Shandi University

Faculty of graduate studies scientific research

Nurse's Competence regarding Initial Management of Patient with Acute Coronary Syndrome in Omdurman Military hospital (CCU/ED)

Thesis submitted for partial fulfillment of master degree in critical and emergency care nursing

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الآية

بسم الله الرحمن الرحم

(يا أيها الناس إن كنتم في ريب من البعث فإنا خلقناكم من تراب ثم من نطفة ثم من علقة ثم من مضغة مخلقة وغير مخلقة لنبين لكم ونقر في الأرحام ما نشاء إلى أجل مسمى ثم نخرجكم طفلا ثم لتبلغوا أشدكم ومنكم من يتوفى ومنكم من يرد إلى أرذل العمر لكيلا يعلم من بعد علم شيئا وترى الأرض هامدة فإذا أنزلنا عليها الماء اهتزت وربت وأنبتت من كل زوج بهيج)

سورة الحج (5)

Dedication,

This is for you , Mom .thank you for always being there for me,,

In the memory of my great father, who left his fingerprint on our life,,

To all brothers and sisters of me for their standing by me,,,

To everyone who encourages me ,supported me ,and prayed for me,,

Nima,,,

Acknowledgement,

My sincere appreciation, thanks and respect provided to Dr. Mohamed jebreldar for being kind to me and patient ,also for his valuable and inspiring guidance and supervision .thanks a lot ,,,

Also, thanks provided to staff member of cardiac care units and emergency departments in study areas for facilitating data collection and being kind to participate.

Finally, thanks to all those who kindly supported encouraged or facilitated me during my study process.

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LIST OF ABBREVIATION

ACC	American College of Cardiology
ACE	Angiotensin-converting enzyme
ACS	acute coronary syndrome
AHA	American Heart Association
AMI	Acute myocardial infarction
ARB	Angiotensin receptor blocker
AV	Atrioventricular
CABG	Coronary Artery Bypass Graft
CCU	Cardiac care unit
CHD	Chronic heart disease
CK	Creatine kinase
CTN1	Cardiac-specific troponin-1
CVD	Cardiovascular disease
DM	Diabetes mellitus
ECG	Electrocardiogram
ED	Emergency Department
EPHESUS	Epleronone Post-Acute Myocardial Infarction Heart Failure Efficacy and Survival Study
ESR	Erythrocyte sedimentation rate
FMH	Federal Ministry of Health
HDL-C	High Density Lipoprotein cholesterol
HDU	High dependent unit
ICDs	implantable cardioverter-defibrillators
IHD	Ischaemic heart diseases
IV	Intravenous
LDL-C	Low Density Lipoprotein cholesterol
LMWH	Low-molecular-weight heparin
LVF	Left ventricular failure
MI	Myocardial Infarction
MONA	Morphine, Oxygen, Nitrates, and Aspirin
NSTMI	Non-ST-segment elevation myocardial infraction
NYH	New York Heart Association
PCI	Percutaneous coronary intervention
`PTSD	Posttraumatic stress disorder
STEMI	ST-segment elevation myocardial infraction
TH	Teaching Hospital
UA	Unstable angina
WHO	World Health Organization

Abstract:

Background: ACS refers to a range of potentially life-threatening conditions that affect coronary artery blood supply to the heart , which requires standardized care policies, as well as it needs qualified and skilled health provider to obtain good outcome of management.

Objectives : To study nurse's competence regarding initial management of patients with acute coronary syndrome.

Methods: This is a descriptive hospital-based study conducted in the Military hospital (Omdurman State) in the period from (April-June2018)at cardiac care unit and emergency department and it included 60 nurses. The assessment was performed by using a questionnaire for knowledge and check list for practice assessment.

Results: Out of 60 participants ,the majority was females most nurses had experience of 5-10 years. Mean of total knowledge was found very good. They showed high knowledge about risk factors of ACS and less knowledge about medications. The practice was very good in regard to connecting monitor and checking vital signs, but poor in the aspect of oxygen administration ,and Inserting urinary catheters. Experience found significantly associated with oxygen administration , giving pain killers and thrombolysis.

Conclusion: The study showed higher percentage of knowledge regarding ACS nature, risk factors, and knowledge regard initial management which identified by vast majority, while they showed less knowledge towards symptoms ,diagnosis, pain assessment, initial drugs and drug side effect, and thrombolysis indication. they showed poor performance regarding assessment of cardiac output, interaction when patient arrive to ED, oxygen administration, giving pain killers and thrombolysis, assessment of pain level ,inserting catheter to reduce exertion of pt.

مستخلص الدراسة:

الخلفيه: متلازمة الشريان التاجي من الامراض المهدده للحياة ومسبب رئيسي للوفاة مما يجعلها تحتاج الى عنايه طبيه فائقه وطاقم خدمة صحيه مؤهل للحصول على النتائج الايجابيه وللوقايه من المضاعفات.

هدف البحث: تقييم الكفاءة العلميه والعملية للطاقم التمريضي في التعامل العاجل مع مرضى متلازمة الشريان التاجي.

منهجيه البحث: عبارة عن دراسه وصفيه مستندة علي العاملين بمستشفى السلاح الطبي بامدرمان في الفترة من (ابريل–يونيو2018) بكل من قسم الطوارئ وقسم العنايه القلبيه وتشمل الدراسه (60) ممرض وممرضه من العاملين بالاقسام حيث تم اختيارهم عن طريق التغطيه الكليه وحسب موافقتهم كما تم تقييم معلوماتهم بواسطة الاستبيان وتم تقييم ادائهم بواسطة استمارة الملاحظة.

نتائج البحث: من بين (60) مشارك ومشاركة الغالبيه كانت لفئه الاناث من الممرضات بخبرات تتراوح ما بين (10–5)سنوات كما ان معدل المعلومات كان جيدا جدا حيث اظهرو درجات عاليه من المعلومات فيما يخص العوامل التي قد تؤدي الى الاصابه بالمرض وكذلك فيما يخص الادويه المنقذة للمريض, كما ان اداء الطاقم كان جيدا في توصيل الاجهزة المراقبه للعلامات الحيويه وكذلك في اخذ العلامات الحيويه للمريض ,ومن ناحيه اخرى كان الاداء ضعيفا تجاه تقديم الاوكسجين ,وادخال القسطرة البوليه للمريض. واثبتت الدراسه ان سنوات الخبرة لها علاقه مصاحبه مع تقديم الاوكسجين للمريض ,واعطاء الادويه المسكنه للالام,وكذلك اعطاء الادويه المذيبه للتجلطات.

خلاصه البحث: اظهرت الدراسه معرفه ومعلومات جيدة عن متلازمة الشريان التاجي,واداء عملي متوسط تجاه الرعايه الصحيه المستعجله للمريض . كما اظهرت اداء ضعيف تجاه بعض المهام مثل تقديم الاوكسجين,التفاعل مع المريض لحظة وصوله لقسم الطوارئ , وتقديم الادويه, وايضا في تقييم النتاج القلبي وكذلك تقييم مستوى الم المريض.

CHAPTER -1

INTRODUCTION

Cardiovascular disease remains a major healthcare problem and one of the most consumers' of the public health resources. Ischemic heart diseases (IHD) remain the commonest cause of death all over the world.

According to measurement criteria of American Association of Critical-Care Nurses (AACN) acquires and maintains current knowledge and competency in the care of acutely and critically ill patients. The nurse participates in ongoing learning activities to acquire and refine the knowledge and skills needed to care and seeks learning opportunities that reflect evidence-based practice in order to maintain clinical skills and competencies. So the nurse engages in a self-assessment and formal performance appraisal on a regular basis own nursing practice in relation to professional practice standards, institutional guidelines, rules, and regulations to identifying areas of strength as well as areas where professional development would be beneficial[9,13].

The overall aim of this study is to assess the knowledge attitude and a skill of nurse's who work at CCU and ER during initial management for patients with acute coronary syndrome. In order to identify the gaps regard nursing care and to search for solutions with expertise and policy makers to construct plans and strategies to fill the gaps and for fulfill men thigh quality nursing care.

PROBLEM STATEMENT:

ACS is a critical health situation which requires standardized care policies, as well as its need qualified and skilled health care providers to obtain good outcome. With the change in life style of Sudanese people, the rate of MI increased since they tend to prefer urban life with low physical efforts. In such circumstance and due to critical status of the patients during the first 24 hours, treatment should be supervised by qualified health care providers among nurse's represents the closest line of care staff. During practice, it was observed that nurse's performance towards ACS in many times do not satisfy the required level, this necessitates more evaluation and workup. The opportunities of training provided for nurses in such field are restricted by many difficulties which expected to be reflected on developing nurse's performance**.**

Justification:

- Critical illness requires life-saving intervention and application of high technology medicine and intensive nursing within a specialist critical care Unit.[15]
- It was observed that many pt came near to death or developed to seriously ill patient because of miss judgment of nurses beginning from triage itself ,and most health care providers does not take it as a serious situation ,which delay patient management and expose him to harmful situation.
- Health care services are provided to patients in an environment with complex Interactions among many factors, such as the disease process itself, clinicians, technology, policies, procedures, and resources. When these complex factors interact, harmful and unanticipated errors can occur. By definition, errors are a cognitive phenomenon because errors reflect human action that is a cognitive activity. Near misses or "good catches.[16]
- Monitoring and evaluation of nurse's competence in Sudan might participate to provide better future performance which can be accomplished by spotting the defects in performance, concepts and settings.
- MI is now single biggest killer in the western world. It is also afflicts and incapacitates many people in their most productive time of life. Sudden death is first manifestation of it. CHD in 20-30 percent of cases two third of deaths from MI are sudden and take place before medical help can be reached .as many die within the first 24 Hours [17].

OBJECTIVES:

1- General objective:

To study nurse's competence regarding initial management of patients with acute coronary syndrome.

2- Specific objectives:

1. To assess nurse's knowledge about acute coronary syndrome .

2.To determine nurse's performance regarding initial management of patient with acute coronary syndrome .

3. To identify the relationships between competence and demographic data .

CHAPTER -2

LITERATURE REVIEW

1.Overview of ACS:

Acute coronary syndrome (ACS) is the umbrella term for the clinical signs and symptoms of myocardial ischemia: unstable angina, non–ST-segment elevation myocardial infarction, and ST-segment elevation myocardial infarction.

coronary artery disease, in which atherosclerotic plaque builds up inside the coronary arteries and restricts the flow of blood (and therefore the delivery of oxygen) to the heart, continues to be the number-one killer. One woman or man experiences a coronary artery disease event about every 25 seconds, despite the time and resources spent educating clinicians and the public on its risk factors, symptoms, and treatment. Coronary artery disease can lead to acute coronary syndrome (ACS), which describes any condition characterized by signs and symptoms of sudden myocardial ischemia—a sudden reduction in blood flow to the heart. The term ACS was adopted because it was believed to more clearly reflect the disease progression associated with myocardial ischemia. Unstable angina and myocardial infarction (MI) both come under the ACS umbrella.[1]

The signs and symptoms of ACS constitute a continuum of intensity from unstable angina to non–Segment elevation MI (NSTEMI) to ST-segment elevation MI (STEMI). Unstable angina and NSTEMI normally result from a partially or intermittently occluded coronary artery, whereas STEMI results from a fully occluded coronary artery. According to the American Heart Association (AHA), 785,000 Americans will have an MI this year, and nearly 500,000 of them will experience another. In 2006 nearly 1.4 million patients were discharged with a primary or secondary diagnosis of ACS, including 537,000 with unstable angina and 810,000 with either NSTEMI or STEMI (some had both unstable angina and MI). The AHA and the American College of Cardiology (ACC) recently updated practice guidelines and performance measures to help clinicians adhere to as standard of care for all patients who present with symptoms of any of the three stages of ACS [2-5] . Nurses not specializing in the care of patients with cardiovascular disease may not be familiar with current practice guidelines and nomenclature, but they nevertheless play significant roles in detecting patients at risk for ACS, facilitating their diagnosis and treatment, and providing education that can improve outcomes. Many patients admitted with a diagnosis of NSTEMI or unstable angina are cared for by physicians other than cardiologists and are therefore less likely to receive evidence-based care. Nurses caring for these patients can be instrumental in promoting adherence to practice guidelines [1]

