International Journal of General Medicine & Surgery

Available Online: https://ijgms.edwiserinternational.com

A Prospective Observational Study on Management of Acute Coronary Syndrome in A Multi-Specialty Hospital

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Article info

Received 11 October 2019
Revised 08 November 2019
Published 15 November 2019

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Abstract

Introduction: Cardiovascular diseases are the leading cause of death in industrialized countries and are expected to become so in emerging countries by 2020. Among this coronary artery disease is the most prevalent cardiovascular disease. ACS may be defined as all clinical syndromes compatible with acute myocardial ischemia resulting from an imbalance between myocardial oxygen demand and supply. ACS comprises of NSTEMI, STEMI, and unstable angina.

Methods: This is a prospective observational study conducted in MAXCURE hospital Hyderabad. Cases were collected who were diagnosed with ACS from inpatients of cardiology department in duration of 6 months.

Results: A total of 198 cases of ACS were observed. Among them 61 cases of NSTEMI, 67 cases of STEMI, 70 cases of unstable angina. It was more common in men when compared to women and was found to be more prevalent in patients under the age of 55. Most common medical history of patients was angina and hypertension. All patients underwent surgery either CABG and PTCA. The drugs used were aspirin, clopidogrel, beta blockers, statins, nitrates, ACEI, heparin.

Conclusion: ACS is one of the most common coronary diseases. The main symptoms includes chest pain and shortness of breath grade II and III. Maximum patients suffer from unstable angina followed by STEMI and then by NSTEMI. The age group most affecting is less than 55. Males are at more risk than females. The common history of patients include angina, hypertension, diabetes and smoking. All the patients arriving at emergency will be prescribed with aspirin, beta blockers, nitrates, statins, clopidogrel and heparin. The use of calcium channel blockers, ACEI, thrombolytics, warfarin, LMWH, is less or almost nil.

Keywords: Acute coronary syndrome; Non-ST elevated myocardial syndrome; ST elevated myocardial syndrome; Unstable angina.

Introduction

Cardiovascular diseases are currently the leading causes of death in industrialised countries and are expected to become so in emerging countries by 2020. Among this coronary artery disease is the most prevalent manifestation and is associated with an increased mortality and morbidity [1].

Acute coronary syndrome includes all clinical syndromes compatible with acute myocardial ischemia resulting from an imbalance between myocardial oxygen demand and supply. The most common symptom is chest pain, often radiating to left shoulder or angle of jaw associated with nausea and vomiting. Many people with acute coronary syndrome also present with symptoms like chest pain, particularly in

women, older patients, and patients with diabetes mellitus [2].

Classification

ACS is classified according the electrocardiographic [ECG] changes into

1. ST segment elevation ACS
2. Non-ST segment elevation myocardial infarction
3. Unstable angina

Definition

STEMI: A clinical syndrome defined by characteristic symptoms of myocardial ischemia in association with persistent ECG ST elevation and subsequent release of biomarkers of myocardial necrosis [3].

NSTEMI: It is an acute process of myocardial ischemia with enough severity and duration to result in myocardial necrosis [4].

Unstable angina: An unprovoked episode of chest pain raising suspension of acute myocardial infarction [4].

Epidemiology

Registry data consistently show that NSTE-ACS is more frequent than STE-ACS.4 The annual incidence is 3 per 1000 inhabitants, but varies between countries.5 Hospital mortality is higher in patients with STEMI than among those with NSTE-ACS (7% *vs*. 3–5%, respectively), but at 6 months the mortality rates are very similar in both conditions (12% and 13%, respectively).Long-term follow-up showed that death rates were higher among patients with NSTE-ACS than with STE-ACS, with a two-fold difference at 4 years.8 This difference in mid- and long-term evolution may be due to different patient profiles, since NSTE-ACS patients tend to be older, with more co-morbidities, especially diabetes and renal failure. The lessons from epidemiological observations are that treatment strategies for NSTE-ACS not only need to address the acute phase but with the same intensity impact on longer term management.

Etiology [5]

Atherosclerosis, Congenital, Embolic, Vasospasm, Trauma, Arteritis, Compression, Drugs, Aortic dissection, Intimal proliferation

Risk factors

History of CAD, Family history of CAD, High blood pressure, Age [Male>33 years, Female>40 years], Blood vessel disease, Diabetes, Smoking, Overweight, High cholesterol, Menopause, No exercise [6].

