MedCrave
Step into the World of Research

Journal of Cardiology & Current Research

Research Article

Open Access

CrossMark

Clinical features and management model of acute coronary syndrome in saudi arabia

Abstract

Background: Acute coronary syndrome (ACS) denotes a variety of conditions related to sudden and reduced blood flow to the heart. The aim of this study was assess the clinical presentation and management model of Acute Coronary Syndrome (ACS) in Northern Kingdom of Saudi Arabia (KSA).

Methods: This study investigated 156 patients with ACS admitted to the intensive care unit (ICU) in 2013, at King Khalid Hospital-cardiac center in Hail city, KSA.

Results: Of the 156 patients, 139/156(89%), 17/156(11%), 83/156(53%) were presented with chest pain (CP), Epigastric pain, and Arrhythmia, respectively. Anterior, Posterior, and Inferior diagnosis were confirmed in 41/156(26%), 3/156(2%) and 40/156(25.6%) patients in this order.

Conclusion: The model of ACS management might be good and dedicated to some new guidelines, thus attained better outcomes and less mortality.

Keywords: acute coronary syndrome, unstable angina, NSTEMI, STEMI

Volume 7 Issue 5 - 2016

Shrafeldin M Alhassan,[1] Rakan Mosa Saleh Algorinees,[1] Bassam Ahmed Almutlaq,[1] Majid Zannon Turkustani,[2] Shaker Hammad Marzouq Alsaadi,[1] Abdullah Abdulaziz Alghuraymil,[1] Hussain Gadelkarim Ahmed[1,3]

[1]College of Medicine, University of Hail, Kingdom of Saudi Arabia

[2]Batterjee Medical College, Kingdom of Saudi Arabia

[3]Molecular Diagnostics and Personalized Therapeutics Unit, University of Ha'il, Kingdom of Saudi Arabia

Correspondence: Hussain Gadelkarim Ahmed, College of Medicine, University of Hail, Kingdom of Saudi Arabia, Tel 966502609300, Email hussaingad1972@yahoo.com

Received: December 08, 2016 | **Published:** December 28, 2016

Introduction

Acute coronary syndromes (ACS), is mainly comprises unstable angina (AU), and acute myocardial infarction. Myocardial infarction is further categorized according to electrocardiographic changes as non-ST elevation myocardial infarction (NSTEMI) and ST elevation myocardial infarction (STEMI).[1] ACS is characterized by reduced blood flow into the coronary arteries rendering the heart muscle unable to function appropriately or dies.[2] The most common symptom is chest pain, repeatedly radiating to the left arm or angle of the jaw, pressure-like in character, and associated with nausea and sweating. ACS commonly arises as a result of one of three problems: ST elevation myocardial infarction (STEMI, 30%), non ST elevation myocardial infarction (NSTEMI, 25%), or unstable angina (38%).[3] ACS should be distinguished from stable angina, which progresses during exertion and resolves at rest. In contrast with stable angina, unstable angina happens suddenly, often at rest or with slight exertion, or at minor degrees of exertion than the individual's previous angina.[4] Several risk factors have been implicated to the etiology of coronary artery disease (CAD) include life style has been recognized as a major factor for the susceptibility to CAD and lack of exercise, smoking, and a diet of energy-dense fast food and Type 2 Diabetes.[5,6]

A number of interventions have been proved to be beneficial for the management of ACS, including the use of medications such as antiplatelet agents, beta-blockers, heparin, glycoprotein IIb/IIIa inhibitors and the use of procedures such as catheterization and thrombolytic therapy such as coronary angioplasty and revascularization.[7] In cases of acute chest pain, the electrocardiogram is the investigation that most reliably distinguishes between various causes of ACS.[8] In cases of chest pain, there are a number of tests can be done, such as a chest X-ray, blood tests (including myocardial markers such as troponin I or T), and telemetry (monitoring of the heart rhythm).[9] ACS are usually treated with aspirin, clopidogrel or ticagrelor, nitroglycerin, and if the chest discomfort persists morphine.[10]

In Saudi Arabia, the incidence of ACS was 8.2%. The most frequent diagnosis at the time of admission was unstable angina, followed by non-STsegment elevation myocardial infarction (NSTEMI) and ST-segment elevation myocardial infarction (STEMI), representing 19.2%, 27.6%, and 53.2% of the patients, respectively.[11] However, the clinical presentation and management of ACS differ from place to another due to different risk factors and available facilities. Therefore, the objective of this study was to assess clinical presentation and management model in ACS in Hail Region, Northern KSA.

