in addition to the other treatments improves life expectancy or decreases heart attack risk [2] .

1.2 Justification

heart disease &stroke statistics update of the American heart association (AHA) has recently reported 15.5 million person >20 years of age in the USA have CHD (in 2016) whilst the reported prevalence increase with age for both women &men &it has been estimated that approximately every 42 seconds, an American will suffer for an MI [3].

Knowledge about modification of risk factors of coronary artery disease is essential part in treatment. However there is still high mortality rate world widely and in our community among low and high socioeconomic status people. So this study conduct to find out how the knowledge of patient with CAD about risk factors and preventive measure (modification ways) that may improve outcome and reduce the mortality rate.

1.3 Objective

1.3.1 General objective

Patient awareness about causes and associated risk factors of coronary artery disease in coronary care unit of Omdurman military hospital.

1.3.2 Specific objective

-To assess patient level of knowledge about causes, and risk factors of coronary artery disease.

-To identify common risk factors of coronary artery disease.

-To determine patient level of knowledge about preventive measure of coronary artery disease.

CHAPTER TWO

2. Literature review

Coronary artery disease (CAD), also known as ischemic heart disease (IHD) is a group of diseases that includes: stable angina, unstable angina, myocardial infarction, and sudden cardiac death. It is within the group of cardiovascular diseases of which it is the most common type [2]. Coronary artery disease (CAD) is a common term for the buildup of plague in the heart's arteries that could lead to heart attack. With coronary artery disease, plague first grows in the coronary arteries until blood flow to the heart's muscle is limited this lead to ischemia it may be chronic, caused by narrowing of the coronary artery and limitation of the blood supply to part of the muscle. Or it can be acute, resulting from a sudden rupture of the plague and formation of a thrombus or blood clot [1].

Coronary artery disease is a term applied to obstructed blood flow through the coronary arteries to the heart muscle. The primary cause of coronary artery disease is atherosclerosis [4].

2.1 Coronary atherosclerosis

The most common heart disease in the United States is atherosclerosis which is an abnormal accumulation of lipid, or fatty substances and fibrous tissue in the vessel wall? These substances create blockages or narrow the vessel in a way that reduces blood flow to the myocardium.

2.2 Pathophysiology of Atherosclerosis

Atherosclerosis begins as fatty streaks, lipids that are deposited in the intima of the arterial wall. Although they are thought to be the precursors of atherosclerosis, fatty streaks are common, even in childhood. Moreover, not all develop into more advanced lesions. The reason why some fatty streaks

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continue to develop is unknown, although genetic and environmental factors are involved. The continued development of atherosclerosis involves

an inflammatory response. T lymphocytes and monocytes (that become macrophages) infiltrate the area to ingest the lipids and then die; this causes smooth muscle cells within the vessel to proliferate and form a fibrous cap over the dead fatty core. These deposits, called atheromas or plaques, protrude into the lumen of the vessel, narrowing it and obstructing blood flow. If the fibrous cap of the plaque is thick and the lipid pool remains relatively stable, it can resist the stress from blood flow and vessel

Movement. If the cap is thin, the lipid core may grow, causing it to rupture and hemorrhage into the plaque, allowing a thrombus to develop. The thrombus may obstruct blood flow, leading to sudden cardiac death or an acute myocardial infarction (MI), which is the death of heart tissue.

2.3 Development of atherosclerosis

Coronary artery disease is thought to begin with damage or injury to the inner layer of a coronary artery, sometimes as early as childhood. The damage may be caused by various factors, including:

- Smoking
- High blood pressure
- High cholesterol
- Diabetes or insulin resistance
- Sedentary lifestyle

Once the inner wall of an artery is damaged, fatty deposits (plaque) made of cholesterol and other cellular waste products tend to accumulate at the site of

injury in a process called atherosclerosis. If the surface of the plaque breaks or ruptures, blood cells called platelets will clump at the site to try to repair the artery. This clump can block the artery, leading to a heart attack.

2.4 Cardiac Ischemia

Cardiac ischemia develops when plaque and fatty matter narrow the inside of an artery so much, it cannot supply enough oxygen-rich blood to the heart. This can cause heart attacks -- with or without chest pain and other symptoms.

Ischemia happens most during:

- Exercise or other exertion
- Eating
- Excitement or stress
- Exposure to cold

2.5 Types of coronary artery disease

2.5.1 ANGINA PECTORIS

Angina pectoris is a clinical syndrome usually characterized by episodes or paroxysms of pain or pressure in the anterior chest. The cause is usually insufficient coronary blood flow. The insufficient flow results in a decreased oxygen supply to meet an increased myocardial demand for oxygen in response to physical exertion or emotional stress. In other words, the need for oxygen exceeds the supply. The severity of angina is based on the precipitating activity and its effect on the activities of daily living.

2.5.1.1 Types of Angina

•Stable angina:

predictable and consistent pain that occurs on exertion and is relieved by rest

•Unstable angina

(also called preinfarction angina or crescendo angina): symptoms occur more frequently and last longer than stable angina. The threshold for pain is lower, and pain may occur at rest.

•Intractable or refractory angina:

severe incapacitating chest pain

•Variant angina

(also called Prinzmetal's angina): pain at rest with reversible ST-segment elevation; thought to be caused by coronary artery vasospasm

•Silent ischemia:

objective evidence of ischemia (such as electrocardiographic changes with a stress test), but patient reports no symptoms.

2.5.2 myocardial infraction

MI refers to the process by which areas of myocardial cells in the heart are permanently destroyed. Like unstable angina, MI is usually caused

by reduced blood flow in a coronary artery due to atherosclerosis and occlusion of an artery by an embolus or thrombus. Because unstable angina and acute   MI are considered to be the same process but different points along a continuum, the term acute coronary syndrome (ACS) may be used for these diagnoses. Other causes of an MI include vasospasm (sudden constriction or narrowing) of a coronary artery; decreased oxygen supply (eg, from acute blood loss, anemia, or low blood pressure); and increased demand for oxygen (eg, from a rapid heart rate, thyrotoxicosis, or ingestion of cocaine). In each case, a profound imbalance exists between myocardial oxygen supply and demand[5] .

2.6 Risk Factors for Coronary Artery Disease

2.6.1 Conventional risk factors

- Older age: getting older increases your risk of damaged and narrowed arteries, over age 45 years in men and over age 55 years in women

- Sex. Men are generally at greater risk of coronary artery disease. However, the risk for women increases after menopause.

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- Family history :A family history of heart disease is associated with a higher risk of coronary artery disease, especially if a close relative developed heart disease at an early age. Your risk is highest if your father or a brother was diagnosed with heart disease before age 55 or if your mother or a sister developed it before age 65.

- Race: Among persons with CAD, the cardiovascular death rate for African Americans is reported to be particularly high; in Asians, low levels of high-density lipoprotein cholesterol (HDL-C), which are considered to be a risk factor for coronary heart disease, appear to be especially prevalent; South Asians appear to have a higher independent risk for cardiovascular disease as well.

2.6.2 Modifiable risk factors

- High blood cholesterol levels (specifically, low-density lipoprotein cholesterol [LDL-C]) High levels of cholesterol in the blood can increase the risk of formation of plaques and atherosclerosis. High cholesterol can be caused by a high level of low-density lipoprotein (LDL), known as the "bad" cholesterol. A low level of high-density lipoprotein (HDL), known as the "good" cholesterol, can be a sign of atherosclerosis.