Pathophysiology [7]

1. Atherosclerosis with superimposed coronary thrombosis.
2. Slowly growing high-grade stenosis can progress to complete occlusion but do not usually precipitate acute STEMI collateral circulation.
3. During development of plaques, abrupt transition can occur, resulting in

a) Platelet activation
b) Thrombin generation
c) Thrombus formation

4. Blood flow occlusion leads to imbalance between supply and demand and could lead to myocardial necrosis
5. Patients with non- transmural infarction more likely to have more significant stenosis in infarct related artery [IRA].
6. Less severe stenosis with lipid laden plaques and fragile caps more likely to rupture and causing thrombosis and STEMI.
7. Initial consequences vary with size, location and duration of obstruction and range from transient ischemia to infarction.
8. Measurement of newer, more sensitive markers indicates that some cell necrosis probably occurs even in mild forms

Myocardial dysfunction [8]

Ischemic [but not infarcted] tissue has impaired contractility and relaxation, resulting in hypo kinetic or a kinetic segment. These segments may expand or bulge during systole [called paradoxical motion]. The size of the affected area determines effects which range from minimal to mild heart failure to cardiogenic shock. Usually large parts of myocardial must be ischemic to cause significant myocardial dysfunction. Some degree of heart failure occurs in about two thirds of hospitalized patients with acute myocardial infarction. It is termed ischemic cardiomyopathy if low cardiac output and heart failure persist. Ischemic involving the papillary muscle may lead to mitral valve regurgitation. Dysfunctional wall motion can allow mural thrombus formation.

Myocardial infarction

It is myocardial necrosis resulting from abrupt reduction in coronary blood flow to part of the myocardium. Infarcted tissue is permanently dysfunction. However, there is a zone of potentially reversible ischemia adjacent to infarct tissue. MI affects predominantly the left ventricle [LV] but damage may extend into the right ventricle or the atria.

Infarction may be as follows:

Transmural: Transmural infarcts involve the whole thickness of myocardium from epicardium the endocardium and are usually characterize by abnormal Q wave on ECG.

Non transmural: Non transmural infarcts do not extend through the ventricular wall and cause only ST segment and T wave abnormalities. Because the transmural depth of necrosis cannot be precisely determined clinically, infarcts are usually classified as STEMI or NSTEMI by the presence or absence of ST segment elevation or Q wave on the ECG (Figure 1).

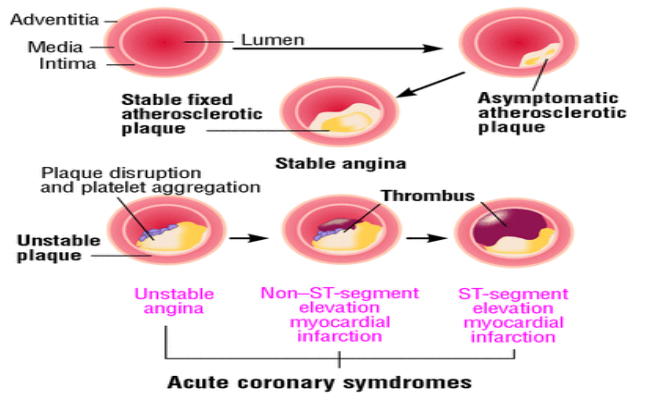


Figure 1: Pathophysiology of Acute Coronary Syndrome [9].

Clinical presentation [10]

The usual symptom of acute coronary syndrome is chest pain. Traditionally, several clinical presentations have been distinguished:

1. Prolonged (20 min) anginal pain at rest;
2. New onset (de novo) angina (Class II or III of the Classification of the Canadian Cardiovascular Society11);
3. Recent destabilization of previously stable angina with at least Canadian Cardiovascular Society Class III angina characteristics
4. Post-MI angina.

General

Chest pain / chest discomfort for at least 20 minutes, Indigestion, Fainting, Nausea and vomiting, Pain in arms, shoulder, neck, back and jaw, Shortness of breath, Sweating, weakness

Diagnosis

Physical examination: They are relatively nonspecific. The heart rate and blood pressure may be elevated due to increased sympathetic tone or blood pressure may be low due to cardiogenic shock depending on extent of the infarction. S4 heart sound may be present due to impaired left ventricular relaxation. S4 sound occurs when non-compliant stiffened left ventricle is not able to relax adequately when it receives blood during atrial contraction. S4 sound is the blood itself striking the non-compliant ventricle [11]. Three major ways to diagnose ACS are:

1. ECG
2. Cardiac biomarkers
3. Coronary angiography

ECG [12]

a. NSTEMI

A 12 Lead ECG should be performed and interpreted within 10 minutes of patient's arrival at an emergency facility to assess for cardiac ischemia or injury. The changes in the ECG for NSTEMI include ST depression, transient ST elevation, or new T wave inversion. Persistent ST elevation or anterior ST depression indicates true posterior myocardial infarction (Figure 2).