Materials and methods

This is a retrospective study carried out in coronary care unit (CCU) at King Khalid Hospital-cardiac Centre, Hail, Kingdom of Saudi Arabia (KSA). One thousand and nine hundred patients were admitted to cardiac Centre during one year time (the period from 1st of January to 30 of December 2013), with different cardiac diseases. Out of 1900 admitted patients, 156 were diagnosed as having acute coronary syndrome (ACS). All records regarding patients with ACS were retrieved from CCU. Data regarding the clinical presentation and diagnosis, subsequent management and outcome were revised.

Statistical analysis

Data management was done using Statistical Package for Social Sciences (SPSS version 16). SPSS was used for analysis and to perform Pearson Chi-square test for statistical significant (P value $P<0.5$). The 95% confidence level and confidence intervals were used.

Ethical consent

The protocol of the present study was approved by the ethical committee at College of Medicine, University of Hail. The informed consent was agreed about by Cardiology Department at King Khalid Hospital. All procedures performed this study were in accordance with the ethical standards of the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Results

The present study examined 156 patients with ACS, their age ranging from 27 to 90 years with a mean age of 59 years old. Of the 156 patients, 130/156 (83.3%) were males and 26/156(16.7%) were females, giving males' females' ratio of 5.00:1.00. Table 1 summarizes the different categories of clinical presentation for the different types of ACS. For Chest Pain (CP), most patients present by STEMI constituting 73/139(52.5%), Followed by NSTEMI and Unstable angina representing 40/139(29%) and 26/139(18.5%), respectively. Of the 17 patients presenting with epigastric pain, 10/17(59%), 4/17(23.5%) and 3/17(17.5%) were found with STEMI, Unstable angina and NSTEMI, respectively. Only two patients presented with symptoms of heart failure and both were with unstable angina, as shown in Figure 1.

In regard to ACS and Electrocardiogram (ECG) Leads involved, of the 41patients presented with anterior, 23/41(56%), 10/41(24.4%) and 8/41(19.5%) patients were found with STEMI, Unstable angina and NSTEMI, respectively. Of the 3 patients with Posterior, two were found with STEMI and on with NSTEMI. For Inferior 24/40(60%), 10/41(25%) and 6/40(15%) patients were found with STEMI, Unstable angina and NSTEMI, respectively, as indicated in Table 2, Figure 2.

Table 1 Distribution of ACS by Clinical presentation

Variable	Category	Unstable	NSTEMI	STEMI	Total
Chest pain	Yes	26	40	73	139
	No	4	3	10	17
Epigastric pain	Total	30	43	83	156
	Yes	2	3	12	17
	No	28	40	71	139
Heart Failure Symptoms	Yes	2	0	0	2
	No	28	43	83	154
Arrhythmia	Yes	30	43	83	156
	No	0	0	0	0

Table 2 Distribution of ACS by Diagnosis and investigations

Variable	Category	Unstable Angina	NSTEMI	STEMI	Total
ECG	Anterior	8	10	23	41
	Posterior	0	1	2	3
	Inferior	6	10	24	40
CXR	Yes	1	3	3	7
	No	29	40	80	149
ECHO	Yes	27	40	82	149
	No	3	3	1	7
Angiography	Yes	8	5	6	19
	No	22	38	77	137
Troponin I	Positive	0	37	70	107
	Negative	30	5	5	40
Troponin T	Positive	0	37	70	107
	Negative	30	6	10	46
CKMB	Positive	0	14	37	51
	Negative	30	29	41	100

Chest X Ray (CXR) was indicated in 7 patients. Echocardiography (ECHO) was indicated in 149 patients, including 82/149(55%) STEMI, 40/149(26.8%) NSTEMI and 27/149(18%) unstable angina. Angiography was done for 19 patients of whom 8/19(42%), 6/19(31.6%) and 5/19(26.3%) were with unstable angina, STEMI and NSTEMI, respectively. Of the 107 patients with positive Troponin I, 70 were with STEMI and the remaining 37 were with NSTEMI. Of the 107 patients with positive Troponin T, 70 were with STEMI and the remaining 37 were with NSTEMI. Of the 51patients with positive creatine kinase-myocardial band (CK-MB), 37 were with STEMI and 14 were with NSTEMI, as indicated in Table 2 & Figure 3.

