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A Study on Clinical and Coronary Angiographic Correlation of Patients with Unstable Angina

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Abstract

Introduction: Unstable Angina (UA) is a clinical syndrome caused by atherosclerotic plaque rupture and thrombosis within a coronary artery. It is defined as angina that is new onset or abruptly increased in intensity, duration or frequency within the past 60 days. It may present as rest angina, new onset severe angina or increasing angina .Initial evaluation includes risk stratification based on history , clinical exam , ECG , cardiac enzymes . Among patients with UA who undergo angiogram, 85% will have significant coronary artery disease (CAD). CABG (coronary artery bypass grafting) confers a survival benefit in patients with > 50% LM stenosis or triple vessel disease with LV dysfunction, importantly patients with no significant lesions at angiography benefit from reorientation of their management. Symptomatic patients with normal coronaries may have significant atherosclerosis by IVUS secondary to coronary artery remodeling.

AIM: 1. Risk stratification based on clinical history & presentation, ECG, Enzymes, 2. To Correlate the clinical profile with Coronary angiographic profile 3. To identify the high risk predictors for early intervention.

Materials and Methods: Study design : Observational and Cross sectional Study ; Study population: Unstable angina patients admitted for coronary angiogram in cardiology ward at Azeezia Medical college and attached hospital, Meeyannoor; Kollam. Inclusion Criteria :Patients admitted with a history of chest pain diagnosed as unstable angina and subsequently underwent CAG in cardiology ward.

Results and Conclusion: Unstable angina commonly affects the age group 45-60yrs in both sexes. 30% of patients in our study were women. Women have normal coronaries compared to men in patients with unstable angina, (30% vs 20%) which suggests a different patho physiological mechanism for their symptoms which leads to difficulty in making a firm diagnosis of UA. Smoking, diabetes, Hyperlipidemia, Hypertension are major risk factors for unstable angina in this study Braunwald class III angina (Rest angina) predicted severity of lesion (left main & triple vessel disease) in our study. Patients who had High TIMI risk scoring had more severe coronary lesions compared to low TIMI risk score which helps in risk stratification and early intervention.

Significant ST-T changes in ECG predicted more extensive disease which helps in decision making regarding treatment strategy (conservative vs invasive) aVR ST elevation in background of unstable angina predicts left main disease &Triple vessel disease in our

study which helps risk stratification and early intervention. ECHO evidence of LV dysfunction predicted Triple vessel disease /LM disease. Out of the 100 pts who underwent coronary angiogram in our study 27 pts had Single Vessel disease (type A lesions predominantly) 24 pts had two Vessel disease .(type B lesions predominantly) 26% had three vessel disease. (type B lesions predominantly) 14 patients had Left Main Coronary artery disease. 23 patients had normal or insignificant coronary artery lesions. 9 patients had thrombus containing lesion who had rest angina, out of whom 6 patients had SVD and 3 patients had multivessel disease. 3 patients had total occlusion with TIMI '0' flow.

Keywords: Unstable, Angina, Coronary, Angiography.

Introduction

Acute Coronary syndrome (ACS) is a useful practical term for referring to any pattern of clinical symptoms that is consistent with acute myocardial ischemia. Two closely related forms of ACS – Unstable angina UA and NSTEMI[1]. Unstable angina and the closely related condition NSTEMI are very common manifestations of CAD[2]. Previously UA and NSTEMI were considered as separate entity but pathophysiological mechanism for both involves the rupture or erosion of atherosclerotic plaque with subsequent thrombus formation that significantly obstruct the coronary artery lumen. There is a complex overlap between the two syndromes[3]. Accordingly patients with either of these syndromes are frequently treated identically with individual variations in a management depending on a classification of high, intermediate and low risk[4-6].

They differ primarily in whether the ischemia is severe enough to cause sufficient myocardial damage to release detectable quantities of a marker of myocardial injury [troponon I, troponin T, etc]. Recently, a plethora of information has come to light concerning the diagnosis and subsequent management of patients with either of these two types of ACS. It came to be recognized that the active plaque was not unique suggesting a more diffuse inflammatory disease[7-9]. These concepts led to a new era of research in cell biology and clinical investigation supported by emerging technologies and procedurally sophisticated clinical research, providing a basis for our current evidence based approaches to therapy. Every year in the United States, ACS is responsible, either as a primary or secondary diagnosis, for approximately 1.36 million hospitalizations of which about 0.81 million are for MI and the remaining for Unstable Angina[10].