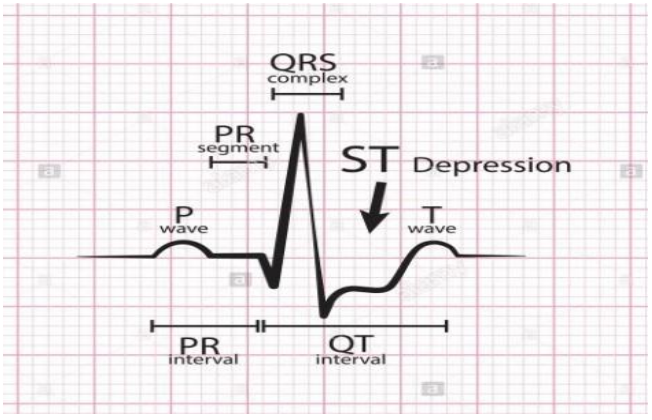


Figure 2: ECG of NSTEMI Description [13].

b. STEMI

The elevation of the ST segment in ECG even may be due to new left bundle branch block equivalent to STEMI. The primary changes observed are hyper acute T waves appear peaks and are related to local hyperkalaemia. These are not seen in ECG as they occur prior to hospital arrival. Anterior STEMI 2mm ST elevation in V2 V3 in men.1.5mm ST elevation in V2 V3 in women and people aged above 40 years

Although ECG is the mainstream diagnostic test for STEMI the above changes are abnormal in the following conditions

1. Left ventricular hypertrophy
2. Early repolarization
3. Pericarditis
4. Left ventricular aneurysm

Unstable angina

It is not diagnosed by only by ECG. When the changes in the ECG are like NSTEMI, it is followed by cardiac biomarkers to confirm unstable angina

Cardiac biomarkers [10]

Myoglobin

It is an enzyme which will be released into the blood when there is damaged muscle tissue. This is produced in skeletal muscles making it nonspecific marker for myocardial infarction. However, there will be a benefit if there is an detectable increase within 30minutes of injury.

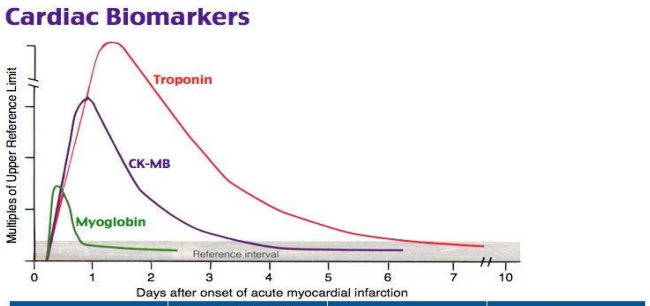
Troponin

Two types of troponin I and T. These are present in the cardiac myocyte. They will be released after 3-4 hours of injury. They will be in the blood for 10hours. This makes it difficult to identify if there was any refraction. But it is more specific than myoglobin and CK MB

Creatinine kinase

It has 2 isoenzymes muscle brain and is classified into MUSCLE and BRAIN

This is further divided into MM, MB, BB. In all the three MB is related to cardiac muscle. It will increase within 3-4 hours after injury and will be in blood for 3-4 days (Figure 3).



	Time Detectable from Onset of Ischemia	Peak	Return to Baseline
Troponin T & I	3-12 hrs	24-48 hrs	5-14 days
Creatine kinase (CK)	3-12 hrs	24 hrs	48-72 hrs
Myoglobin	1-2 hrs	8-10 hrs	1-2 days

Figure 3: Cardiac biomarkers [14]

Treatment of acute coronary syndrome

General recommendations

On admission of patient to hospital there are following recommendations.