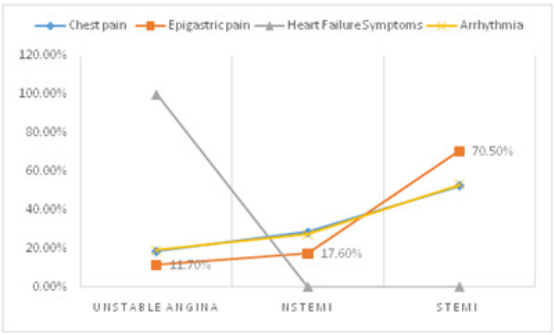

Figure 1 ACS by clinical presentation.

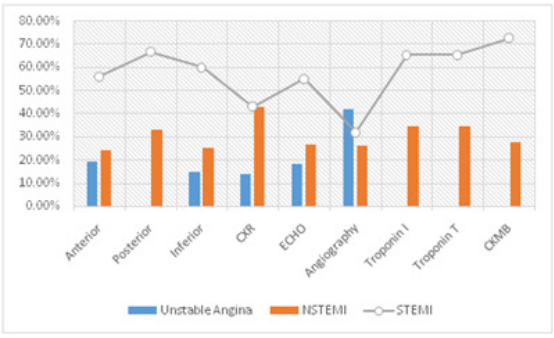

Figure 2 ACS by Diagnosis and investigations.

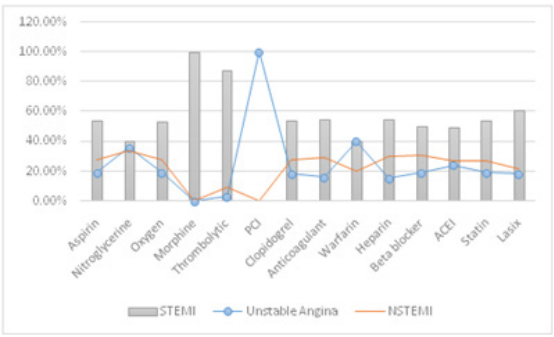

Figure 3 ACS by Treatment.

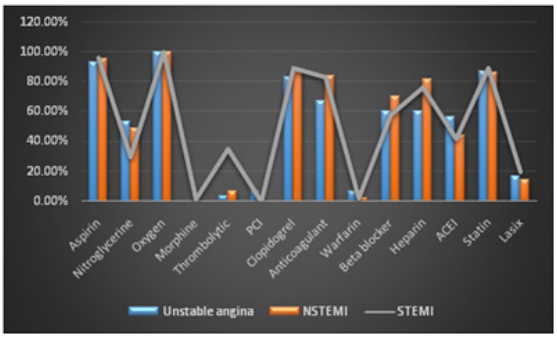

Figure 4 Description of treatment by each ACS type.

Table 3 summarizes, the distribution of ACS with treatment. Aspirin was given to 28/30(93.3%), 41/43(95.3%) and 80/83(96.4%) of the patients with unstable angina, NSTEMI and STEMI respectively. Nitroglycerine was given to 16/30(53.3%), 21/43(49%) and 25/83(30%) of the patients with unstable angina, NSTEMI and