2. PATHOPHYSIOLOGY OF ACS:

ACS begins when a disrupted atherosclerotic plaque in a coronary artery stimulates platelet aggregation and thrombus formation. When the thrombus occlude the vessel that will prevents myocardial perfusion. In the past, researchers supposed that the narrowing of the coronary artery in response to thickening plaque was primarily responsible for the decreased blood flow that leads to ischemia, but more recent data suggest that it's the rupture of an unstable, vulnerable plaque with its associated inflammatory changes. Most cases of infarction are due to the formation of an occluding thrombus on the surface of the plaque [(4-6)]. Myocardial cells require oxygen and adenosine 5b-triphosphate (ATP) to maintain the contractility and electrical stability needed for normal conduction. As myocardial cells are deprived of oxygen and anaerobic metabolism of glycogen takes over, less ATP is produced, leading to failure of the Sodium–potassium and calcium pumps and an accumulation of hydrogen ions and lactate, resulting in acidosis. At this point, infarction—cell death—will occur unless interventions are begun that limit or reverse the ischemia and injury. During the ischemic phase, cells exhibit both aerobic and anaerobic metabolism. If myocardial perfusion continues to decrease, aerobic metabolism ceases and eventually anaerobic metabolism will be significantly reduced. This period is known as the injury phase. If perfusion is not restored within about 20 minutes, myocardial necrosis results and the damage is irreversible. Impaired myocardial contractility, the result of scar tissue replacing healthy tissue in the damaged area, decreases cardiac output, limiting perfusion to vital organs and peripheral tissue and ultimately contributing to signs and symptoms of shock. Clinical manifestations include changes in level of consciousness; cyanosis; cool, clammy skin; hypotension; tachycardia; and decreased urine output. [(7)]

Patients who have experienced an MI are therefore at risk for developing cardiogenic shock. In an attempt to support vital functions, the sympathetic nervous system responds to ischemic changes in the myocardium. Initially, both cardiac output and blood pressure decrease, stimulating the release of the hormones epinephrine and norepinephrine, which in the body's attempt to compensate increase the heart rate, blood pressure, and after load, ultimately increase myocardial demand for oxygen. As oxygen demand increases at the same time that its supply to the heart muscle decreases, ischemic tissue can become necrotic. Low cardiac output also leads to decreased renal perfusion, which in turn stimulates the release of renin and angiotensin, resulting in further vasoconstriction. Additionally, the release of aldosterone and antidiuretic hormone promotes sodium and water reabsorption, increasing preload and ultimately the workload of the myocardium.[(8)]

Mastering the concepts of preload and afterload will guide the nurse in understanding the pharmacologic management of ACS. Preload, the blood volume or pressure in the ventricle at the end of diastole, increases the amount of blood that's pumped fromthe left ventricle (the stroke volume) . Ischemia decreases the ability of the myocardium to contract efficiently; therefore, in a patient with ACS an increase in preload hastens the strain on an already oxygen-deprived myocardium, further decreasing cardiac output and predisposing the patient to heart

failure. As I'll describe in further detail below, medications such as nitroglycerin, morphine, and b-blockers act to decrease preload. These medications, along with angiotensin-converting .enzyme (ACE) inhibitors, also decrease afterload, which is the force the left ventricle has to work against to eject blood.[9] In myocardial ischemia, the weakened myocardium cannot keep up with the additional pressure exerted by an increase inafterload.

3.SIGNS AND SYMPTOMS:

The degree to which a coronary artery is occluded typically correlates with presenting symptoms and with variations in cardiac markers and electrocardiographic findings. Angina, or chest pain, continues to be recognized as the classic symptom of ACS. In unstable angina chest pain normally occurs either at rest or with exertion and results in limited activity. Chest pain associated with NSTEMI is normally longer in duration and more severe than chest pain associated with unstable angina. In both conditions, the frequency and intensity of pain can increase if not resolved with rest, nitroglycerin, or both and may last longer than 15 minutes. Pain may occur with or without radiation to the arm, neck, back, or epigastria area.

In addition to angina, patients with ACS also present with shortness of breath, diaphoresis, nausea, and lightheadedness. Changes in vital signs, such as tachycardia, tachypnea, hypertension, or hypotension, and decreased oxygen saturation (SaO2) or cardiac rhythm abnormalities may also be present.[2]

Atypical ACS symptoms. Many women present with atypical symptoms, resulting in delayed diagnosis and treatment[10] . Women frequently experience shortness of breath, fatigue, lethargy, indigestion, and anxiety prior to an acute MI and may not attribute those symptoms to heart disease.[11] It's also important for clinicians to realize that women tend to experience pain in the back rather than substernally or in the left side of the chest and do not characterize it as pain, but may instead report a numb, tingling, burning, or stabbing sensation[12] in fact, a recent study found that, when compared with men, women diagnosed with ACS more often reported indigestion, palpitations, nausea, numbness in the hands, and atypical fatigue than chest pain.[13]

Silent ischemia. Ischemia can also occur without any obvious signs or symptoms. The classic Framingham Heart Study was initiated in 1948 to explore contributing factors for cardiovascular disease and has provided the scientific community with much of what is known today about heart disease Findings from this longitudinal study of 5,209 participants found that 50% of patients diagnosed with an MI experienced silent ischemia and did not exhibit any of the classic symptoms of ACS [3].Populations more likely to experience a silent MI include people with diabetes, women, older adults, and those with a history of heart failure[3]. As the prevalence of diabetes rises, silent ischemia may also become more common.

Table(1): Unstable angina, non–ST-segment myocardial infarction (NSTEMI), and ST-segment myocardial infarction (STEMI) differ with regard to duration, severity, and treatments.

Unstable Angina	Non–ST-Segment Elevation Myocardial Infarction	ST-Segment Elevation Myocardial Infarction (STEMI)
Cause •Thrombus partially or intermittently occludes the coronary artery	**Cause** • Thrombus partially or intermittently occludes the coronary artery(NSTEMI)	**Cause** • Thrombus fully occludes the coronary artery
Signs and Symptoms • Pain with or without radiation to arm, neck, back, or epigastria region •Shortness of breath, diaphoresis, nausea lightheadedness, tachycardia, tachypnea, hypotension or hypertension, decreased arterial oxygen saturation (SaO2) and rhythm abnormalities • Occurs at rest or with exertion; limits activity	**Signs and Symptoms** • Pain with or without radiation to arm, neck, back, or epigastria region • Shortness of breath, diaphoresis, nausea, lightheadedness, tachycardia, tachypnea, hypotension or hypertension, decreased arterial oxygen saturation (SaO2) and rhythm abnormalities • Occurs at rest or with exertion; limits activity • Longer in duration and more severe than in unstable angina	**Signs and Symptoms** • Pain with or without radiation to arm, neck, back, or epigastria region • Shortness of breath, diaphoresis, nausea, lightheadedness, tachycardia, tachypnea, hypotension or hypertension, decreased arterial oxygen saturation (SaO2), and rhythm abnormalities • Occurs at rest or with exertion; limits activity • Longer in duration and more severe than in unstable angina (irreversible tissue damage[infarction] occurs if perfusion is not restored)
Diagnostic Findings • ST-segment depression or T-wave inversion on electrocardiography • Cardiac biomarkers not elevated	**Diagnostic Findings** • ST-segment depression or T-wave inversion on electrocardiography • Cardiac biomarkers are elevated	**Diagnostic Findings** • ST-segment elevation or new left bundle branch block on electrocardiography • Cardiac biomarkers are elevated
Treatment • Oxygen to maintain oxygen saturation level at > 90% •Nitroglycerin or morphine to control pain • b-blockers, angiotensin-	**Treatment** • Oxygen to maintain SaO2 level at > 90% • Nitroglycerin or morphine to control pain • b-blockers, angiotensin-	**Treatment** • Oxygen to maintain SaO2 level at > 90% • Nitroglycerin or morphine to control pain • b-blockers, angiotensin-

converting enzyme inhibitors, statins (started on admission and continued long term), clopidogrel (Plavix), unfractionated heparin or low molecular-weight heparin, and glycoprotein IIb/IIIa inhibitors	converting enzyme inhibitors, statins (started on admission and continued long term), clopidogrel (Plavix), unfractionated heparin or low molecular-weight heparin, and glycoprotein IIb/IIIa inhibitors • Cardiac catheterization and possible percutaneous Coronary intervention for patients with ongoing chest pain, hemodynamic instability, or increased risk of worsening clinical condition.	converting enzyme inhibitors, statins (started on admission and continued long term), clopidogrel (Plavix), unfractionated heparin or low-molecular weight heparin • Percutaneous coronary intervention within 90 minutes of medical evaluation • Fibrinolysis therapy within 30 minutes of medical evaluation

Differentiations between ACS types.(14)

4. Risk factors:

Risk factors for serious disease and mortality in people have been documented in observational studies. The individual contributions of cigarette smoking, diabetes and hypertension have been noted in the clinical sphere, other factors, such as self-related health, physical disability, marital status, social support and physical activity, have been noted in the socio-demographic sphere.

4.1 Non-Modifiable risk factors:

4.1.1 Age:

Men: The risk increases after age 45

Women: The risk increases after age 55.

4.1.2. Heredity:

Family history of early heart disease -first degree relative- diagnosed before age 55; or a mother or sister diagnosed before age 65.

4.2 Modifiable risk factors:

4.2.1 Smoking:

Cigarette smoking greatly increases the risk of fatal and nonfatal heart attacks in both men and women. It also increases the risk of a second heart attack among survivors. Women who smoke and use oral contraceptives have an even greater risk than smoking alone. The good news is that

quitting smoking greatly reduces the risk of heart attack. One year after quitting the risk drop to about one-half that of current smokers and gradually returns to normal in persons without heart disease. Even among persons with heart disease, the risk also drops sharply one year after quitting smoking and it continues to decline over time but the risk does not return to normal [16].

4.2.2 High blood pressure (Hypertension):

High blood pressure makes the heart work harder. It increases the risk of developing heart disease, as well as kidney disease and stroke, follow a heart healthy eating plan, including foods lower in salt, help prevent or control high blood pressure, and, if a medication was prescribed, compliance is required [18].

4.2.3 High blood cholesterol:

The level of cholesterol in the bloodstream greatly affects the risk of developing heart disease. The higher the level of blood cholesterol, the greater the risk for heart disease or heart attacks. When there is too much cholesterol (a fat-like substance) in the blood, it builds up in the walls of arteries. Over time, this buildup causes arteries to become narrowed, and blood flow to the heart is slowed or blocked. If the blood supply to a portion of the heart is partially or completely cut off, a heart attacks results. Various factors affect cholesterol levels: diet, weight, physical activity, age, gender, and heredity.

High cholesterol is treated with lifestyle changes-a heart healthy eating plan, physical activity, and loss of excess weight-and, if those do not lower it enough, Medications include statins, bile acid sequestrates, nicotinic acid, and fabric acids [18]

4.2.4 Overweight and obesity:

Obesity is an important determinant of CVD. Obese children have an elevated risk of developing CVD in adulthood. The effects of obesity on cardiovascular health and disease are many, one of the most profound of which is hypertension.

Obesity has a strong effect on lipoprotein metabolism regardless of ethnic group. Increased weight is a determinant of higher levels of triglycerides, elevated LDL-C, and low HDL-C. Conversely, weight loss is associated with a healthier lipoprotein profile in both men and women: triglycerides decrease, HDL-C increases, and LDL-C decreases. ―The public health approach (to obesity) requires a systematic education of the public about the dangers of obesity. Various health agencies could work together to promulgate such a message that would reach all population groups [19].

