ABSTRACT
Chest pain is the common reason for presentation in hospital emergency department and outpatient clinic. Some patients presenting with chest pain will have serious life threatening illness with a high short term risk of mortality. The rapid identification of cause of acute chest discomfort and early specific treatment protocol decreases the risk. Cardiac, pulmonary, gastro intestinal, musculoskeletal, abdominal and psychological reasons are the underlying causes of chest pain. Patients with Acute coronary syndromes, pulmonary embolism, aortic dissection and tension pneumothorax can rapidly deteriorate and hence should be identified as early as possible. Regardless of clinical setting, a stepwise approach should be applied. Clinical examination important to assess hemodynamic stability, may point towards a specific diagnosis. ECG is the first and most often used investigation to identify ST elevation myocardial infarction and to initiate reperfusion therapy. Chest radiography, echocardiography, CT and MRI will aid in arriving definitive diagnosis. High sensitive troponin measurements are useful in evaluation of suspected acute coronary syndromes other than ST elevation MI. In most of the cases with clinical, biochemical and imaging the diagnosis can be reached in emergency department and treatment can be initiated.

KEY WORDS
Chest pain, Emergency, Symptom, Electrocardiogram, protocol

INTRODUCTION
Acute chest pain is one of the most common symptom with which patients present to emergency department (ED) or to out-patient clinics. It accounts for 7 million ED visits annually in USA. The evaluation of non traumatic chest pain is often challenging and may include life threatening situations. Rapid identification and triage of life threatening situation is priority, however, routine and liberal use of testing carry potential for adverse effects.

EPIDEMIOLOGY
In the USA, chest pain is the third leading reason for visit to ED and accounts for 6 to 7 million visits each year. Less than 40% are not admitted or observed, however, significant proportion of patients require hospitalization and further evaluation. In most of the series of unselected population 5 to 15% of admitted patients are diagnosed as having acute coronary syndromes. Other life threatening cardiopulmonary conditions are noted in around 10%. Most common cause is attributable to gastro intestinal tract. In significant proportion of cases diagnosis remains unknown, however, the prognosis is good in these cases. In 2 to 6 % of cases who were discharged from ED as non ischemic cause were later found to have had ischaemic injury. These patients with missed diagnosis had worse prognosis. They had 30 day mortality risk double that of hospitalized patients.

ETIOLOGY OF CHEST DISCOMFORT
Cardiac cause (Table 1)
Myocardial causes: Myocardial ischemia is the life threatening chest discomfort. Myocardial ischemia is precipitated by an imbalance between myocardial oxygen requirement and supply. Myocardial oxygen consumption may be elevated by increases in heart rate, ventricular wall stress, and myocardial contractility. Myocardial oxygen supply is determined by coronary blood flow and coronary arterial oxygen content. Coronary artery disease (CAD) is commonly due to atherosclerosis which is a gradual process. Chronic stable angina is characterized by ischemic episodes that are typically precipitated by increase in oxygen demand like exertion, tachyarrhythmia, hypertension etc. The episode is typically relieved upon resting. Rupture or erosion of atherosclerotic plaque leads to unstable coronary syndrome. Acute coronary syndrome is characterized by ST segment elevation in STEMI and either ST depression or T wave changes or none in NSTEMI. Documentation of myocardial injury or necrosis by troponin or other cardiac biomarker establishes the diagnosis of myocardial infarction (MI). Unstable coronary symptoms may also occur because of increased myocardial oxygen demand

Table 1: Causes of chest pain

<table>
<thead>
<tr>
<th>Causes of chest discomfort</th>
<th>%</th>
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<tbody>
<tr>
<td>Gastrointestinal</td>
<td>42</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>31</td>
</tr>
<tr>
<td>Chest wall syndrome</td>
<td>28</td>
</tr>
<tr>
<td>Pericarditis</td>
<td>4</td>
</tr>
<tr>
<td>Pleuritis</td>
<td>2</td>
</tr>
<tr>
<td>Pulmonary embolism</td>
<td>2</td>
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<tr>
<td>Lund cancer</td>
<td>1.5</td>
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<tr>
<td>Aortic aneurysm</td>
<td>1</td>
</tr>
<tr>
<td>Aortic stenosis</td>
<td>1</td>
</tr>
<tr>
<td>Herpes zoster</td>
<td>1</td>
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</tbody>
</table>
or because of decreased oxygen delivery due to anemia, hypoxia, or hypotension. Though atherosclerosis is the underlying cause of ischemia in most of the cases, there are variety of non atherosclerotic cause - Congenital anomalies, myocardial bridging, arteritis, radiation arteriopathy etc. Also non vascular causes like aortic valve disease, cardiomyopathies may cause coronary ischemic symptoms.