STEMI respectively. Oxygen was given to all 156 patients. Morphine was given to only one patient (1.2%) with STEMI. Thrombolytic agent was given to 33/83(40%) of the patients with STEMI respectively. Percutaneous coronary intervention (PCI) was done for two patients (6.7%) with unstable angina. Clopidogrel was given to 25/30(83.3%), 38/43(88.3%) and 74/83(89%) of the patients with unstable angina, NSTEMI and STEMI respectively. Anticoagulant was given to 20/30(66.7%), 36/43(83.7%) and 69/83(83%) of the patients with unstable angina, NSTEMI and STEMI respectively. Warfarin was given to 2/30(6.7%), 1/43(2.3%) and 2/83(2.4%) of the patients with unstable angina, NSTEMI and STEMI respectively. Beta blocker was given to 18/30(60%), 30/43(70%) and 49/83(58%) of the patients with unstable angina, NSTEMI and STEMI respectively. Heparin was given to 18/30(60%), 35/43(81.4%) and 63/83(76%) of the patients with unstable angina, NSTEMI and STEMI respectively. Angiotensin-converting enzyme inhibitors (ACEI) was given to 17/30(56.7%), 19/43(44%) and 35/83(42%) of the patients with unstable angina, NSTEMI and STEMI respectively. Stain was given to 26/30(86.7%), 37/43(86%) and 74/83(89%) of the patients with unstable angina, NSTEMI and STEMI respectively. Lasix was given to 5/30(16.7%), 6/43(14%) and 17/83(20%) of the patients with unstable angina, NSTEMI and STEMI respectively, as indicated in Table 3, Figure 3 & 4.

Table 3 Distribution of ACS by Treatment

Variable	Category	Unstable	NSTEMI	STEMI	Total
Aspirin	Yes	28	41	80	149
	No	2	2	3	7
Nitroglycerine	Yes	16	21	25	62
	No	14	22	58	94
Oxygen	Yes	30	43	83	156
	No	0	0	0	0
Morphine	Yes	0	0	1	1
	No	30	43	83	155
Thrombolytic	Yes	0	0	33	33
	No	29	40	54	123
PCI	Yes	2	0	0	2
	No	28	43	83	154
Clopidogrel	Yes	25	38	74	137
	No	5	5	9	19
Anticoagulant	Yes	20	36	69	125
	No	10	7	14	31
Warfarin	Yes	2	1	2	5
	No	28	42	81	151
Heparin	Yes	18	35	68	121
	No	12	8	15	35
Beta blocker	Yes	18	30	48	96
	No	12	13	35	60
ACEI	Yes	17	19	35	71
	No	13	24	48	85
Statin	Yes	26	37	74	137
	No	4	6	9	19
Lasix	Yes	5	6	17	28
	No	25	37	66	128

In regard to outcomes, 150 patients were referred of whom 28/30(93.3%), 42/43(97.7%) and 80/83(96.4%) were unstable angina, NSTEMI and STEMI, in this order. Death occurred in one patient with STEMI. Two patients with unstable angina and two patients with STEMI and one patient with NSTEMI were discharged.

Discussion

Sometime the clinical presentation of ACS may differ from one population to another according to ethnicity, sex and comorbid conditions. Moreover, diagnosis and subsequent treatment may differ from one region to another and from one center to another according to the facilities available. Therefore, in the present study we focused on the clinical presentation, diagnosis and treatment of patients with ACS in northern KSA.

Approximately, 89% of the patients with ACS presented with chest pain of whom 52.5% were with STEMI, 29% with NSTEMI and 18.5% with unstable angina. In the examination of chest pain patients with suspected ACS in the emergency department, physicians depend on global diagnostic impressions ('gestalt'). Clinical gestalt was superior than its constituents both at ruling in and at ruling out ACS, but overestimated the probability of ACS when patients were assessed as strong suspicion of ACS. Amongst the constituents of the gestalt, TnT and ECG were greater to the chest pain history for ruling in ACS, whereas pain history was superior for ruling out ACS.[12] Although definite components of the chest pain history are associated with increased or decreased likelihoods of a diagnosis of ACS or acute myocardial infarction (AMI), none of them alone or in combination recognize a group of patients that can be safely discharged without further diagnostic testing.[13] It was shown that approximately 10% of cases with acute chest pain are eventually diagnosed with ACS. Initial, precise estimation of the likelihood of ACS in these patients via the clinical examination could prevent many hospital admissions among low-risk patients and confirm that high-risk patients are rapidly treated.[14] In some countries, women experience differences with respect to ACS and MI, particularly when women suffer from more comorbidities than men. They some-times reveal atypical MI presentation symptoms and are complete unlikely to present with chest pain. Women are more likely than men to encounter delays between the beginning of symptoms and influx at the hospital.[15] However, clinical features have very limited value for diagnosing ACS in patients with a normal or non-diagnostic ECG. Radiation of pain to the right arm rises the possibility of ACS.[16]