4.2.5 Physical inactivity:

The risk of heart attack increases if there is a physical in activity or a sedentary lifestyle. Physical activity improves cholesterol levels, helps control high blood pressure and diabetes, and controls

keeps weight. It also increases physical fitness, promotes psychological well-being and self-esteem, and reduces depression and anxiety. Thus exercise and physical activity provides multidimensional benefits, [15].

Those who have already had a heart attack also benefit greatly from being physically active, starting slowly to increase physical activity, and to check with health care provider before starting a physical activity program. This is especially important among patient over age 55, have been inactive, or have diabetes or another medical problem.

4.2.6 Diabetes:

Patients with diabetes have a higher case fatality rate in my MI or stroke than those without diabetes: that is, MI and stroke are more often fatal if diabetes is present. Diabetes mellitus damages blood vessels, including the coronary arteries of the heart. Up to 75 percent of those with diabetes develop heart and blood vessel diseases. Diabetes also can lead to stroke, kidney failure, and other problems [17].

4.3 other risk factors:

1. Use of oral contraceptive pills, cocaine, or amphetamines may also increase chances for a heart attack.

2. Certain psychological factors have been linked to heart attacks and a worse outcome from a heart attack:

a. Depression

b. Anger and hostility

c. Social isolation and lack of social support

d. Chronic (ongoing) stress: Stress can come from any situation or thought that makes you feel frustrated, angry, or anxious. What is stressful to one person is not necessarily stressful to another. Stress is a normal part of life. In small quantities, stress is good – it can motivate you and help you be more productive.

However, too much stress, or a strong response to stress, is harmful. It can set you up for general poor health as well as specific physical or psychological illnesses like infection, heart disease, or depression. Persistent and unrelenting stress often leads to anxiety and unhealthy behaviors like overeating and abuse of alcohol or drugs [18].

5. DIAGNOSING ACS:

The patient's clinical history, presenting symptoms, biomarker levels, and electrocardiographic results are all evaluated.

5.1 Electrocardiographic findings:

The AHA and the ACC recommend that a 12-lead electrocardiogram (ECG) be performed in patients with symptoms consistent with ACS and interpreted by an experienced physician within 10 minutes of ED arrival [20]. Findings on a 12-lead ECG help the practitioner to differentiate between myocardial ischemia, injury, and infarction; locate the affected area; and assess related conduction abnormalities. Electrocardiographic findings reflective of unstable angina or NSTEMI include ST-segment depression and inverted T waves. ST depression will normally resolve when the ischemia or pain has resolved, although T-wave inversion may persist. Providers should review electrocardiographic Findings as well as levels of cardiac biomarkers to ST-segment depression and inverted T waves. ST depression will normally resolve when the ischemia or pain has resolved, although T-wave inversion may persist. Providers should review electrocardiographic findings as well as levels of cardiac biomarkers to distinguish between unstable angina and NSTEMI· [20]On the other hand, ST elevation on a 12-lead ECG in two contiguous leads is diagnostic of STEMI. With STEMI, T-wave inversion may also be present. These changes normally subside within hours of an MI. Abnormal Q waves appear on an ECG in the presence of an MI as a result of alterations in electrical conductivity of the infarcted myocardial cells.

Once an abnormal Q wave has developed it usually remains permanently on the ECG. Therefore, an abnormal Q wave on an ECG does not necessarily signal a current acute MI, but could indicate an old MI. [21]

5.2 Serum cardiac markers:

Certain proteins, called serum cardiac, are released into the blood from necrotic heart muscle after a myocardial infarction. These markers, specifically serum cardiac enzymes and troponin, are important in the diagnosis of MI. The presence of serum cardiac markers that occurs after cellular death indicated cardiac damage. creatine kinase (CK) and troponin are typically measured to diagnose an MI.

CK level begin to rise at about 6 hours after an MI, peak at about 18 hours, and return to normal within 24 to 36 hours .the CK enzymes are fractionated into bands. The CK-MB band is specific to myocardial cells and helps quantify myocardial damage [22].

Cardiac-specific troponin is a myocardial muscle protein released into circulation after myocardial injury. In the heart, there are two subtypes: cardiac-specific troponin T (CTN I) and cardiac –specific troponin I (CTN I).These markers are highly specific indicators to MI and have

greater sensitivity and specificity for myocardial injury than CK-MB troponin is used for diagnostic purposes in conjunction whit CK-MB. Serum levels of CTNI and CTN T increase from 4 to 6 hours after onset the MI, peak at 10 to 24 hours, and return to baseline over 10 to 14 days [(22)].

Myoglobin is released into the circulation within 2hours after MI and peak in 3 to 15 hours. Although it is one of the first serum cardiac markers to appear after an MI, it lacks cardiac specificity .in addition, the kidneys rapidly excite it in urine so that blood levels return to the normal range within 24 hours after MI. [(22)]

5.3 Blood picture:

Usually shows polymorph nuclear leukocytosis and raised ESR

5.4 Chest x ray:

Done to exclude LVF

5.5 Other investigations:

Some other investigations can be done after 24 hours for MI patients to confirm the diagnosis:

5.5.1 Coronary Angiography:

Is carried out in patients with MI who continue to suffer from repeated attacks of angina pain or those who develop cardiac Rupture [(23).]

5.5.2 Technetium:

Technetium scan (T99) is sensitive in the diagnosis of acute infarction .it becomes positive within 24-48 hours of the infarction .the acutely infracted area shows as a hot spot (increased technetium uptake) . The test is usually negative 7-10 days after the infarction [(23)].

5.5.3 Echocardiography:

An echocardiogram may be performed to compare areas of the left ventricle that are contracting normally with those that are not. One of the earliest protective actions of myocardial cells used during limited blood flow is to turn off the energy-requiring mechanism for contraction; this mechanism begins almost immediately after normal blood flow is interrupted. The echocardiogram may be helpful in identifying which portion of the heart is affected by an MI and which of the coronary arteries is most likely to be occluded. Unfortunately, the presence of wall motion abnormalities on the echocardiogram may be the result of an acute MI or previous (old) MI or other myopathy processes, limiting its overall diagnostic utility. Typical rise and fall of cardiac biomarkers following myocardial infarction [(20)]

6. Management:

Aims of management

While the primary concern of physicians is to prevent death, those caring for victims of myocardial infarction aim to minimize the patient's discomfort and distress and to limit the extent of myocardial damage.

The care can be divided conveniently into three phases:

(1) Emergency care: when the main considerations are to relieve pain and to prevent or treat cardiac arrest.
(2) Early care : in which the chief considerations are to initiate reperfusion therapy to limit infarct size and to prevent infarct extension and expansion and to treat immediate complications such as pump failure, shock and life-threatening arrhythmias.
(3) Subsequent care : in which the complications that usually ensue later are addressed, and consideration is given to preventing further infarction and death.

6.1 Emergency care:

6.1.1 Initial diagnosis:

A working diagnosis of myocardial infarction must first be made. This is usually based on the history of severe chest pain lasting for 15 min or more, not responding to nitroglycerine. But the pain may not be severe and, in
the elderly particularly, other presentations such as dyspnoea, faintness or syncope are common. Important clues are a previous history of coronary disease, and radiation of the pain to the neck, lower jaw, or left arm.

There are no individual physical signs diagnostic of myocardial infarction, but most patients have evidence of autonomic nervous system activation (pallor, sweating) and either hypotension or a narrow pulse pressure. Features may also include irregularities of the pulse, bradycardia or tachycardia, a third heart sound and basal rales. An electrocardiogram should be obtained as soon as possible. Even at an early stage, the ECG is seldom normal[11,12].

However, the ECG is often equivocal in the early hours and even in proven infarction it may never show the classical features of ST elevation and new Q waves. Repeated ECG recordings should be obtained and, when possible, the current ECG should be compared with previous records. ECG monitoring should be initiated as soon as possible in all patients to detect life-threatening arrhythmias. When the diagnosis is in doubt, rapid testing of serum markers is valuable. In difficult cases, echocardiography and coronary angiography may be helpful.

6.1.2 Relief of pain, breathlessness and anxiety:

Relief of pain is of paramount importance, not only for humane reasons but because the pain is associated with sympathetic activation which causes vasoconstriction and increases the work of the heart. Intravenous opioids — morphine or, where available, diamorphine are the analgesics most commonly used in this context; intramuscular injections should be avoided. Repeated doses may be necessary. Side-effects include nausea and vomiting, hypotension with

bradycardia, and respiratory depression. Antiemetics may be administered concurrently with opioids. The hypotension and bradycardia will usually respond to atropine, and respiratory depression to naloxone, which should always be available If opioids fail to relieve the pain after repeated administration, intravenous beta-blockers or nitrates are often effective. Paramedics have a limited choice of non-addictive opioids that they may use, the availability of which varies from country to country. Oxygen should be administered especially to those who are breathless or who have any features of heart failure or shock.

Anxiety is a natural response to the pain and to the circumstances surrounding a heart attack. Reassurance of patients and those closely associated with them is of great importance. If the patient becomes excessively
disturbed, it may be appropriate to administer a tranquilliser, but opioids are frequently all that is required.

6.1.3 Cardiac arrest:

BASIC LIFE SUPPORT
Those not trained or equipped to undertake advanced life support should start basic life support as recommended by The European Resuscitation Council[13].
ADVANCED LIFE SUPPORT
Trained paramedics and other health professionals should undertake advanced life support, as described in the guidelines of the European Resuscitation Council[14].

6.2 Early care (Restoring and maintaining patency of the infarct related artery):

6.2.1 DRUG THERAPY:-

Initial drug therapy:

The goals of therapy in acute MI in the first 24 hours are the expedient restoration of normal coronary blood flow and the maximum salvage of functional myocardium. These goals can be met by a number of medical interventions and adjunctive therapies. The primary obstacles to achieving these goals are the patient's failure to recognize MI symptoms quickly and the delay in seeking medical attention. When patients present to a hospital, there are a variety of interventions to achieve treatment goals. ―Time is muscle‖ guides the management decisions in acute STEMI, and an early invasive approach is the standard of care for acute NSTEMI. [(20)]

6.2.1.1 MONA

The ―MONA‖ mnemonic represents the 4 priority interventions for patients suspected to be experiencing a MI. these include morphine, oxygen, nitroglycerin, and aspirin. Keep in mind that while MONA is helpful, it doesn't actually represent the correct order or prioritization of the interventions. The mnemonic that correctly represents the order of actions is ONAM. This can be remembered by On A.M. (as is I work in the mornings: I am ON in the A.M. shift, or I am totally a morning person- I am ON in the A.M.) [(24)].

Oxygen is always given first, allegedly because it's the mos1t important; Nitro is given next in order to dilate the vessels, making it a matter of increased space. Aspirin helps disaggregate the platelets with the goal of clot reduction and prevention of additional ones. Morphine reduces pain and anxiety, which will in turn assist with the psychosocial aspects that contribute to the tachypnea, thereby reducing the oxygen demand [(24)].

MONA Acronym (ON-AM)

- **O: Administer oxygen:** by means of nasal cannula or face mask, oxygen administration provides more concentration for myocardial uptake in order to promote tissue profusion to meet metabolic demands. It can also decrease the discomfort associated with tissue ischemia and prevent additional damage.
- **N: Administer Nitroglycerin**: IV: Nitro has a vasodilatation effect, which promotes perfusion and lowers the myocardial workload and oxygen demand.
- **A: Administer Aspirin**: this works by inhibiting platelet activity and interrupts aggregation at the rupture site, thrombolysis
- **M: Administer morphine:** this relaxes the endothelial lining and promotes profusion of the heart tissue by reducing the myocardial workload. It also controls pain and reduces the risk of developing life-threatening dysrhythmias and other complications[(25)].