- High blood pressure uncontrolled high blood pressure can result in hardening and thickening of the arteries, narrowing the channel through which blood can flow.
- Cigarette smoking: people who smoke have a significantly increased risk of heart disease. exposing others to your secondhand smoke also increases their risk of coronary artery disease. Cessation of cigarette smoking constitutes the single most important preventive measure for CAD

- Diabetes mellitus ;diabetes is associated with an increased risk of coronary artery disease; type 2 diabetes and coronary artery disease share similar risk factors, such as obesity and high blood pressure.

- Obesity ;weight typically worsens other risk factors.

- Lack of physical activity; lack of exercise also is associated with coronary artery disease and some of its risk factors, as well.

- Metabolic syndrome

- Mental stress and depression; high stress. Unrelieved stress in your life may damage your arteries as well as worsen other risk factors for coronary artery disease.

2.6.3 Nontraditional or novel risk factors

High levels of the following are considered to be risk factors for CAD:

- C-reactive protein (CRP): High sensitivity C-reactive protein (hs-CRP) is a normal protein that appears in higher amounts related to the presence of inflammation and, according to some research results, may be associated with an increased risk of CAD development and heart attack.

- Lipoprotein(a)

- Homocysteine: **;** homocysteine is an amino acid in the body uses to make protein and to build and maintain tissue. But high levels of homocysteine may increase your risk of coronary artery disease; In the general population, mild to moderate elevations are due to

insufficient dietary intake of folic acid, but homocysteine levels may also identify people at increased risk for heart disease

- Fibrinogen

2.6.4 Various medical conditions that can contribute to CAD include the following:

- End-stage renal disease (ESRD)
- Chronic inflammatory diseases affecting connective tissues (eg, lupus, rheumatoid arthritis)
- Human immunodeficiency virus (HIV) infection (acquired immunodeficiency syndrome [AIDS], highly active antiretroviral therapy [HAART])
- Xanthelasmata (raised yellow patches around the eyelids.

- Sleep apnea. This disorder causes you to repeatedly stop and start breathing while you're sleeping. Sudden drops in blood oxygen levels that occur during sleep apnea increase blood pressure and strain the cardiovascular system, possibly leading to coronary artery disease.

2.6.5 The following are also considered to be risk factors:

- Tissue plasminogen activator (tPA): An imbalance of the clot dissolving enzymes (eg, tPA) and their respective inhibitors (plasminogen activator inhibitor-1 [PAI-1]) may predispose individuals to myocardial infarctions
- Low serum testosterone levels: Have a significant negative impact on patients with CAD

- Hysterectomy: A study suggests that this becomes a risk factor later in life in women who have the surgery at or before age 50 years [6].

2.7 Clinical manifestation of coronary artery disease

- **Chest pain (angina),** feel pressure or tightness in the chest, as if someone were standing on your chest. This pain, referred to as angina, usually occurs on the middle or left side of the chest. Angina is generally triggered by physical or emotional stress.

  The pain usually goes away within minutes after stopping the stressful activity. In some people, especially women, this pain may be fleeting or sharp and felt in the neck, arm or back.

- **Shortness of breath.** When heart can't pump enough blood to meet your body's needs, develop shortness of breath or extreme fatigue with exertion.
- **Heart attack.** A completely blocked coronary artery may cause a heart attack. The classic signs and symptoms of a heart attack include crushing pressure in the chest and pain in shoulder or arm, sometimes with shortness of breath and sweating.

  Women are somewhat more likely than men are to experience less typical signs and symptoms of a heart attack, such as neck or jaw pain. Sometimes a heart attack occurs without any apparent signs or symptoms[7].

2.8 Diagnostic tests of coronary artery disease

2.8.1 PATIENT HISTORY

The patient history has two parts: the description of the presenting symptom (e.g., pain) and the history of previous illnesses and family health history, particularly of heart disease. Previous history should also include information about the patient's risk factors for heart disease.

2.8.2 ELECTROCARDIOGRAM

The ECG provides information that assists in diagnosing acute MI.
It should be obtained within 10 minutes from the time a patient
reports pain or arrives in the emergency department. By monitoring the ECG over time, the location, evolution, and resolution of an MI can be identified and monitored.

2.8.3 ECHOCARDIOGRAM

The echocardiogram is used to evaluate ventricular function. It
may be used to assist in diagnosing an MI, especially when the ECG is nondiagnostic. The echocardiogram can detect hypokinetic and akinetic wall motion and can determine the ejection fraction .

2.8.4 LABORATORY TESTS

Historically, laboratory tests used to diagnose coronary artery disease included:

2.8.4.1 creatine kinase (CK)

with evaluation of isoenzymes and lactic dehydrogenase (LDH) levels. Newer laboratory tests with faster results, resulting in earlier diagnosis, include myoglobin and troponin analysis. These tests are based on the release of cellular contents into the circulation when myocardial cells die. LDH test is now infrequently ordered because it is not useful in identifying cardiac events .

2.8.4.2 Creatine Kinase and Its Isoenzymes.

There are three CK

isoenzymes: CK-MM (skeletal muscle), CK-MB (heart muscle),

and CK-BB (brain tissue). CK-MB is the cardiac-specific isoenzyme; CK - MB is found mainly in cardiac cells and therefore rises only when there has been damage to these cells. CK-MB assessed by mass assay is the most specific index for the diagnosis of acute MI.,The level starts to increase within a few hours and peaks within 24 hours of an MI.

If the area is reperfused (eg, due to thrombolytic therapy or PTCA), it peaks earlier.

2.8.4.3 Myoglobin.

Myoglobin is a heme protein that helps to transport oxygen. Like CK-MB enzyme, myoglobin is found in cardiac and skeletal muscle. The myoglobin level starts to increase within 1 to 3 hours and peaks within 12 hours after the onset of symptoms.

2.8.4.4 Troponin

  a protein found in the myocardium, regulates the myocardial contractile process. There are three isomers of troponin (C, I, and T). Because of the smaller size of this protein and the increased specificity of the troponins I and T for cardiac muscle, these tests are used more frequently to identify myocardial injury (unstable angina or acute MI). The increase in the level of troponin in the serum starts and peaks at approximately the same time as CK-MB. However, it remains elevated for a longer period, often up to 3 weeks, and it therefore cannot be used to identify subsequent extension or expansion of an MI.

2.9 Management of coronary artery disease

2.9.1 Medical Management

  The goal of medical management is to minimize myocardial damage, preserve myocardial function, and prevent complications.
These goals are achieved by reperfusing the area with the emergency use of thrombolytic medications or PTCA. Minimizing myocardial damage is also accomplished by reducing myocardial oxygen demand and increasing oxygen supply with medications, oxygen administration, and bed rest. The resolution of pain and ECG changes are the primary clinical indicators that demand and supply are in equilibrium; they may also indicate reperfusion. Visualization of blood flow through an open vessel in the catheterization laboratory is evidence of reperfusion.