Definition

The definition of unstable angina is largely based on clinical presentation. Stable angina pectoris typically manifests as a deep ,poorly localized chest or arm discomfort which is precipitated by physical exertion or emotional stress, and relieved within 5- 15 min by rest and or by sublingual nitroglycerin. In contrast, Unstable angina is defined as angina pectoris [or equivalent type of ischemic discomfort] with at least one of the three features[11].

1. Occurring at rest or minimal exertion and usually lasting > 20 min[if not interrupted with nitroglycerine administration

2. Being severe and described as frank pain and of new onset [within 1 month]

3. Occurring with a crescendo pattern [more severe, prolonged or frequent than previously]

Of this group, approximately one half will have proof of myocardial necrosis on the basis of elevated serum cardiac markers, such as creatinine kinase isoenzyme CK-MB and or troponin –I or T and thus confirming a diagnosis of NSTEMI.

Natural History of Unstable Angina

Unstable angina patients have low short-term mortality (1.5-2%) from 0-30 days of presentation than patients with NSTEMI or STEMI .The early mortality risk of the 2 types of MI is same (3-5%). The early mortality risk in UA relates to the extent of myocardial damage and hemodynamic compromise and is less than STEMI[12-13]. In contrast the long term outcome –for mortality and nonfatal events is worse for patients UA compared with STEMI .This finding results from recurrence of ACS in patients with UA as well as old age; great extent of CAD, prior MI and co-morbidities like diabetes & renal dysfunction.

Clinical Presentation

Pain is the main symptom in patients with UA/NSTEMI. It is generally located to the substernal region but it may involve jaw, neck, shoulder, arm back or epigastrium. Some patients present with unexplained dyspnea, fatigue, palpitation, nausea, vomiting and diaphoresis which are called angina equivalent. Such atypical presentations are more common in older adults and women. Rarely patient may present with syncope as primary symptoms[14]. Features which are not usually suggestive myocardial ischemia are sharp stabbing pleuritic pain, reproduction of pain on palpation or with movement, very brief episodes of pain that lasts a few seconds or less, pain that may be localized at the tip of finger, particularly over the LV apex or costochondral junction. Although typical characteristics substantially increase the probability of CAD, atypical features do not totally exclude the possibility of ACS. The chest pain that is relieved by sublingual nitroglycerine does not always predict ACS. In younger patients without any CAD risk factors, one should enquire about cocaine and methamphetamine addiction as these agents cause coronary vasospasm and thrombosis. Urine toxicology should be obtained in such patients[15].

Multivariate Risk Assessment Scores For Nstemi

Integrating all the factors mentioned above, several groups have developed comprehensive risk scores. Of these, TIMI risk score is the one most popularly used. The TIMI risk score identified seven independent risk factors:

1, age > 65 yrs

2, > 3 risk factors for CAD

3, documented CAD at catheterization

4, ST deviation > 0.5 mm

5, > 2 episodes of angina in the last 24 hrs

6, aspirin use within the prior week

7, elevated cardiac markers

Appropriate utilization of TIMI risk score can risk-stratify patients[16] with NSTE-ACS across a 10 fold gradient of risk from 4.7% to 40.9 %(P<0.001). The TIMI risk score was validated internally within the TIMI 11 B trial and two separate cohorts of patients from the ESSENCE trial. Utility of the TIMI risk score lies mainly in the fact that higher TIMI risk scores benefit more from potent and invasive therapies, ie, enoxiparin (in comparison to unfractionated heparin), GP IIb/IIIa inhibitor(in comparison to placebo) and early invasive strategy(in comparison to conservative strategy)[17].

Another well known system is the GRACE risk model. It was developed on the basis of 11,389 patients in the GRACE registry[18] and validated in the GUSTO –II b cohort. It predicts in-hospital mortality in the entire spectrum of ACS, ie STEMI, NSTEMI, UA . The eight variables used in the GRACE risk model include 1, older age 2, Killips class 3, systolic blood pressure 4, STsegment deviation 5, Cardiac arrest during presentation 6,Serum Creatinine level 7, Positive initial cardiac biomarkers and 8, Heart rate. All these variables cause

mortality during the period from hospital discharge to 6 months and the total score can be determined by the application of the sum of scores to a reference nomogram.