Medical Options:

6.2.1.2 Antiplatelet Agents

The use of aspirin has been shown to reduce mortality from MI. Aspirin in a dose of 325 mg should be administered immediately on recognition of MI signs and symptoms. [(20,26)] The nidus of an occlusive coronary thrombus is the adhesion of a small collection of activated platelets at the site of intimal disruption in an unstable atherosclerotic plaque. Aspirin irreversibly interferes with function of cyclooxygenase and inhibits the formation of thromboxane A2. Within minutes, aspirin prevents additional platelet activation and interferes with platelet adhesion and cohesion. This effect benefits all patients with acute coronary syndromes; including those with MI. Aspirin alone has one of the greatest impacts on the reduction of MI mortality. Its beneficial effect is observed early in therapy and persists for years with continued use. The long-term benefit is sustained, even at doses as low as 75 mg/day.

The Clopidogrel and Metoprolol in Myocardial Infarction Trial/Second Chinese Cardiac Study trial evaluated the use of clopidogrel versus placebo in patients who were taking aspirin but notundergoing reperfusion therapy. It demonstrated a benefit in favor of clopidogrel when used with aspirin. [(27, 28)]

6.2.1.3 Supplemental Oxygen:

Oxygen should be administered to patients with symptoms or signs of pulmonary edema or with pulse oximetry less than 90% saturation. [20] The rationale for using oxygen is the assurance that erythrocytes will be saturated to maximum carrying capacity. Because MI impairs the circulatory function of the heart, oxygen extraction by the heart and by other tissues may be diminished. In some cases, elevated pulmonary capillary pressure and pulmonary edema can decrease oxygen uptake as a result of impaired pulmonary alveolar-capillary diffusion. Supplemental oxygen increases the driving gradient for oxygen uptake.[29]

Arterial blood that is at its maximum oxygen-carrying capacity can potentially deliver oxygen to myocardium in jeopardy during an MI via collateral coronary circulation. The recommended duration of supplemental oxygen administration in a MI is 2 to 6 hours, longer if congestive heart failure occurs or arterial oxygen saturation is less than 90%. However, there are no published studies demonstrating that oxygen therapy reduces the mortality or morbidity of an MI.

6.2.1.4 Nitrates:

Intravenous nitrates should be administered to patients with MI and congestive heart failure, persistent ischemia, hypertension, or large anterior wall MI [20, 26] .The primary benefit of nitrates is derived from its vasodilator effect. Nitrates are metabolized to nitric oxide in the vascular endothelium. Nitric oxide relaxes vascular smooth muscle and dilates the blood vessel lumen. Vasodilatation reduces cardiac preload and after load and decreases the myocardial oxygen requirements needed for circulation at a fixed flow rate. Vasodilatation of the coronary arteries improves blood flow through the partially obstructed vessels as well as through collateral vessels. Nitrates can reverse the vasoconstriction associated with thrombosis and coronary occlusion.

When administered sublingually or intravenously, nitroglycerin has a rapid onset of action. Clinical trial data have supported the initial use of nitroglycerin for up to 48 hours in MI. There is little evidence that nitroglycerin provides substantive benefit as long-term post-MI therapy, except when severe pump dysfunction or residual ischemia is present. [27] Low BP, headache, and tachyphylaxis limit the use of nitroglycerin. Nitrate tolerance can be overcome by increasing the dose or by providing a daily nitrate-free interval of 8 to 12 hours. Nitrates must be avoided in patients who have taken a phosphodiesterase inhibitor within the previous 24 hours. [20]

6.2.1.5 Pain Control:

Pain from MI is often intense and requires prompt and adequate analgesia. The agent of choice is morphine sulfate, given initially IV at 5 to 15 minute intervals at typical doses of 2 to 4 mg. [20] Reduction in myocardial ischemia also serves to reduce pain, so oxygen therapy, nitrates,

and beta blockers remain the mainstay of therapy. Because morphine can mask ongoing ischemic symptoms, it should be reserved for patients being sent for coronary angiography.

6.2.1.6 Beta Blockers:

Beta blocker therapy is recommended within 12 hours of MI symptoms and is continued indefinitely. [24,25] Treatment with a beta blocker decreases the incidence of ventricular arrhythmias, recurrent ischemia, reinfarction, and, if given early enough, infarct size and short-term mortality. Beta blockade decreases the rate and force of myocardial contraction and decreases overall myocardial oxygen demand. In the setting of reduced oxygen supply in MI, the reduction in oxygen demand provided by beta blockade can minimize myocardial injury and death.

The use of a beta blocker has a number of recognized adverse effects. The most serious are heart failure, bradycardia, and bronchospasm. During the acute phase of an MI, beta blocker therapy may be initiated intravenously; later, patients can switch to oral therapy for long-term treatment. The COMMIT-CCS 2 trial raised safety concerns about the use of early intravenous beta blockers in high-risk patients. [37] In some patients who are considered high risk due to age or hemodynamic instability, it may be reasonable to hold off on early intravenous therapy [20].

6.2.1.7 Unfractionated Heparin:

Unfractionated heparin is beneficial until the inciting thrombotic cause (ruptured plaque) has completely resolved or healed. Unfractionated heparin has been shown to be effective when administered intravenously or subcutaneously according to specific guidelines. The minimum duration of heparin therapy after MI is generally 48 hours, but it may be longer, depending on the individual clinical scenario. Heparin has the added benefit of preventing thrombus through a different mechanism than aspirin.

6.2.1.8 Low-Molecular-Weight Heparin:

Low-molecular-weight heparin (LMWH) can be administered to MI patients who are not treated with fibrinolysis therapy and who have no contraindications to heparin. The LMWH class of drugs includes several agents that have distinctly different anticoagulant effects. LMWHs are proved to be effective for treating acute coronary syndromes characterized by unstable angina and NSTEMI. [20]Their fixed doses are easy to administer, and laboratory testing to measure their therapeutic effect is usually not necessary.

6.2.1.9 Warfarin:

Warfarin is not routinely used after MI, but it does have a role in selected clinical settings. The latest guidelines recommend the use of warfarin fvgor at least 3 months in patients

with left ventricular aneurysm or thrombus, a left ventricular ejection fraction less than 30%, or chronic atrial fibrillation.

6.2.1.10 Fibrinolysis:

Restoration of coronary blood flow in MI patients can be accomplished pharmacologically with the use of a fibrinolysis agent. Fibrinolysis therapy is indicated for patients who present with a STEMI within 12 hours of symptom onset without a contraindication. Absolute contraindications to fibrinolysis therapy include history of intracranial hemorrhage, ischemic stroke or closed head injury within the past 3 months, presence of an intracranial malignancy, signs of an aortic dissection, or active bleeding. Fibrinolysis therapy is primarily used at facilities without access to an experienced interventionalist within 90 minutes of presentation. [26]

As a class, the plasminogen activators have been shown to restore normal coronary blood flow in 50% to 60% of STEMI patients. The successful use of fibrinolysis agents provides a definite survival benefit that is maintained for years. The most critical variable in achieving successful fibrinolysis is time from symptom onset to drug administration. A fibrinolysis is most effective within the first hour of symptom onset and when the door-to-needle time is 30 minutes or less. [26]

6.2.1.11 Angiotensin-Converting Enzyme Inhibitors and Angiotensin Receptor Blockers:

Angiotensin-converting enzyme (ACE) inhibitors should be used in all patients with a STEMI without contraindications. ACE inhibitors are also recommended in patients with NSTEMI who have diabetes, heart failure, hypertension, or an ejection fraction less than 40%. In such patients, an ACE inhibitor should be administered within 24 hours of admission and continued indefinitely. Further evidence has shown that the benefit of ACE inhibitor therapy can likely be extended to all patients with an MI and should be started before discharge. Contraindications to ACE inhibitor use include hypotension and declining renal function [20,26].

ACE inh:ibitors decrease myocardial after load through vasodilatation. One effective strategy for instituting an ACE inhibitor is to start with a low-dose, short-acting agent and titrate the dose upward toward a stable target maintenance dose at 24 to 48 hours after symptom onset. Once a stable maintenance dose has been achieved, the short-acting agent can be continued or converted to an equivalent-dose long-acting agent to simplify dosing and encourage patient compliance. For patients intolerant of ACE inhibitors, angiotensin receptor blocker (ARB) therapy may be considered [26].

6.2.1.12 Glycoprotein IIb/IIIa Antagonists:

Glycoprotein IIb/IIIa receptors on platelets bind to fibrinogen in the final common pathway of platelet aggregation. Antagonists to glycoprotein IIb/IIIa receptors are potent inhibitors of platelet aggregation. The use of glycoprotein IIb/IIIa inhibitors during percutaneous coronary intervention (PCI) and in patients with MI and acute coronary syndromes has been shown to reduce the composite end point of death, reinfarction, and the need to revascularize the target lesion at follow-up. The current guidelines recommend the use of aIIb/IIIa inhibitor for patients in whom PCI is planned. For high-risk patients with NSTEMI who do not undergo PCI, aIIb/IIIa inhibitor may be used for 48 to 72 hours. [20]

6.2.1.13 Statin Therapy:

A statin should be started in all patients with a myocardial infarction without known intolerance or adverse reaction prior to hospital discharge. Preferably, a statin would be started as soon as a patient is stabilized after presentation. The Pravastatin or Atorvastatin Evaluation and Infection—Thrombolysis in MI-22 trial suggested a benefit of starting patients on high-dose therapy from the start (e.g., atorvastatin 80 mg/day). [20,30]

6.2.2 Percutaneous Coronary Intervention:-

PCI refers to invasive procedures in which a catheter is inserted, normally through the femoral artery, into the occluded coronary artery in order to open blockages and restore blood flow. Percutaneous transluminal coronary angioplasty (PTCA) is the insertion of a catheter with a balloon tip that's inflated to open the artery.[31]

A metal mesh device known as a coronary stent can also be inserted after angioplasty to keep the artery open. Drug-eluting stents are coated with medications that prevent restenosis by reducing inflammation and the formation of thrombin. Blockages can also be destroyed in a procedure known as an arthrectomy, in which a mechanical device or rotational technology is used to cut or shave the plaque. Once the artery is opened with PTCA or a coronary stent, radiation is delivered to the lesion (through brachytherapy), which helps prevent narrowing or reclusion. PCI is indicated if the onset of ACS symptoms occurred more than three hours earlier, if fibrinolysis therapy is contraindicated, if the patient is at high risk for developing heart failure, or if the STEMI diagnosis is not absolute. PCI should be performed within 90 minutes of medical evaluation.[31]

The degree of coronary occlusion and the structure and viability of the affected vessel may exclude candidates from consideration for PCI.18Possible complications include bleeding or hematoma from the arterial insertion site, decreased peripheral perfusion, retroperitoneal bleeding, cardiac arrhythmias, coronary spasm or MI, acute renal failure, stroke, and cardiac arrest. Post procedure care should include frequent monitoring of vital signs and cardiac rhythm as well as assessment of Peripheral pulses, arterial insertion site, pain, and intake and output[31].

Patients with STEMI or MI with new left bundle branch block should have PCI within 90 minutes of arrival at the hospital if skilled cardiac catheterization services are available. [28] Patients with NSTEMI and high-risk features such as elevated cardiac enzymes, ST-segment depression, recurrent angina, hemodynamic instability, sustained ventricular tachycardia, diabetes, prior PCI, or bypass surgery are recommended to undergo early PCI (<48 hours). PCI consists of diagnostic angiography combined with angioplasty and, usually, stenting. It is well established that emergency PCI is more effective than fibrinolysis therapy in centers in which PCI can be performed by experienced personnel in a timely fashion [31] an operator is considered experienced with more than 75 interventional procedures per year. A well-equipped catheterization laboratory with experienced personnel performs more than 200 interventional procedures per year and has surgical backup available. Centers that are unable to provide such support should consider administering fibrinolysis therapy as their primary MI treatment.