2.9.1.1 Oxygen Administration

Oxygen therapy is usually initiated at the onset of chest pain in an attempt to increase the amount of oxygen delivered to the myocardium and to decrease pain. Oxygen inhaled directly increases the amount of oxygen in the blood. The therapeutic effectiveness of oxygen is determined by observing the rate and rhythm of respirations. Blood oxygen saturation is monitored by pulse oximetry; the normal oxygen saturation (SpO2) level is greater than 93%. Studies are being conducted to assess the use of oxygen in patients without respiratory distress and its effect on outcome.

2.9.1.2 PHARMACOLOGIC THERAPY

The patient with an acute MI receives the same medications as the patient with unstable angina, with the possible additions of thrombolytics, analgesics, and angiotensin converting enzyme (ACE) inhibitors. Patients should receive a beta-blocker initially, throughout the hospitalization, and a prescription to continue its use after hospital discharge.

2.9.1.2.1 Nitroglycerin

Nitrates remain the mainstay for treatment of angina pectoris. Avasoactive agent, nitroglycerin (Nitrostat,Nitrol, Nitrobid IV) is administered to reduce myocardial oxygen consumption, which decreases ischemia and relieves pain. Nitroglycerin dilates primarily the veins and, in higher doses, also dilates the arteries. It helps to increase coronary blood flow bypreventing vasospasm and increasing perfusion through the co-llateral vessels.

Dilation of the veins causes venous pooling of blood through out the body. As a result, less blood returns to the heart, and filling pressure (preload) is reduced. If the patient is hypovolemic (does not have adequate circulating blood volume), the decrease in filling pressure can cause a significant decrease in cardiac out put and blood pressure.

Nitrates in higher doses also relax the systemic arteriolar bed and lower blood pressure (decreased afterload). Nitrates may increase blood flow to diseased coronary arteries and through co-llateral coronary arteries, arteries that have been underused until the body recognizes poorly perfused areas. These effects decrease myocardial oxygen requirements and increase oxygen supply, bringing about a more favorable balance between supply and demand.

Nitroglycerin may be given by several routes: sublingual tablet or spray, topical agent, and intravenous administration. Sublingual nitroglycerin is generally placed under the tongue or in the cheek (buccal pouch) and alleviates the pain of ischemia within 3 minutes. Topical nitroglycerin is also fast acting and is a convenient way to administer the medication. Both routes are suitable for patients who self administer the medication.

A continuous or intermittent intravenous infusion of nitroglycerin may be administered to the hospitalized patient with recurring signs and symptoms of ischemia or after a revascularization procedure. The amount of nitroglycerin administered is based on the patient's symptoms while avoiding side effects such as hypotension. It usually is not given if the systolic blood pressure is 90 mm Hg or less. Generally, after the patient is symptom free, thenitroglycerin may be switched to a topical preparation within 24 hours.

2.9.1.2.2 Beta-Adrenergic Blocking Agents.

Beta-blockers such as propranolol (Inderal), metoprolol (Lopressor, Toprol), and atenolol (Tenormin) appear to reduce myocardial oxygen consumption by blocking the beta-adrenergic sympathetic stimulation to the heart. The result is a reduction in heart rate, slowed conduction

of an impulse through the heart, decreased blood pressure, and

reduced myocardial contractility (force of contraction) that establishes a more favorable balance between myocardial oxygen needs (demands) and the amount of oxygen available (supply). This helps to control chest pain and delays the onset of ischemia during work or exercise. Beta-blockers reduce the incidence of recurrent angina, infarction, and cardiac mortality. The dose can be titrated to achieve a resting heart rate of 50 to 60 beats per minute.

Cardiac side effects and possible contraindications include hypotension, bradycardia, advanced atrioventricular block, and decompensated heart failure. If a beta-blocker is given intravenously for an acute cardiac event, the ECG, blood pressure, and heart rate are monitored closely after the medication has been administered.

Because some beta-blockers also affect the beta-adrenergic receptors in the bronchioles, causing bronchoconstriction, they are contraindicated in patients with significant pulmonary contrictive diseases, suchasasthma. Other side effects include worsening of hyperlipidemia, depression, fatigue, decreased libido, and masking of symptoms of hypoglycemia. Patients taking beta blockers are cautioned not to stop taking them abruptly, because angina may worsen and MI may develop. Beta-blocker therapy needs to be decreased gradually over several days before discontinuing it.

Patients with diabetes who take beta-blockers are instructed to assess their blood glucose levels more often and to observe for signs and symptoms of hypoglycemia.

2.9.1.2.3 Antiplatelet and Anticoagulant Medications

Antiplatelet medications are administered to prevent platelet aggregation, which impedes blood flow.

2.9.1.2.3.1 Aspirin.

Aspirin prevents platelet activation and reduces the incidence of MI and death in patients with CAD. A 160- to 325-mg dose of aspirin should be given to the patient with angina as soon as the diagnosis is made (e.g., in the emergency room or physician's office) and then continued with 81 to 325 mg daily. Although it may be one of the most important medications in the treatment of CAD, aspirin may be overlooked because of its low cost and common use. Patients should be advised to   continue aspirin even if concurrently taking nonsteroidal antiinflammatory drugs (NSAIDs) or other analgesics. Because aspirin may cause gastrointestinal upset and bleeding, treatment of

Helicobacter pylori and the use of H2-blockers (eg, cimetidine [Tagamet], famotidine [Mylanta AR, Pepcid], ranitidine [Zantac]) or misoprostol (Cytotec) should be considered to allow continued aspirin therapy.

2.9.1.2.3.2 Clopidogrel and Ticlopidine

Clopidogrel (Plavix) or ticlopidine (Ticlid) is given to patients who are allergic to aspirin or given in addition to aspirin in patients at high risk for

MI. Unlike aspirin,these medications take a few days to achieve their antiplatelet effect. They also cause gastrointestinal upset, including nausea,vomiting, and diarrhea, and they decrease the neutrophil level.

2.9.1.2.3.3 Heparin.

Unfractionated heparin prevents the formation of new blood clots. Use of heparin alone in treating patients with unstable angina reduces the occurrence of MI. If the patient's signs and symptoms indicate a significant risk for a cardiac event, the patient is hospitalized and may be given an intravenous bolus of heparin and started on a continuous infusion or given an intravenous bolus every 4 to 6 hours. The amount of heparin administered is based on the results of the activated partial thromboplastin time (aPTT).

Heparin therapy is usually considered therapeutic when the aPTT

is 1.5 to 2 times the normal aPTT valuea   subcutaneous injection of lowmolecular-weight heparin (LMWH; enoxaparin [Lovenox] or dalteparin [Fragmin]) may be used instead of intravenous unfractionated heparin to treat patients with unstable angina or non ST-segment elevation MIs.

LMWH provides more effective and stable anticoagulation, potentially reducing the risk of rebound ischemic events, and it eliminates the need to monitor aPTT results LMWH may be beneficial before and during PCIs and for ST-segment elevation MIs .

2.9.1.2.3.4 Thrombolytics

Thrombolytics are medications that are usually administered intravenously, although some may also be given directly into the coronary artery in the cardiac catheterization laboratory.

The purpose of thrombolytics is to dissolve and lyse the thrombus in a coronary artery (thrombolysis), allowing blood to flow through the coronary artery again (reperfusion), minimizing the size of the infarction, and preserving ventricular function.