1. Oxygen supply [if SpO2 decreased by 90%]
2. ST segment monitoring [arrhythmias or ischemia]
3. Glycemic control
4. Check vitals and bed rest [12 hours]
5. Blood chemistry tests

a) K+, Mg +2 [heart rhythm]
b) Glucose [risk of mortality and morbidity]
c) Serum creatinine
d) CBP, coagulation tests [bleeding risk]
e) Serum cholesterol level with 24 hours of hospitalization

For ST elevated ACS

There is a high of death and we should reestablish coronary perfusion immediately.

For NonST elevated ACS and unstable angina

The risk of death is comparatively low; but may be high depending on increased risk of the patient. When there is a low risk of death, we obtain serial biomarkers and TIMI [thrombolysis in MI] score. When biomarkers are negative, we monitor the ECG. We perform stress test and then discharge with management. But when the biomarkers are positive and there is high risk of death, we perform coronary angiogram within 24-48 hours and find the coronary stenosis. We perform the revascularisation procedures.

Treatment for stemi

All patients with STEMI and without contradiction should receive within the first day of hospitalization and preferably in the emergency department.

1. Intranasal oxygen [If oxygen saturation is low].
2. Sublingual nitroglycerin.
3. Aspirin.
4. P2Y12 platelet inhibitor.
5. Anti-coagulation with bivalirudin, unfractionated heparin [UFH] or enoxaparin.

Administer a Gp 2b or 3a inhibitor with UFH to patient and undergoing primary PCI give BETA blockers IV and IV nitroglycerin to select patients.

Oral beta blockers the first day in patient without cardiogenic shock

administer morphine to patients with refractory angina as an analgesic and vasodilator that lower preload.

Start ACE inhibitor within 24 hours in patient who have either AWMI or LVEF of 40% or less and no contradiction to

1. Fibrinolytic therapy
2. Aspirin
3. Platelet p2y12 inhibitor
4. Glycoprotein IIb or IIIa receptor inhibitor
5. Anticoagulants
6. Beta adrenergic blockers
7. Statins
8. Nitrates
9. Calcium channel blockers

Treatment for NSTEMI

Early pharmacotherapy for NSTE ACS is similar to STE ACS. In absence of contraindication, treat all patients in the emergency department with

1. Intranasal O_2.
2. NTG.
3. Aspirin.
4. Anticoagulant (UFH, Enoxaparin, fondaparinux).

High risk patient should proceed to early angiography and may receive a GpIIb/IIIa inhibitor. Patients can be administered with a p2y12 inhibitors like clopidogrel. Give IV beta blocker and IV NTG to selected patients. Initiate oral Beta blocker within the first 24 hrs in patients without cardiogenic shock. Give morphine to patient with refractory angina as described previously.

Fibrinolytic therapy should never be administered in patients with NSTEMI.

Aspirin

Aspirin reduces the risk of death or MI by approximately 50% compared with no antiplatelet therapy in patients with NSTEMI.

DOSE: Loading Dose – 162 – 325mg

Maintenance Dose – 75-162mg

Anticoagulant therapy

For patients treated by an early invasive approach with early coronary angiography and PCI administer UFH, enoxapirin. If an initial conservative strategy is planed enoxapirin, UFH or low dose Fondaparinux is recommended. Continue the therapy for at least 48 hrs for UFH, until the patient is discharged from hospital for either enoxapirin or fondaparinux, and until end of PCI or angiography procedure.

DOSE of UFH: 60units/kg IV bolus (max. 4000 units) followed by a continuous IV infusion of 12 units/kg/hr(max. 1000units/hr).

P2y12 Inhibitor

On the first day of hospital admission a loading dose of 300-600 mg should be administered followed by the next day with a maintenance dose of 75 mg per day orally. Clopidogrel plus aspirin combination can be added with GpIIb/IIIa in patients undergoing PCI. Following PCI use clopidogrel up to 12 months, or at least for 1 month when bear metal stent, or at least for 12 months for DES (sirolimus / paclitaxel stent). For patients undergoing CABG stop clopidogrel for at least 5 – 7 days before procedure.

Glycoprotein 2b3a receptor inhibitor

The role of this drug in NSTE ACS is diminishing as p2y12 inhibitor used earlier and bivalirudin is often selected as the anticoagulant. Routine administration of eptifibatide (added to aspirin + clopidogrel) prior angiography and PCI in NSTEMI does not reduce ischemia events and increase bleeding risks.

Nitrates

Administer SL NTG followed by IV NTG to patients with NSTEMI and ongoing ischemia, heart failure or

uncontrolled hypertension. Continue IVNTG for approximately 24 hrs after ischemia relief.

