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Acute Coronary Syndrome in Critical Care

Aim To provide guidance on risk stratification and initial management of suspected acute coronary syndrome in Critical Care

Scope All adult patients in Critical Care with suspected Acute Coronary Syndrome

Suspected Acute Coronary Syndrome (ACS) in Critical Care

Unlikely primary cardiac cause for overall critical illness

Are new ECG changes present?

- ST elevation
- ST depression
- T wave inversion
- LBBB

Yes

Are there other more likely causes for these ECG changes?

For example:
- Sepsis
- Hypoxaemia
- PE
- Stress-induced cardiomyopathy

Yes → **Treat as non-ACS**
Treat other possible underlying causes
Keep monitoring for features of ACS

No

No

Likely or definite primary cardiac cause for overall critical illness

Any of these features present?

VF or VT
ST elevation >1mm
ST depression >2mm

Yes → **Treat as ACS**
Emergency Cardiology Review
Consider dual antiplatelet therapy

Consider clinical status and risk factors

Change in Clinical Status

- New pulmonary oedema
- New cardiac murmur or mitral regurgitation
- 3rd heart sound
- New haemodynamic compromise
- New arrhythmia

Coronary Artery Disease Risk Factors

- Known ischaemic heart disease
- Older age
- Recent history suggestive of undiagnosed angina
- Diabetes, smoking, or peripheral vascular disease
- Hypertension or family history of heart disease

Low Risk
No change in clinical status
No risk factors

- Check Troponin I twice (12hrs apart)
- Monitor for ECG changes

Intermediate Risk
Either change in clinical status
or risk factors present

- Transthoracic Echo
- Serial Troponin I (at 3, 6, and 12 hours)
- Monitor for ECG changes
- **If dynamic ECG changes; Q waves; raised Troponin; or new regional wall motion abnormality, teat as High Risk**

High Risk
Change in clinical status
and risk factors present

- Get urgent Cardiology review
- Consider starting dual antiplatelet therapy

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Explanatory Notes

Acute Coronary Syndrome (ACS) is a difficult diagnosis to make in Critical Care as there are often multiple possible causes for a raised troponin or ECG changes. In addition, there is often a lack of clinical history to put these results into context. Establishing the type of myocardial infarction (see below) and reason for the troponin rise is especially important in Critical Care.[1] Dual antiplatelet therapy with 48 hours of therapeutic heparin is only indicated in ACS suspected to be secondary to coronary artery disease (in specific rupture, erosion or dissection of an atherosclerotic plaque with superimposed thrombus). Dual antiplatelet therapy started inappropriately can have serious consequences.

Definitions and Evidence

Universal Definition of MI adopted by the European Society of Cardiology, American Heart Association, American College of Cardiology and the World Health Federation:

•Diagnosis requires rise and/or fall in cardiac biomarkers + defined evidence of myocardial ischaemia (patient history, ECG changes – ST/T wave changes, new LBBB, pathological Q waves or imaging evidence of new regional wall motion abnormality). [2]

There are 5 described types of MI:

•Type 1 - Spontaneous myocardial infarction related to ischaemia due to a primary coronary event such as plaque erosion and/or rupture, fissuring, or dissection. [2]

•Type 2 - Myocardial infarction secondary to ischaemia due to either increased oxygen demand or decreased supply, e.g. coronary artery spasm, coronary embolism, anaemia, arrhythmias, hypertension, or hypotension. [2]

•Type 3, 4 , and 5 – Relate to Sudden Cardiac Death, PCI and CABG.

Differentiating between Type 1 and 2 MI is very important in Critical Care: as stated above, dual antiplatelet therapy is only indicated in Type 1 MI. Factors that point to a diagnosis of Type 2 MI include presence of another definitive diagnosis known to be associated with an increase in troponin levels (sepsis, PE, intracranial bleeding etc.), lack of sufficient criteria for diagnosis of ACS and troponin T levels constantly elevated at the same level. [3]

Other tools that aid the clinician in differentiating between the two types of MI include early TTE (Regional Wall Motion Abnormality (RWMA) often precedes ECG changes)[4] and establishing the patient's risk factors for having coronary artery disease.[1]

This algorithm incorporates the above guidance and aims to aid clinicians in this difficult area of practice.

References

1. Jeffrey C et al. Treatment of acute coronary syndrome: Part 1 : Non ST-segment acute coronary syndrome. *Critical Care Medicine.* 2011; 39 (10) 2346-2353

2. Thygesen K et al.: Universal definition of myocardial infarction. Circulation 2007, 116:2634-2653.

3. Alcalai R et al: Acute Coronary Syndrome vs Nonspecific Troponin Elevation. *Archives of internal Medicine.* 2007; 167: 276- 281

4. British Society of Echocardiography. *Systolic Function of LV.* http://www.bsecho.org/evaluation-of-systolic-function-of-the-left-ventricle/. (Date Accessed 6/09/15)