Even though thrombolytics may dissolve the thrombus, they do not affect the underlying atherosclerotic lesion. The patient may be referred for a cardiac catheterization and other invasive intervention. hrombolytics dissolve all clots, not just the one in the coronary artery. They should not be used if the patient has formed aprotective clot, such as after major surgery or hemorrhagic stroke.

Because thrombolytics reduce the patient's ability to form a stabilizing clot, the patient is at risk for bleeding. Thrombolytics should not be used if the patient is bleeding or has a bleeding disorder. All patients who receive thrombolytic therapy are placed on bleeding precautions to minimize the risk for bleeding. This means minimizing the number of punctures for inserting intravenous lines, avoiding intramuscular injections, preventing tissue trauma, and applying pressure for longer than usual after any puncture.

To be effective, thrombolytics must be administered as early as possible after the onset of symptoms that indicate an acute MI.

They are not given to patients with unstable angina. Hospitals monitor their
ability to administer these medications within 30 minutes
from the time the patient arrives in the emergency department. This
is called door-to-needle time.

The thrombolytic agents used most often are :
streptokinase(Kabikinase, Streptase), alteplase (Activase), and
reteplase (r-PA, TNKase). Anistreplase (Eminase) is another thrombolytic
agent that may be used.

Streptokinase increases the amount of plasminogen activator,
which then increases the amount of circulating and clot bound
plasmin. Because streptokinase is made from a bacterium, its use
also entails a risk of an allergic reaction. vasculitis has occurred
up to 9 days after administration. streptokinase is not used if the
patient has been exposed to a recent streptococcus infection or has
received streptokinase in the past 6 to 12 months.

Alteplase is a type of tissue plasminogen activator (t-PA). In
contrast to streptokinase, alteplase activates the plasminogen on
the clot more than the circulating plasminogen. Because it does
not decrease the clotting factors as much as streptokinase, un-
fractionated or low molecular weight heparin is used with t-PA to
prevent another clot from forming at the same lesion site. Because
t-PA is a naturally occurring enzyme, allergic reactions are minimized, but t-
PA costs considerably more than streptokinase.

Reteplase is structurally very similar to alteplase and has similar

effects. Anistreplase is similar to streptokinase and has similar effects.

2.9.1.2.4 Analgesics.

The analgesic of choice for acute MI is morphine sulfate (Duramorph, Astramorph) administered in intravenous boluses. Morphine reduces pain and anxiety. It reduces preload, which decreases the workload of the heart. Morphine also relaxes bronchioles to enhance oxygenation. The cardiovascular response to morphine is monitored carefully, particularly the blood pressure, which can be lowered, and the respiratory rate, which can be depressed. Because morphine decreases sensation of pain, STsegment monitoring may be a better indicator of subsequent ischemia than assessment of pain.

2.9.1.2.5 Angiotensin-Converting Enzyme Inhibitors (ACE-I).

Angiotensin I is formed when the kidneys release renin in response to decreased blood flow. Angiotensin I is converted to angiotensin II by ACE, a substance found in the lumen of all blood vessels, especially the pulmonary vasculature. Angiotensin II causes the blood vessels to constrict and the kidneys to retain sodium and fluid while excreting potassium. These actions increase circulating fluid and raise the pressure against which the heart must pump, resulting in significantly increased cardiac workload.

ACE inhibitors(ACE-I)prevent the conversion of angiotensin from I to II. In the absence of angiotensin II, the blood pressure decreases and the kidneys excrete sodium and fluid (diuresis), decreasing the oxygen demand of the heart. Use of ACE inhibitors in patients after MI decreases the mortality rate

and prevents the onset of heart failure. It is important to ensure that the patient is not hypotensive, hyponatremic, hypovolemic, or hyperkalemic before ACE-I administration. Blood pressure, urine output, and serum sodium,potassium, and creatinine levels need to be monitored closely.

2.9.2 Surgical management

2.9.2.1 EMERGENT PERCUTANEOUS CORONARY INTERVENTION (PCI)

The patient in whom an acute MI is suspected may be referred for ani immediate PCI. PCI may be used to open the occluded coronary artery in an acute MI and promote reperfusion to the area that has been deprived of oxygen. PCI treats the underlying atherosclerotic lesion. Because the duration of oxygen deprivation is directly related to the number of cells that die, the time from the patient's arrival in the emergency department to the time PCI is performed should be less than 60 minutes (time is muscle). This is frequently referred to as door-to-balloon time .

To perform an emergent PCI within this short time, a cardiac catheterization laboratory and staff must be available.

2.9.2.2 Percutaneous Transluminal Coronary Angioplasty (PTCA)

PTCA may be used to treat patients who do not experience angina but are at high risk for a cardiac event as identified by non invasive testing, with recurrent chest pain that is unresponsive to medical therapy, with a

significant amount of myocardium at risk but are poor surgical candidates, or with an acute MI (as an alternate to thrombolysis and after thrombolysis) . The procedure is attempted when the cardiologist believes that PTCA can improve blood flow to the myocardium. PTCA alone is seldom attempted in the patient with occlusions of the left main coronary artery that do not demonstrate collateral circulation to the left anterior descending and circumflex arteries.

The purpose of PTCA is to improve blood flow within a coronary artery by "cracking" the atheroma.

This invasive interventional procedure is carried out in the cardiac catheterization laboratory. The coronary arteries are examined by angiography, as they are during the diagnostic cardiac catheterization, and the location, extent, and calcification of the atheroma are verified. Hollow catheters, called sheaths, are inserted, usually in the femoral vein or artery (or both), providing a conduit for other catheters. After the presence of atheroma is verified, a balloon-tipped dilation catheter is passed through the sheath along a guide catheter and positioned over the lesion. The physician determines the catheter position by examining markers on the balloon that can be seen with fluoroscopy. When the

Catheter is properly positioned, the balloon is inflated with a radiopaque contrast agent (commonly called dye) to visualize the blood vessel and to provide a steady or oscillating pressure within the balloon. The balloon is inflated to a certain pressure for several seconds and then deflated. The pressure "cracks" and possibly compresses the atheroma . The coronary artery's media and adventitia are also stretched.Several inflations and several balloon sizes may be required to achieve the desired goal, usually defined as an improvement in blood flow and a residual stenosis of less than

20%. Other gauges of the success of a PTCA are an increase in the artery's lumen, difference of less than 20 mm Hg in blood pressure from one side of the lesion to the other, and no clinically obvious arterial trauma. Because the blood supply to the coronary artery decreases while the balloon is inflated, the patient may complain of chest pain (often called stretch pain), and the ECG may display significant ST-segment changes.

2.9.2.3 Coronary Artery Stent

After PTCA, a portion of the plaque that was not removed may block the artery. The coronary artery may recoil (constrict) and the tissue remodels, increasing the risk for restenosis.