In the present study 89% of the patients with ACS presented with epigastric pain. Patients who present with pressure-type or heavy chest pain or tightness must be expected to have likely ACS. Furthermore, the patient who complains of "indigestion" or "heartburn" as well as belching, cramping, epigastric pain should also be assessed for ACS.[17] Symptoms of heart failure were detected in only two patients in this study. However, there is intersection between heart failure and ACS. The bulk of patients admitted with heart failure have coronary artery disease (CAD), which independently has an adversative effect on prognosis. Patients with underlying CAD or ACS usually complicated by heart failure.[18]

Arrhythmia was indicated in all patients in the present study. Atrial fibrillation and sustained ventricular arrhythmias are the most shared arrhythmias associated ACS. Arrhythmias are related to worse clinical sequence and greater risk of in-hospital, short-term and long-term mortality in patients with ST-elevation myocardial infarction.[19] However, in the present study, most of patients in this study presented with anterior and inferior ACS. CXR was only performed for 7 patients. ECHO was done for 149 patients most of them with STEMI. Angiography was performed for 19 patients most of them were with unstable angina. Troponin I & T were positive in 68.6% of patients with ACS of whom 65.4% were STEMI and the remaining 34.6% were NSTEMI. Also CK-MB was positive in 32.7% of the patients with ACS, of whom, 72.5% were STEMI and the remaining 27.5%

Citation: Alhassan SM, Algorinees RMS, Almutlaq BA, et al. Clinical features and management model of acute coronary syndrome in saudi arabia. *J Cardiol Curr Res.* 2016;7(5):1–6. DOI: 10.15406/jccr.2016.07.00263

were NSTEMI. Several cardiac biomarkers are frequently measured during evaluation of patients suspected of ACS. Cardiac troponin is the preferred biomarker and is more sensitive than creatine kinase isoenzyme (CK-MB). Troponins are valuable in diagnosis, risk stratification, and characterization of prognosis. A high level of *troponin* associates with an increased risk of death, and more rises predict superior risk of adverse outcome.[20]

Furthermore, a diverse of treatment means were used in the current study. Aspirin was used for 89% of the patients. Aspirin blocks the synthesis of thromboxane A2 by irreversibly inhibiting cyclooxygenase 1, thus weakening platelet aggregation. Aspirin decreases the risk of death or MI by more than 50% for patients presenting with UA/NSTEMI.[21] Nitroglycerine was used in 39.7% of the patients in the current study. Nitroglycerin ointment is usually used in the treatment of emergency department (ED) patients with suspected acute heart failure or suspected ACS, but its hemodynamic properties in this population are not well designated.[22] Nitroglycerin is a vasodilator that reduces myocardial oxygen demand by declining ventricular preload via venodilation. Nitroglycerine improves myocardial oxygen delivery by dilating large coronary arteries and enhancing collateral flow to ischemic areas.[23]

Oxygen was given to almost all patients. Oxygen therapy will improve oxygen supply to the threatened heart and may decrease the volume of heart muscle that perishes. The addition of oxygen therapy to standard treatment may reduce death rate and other major adversative outcomes.[24] However, several studies propose that the use of oxygen should be more restrictive. Unfavorable special effects of normobaric oxygen therapy in patients suffering from hypercapnic respiratory diseases have been validated, mainly because of the suppression of the hypoxic initiative. Distant from this particular condition, correction of hypoxemia is still an extensively accepted treatment target, although there is increasing evidence that hyperoxemia could be dangerous in ACS and cardio-respiratory arrests.[25]