Another risk model (the PURSUIT risk model) was developed on the basis of the PURSUIT trial and it is another useful tool to predict the 30-day incidence of death and composite of death or MI. The markers included in the risk model(in order of strength) are 1, age 2, heart rate 3, systolic blood pressure 4,ST segment depression 5, signs of heart failure, and 6, cardiac biomarkers. All three risk scores (TIMI, GRACE, and PURSUIT) demonstrated good predictive accuracy for death and MI at 1 yr[19-20] and they help us immensely in identifying those patients of NSTE-ACS who are likely to benefit from aggressive therapy, including early invasive strategy. However, with the emergence of new biomarkers, it is likely that these too will be included in comprehensive risk scores in future, after they become established in clinical practice[21].

Methodology

Study subjects 100 patients admitted in intensive care unit of department of cardiology, Azeezia Institute of Medical Sciences and hospital; Meeyannoor; Kollam, clinically diagnosed as unstable angina were studied. This is a prospective study Risk stratification was made clinically, ECG, Enzymes, TIMI RISK score and all these patients were subjected to coronary angiogram. This study was a cross sectional analytical study planned from December 2017 to May 2018.

Clinical Examination Patients were risk stratified into low/intermediate /high risk based on Braunwalds classification – class I, 2, 3 TIMI Risk score was done for all patients by including 7 parameters

1, age>65

2, >3 CAD risk factors (high lipids, F/h, HTN, DM, Smoking)

3, Prior coronary stenosis >50%

4, Aspirin in last 7 days

5, > 2 anginal events in <24 hrs.

6, ST segment deviation

7, Elevated cardiac markers

Total score of 0-7/7 was given.

Electrocardiogram

12 lead ECG was done for all patients at the time of admission, depending on the ECG changes (normal or unchanged ECG , T wave inversion >0.2 mV, ST-segment changes) the patients were risk stratified into low, intermediate and high risk

Cardiac Markers

Qualitative Troponin tests were used to risk stratify into low, intermediate and high risk categories

Coronary Angiography

All patients underwent coronary angiogram through femoral or radial access using judkins technique The procedure was performed within 72 hrs of admission to hospital . All obstructive lesions were visualized in multiple planes . Qualitative morphological analysis of all angiograms were performed . In each case we attempted to identify the ischemia related artery and a culprit lesion with a visual diameter stenosis of > 70 % on the basis of anatomy (LAD, LCx, RCA lesions), 50% for LMCA or LM equivalent lesions which was taken as significant anatomical stenosis.

All the lesions are characterized into Type A (high success, >85%; low risk), Type B lesions (moderate success, 60% - 85%; moderate risk) Type C lesions (low success, <60% high risk) according to ACC/AHA classification.

Exclusion Criteria

1. Doesn't opt for inclusion in the study
2. Chronic renal insufficiency GFR

3. History of allergy
4. STEMI / CSA.

Statistical Analysis

The information collected regarding all the selected cases were recorded in a master chart. Data analysis was done with the help of computer by using SPSS software and Sigma Stat 3.5 version (2012). Using this software, frequencies, percentage, mean, standard deviation and 'p' value were calculated through Chi square and P value of < 0.05 was taken as significant.

Results

The age distribution were as follows: Patients within the age group of 46-60 years formed 56% of cases with significant lesion in 54 patients. Followed by age group 61-75 yrs, 36-45 yrs and least with 25-35 years.

The sex distribution was plotted in table 2. Male outnumber females in this study. 70 out of 100 cases are males. 66 patients out of 70 patients had significant lesions whereas only 15 patients of the total of 30 patients in female population had significant lesion.

Almost all patients presented with H/o chest pain. Other symptoms like dyspnoea, syncope, palpitation was present in less than 10% of patients. Smoking, diabetes, hyperlipidemia, hypertension formed the major risk factors in this study. Majority of the patients are smokers and diabetes. 3% of patients had a family history of heart diseases.

46 patients presented with class II angina and significant lesion was seen in 40 patients. 31 patients presented with class III symptoms for whom there were significant 3VD and LMCA diseases. As Braunwald class increased the severity of lesion increased 32 patients presented with TIMI-3 score for whom 31 patients had TVD 3 had LMCA diseases. 16 patients presented with TIMI-4 score for whom 13 patients had significant CAD 3 patient 14 patients who presented with TIMI score 1 had lesions in 4 patients.