A coronary artery stent is placed to overcome these risks. A stent is awoven mesh that provides structural support to a vessel at risk of acute closure. The stent is placed over the angioplasty balloon. When the balloon is inflated, the mesh expands and presses against the vessel wall, holding the artery open. The balloon is withdrawn, but the stent is left permanently in place within the artery Eventually, endothelium covers the stent and it is incorporated into the vessel wall. Because of the risk of thrombus formation in the stent, the patient receives antiplatelet medications (eg, clopidogrel [Plavix] therapy for 2 weeks and lifetime use of aspirin). Some stents have medication which may minimize the formation of thrombi or excessive scar tissue. It is estimated that 50% to 80% of all PCIs in-volve implanting at least one stent (Braunwald et al.. Stents may be used in conjunction with PTCA or independently as a PCI. Use of stents without PTCA may decrease procedure time, use of the potentially nephrotoxic contrast agent,radiation exposure, and cost . Care of the

patient after coronary artery stent placement is the same as for a patient after PTCA.

2.9.2.4 Atherectomy

Atherectomy is an invasive interventional procedure that involves the removal of the atheroma, or plaque, from a coronary artery. Directional (DCA) and transluminal extraction (TEC) coronary atherectomy procedures involve the use of a catheter that removes the lesion and its fragments. Rotational atherectomy uses a catheter with diamond chips impregnated on the tip (called a burr) that rotates like a dentist's drill at 130,000 to 180,000 rpm, pulverizing the lesion (Braunwald et al., 2001). Usually, several passes of these catheters are needed to achieve satisfactory results. Postprocedural patient care is the same as for a patient after PTCA

2.9.2.5 Transmyocardial Revascularization

Patients who have cardiac ischemia and who are not candidates for CABG may benefit from transmyocardial laser revascularization (TMR) The procedure may be performed percutaneously in the cardiac catheterization laboratory (percutaneous transmyocardial revascularization [PTMR]) or through a midsternal or thoracotomy incision in the operating room The tip of a fiberoptic catheter is held firmly against the ischemic area of the heart while a laser burns a channel into but not through the muscle. If the procedure is percutaneous, the catheter is positioned inside the ventricle. If the procedure is surgical, the catheter is positioned on the outer surface of the ventricle. Each procedure usually involves making 20 to 40

channels. It is thought that some blood flows into the channels, decreasing the ischemia directly. Within the next few days to months, the channels close as a result of the body's inflammatory process of healing a wound long-term result is the formation of new blood vessels (angiogenesis) during the inflammatory process that follows the laser burns

The new blood vessels provide enough blood to decrease the symptoms of cardiac ischemia. Nursing care before, during, and after the procedure depends on the approach: if the approach was percutaneous, the patient care is the same as following a PTCA; if the approach was surgical, the patient care is the same as following CABG.

2.9.2.6 Cardiopulmonary Bypass (CPB).

Many cardiac surgical procedures are possible because of CPB (ie, extracorporeal circulation).

The procedure mechanically circulates and oxygenates blood for

the body while bypassing the heart and lungs. CPB uses a heart lung machine to maintain perfusion to other body organs and tissues while the surgeon works in a bloodless surgical field.

CPB, a common but complex technique, is accomplished by placing a cannula in the right atrium, vena cava, or femoral vein to withdraw blood from the body. The cannula is connected to tubing filled with an isotonic crystalloid solution (usually 5% dextrose in lactated Ringer's solution). Venous blood removed from the body by the cannula is filtered, oxygenated, cooled or warmed, and then returned to the body. The cannula used to return the oxygenated blood is usually inserted in the ascending aorta, but it may be inserted in the femoral artery

The patient receives heparin, an anticoagulant, to prevent thrombus formation and possible embolization that may occur when blood contacts the foreign surfaces of the CPB circuit and is pumped into the body by a mechanical pump (not the normal blood vessels and heart). After the patient is disconnected from the bypass machine, protamine sulfate is administered to reverse the effects of heparin.

During the procedure, hypothermia is maintained, usually28°C to 32°C (82.4°F to 89.6°F). The blood is cooled duringCPB and returned to the body. The cooled blood slows the body's basal metabolic rate, thereby decreasing its demand for oxygen. Cooled blood usually has a higher viscosity, but the crystalloid solution used to prime the bypass tubing dilutes the blood When the surgical procedure is completed, the blood is rewarmednas it passes through the CPB circuit. Urine output, blood pressure, arterial blood gas measurements, electrolytes, coagulation studies, and the ECG are monitored to assess the patient's status during CPB[4].

2.10 Complication of coronary artery disease

Coronary artery disease can lead to:

- **Chest pain (angina).** When your coronary arteries narrow, your heart may not receive enough blood when demand is greatest — particularly during physical activity. This can cause chest pain (angina) or shortness of breath.
- **Heart attack.** If a cholesterol plaque ruptures and a blood clot forms, complete blockage of your heart artery may trigger a heart attack. The lack of blood flow to your heart may damage your heart muscle. The

amount of damage depends in part on how quickly you receive treatment.

- **Heart failure.** If some areas of your heart are chronically deprived of oxygen and nutrients because of reduced blood flow, or if your heart has been damaged by a heart attack, your heart may become too weak to pump enough blood to meet your body's needs. This condition is known as heart failure.

- **Abnormal heart rhythm (arrhythmia).** Inadequate blood supply to the heart or damage to heart tissue can interfere with your heart's electrical impulses, causing abnormal heart rhythms [7].

2.11 prevention

Up to 90% of cardiovascular disease may be preventable if established risk factors are avoided.Prevention involves: exercise, decreasing obesity, treating hypertension, a healthy diet, decreasing cholesterol levels, and stopping smoking. Medications and exercise are roughly equally effective.High levels of physical activity reduce the risk of coronary artery disease by about 25%.In diabetes mellitus, there is little evidence that very tight blood sugar control improves cardiac risk although improved sugar control appears to decrease other problems like kidney failure and blindness. The World Health Organization (WHO) recommends "low to moderate alcohol intake" to reduce risk of coronary artery disease while high intake increases the risk.

A diet high in fruits and vegetables decreases the risk of cardiovascular disease and death Vegetarians have a lower risk of heart disease, possibly due to their greater consumption of fruits and vegetables Evidence also

suggests that the Mediterranean diet and a high fiber diet lower the risk. The consumption of Trans fat (commonly found in hydrogenated products such as margarine) has been shown to cause a precursor to atherosclerosis and increase the risk of coronary artery disease. Evidence does not support a beneficial role for omega-3 fatty acid supplementation in preventing cardiovascular disease (including myocardial infarction and sudden cardiac death). There is tentative evidence that menaquinone (Vitamin K_2), but not phylloquinone (Vitamin K_1), intake may reduce the risk of CAD mortality.

Secondary prevention

Secondary prevention is preventing further sequelae of already established disease. Lifestyle changes that have been shown to be effective to this goal include:

- Weight control
- Smoking cessation
- Avoiding the consumption of trans fats (in partially hydrogenated oils)
- Decrease psychosocial stress.
- Exercise. Aerobic exercise, like walking, jogging, or swimming, can reduce the risk of mortality from coronary artery disease. aerobic exercise can help decrease blood pressure and the amount of blood cholesterol (LDL) over time. It also increases HDL cholesterol which is considered as "good cholesterol".Separate to the question of the benefits of exercise; it is unclear whether doctors should spend time counseling patients to exercise. The U.S. Preventive Services Task Force, found "insufficient evidence" to recommend that doctors

counsel patients on exercise, but "it did not review the evidence for the effectiveness of physical activity to reduce chronic disease, morbidity and mortality", it only examined the effectiveness of the counseling itself The American Heart Association, based on a non-systematic review, recommends that doctors counsel patients on exercise [2].