Beta blockers

In absence of contraindication, administer oral beta blocker to all patients with NSTEMI within 24 hrs of hospital admission. Continue beta blocker indefinitely in patients with LVEF of 40% or less and for patients with normal LV dysfunction.

Calcium channel blockers

Calcium channel blockers should not be administered to patients with ACS as they may increase heart rate increasing the risk of death of the patient.

Treatment of unstable angina

Patients with unstable angina should be given the following [Unless contraindicated]
Anti-platelets: aspirin, clopidogrel, or both
Anti-coagulants: heparin, it may be UFH or LMWH, or bivalirudin.
Glycoprotein IIb/IIIa inhibitor specially for high risk patients
Anti-angina therapy specially nitroglycerin.
Beta blocker.
ACE inhibitor.
Statin.

Surgical management of acute coronary syndrome
In general, there 2 major surgeries for ACS are CABG. PTCA.

CABG [12]

Coronary artery bypass grafting or simply called as a bypass surgery is an open heart surgery that improves blood flow to heart.

Unstable angina and NSTEMI

- ✓ It is recommended for patients with
- ✓ Significant left main coronary artery disease (>50% stenosis)
- ✓ Double vessel disease (DVD)
- ✓ Triple vessel disease (TVD)
- ✓ Abnormal LV function

STEMI

Mechanical circulatory support is reasonable in patients with STEMI who are hemodynamically unstable and require urgent CABG.

Emergency CABG within 6 hrs. of symptoms on set maybe considered in patients with STEMI who don't have cardiogenic shock and are not recommended for PCI or fibrinolytic therapy.

Number of Bypass

Double Bypass–Means Two coronary Arteries are bypassed (Left Anterior Descending (LAD) CA and Right Coronary Artery (RCA)

Triple Bypass–Means Three Arteries are bypassed (ex-LDA, RCA, Left Circumflex Artery(LCX)

Quadruple- Means Four Vessels are bypassed (ex-LDA, RCA, LCX, First Diagonal Artery of the LAD)

Purposes

1. Restore Blood flow to the heart.

2. Relieves Chest pain and ischemia.

3. Improve patient Quality of life.

4. Enable the patient to resume normal lifestyle.

5. Lower the risk of Heart attack.

Indication

Patient with Blockages in coronary Arteries

Patient with angina

Patient who cannot Tolerate PTCA and Do not Response will to Drug Therapy

PTCA [12]

Percutaneous transluminal coronary angioplasty (PTCA) is a minimally invasive procedure to open blocked or narrowed coronary arteries and to restore arterial blood flow to the heart tissue without an open-heart surgery. PTCA is majorly of 2 types

Bear metal stents: The balloon is inflated once the catheter is placed into a narrowed area of the coronary artery. The inflation of the balloon compresses the fatty tissue in the artery and make a large opening inside the artery for blood flow.

Drug eluting stents: They are coated with medication that is released to help prevent the growth of scar tissue in the artery lining. This helps the artery remain smooth and open, ensuring good blood flow and reduces the chance of the artery re-narrowing or restenosis. The commonly used drugs in this stent are Sirolimus and Paclitaxel

Methodology

Study design

The study is prospective, open level, observational study.

Source of data and materials

Patient consent form
Patient data collection form
Patient case note/prescription

Inclusion criteria

Inpatients of department of cardiology diagnosed with acute coronary syndrome. Any patient with a chief complaint of chest pain and is at a risk of acute coronary syndrome.

Exclusion criteria

Patients who are not willing to give the consent. Patients who were not diagnosed with acute coronary syndrome

Method of data collection

Patient questionnaire/interview.

Study procedure

This is a prospective observational study where patients eligible are enrolled into the study after obtaining the consent. The data collection form will be prepared and used. This form mainly contains the demographic details of the patient and medication chart. Study will be conducted at MAXCURE hospital. All information relevant to the study will be collected at the time of admission till the date of discharge and the data will be analyzed using suitable method for statistical analysis. The study does not require any investigation or intervention to be conducted on patients. The ethical committee clearance will be obtained from the Institutional Ethical Committee of MAXCURE HOSPITAL before initiating the study. The study was conducted for a period of 6 months. The study was conducted at MAXCURE HOSPITAL

Results

Patients and their characteristics

198patient's details were collected and assessed. The types of patients were NSTEMI, STEMI, and UNSTABLE ANGINA. The details of the distribution were illustrated in the table 1.