Restoration of coronary blood flow in a MI can be accomplished mechanically by PCI. PCI can successfully restore coronary blood flow in 90% to 95% of MI patients. Several studies have demonstrated that PCI has an advantage over fibrinolysis with respect to short-term mortality, bleeding rates, and reinfarction rates. However, the short-term mortality advantage is not durable, and PCI and fibrinolysis appear to yield similar survival rates over the long term. PCI provides a definite survival advantage over fibrinolysis for MI patients who are in cardiogenic shock. The use of stents with PCI for MI is superior to the use of PCI without stents, primarily because stenting reduces the need for subsequent target vessel revascularization. [32]

6.2.3 Other Treatment Options:-

6.2.3.1 Surgical Revascularization:

Emergent or urgent coronary artery bypass grafting (CABG) is warranted in the setting of failed PCI in patients with hemodynamic instability and coronary anatomy amenable to surgical grafting.[26] Surgical revascularization is also indicated in the setting of mechanical complications of MI, such as ventricular septal defect, free wall rupture, or acute mitral regurgitation. Restoration of coronary blood flow with emergency CABG can limit myocardial injury and cell death if performed within 2 or 3 hours of symptom onset. Emergency CABG carries a higher risk of perioperative morbidity (bleeding and MI extension) and mortality than elective CABG. Elective CABG improves survival in post-MI patients who have left main artery disease, three-vessel disease, or two-vessel disease not amenable to PCI.[31]

6.2.3.2 Implantable Cardiac Defibrillators:

The results of a multicenter automatic defibrillator implantation trial have expanded the indications for automatic implantable cardioverter-defibrillators (ICDs) in post-MI patients. The trial demonstrated a 31% relative risk reduction in all-cause mortality with the prophylactic

use of an ICD in post-MI patients with depressed ejection fractions. [33]The current guidelines recommend waiting 40 days after an MI to evaluate the need for ICD implantation. ICD implantation is appropriate for patients in NYHA functional class II or III with an ejection fraction less than 35%. For patients in NYHA functional class I, the ejection fraction should be less than 30% before considering ICD placement. ICDs are not recommended while patients are in NYHA functional class IV. [25]

6.3 Management of the later in-hospital course:

Most patients should rest in bed for the first 12–24 h , by which time it will be apparent whether the infarction is going to be complicated. In uncomplicated cases, the patient can sit out of bed late on the first day, be allowed to use a commode and undertake self-care and self feeding. Ambulation can start the next day and such patients can be walking up to 200 m on the flat, and walking up stairs within a few days. Those who have experienced heart failure, shock or serious arrhythmias should be kept in bed longer, and their physical activity increased slowly, dependent upon their symptoms and the extent of myocardial damage.

6.3.1 Treatment Outcomes:

An individual patient's long-term outcome following an MI depends on numerous variables, some of which are not modifiable from a clinical standpoint. However, patients can modify other variables by complying with prescribed therapy and adopting lifestyle changes.

6.3.2 Stress Testing:

Cardiac stress testing after MI has established value in risk stratification and assessment of functional capacity. [27]The timing of performing cardiac stress testing remains debatable. The degree of allowable physiologic stress during testing depends on the length of time from MI presentation. Stress testing is not recommended within several days after a myocardial infarction. Only sub maximal stress tests should be performed in stable patients 4 to 7 days after an MI. Symptom-limited stress tests are recommended 14 to 21 days after an MI. Imaging modalities can be added to stress testing in patients whose electrocardiographic response to exercise is inadequate to confidently assess for ischemia (e.g., complete left bundle branch block, paced rhythm, accessory pathway, left ventricular hypertrophy, digitalis use, and resting ST-segment abnormalities). [34]

From a prognostic standpoint, an inability to exercise and exercise-induced ST-segment depression are associated with higher cardiac morbidity and mortality compared with patients able to exercise and without ST-segment depression. [20]Exercise testing identifies patients with residual ischemia for additional efforts at revascularization. Exercise testing also provides prognostic information and acts as a guide for post-MI exercise prescription and cardiac rehabilitation.

6.3.3 Smoking Cessation:

Smoking is a major risk factor for coronary artery disease and MI. For patients who have undergone an MI, smoking cessation is essential to recovery, long-term health, and prevention of reinfarction. In one study, the risk of recurrent MI decreased by 50% after 1 year of smoking cessation.[35] All STEMI and NSTEMI patients with a history of smoking should be advised to quit and offered smoking cessation resources, including nicotine replacement therapy, pharmacologic therapy, and referral to behavioral counseling or support groups. [20, 26] Smoking cessation counseling should begin in the hospital, at discharge, and during follow-up.

6.3.4 Long-Term Medications:

Most oral medications instituted in the hospital at the time of MI will be continued long term. Therapy with aspirin and beta blockade is continued indefinitely in all patients. ACE inhibitors are continued indefinitely in patients with congestive heart failure, left ventricular dysfunction, hypertension, or diabetes. [20, 26] A lipid-lowering agent, specifically a statin, in addition to diet modification, is continued indefinitely as well. Post-MI patients with diabetes should have tight glycolic control according to earlier studies. The latest ACC/AHA guidelines recommend a goal HbA1c of less than 7%.

6.3.5 Cardiac Rehabilitation:

Cardiac rehabilitation provides a venue for continued education, reinforcement of lifestyle modification, and adherence to a comprehensive prescription of therapies for recovery from MI including exercise training. Participation in cardiac rehabilitation programs after MI is associated with decreases in subsequent cardiac morbidity and mortality. Other benefits include improvements in quality of life, functional capacity, and social support. However, only a minority of post-MI patients actually participate in formal cardiac rehabilitation programs because of several factors, including lack of structured programs, physician referrals, low patient motivation, noncompliance, and financial constraints.

7.Complications of ACS:-

Severe left ventricular dysfunction or one of the other mechanical complications of acute myocardial infarction (AMI) causes most of the deaths following AMI. Complications of AMI include: [36, 37]

- Ischemic (including failure of reperfusion): angina, re-infarction, infarct extension.
- Mechanical: heart failure, cardiogenic shock, mitral valve dysfunction, aneurysms, cardiac rupture.
- Arrhythmic: atrial or ventricular arrhythmias, sinus or atrioventricular (AV) node dysfunction.
- Thrombosis and embolic: central nervous system or peripheral embolization.

- Inflammatory: pericarditis.
- Psychosocial complications (including depression).

7.1 Ischemic complications:-

Failure of reperfusion is less likely with the availability of primary percutaneous coronary intervention (PCI). Reperfusion should reduce ST elevation to less than 50% within one hour[38].

7.1.1 Re-occlusion of an infarct-related artery:

- Occurs in a minority but significant number of patients following fibrinolysis therapy. These patients also tend to have a poorer outcome.
- Can be difficult to diagnose[38].

7.1.2 Infarction in a separate territory (recurrent infarction):

- May be difficult to diagnose within the first 24 to 48 hours after the initial event.
- Multivessel coronary artery disease is common in patients with AMI[38].

7.1.3 Post infarction angina:

- Angina may occur from a few hours to 30 days after AMI.
- the incidence is highest in patients with non-ST-elevation myocardial infarction and those treated with fibrinolysis compared with PCI[38].

7.2 Mechanical complications:-

7.2.1 Left ventricular dysfunction and heart failure:

- Pulmonary edema is common following a myocardial infarction. Overt cardiac failure following a myocardial infarction is a poor prognostic feature.
- The severity of the heart failure depends on the extent of the infarction and the presence of any other complications - e.g. acute mitral regurgitation.
- Cardiogenic shock occurs in 5-20% of patients following myocardial infarction.
- Killip's classification is one method used to assess the severity of cardiac failure following a myocardial infarction: [39]
- Cardiac failure usually responds well to oxygen, diuretics and ACE inhibitors/angiotensin receptor antagonists (and intravenous nitrates if no hypotension[39].

7.2.2 Ventricular septal rupture and free wall rupture:

- Risk factors: older age, female gender, non-smoker, anterior infarction, worse killed class on admission, increasing heart rate on admission, first myocardial infarction and hypertension. [40]
- Post infarction VSD is relatively infrequent but life-threatening. [41] The incidence has dramatically decreased with reperfusion therapy.
- May develop as early as 24 hours after myocardial infarction but often presents 2-7 days afterwards. Mortality rates are greater than 90%.
- Ventricular septal rupture:
- Free wall rupture:
- Pseudo aneurysm (false aneurysm)[41]

7.2.3 Acute mitral regurgitation:

- Most common with an inferior-posterior infarction and may be due to ischemia, necrosis, or rupture of the papillary muscle.
- Mitral regurgitation following myocardial infarction predicts a poor prognosis but is often transient and asymptomatic.
- Rupture of papillary muscle or chordate tendone [37, 42]
- Mitral regurgitation is often accompanied by a pan systolic murmur, but the murmur may be inaudible if left a trial pressure rises sharply.
- Echocardiogram is required to confirm the diagnosis, especially to differentiate from rupture of the interventricular septum, and to assess severity.

7.2.4 Left ventricular aneurysm:

The vulnerable myocardium following an AMI is susceptible to wall stress, resulting in infarct expansion. Sub-acute cardiac rupture is an extreme form of infarct expansion, whereas ventricular aneurysm is its chronic form. [43] Occurs after 2-15% of infarcts. Patients who do not receive reperfusion therapy are at greatest risk (10% to 30%). Five-year survival is 10-25%. May be clinically silent or cause recurrent tachyarrhythmia, heart failure or systemic emboli[37].

7.2.5 Right ventricular failure:

Right ventricle myocardial infarctions accompany inferior wall ischemia in up to one half of cases.Mild right ventricular dysfunction is common after inferior-posterior infarcts but right heart failure only occurs in a minority of these patients. May present with hypotension, jugular venous distention with clear lungs and no dyspnea. Severe right ventricular failure may present with a low cardiac output state, including oliguria and altered mental state. Diagnosis is made by echocardiography[44].

7.2.6 Left ventricular outflow tract obstruction :

Dynamic left ventricular outflow tract obstruction can independently result from various causes such as left ventricular hypertrophy, reduced left ventricular chamber size (dehydration, bleeding, or dieresis), mitral valve abnormalities, and hyper contractility (stress, anxiety, or isotropic agents such as dobutamine).[45]

7.2.7 Arrhythmias:

A life-threatening arrhythmia (e.g., ventricular tachycardia, ventricular fibrillation and total AV block) may be the first manifestation of ischemia. These arrhythmias may cause many of the reported sudden cardiac deaths in patients with acute coronary syndromes. Ventricular fibrillation or sustained ventricular tachycardia has been reported in up to 20% of patients. The risk of arrhythmic death in survivors of acute myocardial infarction is highest in the first six months after myocardial infarction and remains high for the subsequent two years. [46]

Anti arrhythmic agents are negatively isotropic and may encourage arrhythmias in acute coronary ischemia. Minor arrhythmias should not be treated.

7.2.7.1 Ventricular arrhythmias:

- Defibrillation should be administered for patients with ventricular fibrillation or pulse less ventricular tachycardia.
- Intravenous adrenaline (epinephrine) should be used (as a last resort) for patients with refractory ventricular tachycardia or ventricular fibrillation.[47]

7.2.7.2 Bradycardia, sinoatrial dysfunction or heart block:

- Sinus bradycardia may be due to drugs, ischemia or a vagal response.
- Atropine should be used for patients with symptomatic bradycardia.
- Temporary transcutaneous pacing should be initiated for patients not responding to atropine. Temporary transcutaneous pacing is only an interim measure until a more permanent method can be employed.[47]

7.2.7.3 Sinus tachycardia may be due to pain, anxiety, or drugs

7.2.7.4 Atrial fibrillation and other supraventricular tachycardia may also occur. Atrial fibrillation complicates 10-20% of AMIs but other supraventricular tachycardia is rare and usually self-limited.[47]

7.3 Thrombosis and embolic complications:-

- Deep vein thrombosis and pulmonary embolism are now relatively uncommon after infarction, except in patients kept in bed because of heart failure.
- Prophylactic doses of a low molecular weight heparin (LMWH) and compression stockings should be used for prevention.
- Treatment should be with therapeutic doses of LMWH, followed by oral anticoagulation for 3-6 months

.