CHAPTER THREE

3. Research methodology

3.1 Study design:

Descriptive cross sectional study hospital base, to assess Patient awareness about causes &associated risk factors of coronary artery diseas e.

3.2 Study area and setting:

Omdurman military hospital located in Omdurman in mowrada street bounded by youth palace and national council from the north, white river from the east and Banat from the west, it is general hospital contain all department of medicine, surgery, obs &gyna ,pediatric. coronary care unit is one of medicine department all coronary artery disease and other cardiovascular disease were admitted in it contain 7 beds all beds behind it source of oxygen supply, suction machine ,monitor ,infusion syringe pump. It's ready for admission to all cardiovascular disease from all Sudan state.

3.3 study population:

All Coronary artery disease patients in Coronary care unit at Omdurman military hospital from May to July 2017.

3.4 inclusive criteria:

Diagnosed of coronary arteries disease patients those admitted to coronary care unit at Omdurman military hospital during study period and those who agree to be included.

3.5 exclusive criteria:

Those who are disagree to be included in this study and those who was very ill or comatose patient and other pt with other cardiovascular disease.

3.6 Sampling and Sample size:

Sample technique: convenience sample calculated in a period from may to July   that equal 35 patient.

3.7 data collection tool:

 Interview administrative questioner

3.8 Data collection technique:

Structured Interview administrative questioner collected in a period of six weeks consists of two sections:

- Section one: about demographic data (gender, age, educational level.
- Section two: knowledge about :

-The development of coronary artery disease

 - The primary cause of coronary artery disease

- the risk factors for developing coronary artery disease

- Characteristics of chest pain of acute coronary syndrome related to coronary artery disease

- Factors assist recurrent of chest pain

- The common risk factor for developing coronary artery disease

- Complication of coronary artery disease

- Prevention for coronary artery disease

3.9 data analysis:

Computerized by using SPSS program version 16.

3.10 ethical consideration:

Approval was taken from Faculty of nursing science Shendi University

Approval from Administration of Omdurman military hospital

Verbal consent from all Participants before the beginning of interview ,after explained research objectives and purpose to participants in clear simple words, and they had right to leaf at any time, and privacy and confidentiality were insured.

CHAPTER

FOUR

Table (1): Gender of study population

ITEM	Frequency	Percent
male	20	57.1
female	15	42.9
Total	35	100.0

Table (2): Gender VS Age of study population

ITEM		Age				Total
		40-50	51-60	61-70	>70	
Gender	Male	4	7	6	3	20
	Female	9	6	0	0	15
Total		13	13	6	3	35

Table (3): Educational level of study population

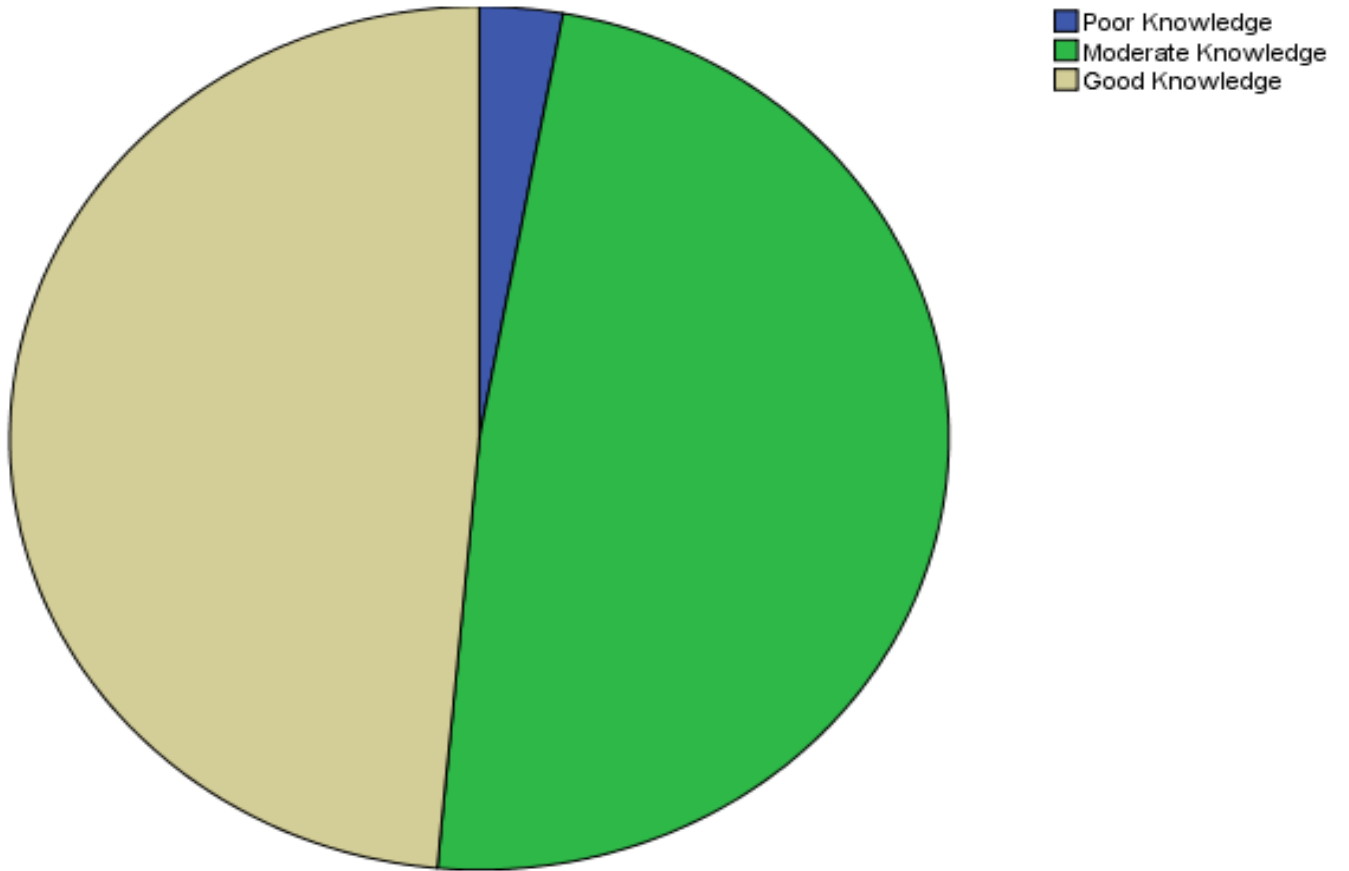| ITEM	Frequency	Percent
Khalwa	7	20.0
Primary	5	14.3
Secondary	19	54.3
University	4	11.4
Total	35	100.0
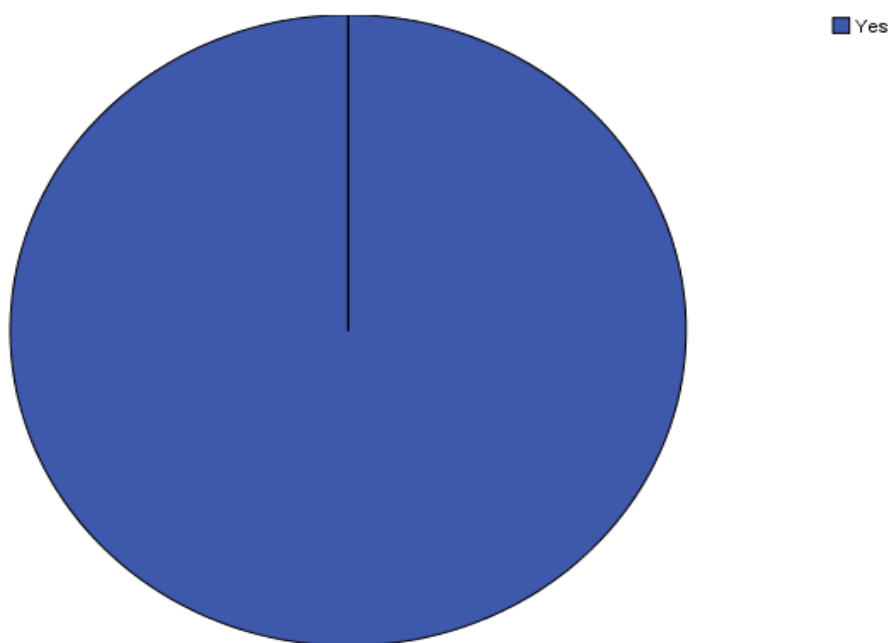
Figure (1): knowledge about the development of coronary artery disease
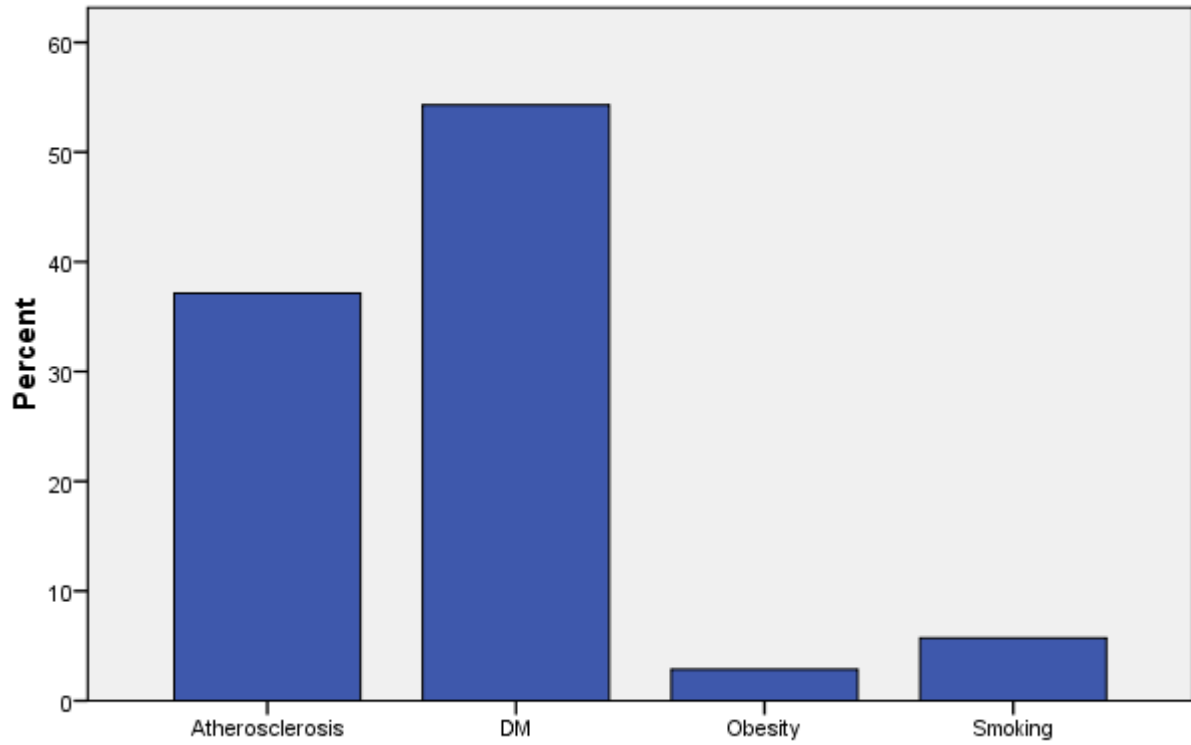
Figure (2): knowledge about the primary cause of coronary artery disease