Clopidogrel was used in 87.8% of the patients with ACS in this study. Clopidogrel blocks the P2Y12 adenosine diphosphate (ADP) receptor on platelets. This action losses platelet activation and aggregation, rises bleeding time, and decreases blood viscosity. Therapy with clopidogrel and aspirin is suggested for basically all patients with UA/NSTEMI.[26] Morphine was only used in one patients. Morphine acts as a powerful analgesic and anxiolytic; in addition, its hemodynamic effects and it may be beneficial in treating UA/NSTEMI.[27] Thrombolytic was used in 21**%** of the patients with ACS in this study. Over the past decade, more men than women have revealed better outcomes from antithrombotic therapies after ACS, which raises the query of whether there are sex-specific variances in treatment designs and response to therapy. Large trials and registry data find that male and female ACS patients experience similar benefits from antithrombotic therapy without significant difference in treatment utilization rates, nevertheless women are steadily at greater risk of bleeding than men. Bleeding may result in antithrombotic treatment interruption, which rises the risk of long-term thrombotic actions. Moreover, female ACS patients are more feasible to receive suboptimal medication dosing and have lower rates of long-term medication adherence. These variances have significant clinical consequences for women, indicating the necessity for strategies that will improve initial treatment and long-term management adjusted to these accepted sex-specific gaps.[28]

Anticoagulant was used in 80% of the patients with ACS in this study. With the development of techniques and pharmacological strategies in percutaneous coronary intervention, significant progresses have been done towards dropping the risk of in-stent restenosis and enhancing patient outcomes. However, yet, stent thrombosis still a deadly complication of stent implantation. The vital challenge in implementing a combined anticoagulant and antiplatelet strategy is balancing the risk of bleeding with the improved ability of therapy on both pathways [29]. Heparin was used in 77.6% of the patients with ACS in this study. Heparin is an indirect inhibitor of thrombin that has been broadly used in ACS as adjunctive therapy for fibrinolysis and in combination with aspirin and other platelet inhibitors for the treatment of NSTEMI.[10]

Beta blocker was used in 77.6% of the patients with ACS in this study. Beta-blocker treatment in patients with ACS and conserved left ventricular ejection fraction is associated with lower long-term mortality [30]. Beta-blockade is presently suggested in the initial management of patients with ACS and early beta-blockade improves the outcome of patients with ACS.[31]

ACEI was used in 45.5% of the patients with ACS in this study. ACE-inhibitor therapy appears to be an effective first-line treatment for preventing the occurrence of mortality in patients with non-obstructive coronary artery disease.[32] Some studies have shown that previous use of an ACE inhibitor is not independently associated with better in-hospital outcomes after an ACS.[33]

Statin was used in 87.8% of the patients with ACS in this study. In several clinical trials, reducing LDL-C with statin therapy has been verified to reduce the risk of cardiovascular disease (CVD) in primary and secondary prevention settings. Guidelines recommend statins for first-line therapy in cholesterol-dropping management of patients with CVD risk. In spite of increased statin monotherapy use over the last decade, a number of patients with high CVD risk do not accomplish optimal LDL-C lowering. Guidelines recommend consideration of statin combination therapy with non-statin agents for these patients.[34]

Nevertheless, the predictors of mortality and morbidity greatly vary worldwide according to the present risk factors.[35,36,11] There are several risk factors for CAD in Saudi Arabia, but there is a lack of literature regarding the mortality and morbidity burden associated with these risk factors and CAD. However, other treatment models have been used in the present study include, Lasix, Warfarin and PCI. Since, all patients received several treatment parameters, no complication was fixed for a unique type of treatment given. However, there might be some complications for the whole model of treatment, but might not be documented. In the present study, only documented variables were used regarding these patients, since the data were collected retrospectively. Nevertheless, the outcome of these patients is good. Notability the majority of patients were referred to other bigger centers and this affect the assessment of outcomes in this study, and this represent a major limitation this study.

Conclusion

As the term ACS related to unstable angina (UA), non-ST-segment elevation myocardial infarction (NSTEMI), and ST-segment elevation myocardial infarction (STEMI). However, assessment of the patient's history and results on physical examination, electrocardiography, radiologic studies, and cardiac biomarker tests permit precise diagnosis and help in early risk stratification, which is important for guiding treatment. Clinical outcomes can be optimized by revascularization coupled with aggressive medical therapy that includes anti-ischemic, antiplatelet, anticoagulant, and lipid-lowering drugs. Evidence-based guidelines provide recommendations for the management of ACS; however, therapeutic approaches to the management of ACS continue to change at a fast pace determined by a multitude of large-scale

randomized controlled trials. Consequently, clinicians are often faced with the problem of deciding which drug or therapeutic strategy will attain the best results. The model of ACS management might be good and dedicated to some new guidelines, thus attained better outcomes and less mortality.