Pericardial: Pericarditis due to infective or non infective causes can produce acute chest discomfort. Most of the pericardium is insensitive to pain. It is postulated that associated pleurisy is the cause of pain and it typically referred to shoulder and neck due to overlapping supply of central diaphragm via the phrenic nerve with somatic sensory fibers originating from 3rd and 4th cervical segments.

**OTHER CARDIO-PULMONARY CAUSES**

Acute aortic syndromes: This includes a spectrum of diseases related to disruption of the media of the aortic wall. Aortic dissection, penetrating ulcer and intramural hematoma are the prototypes of this spectrum. Though less common (estimated annual incidence, 3 cases per lakh population) these can be catastrophic when recognized late or left untreated. Aortic aneurysms can also produce chest discomfort.

Pulmonary disorders: Pleurisy either infective or non
infective is an important cause of chest discomfort. It is typically knifelike pain that is worsened by inspiration or coughing. Pulmonary embolism may be associated with chest discomfort. The pain may be because of pulmonary infarction with pleurisy, distension of pulmonary artery or RV wall stress and / or RV sub endocardial ischemia. Pneumothorax may present with chest pain. Most the pulmonary disorders presenting with chest pain are usually associated with other symptoms like dyspnoea, cough, syncope etc.

**NON CARDIOPULMONARY CAUSES**

GI disorders: This is a most common cause of non traumatic chest pain. The discomfort may mimic serious conditions like MI. GERD, motility disorders of esophagus and tear should be considered in evaluation of chest pain.

Hepatobiliary disorders like cholecystitis and biliary colic may also mimic acute coronary syndromes. Musculoskeletal and others: Musculoskeletal disorders are an important cause of chest pain. Pain may be due to involvement of chest wall, nerves of the chest wall or upper limbs. Costochondritis and cervical radiculitis may produce acute or chronic pain. Herpes zoster before the eruptions may confuse with other causes.

Emotional and psychological causes: Significant proportion of patients presenting ED may have emotional or psychological cause for chest pain. There may be other clues to suggest psychological origin, however, at times it may be difficult differentiate it from common organic causes.

Approach to chest pain (Figure 1): As there are many causes of chest pain it is challenging in the ED to triage patients with chest pain. The most important questions to be answered on arrival are clinical stability of the patient and likelihood of a underlying cause of chest to life threatening. The high risk life threatening conditions

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**Fig. 2: Risk stratification in acute cardiac chest pain**
include ACS, aortic syndromes, tension pneumothorax, pulmonary embolism. A rapid targeted assessment for a serious cardiopulmonary cause is of particular relevance for patients with acute ongoing chest pain. Mortality for patients with AMI differs greatly between admitted and discharged patients (6% vs 25%). Missed MI accounts for 20% of US emergency medicine related litigation dollars. Task force report on the management of chest pain is available. Following questionnaire is useful in the assessment of acute chest pain.

1. Could the chest pain be due to potentially life threatening conditions?
2. Could the chest pain be due to a chronic condition likely to lead to serious complications?
3. Could the chest pain be due to an acute condition that warrants specific treatment?
4. Could the chest pain be due to another treatable condition?

HISTORY AND CLINICAL EVALUATION

Careful history is useful aid in diagnosis of chest pain. Past history of MI may point towards ACS while history suggestive connective tissue disorders or Marfan’s syndrome may indicate aortic dissection or pneumothorax. Risk factors for atherosclerosis may be present. Past history of cholelithiasis or evidence of peptic ulcer on previous endoscopy may help in arriving diagnosis.