Legend:
- Poor Knowledge
- Moderate Knowledge
- Good Knowledge

Figure (3): knowledge about risk` factor for coronary artery disease

Table (4): Educational level vs. Characteristics of chest pain of acute coronary syndrome related to coronary artery disease

ITEM	Poor Knowledge	Moderate Knowledge	Good Knowledge	Total
Khalwa	3	2	2	7
Primary	1	2	2	5
Secondary	0	6	13	19
University	0	0	4	4
Total	4	10	21	35

Table (5): Gender * Age * knowledge about Factors that assist recurrent of chest pain includes: Cross tabulation

ITEM			Age				Total
			40-50	51-60	61-70	>70	
Poor Knowledge	Gender	male	3	3	5	3	14
		female	4	3	0	0	7
		Total	7	6	5	3	21
Moderate Knowledge	Gender	male	1	4	1	0	6
		female	5	2	0	0	7
		Total	6	6	1	0	13
Good Knowledge	Gender	female	0	1	0	0	1
		Total	0	1	0	0	1

Table (6): Gender vs. Age the common risk factor for developing coronary artery disease

ITEM			Age				Total
			40-50	51-60	61-70	>70	
HTN	Gender	male	0	1	4		5
		female	2	2	0		4
		Total	2	3	4		9
HTN & Increasing age	Gender	male			1	2	3
		Total			1	2	3
HTN & Smoking	Gender	male	0	1	0	0	1
		Total	0	0	0	0	0
HTN & Smoking & DM	Gender	0	1	0	0	0	1
		Total	1	0	0	0	0
Hyperlipidemia& Family history & Increasing weight	Gender	female	0	0	0	0	2
		Total	1	1	0	0	0
Hyperlipidemia& Increasing weight	Gender	female	1	0	0	0	1
		Total	1	0	0	0	0
Increasing weight	Gender	female	0	0	0	0	4
		Total	3	1	0	0	0
Smoking	Gender	male	2	0	0	0	2
		Total	2	0	0	0	0
Increasing Age	Gender	male	0	0	0	1	2
		Total	0	0	0	1	2
DM	Gender	0	0	2	0	0	3
		Total	1	2	0	0	0
HTN & Family history	Gender	male	0	0	0	0	2
		female	1	0	0	0	1
		Total	1	2	0	0	0
Family history & Smoking	Gender	male	1	0	0	0	2
		Total	1	1	0	0	0
Hyperlipidemia& Increasing weight & DM	Gender	male	0	0	0	0	1
		Total	0	1	0	0	1
Smoking & Increasing weight	Gender	male	0	1	0	0	1
		Total	0	1	0	0	1