Acknowledgements

Authors would like to thank people at coronary care unit (CCU) at King Khalid Hospital-cardiac Centre, Hail, Kingdom of Saudi Arabia (KSA) for their help in data collection.

Conflicts of interest

author declares there are no conflicts of interest.

Funding

None.

References

1. Adam Timmis. Acute coronary syndromes.*BMJ*. 2015;351:h5153.
2. Torres M, Moayedi S. Evaluation of the acutely dyspneic elderly patient. *Clin Geriatr Med*. 2007;23(2):307–325.
3. Grech ED, Ramsdale. Acute coronary syndrome: unstable angina and non-ST segment elevation myocardial infarction. *BMJ.* 2003;326(7401):1259–1261.
4. Achar SA, Kundu S, Norcross WA. Diagnosis of acute coronary syndrome. *Am Fam Physician*. 2005;72(1):119–126.
5. Chow CK, Lock K, Teo K, et al. Environmental and societal influences acting on cardiovascular risk factors and disease at a population level: a review. *Int J Epidemiol* . 2009;38(6):1580–1594.
6. De Kovel CG, Mulder F, van Setten J, et al. Exome-Wide Association Analysis of Coronary Artery Disease in the Kingdom of Saudi Arabia Population. *PLoS ONE* . 2016;11(2):e0146502.
7. Pesaro AE, Campos PC, Katz M, et al. Acute coronary syndromes: treatment and risk stratification. *Rev Bras Ter Intensiva*. 2008;20(2):197–204.
8. Chun AA, McGee SR. Bedside diagnosis of coronary artery disease: a systematic review. *Am J Med.* 2004;117 (5):334–343.
9. Shah AS, Anand A, Sandoval Y, et al. High-sensitivity cardiac troponin I at presentation in patients with suspected acute coronary syndrome: a cohort study. *Lancet.* 2015;386(10012):2481–2488.
10. O'Connor RE, Brady W, Brooks SC, et al. Part 10: acute coronary syndromes: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation.* 2010;122(18 Suppl 3):S787–S817.
11. Alhassan SM, Alharbi MS, Algorinees RMS, et al. Incidence of Acute Coronary Syndrome (ACS) in Northern Saudi Arabia. *European Academic Research* . 2016;3(12):12982–12993.
12. Mokhtari A, Dryver E, Söderholm M, et al. Diagnostic values of chest pain history, ECG, troponin and clinical gestalt in patients with chest pain and potential acute coronary syndrome assessed in the emergency department. *Springerplus.* 2015;4:219.
13. Swap CJ, Nagurney JT. Value and limitations of chest pain history in the evaluation of patients with suspected acute coronary syndromes. JAMA. 2005;294(20):2623–2629.
14. Fanaroff AC, Rymer JA, Goldstein SA, et al. Does This Patient With Chest Pain Have Acute Coronary Syndrome?: The Rational Clinical Examination Systematic Review. *JAMA.* 2015;314(18):1955–1965.
15. Graham G. Acute Coronary Syndromes in Women: Recent Treatment Trends and Outcomes. *Clin Med Insights Cardiol* . 2016;10:1–10.
16. Goodacre S, Pett P, Arnold J, et al. Clinical diagnosis of acute coronary syndrome in patients with chest pain and a normal or non-diagnostic electrocardiogram. *Emerg Med J*. 2009;26(12):866–870.
17. The American College of Cardiology and the American Heart Association. New Guidelines for Evaluating Acute Coronary Syndrome. *Clinician Reviews* . 2001;11(1):73–86.
18. Flaherty JD, Bax JJ, Luca LD, et al. Acute Heart Failure Syndromes in Patients With Coronary Artery Disease early Assessment and Treatment. *J Am Coll Cardiol.* 2009;53(3):254–263.
19. Gorenek B, Kudaiberdieva G. Arrhythmic emergencies in ICCU. *Minerva Med.* 2013;104(4):383–390.
20. Antman EM, Tanasijevic MJ, Thompson B, et al. Cardiac-specific troponin I levels to predict the risk of mortality in patients with acute coronary syndromes. *N Engl J Med*. 1996;335(18):1342–1349.