Quality of pain

Tightness or pressure is consistent with typical presentation of myocardial ischemic pain. However, many times patients may have atypical chest discomfort. Pain similar to earlier angina episodes points towards ACS. Tearing or ripping pain is often noted in aortic dissection. A burning quality may suggest acid reflux or peptic ulcer disease but may occur with MI. Esophageal spasm is usually severe squeezing type. Pleurisy is typically described as a knife like pain.

Location and radiation

A substernal location with radiation to neck, jaw, or arms is typical of myocardial ischemia. Highly localized pain are unlikely angina episodes. Esophageal disorders most commonly present with substernal pain. Severe pain radiating to back should suggest aortic dissection. Radiation to ridge of trapezius is characteristic of pericarditis. Hepatobiliary, pancreatic and peptic ulcer pains are predominantly epigastric.

Pattern of pain

Many patients present to ED repeatedly with severe very short lasting migratory pain. This is unlikely to be of ischemic origin. Myocardial ischemic discomfort usually builds over minutes. Constant and prolonged pains are also unlikely to represent ACS. They are more likely to due to hepatobiliary or pancreatic in origin.

Aggravating and relieving factors

Alteration in the severity of pain with changes in position or movement are more likely to be musculoskeletal. Pain getting worsened in supine and relieved by sitting upright or leaning forward suggest pericarditis. GERD symptoms are also aggravated by supine position and may be relieved by sitting. Angina during stable patients are noted during exertion and are relieved by rest. The similar discomfort at rest may suggest life threatening ACS. Post prandial angina also suggest severe underlying CAD.

Associated symptoms

Presence of associated symptoms may indicate specific diagnosis. Diaphoresis, dyspnea, nausea, fatigue, pre syncope or syncope may accompany angina chest pain. Hemo dynamically significant pulmonary embolism is associated with dyspnea and / or syncope. Hemoptysis suggest paranchymal pathology, pulmonary edema, pulmonary embolism. Predominant nausea, vomiting and eructation generally indicate gastro intestinal pathology. Clinical examination: General and systemic examination may reveal the origin of chest pain.

General physical examination and vital signs

Patients with ACS are generally anxious, uncomfortable, pale, cyanotic or diaphoretic. Levine’s sign (clenched fist against the sternum) suggest angina pain. Hyperventilation is sign of psychological origin. Peripheral signs of shock are hallmark of severe organic pathology and needs emergent attention. Hypertension and pulse asymmetry are noted in aortic dissection. Hypoxemia indicating severe nature may occur both in pulmonary and cardiac conditions. Evidence of DVT should arose strong suspicion of pulmonary embolism. There may be tell tale signs of peripheral arterial disease.

Chest

Specific signs like pleural rub (pleurisy), bronchial breathing (pneumonia), absent lung sounds (pneumothorax), basal rales (pulmonary edema) are useful in arriving clinical diagnosis.

Cardiovascular system

Third heart sound indicate heart failure, fourth sound is common in hypertension and MI. Murmurs due to valvular pathology and VSD are important clinical indicators of cardiac pathology. Pericardial friction rub indicate pericardial inflammation.

Abdomen

Organomegaly and local signs point towards local causes. Congestive hepatomegaly may be significant in congestive heart failure.

Investigations

Rapid access to investigative tools is a key while triaging patients with acute chest.

Electrocardiography

The ECG should be recorded as early as possible. Guidelines recommend that an ECG be obtained within 10 minutes of presentation. The ECG is an excellent tool for identifying ACS especially STEMI. It is highly specific
(77 to 100%) depending on the criteria used. However, the sensitivity of ECG is poor (28 to 54%) in the first 12 hours.7,9 Presence of a normal ECG neither excludes MI nor provides sufficient assurance to discharge the patient from ED. Reperfusion therapy is mainly based on recognition of STEMI on ECG. ST segment depression and symmetric T wave inversions at least 0.2 mV in depth are useful for detecting myocardial ischemia in the absence of STEMI and are also indicative of higher risk of death or recurrent ischemia. Serial ECGs is recommended in the ED for suspected ACS. ST and T wave changes may be seen in variety of non ischemic conditions like pericarditis, myocarditis, metabolic abnormalities, pulmonary embolism, hyperventilation, ventricular hypertrophy etc.