Table (7): knowledge about complication of Coronary artery disease

ITEM	Frequency	Percent
Poor Knowledge	8	22.9
Moderate Knowledge	20	57.1
Good Knowledge	7	20.0
Total	35	100.0
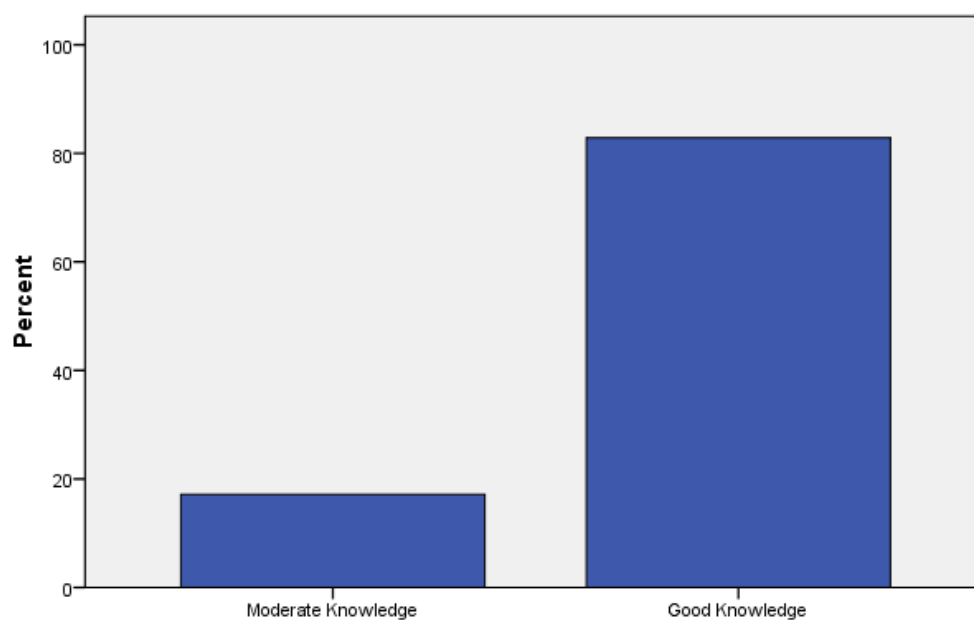
Figure (4): knowledge about prevention for Coronary artery disease

CHAPTER

FIVE

5.1 discussions

This is cross-sectional study was conducted to assess patient awareness about causes & associated risk factors of coronary artery disease in coronary care unit in Omdurman military hospital , include 35 of participant ,more than half were male ,and their age above 50 years old ,this result is expected because age considered as major risk factors of CAD & men have a greater risk for CAD than women , majority of them their education was secondary level this also impact on their knowledge .

This is agree with another study which was conducted in alshab hospital to assess the knowledge of CAD patient about risk factor and preventive measure in most of them are male except in (their age 61-70).

The response rate of participant about coronary artery disease developing gradually event many Years and cannot easily detected is 100% this indicate that the participant had good knowledge about development of CAD.

This result disagree with alshab study about development of coronary artery disease only 40,9% know that CAD developing gradually event many Years and cannot easily detected

The response rate about the primary cause of coronary artery disease only 32% of study population were answer that atherosclerosis is the primary cause for coronary artery disease, this indicate the participant had poor knowledge about the primary cause of CAD.

The distribution of knowledge about risk factor for coronary artery disease is range between good & moderate knowledge.

this result disagree with another study was conducted in Oman about assessment of knowledge of coronary artery disease risk factors which was revealed that only less than half (40%) had a good knowledge   about risk factors for CAD.

More than half of study population 60% was good about their knowledge of characteristics of chest pain of acute coronary syndrome related to coronary artery disease this can improve the prognosis.

Most study population was poor knowledge about factors that assist recurrent of chest pain this limits the participant ability to engage in preventive practice.

The common risk factors between the study populations is HTN common for one select, family history and smoking for two selects (options). Hyperlipidemia & Increasing weight & DM for three select.

More than half of study population 57% has a moderate Knowledge about the complication of coronary artery disease

The knowledge about the prevention of coronary artery disease is good Majority of study population 92.9% was knowledgeable about the prevention methods.

This result disagree with alshab study that concluded most of participant had poor knowledge regarding prevention of CAD.

5.2 conclusions

From the study that has been carried out, the researcher can concluded that study population had a fair knowledge about the awareness of coronary artery disease development, risk factors, complication, prevention methods and they had poor knowledge regarding the primary causes, so the study recommended comprehensive education about causes &risk factors.

5.3 recommendation

According to the result of the study the researcher recommended that:

- Establish of health educational program to patient and their families through primary, secondary and tertiary prevention to increase the awareness about coronary artery disease especially in the community of health persons to minimize the risk for developing coronary artery disease, education about life style changes(health diet, regular exercise, decrease weight, cigarette and tobacco cessations).
- Increase control on all modifiable risk factors for those whom at risk for developing coronary artery disease.
- Optimum medical treatment and follow up to decrease recurrent attack of CAD.

ANNEX

Reference

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Date: 6/4/2017   time: 6:32pm

COLLAGE OF NURSING SCIENCE

QUESTIONNAIRE ABOUT

PATIENT AWARENESS ABOUT CAUSES & ASSOCIATED RISK FACTORS OF CORONARY ARTERY DISEASE IN CORONARY CARE UNIT AT OMDURMAN MILITARY HOSPITAL

This questionnaire contains information about risk factors of coronary artery disease (heart disease) and preventive measures for purpose of research only.

Section (A)

1- Gender:-

A- male ☐   B- female ☐

2-Age:-

A- 40-50 ☐   B- 51-60 ☐   C- 61-70 ☐

D- above 70 ☐

3- Educational level:-

A- Illiterate ☐   B- khalwa ☐   C- primary ☐

D- Secondary ☐   E- university ☐

Section (B)

4 – Coronary artery disease Developing Gradually Event Many Years and Cannot Easily Detected?

A- Yes ☐   B- NO ☐

5-The primary cause of coronary artery disease is?

☐

A- Atherosclerosis [ ] B- DM

C- Obesity [ ] D- smoking [ ]

6- The risk factors for developing coronary artery disease are?

A- Hyperlipedemia [ ] B- HTN &DM [ ]

C - Family history [ ] D- smoking [ ]

E- Increasing weight [ ] F-increasing age [ ]

7- Characteristics of chest pain of acute coronary syndrome related to coronary artery disease are ?

A- central or epigastric chest pain [ ]
B- Substernal pressure ,burning ,aching, or sharp pain [ ]
C- Radiating to the arm, shoulder, neck and jaw [ ]
D- Associated with nausea ,vomiting, dyspnea and palpitation [ ]

8-factors assist recurrent of chest pain include:

A- Stress [ ] B- heavy exercise [ ]
B- Infection [ ] D- bed rest [ ]
E- others [ ]

9- What is your risk factor for developing coronary artery disease?

A- Hyperlipedemia [ ] B- HTN [ ]

C - Family history [ ] D- smoking [ ]

E- Increasing weight [ ] F- increasing age [ ]

G- DM [ ] H- alcohol intake [ ]

10- Coronary artery disease can lead to:

A- DCM [ ] B- sudden cardiac death [ ]

C- Heart failure [ ] D- Abnormal heart rhythm [ ]
(arrhythmia).

55

11-coronary artery disease can be prevented by:

A- Physical exercise ☐ B- cigarette cessation ☐

C- Decrease weight ☐ D- high fiber diet intake ☐

E- control of DM&HTN ☐