21. The RISC Group. Risk of myocardial infarction and death during treatment with low dose aspirin and intravenous heparin in men with unstable coronary artery disease. *Lancet* . 1990;336(8719):827–830.
22. Mumma BE, Dhingra KR, Kurlinkus C, et al. Hemodynamic effects of nitroglycerin ointment in emergency department patients. *J Emerg Med*. 2014;47(2):192–197.
23. Cheitlin MD, Hutter AM, Brindis RG, et al. ACC/AHA expert consensus document. Use of sildenafil (Viagra) in patients with cardiovascular disease. American College of Cardiology/American Heart Association. *J Am Coll Cardiol*. 1999;33(1):273–282.
24. Bennett MH, Lehm JP, Jepson N. Hyperbaric oxygen therapy for acute coronary syndrome. *Cochrane Database Syst Rev*. 2015;8:CD004818.
25. Von Düring S, Bruchez S, Suppan L, et al. Normobaric oxygen therapy in acute medical care: myths versus reality. *Rev Med Suisse.* 2015;11(482):1476–1485.
26. Mehta SR, Yusuf S, Clopidogrel in Unstable angina to prevent Recurrent Events (CURE) Study Investigators. The Clopidogrel in Unstable angina to prevent Recurrent Events (CURE) trial programme; rationale, design and baseline characteristics including a meta-analysis of the effects of thienopyridines in vascular disease. *Eur Heart J*. 2000;21(24):2033–2041.
27. Meine TJ, Roe MT, Chen AY, et al. Association of intravenous morphine use and outcomes in acute coronary syndromes: results from the CRUSADE Quality Improvement Initiative. *Am Heart J.* 2005;149(6):1043–1049.
28. Wang WT, James SK, Wang TY. A review of sex-specific benefits and risks of antithrombotic therapy in acute coronary syndrome. *Eur Heart J pii: ehv*. 2016;758.
29. Welsh RC, Zeymer U, Tarrantini G. Direct oral anticoagulant use and stent thrombosis following an acute coronary syndrome: A potential new pharmacological option? *Arch Cardiovasc Dis.* 2016;109(5):359–369.
30. Raposeiras-Roubín S, Abu Assi E, Redondo Diéguez A, et al. Prognostic Benefit of Beta-blockers After Acute Coronary Syndrome With Preserved Systolic Function. Still Relevant Today? *Rev Esp Cardiol* . 2015;68(7):585–591.
31. Brandler E, Paladino L, Sinert R. Does the early administration of beta-blockers improve the in-hospital mortality rate of patients admitted with acute coronary syndrome? *Acad Emerg Med.* 2010;17(1):1–10.
32. Manfrini O, Morrell C, Das R, et al. Effects of angiotensin-converting enzyme inhibitors and beta blockers on clinical outcomes in patients with and without coronary artery obstructions at angiography (from a Register-Based Cohort Study on Acute Coronary Syndromes. *Am J Cardiol*. 2014;113(10):1628–1633.

33. Singh SM, Goodman SG, Yan RT, et al. Relation between previous angiotensin-converting enzyme inhibitor use and in-hospital outcomes in acute coronary syndromes. *Am J Cardiol.* 20122;109(3):332–336.

34. Toth PP, Farnier M, Tomassini JE, et al. Statin combination therapy and cardiovascular risk reduction. *Future Cardiol*. 2016;12(3):289–315.

35. Kolansky DM. Acute coronary syndromes: morbidity, mortality, and pharmacoeconomic burden. *Am J Manag Care.* 2009;15(2 Suppl):S36–S41.

36. Pizzi C, Xhyheri B, Costa GM, et al. Nonobstructive Versus Obstructive Coronary Artery Disease in Acute Coronary Syndrome: A Meta-Analysis. *JAm Heart Assoc*. 2016;5(12):e004185.

Citation: Alhassan SM, Algorinees RMS, Almutlaq BA, et al. Clinical features and management model of acute coronary syndrome in saudi arabia. *J Cardiol Curr Res.* 2016;7(5):1–6. DOI: 10.15406/jccr.2016.07.00263