Chest Radiography
Chest radiograph is useful in evaluating chest pain due to pulmonary causes. It may reveal pneumonia, pneumothorax, pleural effusion. Mediastinal widening may be present in acute aortic syndromes. Hampton’s hump or Westmark’s sign in pulmonary embolism or pericardial calcification in chronic pericarditis are some specific findings.

Cardiac biomarkers
Assessment of cardiac biomarkers is vital during the initial and subsequent evaluation of patient with chest pain suggestive of ACS. Elevated biomarkers indicate myocardial injury and high risk. These are circulating proteins released from injured myocardial cells and the rise is proportional to degree of injury. However, owing to the time necessary for the release, initial evaluation may reveal normal range of biomarkers despite extensive STEMI. Over the years cardiac specific troponin assessment has become gold standard in the evaluation of ACS patients. High sensitivity troponin assay are available to rule in or rule out MI based on serial assay. High sensitivity assay have revolutionized evaluation of ACS patients. This permits earlier detection of myocardial injury and enhanced overall accuracy and improved risk stratification. The diagnosis of MI should be reserved for acute myocardial injury as shown by a rising and / or falling pattern with at least on value exceeding 99th percentile of reference limit due to ischemia.

D-dimer assessment is useful in the evaluation of suspected pulmonary embolism. It has a very high negative predictive value. NT-proBNP is useful in diagnosis and prognostication of heart failure.

Echocardiography
Trans-thoracic echocardiography is widely available now. It is useful tool to assess overall ventricular function, valvular morphology, presence of regional wall motion abnormality, pericardial effusion etc. Trans-esophageal echocardiography can accurately detect acute aortic syndromes. Mechanical complications of MI like mitral regurgitation, VSD or cardiac tamponade can be quickly appreciated by echocardiography. Right sided chamber assessment can reveal possibility of pulmonary embolism.

CT angiography
The technology is very useful in certain subgroup of patients when diagnosis in uncertain. CT coronary angiography can detect presence or absence of significant obstructive coronary artery disease. CT pulmonary angiography can identify pulmonary embolism. CT angiography is very useful in evaluating aortic dissections. CT scan can detect other pulmonary paranchymal or pleural pathology.

MRI imaging
Cardiac MRI (CMR) can accurately predict structural and functional abnormalities of heart and vasculature. Gadolinium enhanced CMR can provide early detection of MI. MR angiography is very accurate in detecting aortic dissections.

Clinical decision making
Clinical decision making can be challenging in ED. The most important is to identify life threatening conditions and to start early interventions specific to diagnosis. Several clinical algorithms are available as decision aids in ED. Goldman and Lee developed one of the first such decision aids, using only the ECG and risk indicators – hypotension, pulmonary rales, and known CAD – to categorize patients into four risk categories ranging from a <1% to a >16% probability of a major CV complications. The Acute Cardiac Ischemia Time-Insensitive Predictive Instrument (ACI-TIPI) combines age, sex, chest pain presence, and ST –segment abnormalities to define a probability of ACS. Heart Score and North American Chest pain Rule can also be used as decision aids.

Treatment of acute chest pain: Acute chest pain treatment depends on the diagnosis made after initial clinical evaluation, ECG, biochemical and other imaging modalities. ACS, pulmonary embolism, aortic dissection and tension pneumothorax which have high morbidity and mortality should be treated early as per the standard guidelines. Low risk group can be discharged from the ED and can be followed on out-patient basis (Figure 2).

CONCLUSIONS
Acute chest pain is the commonest symptom for attending emergency and out-patient departments. Acute chest pain can be manifestation of a life threatening situation. However, it can also be a manifestation insignificant cause. Clinical, biochemical and other investigations should be used to diagnosis of chest pain as early as possible. Early initiation of targeted therapy reduces mortality and morbidity. Although it is essential to identify all patients with serious conditions, it is also important to control costs and not to subject to unnecessary investigations, in patient care and resultant psychological stress.

REFERENCES
2. Marrow DA. Chest discomfort in Harrison’s principles of Internal Medicine 19th ed Kasper DL, Fauci AS, Hauser SL,