Table 1: Types of acute coronary syndrome.

Types of ACS	No of patients
NSTEMI	62
STEMI	67
Unstable angina	71

Interpretation of age

As per the age the patients were categorized into 4 categories starting from less than the age of 55 to more than the age of 75. The distribution of patients were illustrated table 5.2 (Table 2).

Table 2: Interpretation of age.

Group	NSTEMI	STEMI	Unstable angina
<55	22	29	27
55-64	20	11	26
65-74	13	22	11
>75	6	5	6

Gender wise distribution of acute coronary syndrome

As per the gender the patients were divided as male and female separately for NSTEMI, STEMI, Unstable Angina. The details are in table 5.3 (Table 3).

Table 3: Gender wise distribution of cases.

Gender	NSTEMI	STEMI	Unstable angina
Male	40	44	42
Female	22	23	29

Medical history of patients with ACS

The medical history of patients was collected. The information includes a history of angina, myocardial infarction, hypertension, stroke, heart failure. Surgical history of PCI, CABG was also collected. Social history of the patients like smoking and alcohol was collected Table 4.

Table 4: Medical history of patients with ACS.

Medical history	NSTEMI	STEMI	Unstable angina
Angina	42	45	56
Hypertension	32	25	40
Diabetes	35	36	32

Distribution of cases based on social history

Table 5: Distribution of cases based on social history.

Social history	NSTEMI	STEMI	Unstable angina
Smoker	25	22	23
Alcoholic	11	14	16

Estimation of average laboratory tests & blood pressure in patients with ACS

Investigations assessed

Laboratory investigations like creatinine, cholesterol, white blood cells, blood pressure were collected, and the averages were tabulated in table 6.

Table 6: Estimation of avg lab tests and B.P in patients with ACS.

Lab tests	NSTEMI	STEMI	Unstable angina
Creatinine	2	1.7	1.5
Cholesterol	40 mg/dl	67 mg/dl	40 mg/dl
AVG systolic pressure	170	140	140
AVG diastolic pressure	90	90	90

Pharmacological Management of ACS

TREATMENTS GIVEN: Two types of management were included which is drug therapy and surgical procedures. The drugs included were aspirin, ACEI, beta blockers, calcium channel blockers, low molecular weight heparin, nitrates, statins, thrombolytics, heparin and warfarin. All the classes of drugs used were listed in table 5.6.1. The treatments given for NSTEMI, STEMI and UNSTABLE ANGINA were tabulated in table 7.

Table 7. Classes of drugs used.

Drugs	Percentage given
Anti-hypertensives	89.20%
Hyperlipidaemic	94.20%
Thrombolytic	92.40%

Antiplatelet	90.20%
Nitrates	87.60%
Anti-coagulant	96.40%
Anti-emetic	9.03%
Antibiotic	65.34%
Insulin	73.20%
Analgesic	70.20%
Proton pump inhibitors	89.25%

Table 8. Pharmacological management of ACS.

Ace inhibitors	56.20%	4.20%	42.30%
Aspirin	82%	79-3%	93%
Beta blockers	82.20%	74%	86.20%
Calcium channel blockers	10%	4.02%	19%
Low mol wt. heparin	-	-	-
Nitrates	76%	62.20%	64.20%
Statins	97.70%	93.20%	93%
P2y12 inhibitors	90.20%	94%	89.30%
Heparin	96.60%	76%	66.20%
Warfarin	-	-	-

Surgery options opted for patients with ACS

Surgical procedures include cardiac cathertisation, PCI, CABG and PTCA. Treatments given for NSTEMI, STEMI and UNTABLE ANGINA were tabulated in table 9

The surgical options considered was based on age and the number of blood vessels damaged. CABG was most frequently used in patients with NSTEMI. PTCA was considered in patients with STEMI. Both CABG and PTCA were used in equal proportions in unstable angina patients. The usage of cardiac cathertisation and per-cutaneous intervention was not observed in this area

Table 9: Surgery NSTEMI STEMIunstable angina.

