Acute Myocardial Infarction

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KEYWORDS

- Myocardial infarction • Coronary artery disease • Unstable angina
- Fibrinolytic therapy • Primary percutaneous coronary intervention

KEY POINTS

- Cases of acute coronary syndrome demand rapid treatment which varies depending on the underlying type.
- In the intensive care unit setting, chest pain demands immediate evaluation.
- The differential diagnoses to consider with chest pain are many, some of which are benign; but morbid consequences can ensue if high-risk features are not recognized.
- Many causes may be stratified by a focused history alone; but most presentations of chest pain will still require additional testing, such as electrocardiograms, imaging, and laboratory data for a reliable diagnosis.

APPROACH TO PATIENTS WITH ACUTE CHEST PAIN IN THE INTENSIVE CARE UNIT

In the intensive care unit (ICU) setting, chest pain demands immediate evaluation. The differential diagnoses to consider with chest pain are many, some of which are benign; but morbid consequences can ensue if high-risk features are not recognized. Many causes may be stratified by a focused history alone; but most presentations of chest pain will still require additional testing, such as electrocardiograms (ECG), imaging, and laboratory data for a reliable diagnosis.

Acute myocardial infarction (AMI) is the most feared cause of chest pain, but a wide range of cardiac and noncardiac pathophysiology may explain new-onset chest discomfort. It is important to review the common causes of chest pain in the ICU setting not related to AMI.

Common causes of chest pain in ICU setting

- Pericarditis
- Aortic dissection

- Pulmonary embolism
- Pneumothorax
- Musculoskeletal pain
- Acute myocarditis
- Recommendation for initial assessment
  - A focused history and physical
  - Vital signs (including invasive hemodynamic monitoring if necessary)
  - ECG
  - Chest radiograph or further imaging modality
  - Cardiac biomarkers

Pericarditis

Acute pericarditis refers to inflammation of the pericardial sac, which consists of 2 tissue layers encasing the heart and great vessels in the mediastinum. The potential causes of pericarditis are many and may present as an isolated disorder or part of a systemic process. Although most episodes of pericarditis can be effectively managed with medications, certain high-risk features should be noted in the ICU setting[1]:
High-risk features of acute pericarditis

- Fever and leukocytosis
- Large pericardial effusion or evidence of cardiac tamponade
- Acute trauma
- Recent thoracic surgical procedure or instrumentation
- Anticoagulant therapy
- Immunosuppressed state

Certain specific auscultatory phenomena may occur in acute pericarditis. The pericardial friction rub is the most common finding and is caused by friction between the 2 inflamed pericardial layers.[2] The murmur is generally loudest along the left sternal border and consists of scratching or scraping sounds between S1 and S2.[3]

ECG findings of pericarditis are varied and change with the acuity of the inflammatory process. Typical ECG findings are listed next[4]:

Stage 1 is seen in the first few hours to days and is characterized by diffuse ST elevation (typically concave up) with reciprocal ST depression in leads aVR and V1. There is also an atrial current of injury, reflected by the elevation of the PR segment in lead aVR and depression of the PR segment in other limb leads and in the left chest leads, primarily V5 and V6 (**Fig. 1**).

Stage 2, typically seen in the first week, is characterized by normalization of the ST and PR segments.

Stage 3 is characterized by the development of diffuse T-wave inversions, generally after the ST segments have become isoelectric. However, this stage is not seen in some patients.

Stage 4 is represented by the normalization of the ECG or indefinite persistence of T-wave inversions.

Important distinctions must be made in this setting between ECG features of acute pericarditis and ST-elevation MI (STEMI). The first is that ST elevation in acute pericarditis usually begins at the J point (at the end of the QRS complex and the beginning of the ST segment) and remains concave (see ECG discussed earlier). In STEMI, the ST segments are generally more convex (dome shaped). The ST elevation in pericarditis tends to be widespread, whereas in STEMI the ST-elevation pattern will follow the distribution of a blocked coronary artery. Lastly, the classic PR depression