Conclusion

This is a single center observational cross sectional study where 100 patients who were admitted as unstable angina over a period of 6 months at our hospital who subsequently underwent coronary angiogram, to study the correlation of the clinical presentation and coronary angiographic profile. After clinical evaluation, braunwald clinical classification, TIMI risk score, ECG, biomarkers, ECHO the patients were risk stratified into low, intermediate and high risk. The clinical presentation and angiographic profile of the patients are correlated. The following conclusions are derived. Unstable angina commonly affects the age group 45-60yrs in both sexes. 30% of patients in our study were women Women have normal coronaries compared to men in patients with unstable angina, (30% vs 20%) which suggests a different patho physiological mechanism for their symptoms which leads to difficulty in making a firm diagnosis of UA. Smoking, diabetes, Hyperlipidemia, Hypertension are major risk factors for unstable angina in this study Braunwald class III angina (Rest angina) predicted severity of lesion (left main & triple vessel disease) in our study.

Patients who had High TIMI risk scoring had more severe coronary lesions compared to low TIMI risk score which helps in risk stratification and early intervention Significant ST-T changes in ECG predicted more extensive disease which helps in decision making regarding treatment strategy (conservative vs invasive) aVR ST elevation in background of unstable angina predicts left main disease &Triple vessel disease in our study which helps risk stratification and early intervention

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Recommendations

The results of this study gives us an idea about the clinical presentation of the patients with unstable angina correlated with coronary angiographic findings Higher Braunwald class (rest angina), higher TIMI risk score, Significant ECG changes (ST-T) changes & aVR ST elevation ,features of LV dysfunction signifies significant and extensive lesions which helps in risk stratification & early intervention. These clinical and other parameters helps us to predict in the bedside the severity of the lesion and the culprit vessel involved so that early invasive intervention can be planned. When practicing in an remote area without cathlab facilities , based on these clinical parameters it helps us to identify high risk patient who will benefit the most from early intervention ,by timely referral or by aggressive use of antithrombotics. Also in the precath evaluation we can be mentally prepared about the anatomy of the culprit vessel involved so that utmost precaution can be carried out during the procedure to prevent complications.

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List of Figures and Tables

RISK STRATIFICATION

TIMI SCORE		
	0-2 points	Low risk
	3-4 points	Intermediate risk
	5-7 points	High risk
GRACE RISK SCORE	**Inhospital risk stratification**	
<108	Low	
109-140	intermediate	
>140	High	
GRACE RISK SCORE	**Predischarge to 6 months risk stratification**	
<108	Low	
89-118	Intermediate	
>118	High	

AGE DISTRIBUTION

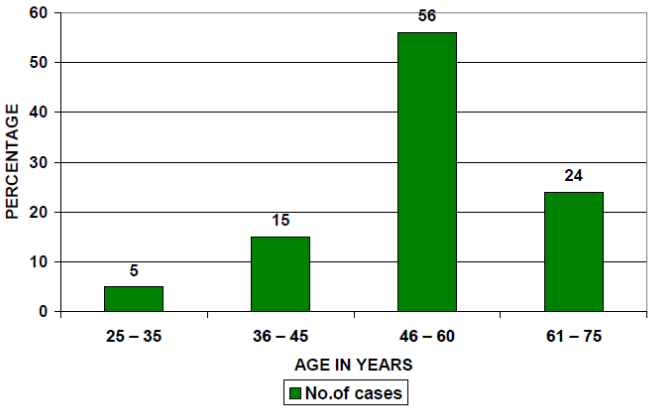


Table - 2

Sex Distribution

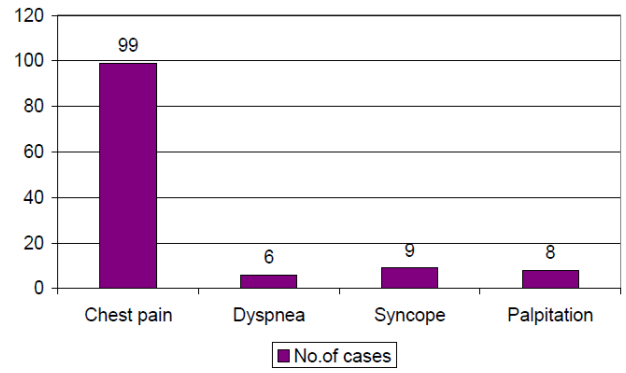
		CAG			
Sex	No.of cases	LMCA	SVD	DVD	TVD
Male	70	10	19	17	20
Female	30	4	8	7	6
Total	100	14	27	24	26

Chi square value - 10.061

'p' value - 0.002 Significant

(Male)

COMPLAINTS



RISK FACTORS