Mural thrombosis and systemic embolism:

- Echocardiography may reveal intraventricular thrombi. Left ventricular thrombus occurs in 20% after infarction but in up to 60% of those after a large anterior infarction.
- The thrombus may be large and may be associated with embolization.
- The rate of thrombus formation is similar for patients treated with primary percutaneous coronary intervention when compared with patients currently treated conservatively or with thrombolysis.
  Left ventricular mural thrombus has not been shown to be related to increased intermediate-term mortality when patients are treated with warfarin.[48]

7.4 Pericarditis:

Pericarditis is most common following an anterior infarction. The incidence of early pericarditis after AMI is approximately 10%. Pericarditis usually develops between 24 and 96 hours after AMI. [37]

7.5 Dressler's syndrome:

Dressler's syndrome presents as pericarditis 2-5 weeks after AMI, often accompanied by pleural and pericardial effusions. The incidence is between 1% and 3 %.[39] Dressler's syndrome typically presents 2-5 weeks after a myocardial infarction with a self-limiting febrile illness accompanied by pericardial or pleural pain.

7.6 Depression:

- Significant depression occurs in about 20% of patients following myocardial infarction.
- Myocardial infarction increases the risk of suicide, and depression following myocardial infarction impairs the overall prognosis[49].

8. ACS nursing management:

8.1 Initial management of ACS:

The primary goals of immediate interventions are to decrease the myocardial workload while increasing the available oxygenation. Rapid identification and actions will minimize the damage caused to the heart muscle and reduce tissue necrosis. Chances of survival are increased and long-term disabilities are reduced if tissue necrosis can be prevented or minimized [(24)].

- Put patient in cardiac bed.
- Obtain vitals.
- Assess level of pain.
- Establish venous access.
- Administer medications (oxygen, nitroglycerin, aspirin, morphine, ACE-inhibitor)
- Draw laboratory samples.
- Perform ECG
- Connect cardiac monitor.

ECG:

- An ECG should be done within 10 minutes of admission so that the health care team can gather data to treat the patient.
- The ECG will help to confirm that the patient is experiencing any type of ACS.
- The ECG will also help to determine the location of the occlusion, which is crucial in predicting the prognosis and guiding the course of treatment.
- Classic presentation: The ECG reading for a patient with a MI often demonstrates inverted T wave. However, this pattern can also be seen in patients who formally had a MI.
- Presentation of an Anterior Wall MI: On a 12 led ECG of an Anterior Wall MI, the characteristic findings present with a ST-segment elevation MI (STEMI). There will be changes in leads V1 through V4 with a loss of the normal R-wave progression and reciprocal changes in the lateral leads [(50)].

8.2 Late nursing intervention:

- Assess level of pain after administration of drugs.
- Insert urinary catheter to minimize efforts.
- Re assurance of patient and provide information to patient family and support them.
- Restrict pt visits .
- Provide good nutrition for pt according to his situation.
- Prepare pt either for discharge or admission.
- Provide counseling about drugs, diet, rest and activity…

8.3 Discharge Preparation:

8.3.1 Medication Management

- The medication regimen the patient will need must be established prior to discharge
- The provider uses the nurse's assessment findings to determine what medications to prescribe.

8.3.2 Possible Medication Regimen

ACE Inhibitor: Maintains blood pressure within optimal range and promotes vascular health by reducing cardiac workload. This prevents (or slows down the process of) ventricular remodeling and reduces the risk of future cardiac events

- Beta-blockers: Maintains blood pressure within optimal range. Common beta-blockers include atenolol (Tenormin), pindolol (Visken), propranolol (Inderal), nadolol (Corgard), and metoprolol (Lopressor)
- Aspirin: A low daily dose reduces the risk of subsequent cardiac events
- A different ant platelet agent, such as clopidogrel (Plavix), may be given instead
- Nitroglycerin: A vasodilator that's ordered as a sublingual tablet for the patient to use at home in the event of chest pain related to angina
- Wilburton or Chantix: For smoking cessation, as smoking is a known risk factor for MI.**(24)**

8.3.3 Nursing counseling

Following a MI, it's important to educate patients on reducing preventable risks factors. This includes smoking cessation, weight control, and stress reduction, dietary changes, reducing LDL while keeping HDL high, and lowering blood pressure. The patient should understand the treatment regimen, such as how many times to take nitro before seeking health care provider **(50)**.

8.3.4 Diet:

Initially, keep the patient on nothing by mouth until his or her condition has been stabilized and treated. Following the patient's initial therapy and admission, a dietitian should instruct the patient regarding appropriate diet. A low-salt, low-fat, and low-cholesterol diet is generally recommended.

8.3.5 Activity:

Confine patients to bed rest to minimize oxygen consumption until reperfusion and initial therapy are complete. This usually lasts about 24-48 hours; after that, the patient's activity may be accelerated slowly as tolerated and as the clinical situation allows. Initiate cardiac rehabilitation prior to discharge.**(50)**

8.4 Transfer:

A study showed that the transfer of patients to an invasive-treatment center for primary PCI is superior to on-site fibrinolysis provided that the transfer can be accomplished within 2 hours. Transfer should be considered for those patients who are likely to benefit from PCI or cardiac surgery but who are in an institution where access to such interventions is not immediate. The benefits of transferring such a patient must outweigh the risks. Patients for whom transfer might be considered include the following:

- Patients with new or worsening hemodynamically significant mitral regurgitant murmurs
- Patients with known or suspected critical aortic stenosis and either ongoing ischemia or hemodynamic instability
- Patients who have received thrombolysis and fail to reperfusion
- Patients with significant LV dysfunction or cardiogenic shock in an aforementioned study by Cantor et al, a significant decrease in ischemic complications was observed in high-risk patients with STEMI who were treated with fibrinolysis and transferred for PCI within 6 hours following fibrinolysis [(51)].

CHAPTER -3

METHODOLGY

Study design and period:

In hospital descriptive-based study conducted in the period between (April – June 2018).

Study area:

This study will be conducted in Omdurman emergency and accidental military Hospital , which located in Khartoum state, Omdurman city, west blue Nile bridge, south youth and children palace, near to Aliaa hospital .it consist from three floor , ground floor it consist from(A,B)resuscitation and cardiology rooms (C1,C2)regular rooms and (trauma ,asthma) and cold clinic, minor theater ,laboratory and X-ray and CT- department ,And first floor which consist of medical and surgical word, and matron office and major theater, CCR(1).Second floor consist of VIP rooms, CCR(2), administration office and medical director office and in the roof we found cafeteria and archive files office.

ER hospital it received military patients and non military in case of emergency and also received war traumatic patients .

Study period: This study was covered the period from (10of April – 30July of 2017).

SETTING:

This study conducted in room(A,B)resuscitation and cardiology emergencies consist of 4senior staff nurse and one charge nurse per shift , two shift per day (12hours),and one resident doctor .both rooms are well prepared they consist of four emergency beds with monitors and suction machines,one sanitation pool , defebrilator ,emergency drug trolley ,equipment trolley ,file and paper cupboard,ambubag ,laryngeoscope,and all needed equipments for emergency interventions,ana bed side tables.

Pt arrive from triage who have a score under (9) or who arrived by typical chest pain , uncounsious pt, and arrested pt ,management started immediately in the rooms.

study population:

All nurses working in cardiac care unit & emergency department in the hospital under study.

Inclusion criteria:

- Nurses with bachelor degree.
- Nurses have experience of more than 6 months in cardiac care unit and cardiac emergency care units.

Exclusion criteria:

- Nurses who refused to participate.
- Nurses who were in duty of ―diploma.

Sample size & sampling:

Sample size: 60 nurses'

Sampling: total coverage

Data collection tools

The data was collected by a standardized close-ended questionnaire and check list . The close-ended questionnaire included 28 questions; every question had 4 options answers. The practice check list contained 18 criteria, classified into ―done and not done.

Variables under Study:

Un Dependant variables:(Age, sex, total work experience ,working area)

Dependant variables:(Knowledge about ACS, management of ACS ,nurses practice toward ACS pt).

Data collection technique:

The data of the questionnaire was fulfilled by the respondents. The check list will be filled by observing nurse's practice with informed respondents starting a when a patients hospitalized up to the first 4 hours at the CCU and ED. All data collection was managed by the writer. The data was collected during the period of April to June, 5 times per week, in the morning and noon shifts.

Data management and analysis:

Data was coded then inter by using simple statistical method and analyzed by using package (spss) program.

Ethical clearance:

- Ethical Approval was taking from Shandi University
- Verbal consent will be taken from participants.
- Research purpose and objectives will be explained to participant in clear simple words.

CHAPTER -4

The result

Table (2): Demographic data of nurses participant:

	Frequency	%
Age:		
Less than 25	8	13.3
26-30	25	41.7
31-35	22	36.7
More than 35	5	8.3
Gender:		
Male	11	18.3
Female	49	76.7
Qualification:		
Bachelor	52	86.7
MCs	8	13.3
PHD	0	0
Years of experience:		
0-1 year	17	28.3
1-5 year	34	56.7
6-10 year	7	11.7
More than 10 years	2	3.3
Working area:		
CCU	40	66.7
ED	20	33.3

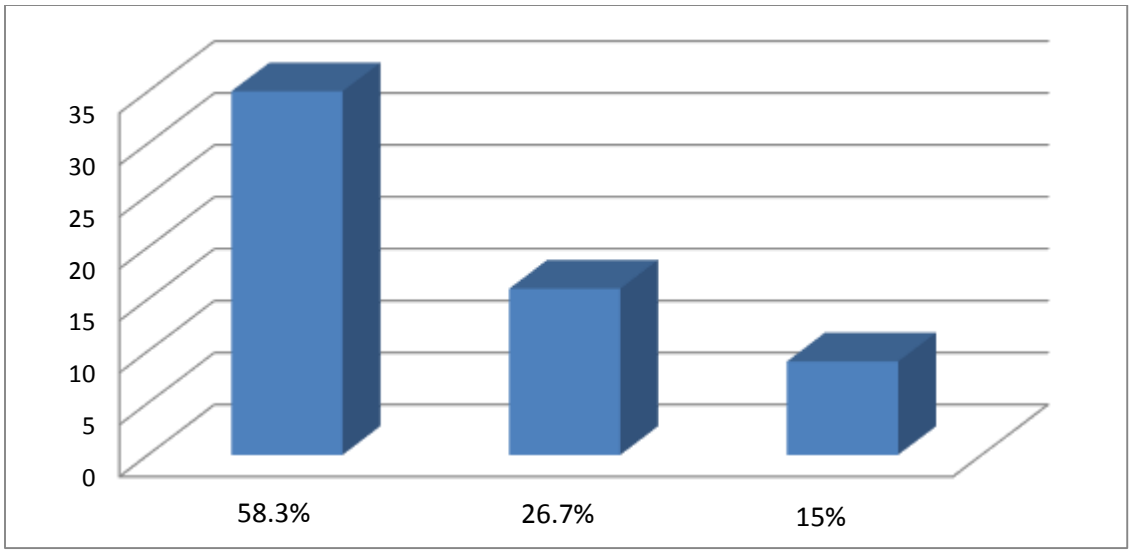

Figure (1):nurses Knowledge about nature of ACS

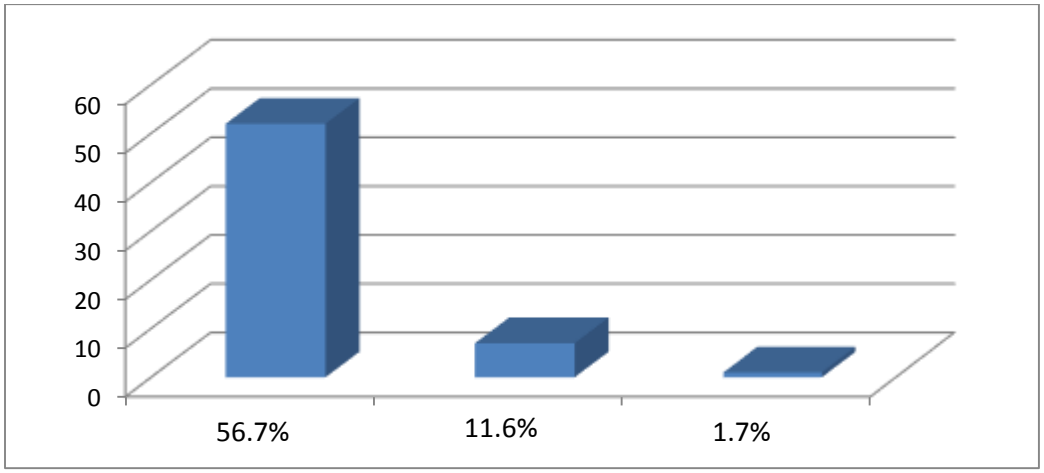

Figure (2):nurses knowledge about types of ACS

Table(3): nurses knowledge regard symptoms of ACS:

Level of knowledge regard symptoms of ACS	Frequency	%
Good	36	60
Fair	17	28.3
Poor	7	11.7
Total	**60**	**100**

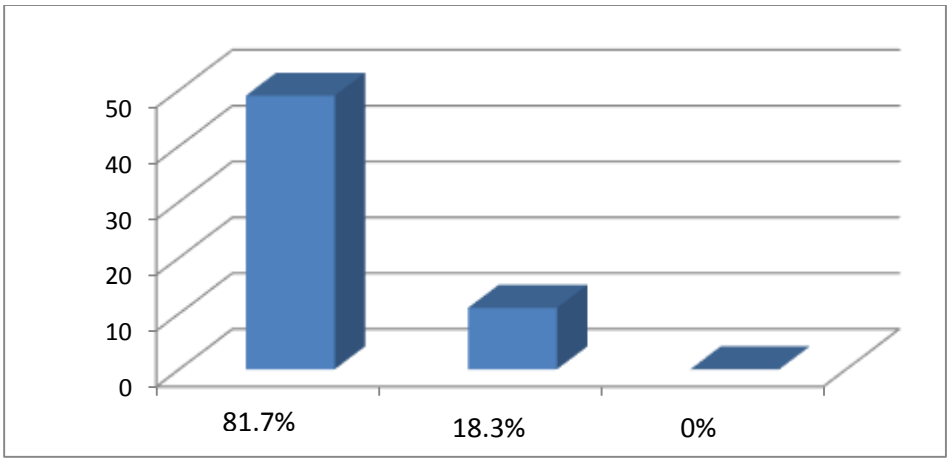

Figure (3):knowledge about risk factor of ACS

Table(4): assessment of nurses knowledge regarding diagnosis of ACS:

	Frequency	**%**
Diagnostic measure to confirm ACS:		
Good	38	63.3
Fair	15	25
Poor	7	11.7
Total	**60**	**100**
Totally occluded coronary artery on ECG reveals:		
Good	33	55
Fair	19	31.7
Poor	8	13.3
Total	**60**	**100**
Lab investigation that should be taken:		
Good	36	60
Fair	20	33.3
Poor	4	6.7
Total	**60**	**100**

Table (5): assessment of nurses knowledge regarding characteristic , radiation ,assessment and management of ACS chest pain:

	Frequency	%
ACS chest pain characterized by:		
Good	47	78.3
Fair	9	15
Poor	4	6.7
Total	**60**	**100**
The pain is radiated to:		
Good	51	85
Fair	8	13.3
Poor	1	1.7
Total	**60**	**100**
Assessment of pain includes:		
Good	29	48.3
Fair	24	40
Poor	7	11.7
Total	**60**	**100**
Pain management includes:		
Good	34	56.7
Fair	24	40
Poor	2	3.3
Total	**60**	**100**

Table(6): assessment of nurses knowledge regarding initial management and drugs when patients arrive to ED:

	Frequency	%
initial management when patients arrive to ED:		
Good	51	85
Fair	6	10
Poor	3	5
Total	**60**	**100**
Initial Drugs given when patient arrive to ED:		
Good	43	71.7
Fair	5	8.3
Poor	12	20
Total	**60**	**100%**

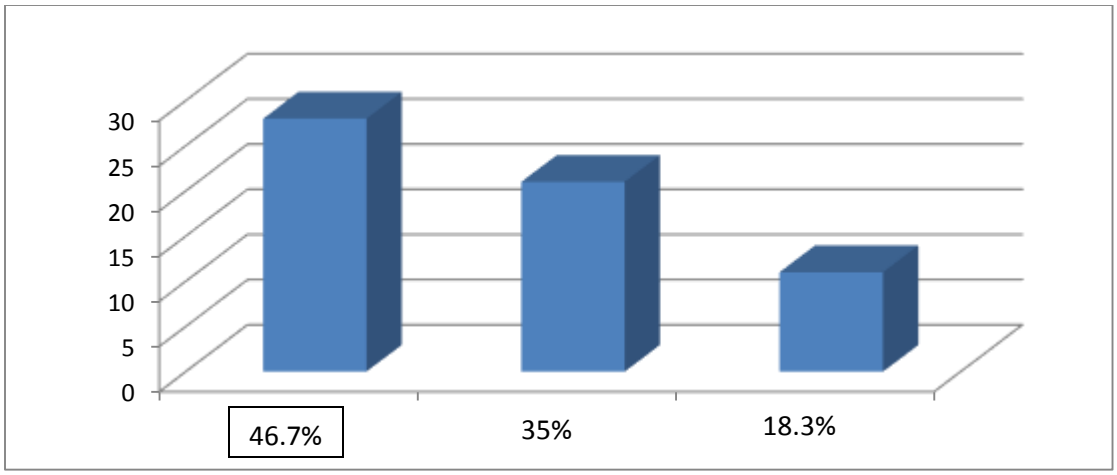

Figure (4):nurses knowledge about typical indication for thrombolysis in pt with ACS

Table(7): assessment of nurses knowledge regarding other management of STEMI pt:

	Frequency	**%**
Good	31	51.7
Fair	24	40
Poor	5	8.3
Total	**60**	**100**

Table(8): nurses knowledge regarding other drugs given to ACS patient:

Knowledge regarding drugs	**Frequency**	**%**
Good	42	70
Fair	15	25
Poor	3	5
Total	**60**	**100**

Table (9): assessment of nurses knowledge in regard to drugs side effect:

	Frequency	%	
Streptokinase side effect:			
Good	34	56.7	
Fair	14	23.3	
Poor	12	20	60 (100%)
Nitrate side effect:			
Good	18	30	
Fair	29	48.3	60 (100%)
Poor	13	21.7	

Table(10):assessment of nurses knowledge regarding importance of vital sign:

Knowledge regarding v\s	**Frequency**	**%**
Good	44	73.3
Fair	12	20
Poor	4	6.7
Total	**60**	**100**

Table(11): nurses knowledge regard assessment of cardiac output:

Assessment of cardiac out put	**Frequency**	**%**
Good	24	40
Fair	9	15
Poor	27	45
Total	**60**	**100**

Table(12): assessment of nurses knowledge in regard to complication of ACS:

	Frequency	%
Complication of ACS:		
Good	31	51.7
Fair	26	43.3
Poor	3	5
Total	**60**	**100**
Complication of ACS observed on monitor:		
Good	29	48.33
Fair	17	28.33
Poor	14	23.33
Total	**60**	**100**

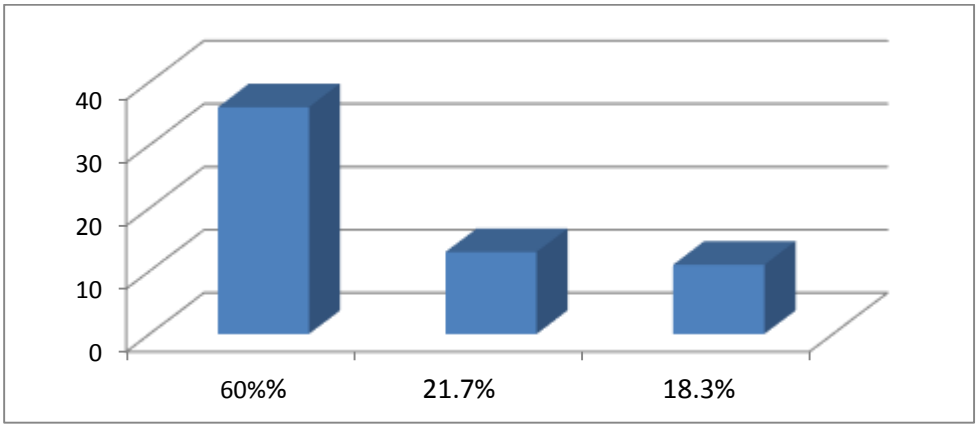

Figure (5): nurses knowledge to ward precaution of transferring ACS patient

Table (13): Evaluating of practice regarding initial nurses care towards acute ACS patients, among nurses working in the CCU and ED(N=60)

	Done		Not Done		Total
	Freq	%	Freq	%	
Initial management :					60(100%)
Interaction when patient arrive	28	46.7	32	53.3	
Put in cardiac bed	18	30	42	70	
Check vital signs	35	58.3	25	41.7	
Insert IV access and drawing samples	50	83.2	10	16.7	
Connect monitor	47	78.3	13	21.7	
Perform ECG	41	68.3	19	31.7	
Administer of oxygen	25	41.7	35	58.3	
Give prescribed drugs (aspirin ,clopderil,nitrate)	54	90	6	10	
Give pain killers	52	86.7	8	13.3	

Table(14): Evaluating of practice regarding late management of acute ACS patients, among nurses working in the CCU and ED(N=60)

Criteria	F	%	F	%	Total
Insert urinary catheters	15	25	45	75	60(100%)
Give thrombolysis	39	65	21	35	
Assessment of pain level before giving pain killer	13	21.7	47	78.3	
Assessment of pain level after giving pain killer	26	43.3	34	56.7	
Connect monitor and reassess the patient	45	75	15	25	
Reduce exertion of the patient	41	68.3	19	31.7	
Check serial investigation	40	66.7	20	33.3	
Psychological support and education for the family	39	65	21	35	
Documentation	38	63.3	22	36.7	

Table (15):correlation between qualification and knowledge about ACS nature ,symptoms ,initial management ,and typical indication for thrombolytic:

		Nature of ACS	symptoms of ACS	Initial Management	Typical Indication for thrombolysis	Total
Qualification	**Freq**	26	22	16	4	68
	%	43.3%	36.7%	26.7%	6.67%	113.37
Correlation		-0.684	0.138	0.096	0.018	0.432

P. value 0.0086

Table (16):correlation between years of experience and nurses practice regarding initial , and late management for a pt with ACS:

Evaluation of management		Initial management	Late management	Total
Years of experience	**Freq**	56	24	80
	%	93.3	40	133.3
Correlation		0.02	0.013	0.033

p-value=0.14

CHAPTER -5

The discussion

The study was conducted at cardiac care unit and emergency department in the Military hospital (Omdurman)among 60 nurses, and they were assessed for their knowledge and practices towards initial management of patient with acute coronary syndrome.

Age distribution showed that less than half of nurses(41.7%)have age between 26-30 year, More than two third of them were a female(76.7%)This is compatible with findings reported by Elbashir H and colleagues in Sudan, who found that, females represented (84%) while males represented (16%) [52]. majority of nurses(86.7%)having a bachelor degree, And two third of them(66.7%) are working in CCU . As shown in table(1).

more than half nurses(58.3%)have a good knowledge about ACS nature .as demonstrated in figure(1) and its significant with their qualification with p-value(-0.684) as shown un tablec(14).

assessment of knowledge regarding types of ACS the majority was for nurses who revealed a good knowledge about it(86.7%),as Shown in figure(2).