Surgery	NSTEMI	STEMI	Unstable angina
CABG	70.20%	26.30%	49.20%
PTCA	42.20%	60%	62.40%

Discussion

In the present 6 months study 198 patients were observed in 30.80% of patients were of NSTEMI, 33.83% of patients were of STEMI, 30.35% of patients were of unstable angina. According to global registry of acute coronary events, the incidence of NSTEMI was 47.69% and STEMI is 52.31% [15]. Age has an important influence on occurrence of coronary events. In this study we observed patients between the age group as below 55 to above 75. In current study maximum numbers of patients were under the age of 55. In our study majority of patients were men with 63.64% and female were 36.36%. in earlier studies male were 57.4% and female were 42.6% [13]. In this study the medical history of patients was in NSTEMI

68% of angina, 52% hypertension, 52% diabetes and 40% were smokers. In STEMI 67% patients have angina, 37% hypertension, 53% diabetes and 32% were smokers. In unstable angina 78% angina, 57% hypertension, 45% diabetes and 32% were smokers. In earlier studies in NSTEMI patients 46.8% angina, 50% hypertension, 21% diabetes, 62.5% were smokers. In STEMI 62.2% angina, 61% hypertension, 27% diabetes and 57.43% were smokers [15]. Surgery is one of the important treatment options in patients suffering from ACS. This study has shown that CABG was performed in 70.2% patients of NSTEMI, 26.3% of patients of STEMI, 49.2% of patients of unstable angina. In earlier study CABG was performed in 10.6% of patients of STEMI and 23% of NSTEMI patients. In our study PTCA was performed in 42.2% of patients of NSTEMI, 62% of patients of STEMI, 62.4% of unstable angina. In previous studies PTCA was performed in 22.9% patients [16]. This study shows that drugs used for NSTEMI include aspirin [82%], ACEI [56%], calcium channel blockers [10%], beta blockers [82.2%], nitrates [76%], statins [97.7%], clopidogrel [90.2%], heparin [96.6%]; for STEMI aspirin [79.3%], ACEI [40.2%], beta blockers [74%], calcium channel blockers [4.02%], nitrates [62.2%], statins [93.2%], heparin [76%]; for unstable angina aspirin [93%], ACEI [42.3%], beta blockers [86.2%], calcium channel blockers [19%], nitrates [64.2%], statins [93%], clopidogrel [89%], heparin [66.2%]. In previous studies shows that aspirin [94%9, low molecular weight heparin [40.6%], clopidogrel [36.2%], gpIIb/ IIIb inhibitors [19.5%], statins [39.6%], betablockers [86.5%], calcium channel blockers [16.6%], ACEI [63.2%], thrombolytics [54.1%][15].

Summary and conclusion

This is a prospective observational study conducted for a period of 6 months. A total of 198 patients suffering from ACS were included in the study. There were 70 patients with unstable angina, 67 were STEMI, and 61 patients were of NSTEMI. All information about demographics, medical history, diagnostic tests, drugs prescribed and surgical details were collected. The incidence of ACS was below the age of 55 years. Men are more at risk than women. Most of the patients were suffering from hypertension, diabetes and angina and were on medications. The common classes of drugs used include anti hypertensive's [89.2%], hyperlipedemics [94.2%], thrombolytic [92.4%], ant platelets [90.2%], anticoagulants [96.4%], nitrates [87.6%], antibiotics [65.3%], insulin [73.2%] and proton pump inhibitors [89.2%], antiemetic [9.03%], analgesics [70.2%]. Drugs used for NSTEMI are statins [97.7%], heparin [96%], P2Y12 inhibitors [90%], beta blockers [82.2%], aspirin [82%], nitrates [76%], calcium channel blockers [10%], ACEI [56.2%]. Drugs given for STEMI are P2Y12 inhibitors [94%], statins [93.2%], aspirin [79.3%], heparin [76%], beta blockers [74%], nitrates [62%]. In unstable angina statins [93%], aspirin [93%], P2Y12 inhibitors [89.2%], beta blockers [86.2%], heparin [66.2%], nitrates [64.2%], ACEI [42.3%]. The surgical manifestations under went by patients were PTCA and CABG.

In this study we studied the management of ACS including NSTEMI, STEMI, unstable angina. It was observed that the treatment given to patients was in compliance with guidelines. All patients underwent surgeries. Early diagnosis of NSTEMI and unstable angina can help prevent surgical procedure. It is important to create awareness about lifestyle management to help prevent this disease. Management of ACS is a multidisciplinary approach where in we should be able to treat the disease and the co-morbidities.

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This manuscript was peer-reviewed
Mode of Review: Single-blinded
Academic Editor: Srinivas P