More than half of nurses(60%), in study shows good knowledge about symptoms of ACS ,with a p-value(0.138)which means that there is no signification between qualification and knowledge about ACS symptoms as shown in table (14). Knowledge about symptoms enabling early diagnosis and accordingly early management as well as it prevents complications and safe patients' life. This disagrees with findings reported by Newens AJ1, and colleagues earlier who have shown that, the percentage of nurses who correctly estimated the incidence of symptoms was low(25%)[53]. as shown in table(2).

the majority of nurses (81.7%)reflect good knowledge about risk factor of ACS, This finding was higher than the rate of knowledge among nurses reported by Steffeninog et al in Italy in which risk factors were answered by only15% of staff nurses [54]. as demonstrated in figure(3).

The study shows that more than half(63.3%)nurse revealed good knowledge about diagnostic measures .more than half of them(55%)shows a good knowledge about that totally occluded coronary artery reveal STEMI on ECG.(60%) above to half of nurses have a good knowledge about the important lab investigation that should be taken , This is higher than degree of knowledge shown in the Indian study in Madurai which reported that knowledge regarding electrocardiogram among bachelor degree nurses was poor (33.3%) [55]. as illustrated in table(3).

More than two third(78.3%)nurse have good information about characteristic of ACS chest pain , While the majority(85%)of them gets good knowledge regarding radiation of it .In another hand less than half of them(48.3%)shows a good knowledge about how to assess the chest pain, And greater than half of nurses(56.7%)have a good knowledge about management of pain it, A similar finding were reported in Uganda which showed that, majority of the participants (91.2%) had a good knowledge [56]. as shown in table(4).

Regarding information about initial management and drugs for a patient with ACS the study found that there is no signification between it and nurses qualification with a p-value(0.096)as shown in table(14), The majority of nurses(85%) have a good information about

it, While more than two third of them(71.7%)shows a good information about initial drugs that given to ACS patient immediately . as demonstrated in table(5).

Regarding knowledge about typical indication for thrombolysis there is high signification between it and qualification of the nurses with a p-value(0.018)as demonstrated in table(14), and the study shows that (46.7%)below the half of nurses reveals a good knowledge about it .Administration of thrombolysis usually depend on doctor's instructions. as reflected in figure(4).

In regard to knowledge about other management that can be carried out for an ST-EMI patient more than half of them(51.7%)having a good knowledge. as shown in table(6).

Out of total (70%)of nurses have a good information about other drugs that can be given to ACS patient . However studies conducted in Turkey have shown that nurses in general do not satisfactorily meet this responsibility [57]. as demonstrated in table(7).

In regard information about drugs side effect: more than half of nurses(56.7%) shows good information about streptokinase side effect, While less than half of nueses(48.3%) having a fair knowledge about it. As shown in table (8).

To ward assessment of vital sign need to be checked for ACS patient ,more than two third of nurses(73.3%)shows a good knowledge, as shown in table(9).

less than half of nurses(45%) which represent the major participant have a poor knowledge in assessment of cardiac output .An study conducted in Turkey have shown that nurses do not satisfactorily meet this responsibility [57]. as illustrated in table(10).

Regarding complication of ACS:above the half of nurses(51.7%)shows good information about it , While less than half of them (48.33%) demonstrate good knowledge about ACS complication that revealed on the cardiac monitor. as shown in table(11).

Less than two third of nurses(60%) have a good knowledge about precautions of ACS patient transferring . as shown in figure(5)

In regard to assessment practice of initial management toward ACS patient it was found that there is high signification between practice and nurses experience with p-value (0.02) as shown in table (15) , more than half nurses (53.3%) having a good interaction with pt , And more than two third of them (70%) are poor in performing cardiac bed to the patient , in another hand more than half (58.3%) have good practice in vital signs checking , And majority of nurses (83.3%)perform good practice in cannulation and drawing blood samples , While more than third (68.3%)of nurses have good practice regard ECG performance, And more than half of the participates (58.3%)shows poor performance in oxygen administration, while the majority of them(90%) are good in giving prescribed medication ,also the majority of nurses participant (86.7%)are good in administration pain killers to ACS pt. As shown in table(12).

While assessing nurses performance regarding late management of ACS patient p-value was (0.013) between it and years of experience which means that we have a relationship between them as shown in table (15), And the study shows that more than two third(75%)nurse are poor in inserting catheter, less than two third(65%) nurses are good in giving thrombolysis as order , more than two third of them(78.3%)poor in assessment of pain in ACS pt , And more than

half (56.7%)also have poor practice in assessing ACS pain after administration of pain killer ,And more than two third (75%)nurse have a good practice in connecting pt with monitor . While more than two third (68.3%)of nurses are good in reducing pt exertion ,Also two third of them (66.3%) perform good practice in checking serial investigation of ACS pt, And less than two third are good in performing psychological support for pt and pt family, also less than two third of nurses(63.3%)revealed good practice toward documentation of information . The study by Kizza IB in Uganda showed poor performance in different aspects of practice; he reported that, nurses in this study had lack of education on assessment tools with percentage of 82.4%, poor documentation of pain assessment and management was (77.6%) [56]. as shown in table(13)

Conclusion:

According to findings of the current study, it was concluded that, the total evaluation of knowledge was found good representing estimated by 66.9% , while the total evaluation of practice was found moderate with percentage of 60%, suggesting that, practice was incompatible with the knowledge

The study showed higher percentage of knowledge regarding ACS nature, risk factors, and knowledge regard initial management which identified by vast majority, while they showed less knowledge towards symptoms ,diagnosis, pain assessment, initial drugs and drug side effect, and thrombolysis indication which were moderate and good respectively. They showed poor performance regarding assessment of cardiac output.

Also, the study showed very good practice in connecting monitor, checking vital signs and , but they showed poor practice the aspect of interaction when patient arrive to ED, oxygen administration, giving pain killers and thrombolysis, assessment of pain level ,inserting catheter to reduce exertion of pt .

Recommendations

- Routine supportive supervision of nursing staff working in the coronary care unit (CCU) and Emergency department (ED) units after training and focusing on medication and practice (inserting urinary catheters, reassurance and psychological support for patient and family and educating patients.).
- Educational sessions are necessary to improve the nurse's ability dealing with management with such cases.
- Increase the number of training sessions to nurses as general and special training session to nurse work in cardiac unit and emergency care unit , with encouraging nurses with good level of knowledge to join the cardiac units
- Further studies are needed to elaborate the effects of different variables on the knowledge and practice of nursing.

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Appendix

Questionnaire

Nurse's competence regarding initial management of patient with acute coronary syndrome

Note: please put ( ) in the right answer, and (X) in wrong answer,

Many questions require more than one correct answer to be chosen

……..

No.: …….

1. Age:	less than 25		26-30	
	31-35		More than 35	
2. Gender:	Male		Female	
3.qualificatons:	bachlor		MCs	
4. Years of experience:	0-1years		1-5years	
	6-10years		More than 10 years	
5. Working area:	CCU		ED	

6.nature of ACS:

1-ACS begins when a disrupted atherosclerotic plaque in a coronary artery stimulates platelet aggregation and thrombus formation. ☐

2-Most cases of infarction are due to the formation of an occluding thrombus on the surface of the plaque. ☐

3-During the ischemic phase, cells exhibit both aerobic and anaerobic metabolism ☐

4-If myocardial perfusion continues to decrease, aerobic metabolism ceases and eventually anaerobic metabolism will be significantly reduced. This period is known as the injury phase. ☐

7-types of ACS:

1-unstable angina ☐

2- non–ST-segment elevation myocardial infarction ☐

3-ST-segment elevation myocardial infarction. ☐

4- Angina ☐

8. Symptoms of ACS include:

1. Chest pain ☐

2. Epigastria pain ☐

3. Shortness of breath ☐

4. Sweating ☐

9- Risk factors of ACS include:

1. Smoking ☐

2. Hyperlipidemia & Obesity ☐

3. Hypertension ☐

4. Family history ☐

10. The ACS chest pain characterized by:

1. crushed ☐

2. Prolong ☐

3. Heaviness ☐

4. Severe ☐

11. The pain is radiated to:

1. Arm and shoulder ☐

2. Neck ☐

3. Back and epigastria ☐

4. Posterior intrascapular area ☐

12. Assessment of pain includes:

1. Duration ☐

2. Location and pain radiation ☐

3. Pain nature

4. Associated manifestations

13. Pain management includes:

1. Put patients in comfortable position

2. Give IV morphine

3. Supplemental oxygen

4. Nitroglycerines (GTN)

14. The diagnostic measures to confirm ACS include:

1.ECG

2. Cardiac enzymes

3. Angiography

4. Echocardiography

15.totally occluded coronary artery on ECG reveals:

1.STEMI

2.NSTEMI

3.un stable angina

4.i don't know

16- Complications of ACS include:

1. Cardiogenic shock

2. Heart failure

3. Arrhythmias

4. Cardiomegaly

17. Initial management when patients arrive to ED:

1. Put patient in cardiac bed

2. Insert IV lines and take investigations

3. Administration of oxygen therapy ☐

4. Connect monitors an ECG ☐

18.initial Drugs given when patient arrive to ED:

1. Aspirin , Clopidogrel & Nitrate ☐

2.statin ☐

3. Morphine and antiemetic ☐

4. Thrombolytic (Streptokinase & heparin) ☐

19. Side effects of streptokinase include:

1. Bleeding ☐

2. Hypotension ☐

3. Skin rash ☐

4. Drowsiness ☐

20. Nitrate side effects:

1. Flushing ☐

2. Tachycardia ☐

3. Postural hypotension ☐

4. Headache ☐

21. Typical indication for immediate thrombolytic therapy :

1. Presentation within 12 hrs of onset of chest pain ☐

2. STEMI ☐

3.ST depression MI ☐

4. New onset LBBB (Left Bundle Branch Block) ☐

22.Other management for STEMI:

1. Re-perfusion therapy ☐

2. Immediate *Percutaneous coronary intervention* (PCI) ☐

3. Rescue Angioplasty ☐

4. Cardiac pacing ☐

23. The most important vital signs need to be checked for ACS patient include:

1. Blood pressure and Pulse rate ☐

2. Oxygen saturation ☐

3. pain ☐

4. I don't know ☐

24.Lab investigations should be taken:

1. Lipid profile ☐

2.Bleeding profile ☐

3. Renal profile ☐

4.troponin serum ☐

25. Other drugs can be given to MI patient include:

1. Beta blockers ☐

2. ACE inhibitors ☐

3. Anti-arrhythmias ☐

4. Anticoagulant ☐

26. Assessment of cardiac output can be done through:

1.Heart beats ☐

2. Blood pressure ☐

3. echocardiography ☐

4.i don't know ☐

27. The complications ACS observed On-monitor:

1. Arrhythmias ☐

2.hypotension ☐

3. cardiac arrest ☐

4.i don’t know ☐

28.precaution during transferring ACS patient:

1.ambulance ☐

2.couch ☐

3.wheel chair ☐

4.i don’t know ☐

Check List

Assessment of nurse's practice in ER and CCU

Initial management of pt with ACS:	**DONE**	**NOT DONE**
Interaction when patient arrive		
Put in cardiac bed		
Check vital sign		
Insert IV access		
Draw blood samples		
Connect monitor		
Per form ECG		
Administer of oxygen		
Give prescribed drugs(aspirin ,clopidogrel ,nitrate)		
Give pain killers		
Late management of pt with ACS:		
Insert urinary catheters		
Given thrombolysis as order		
Assessment of pain level before give medication		
Assessment of pain level after give medication		
Connect monitor and reassess the patient		
Reduce exertion for patient		
Check of serial investigation		
Psychological support and education for the family		
Documentation		