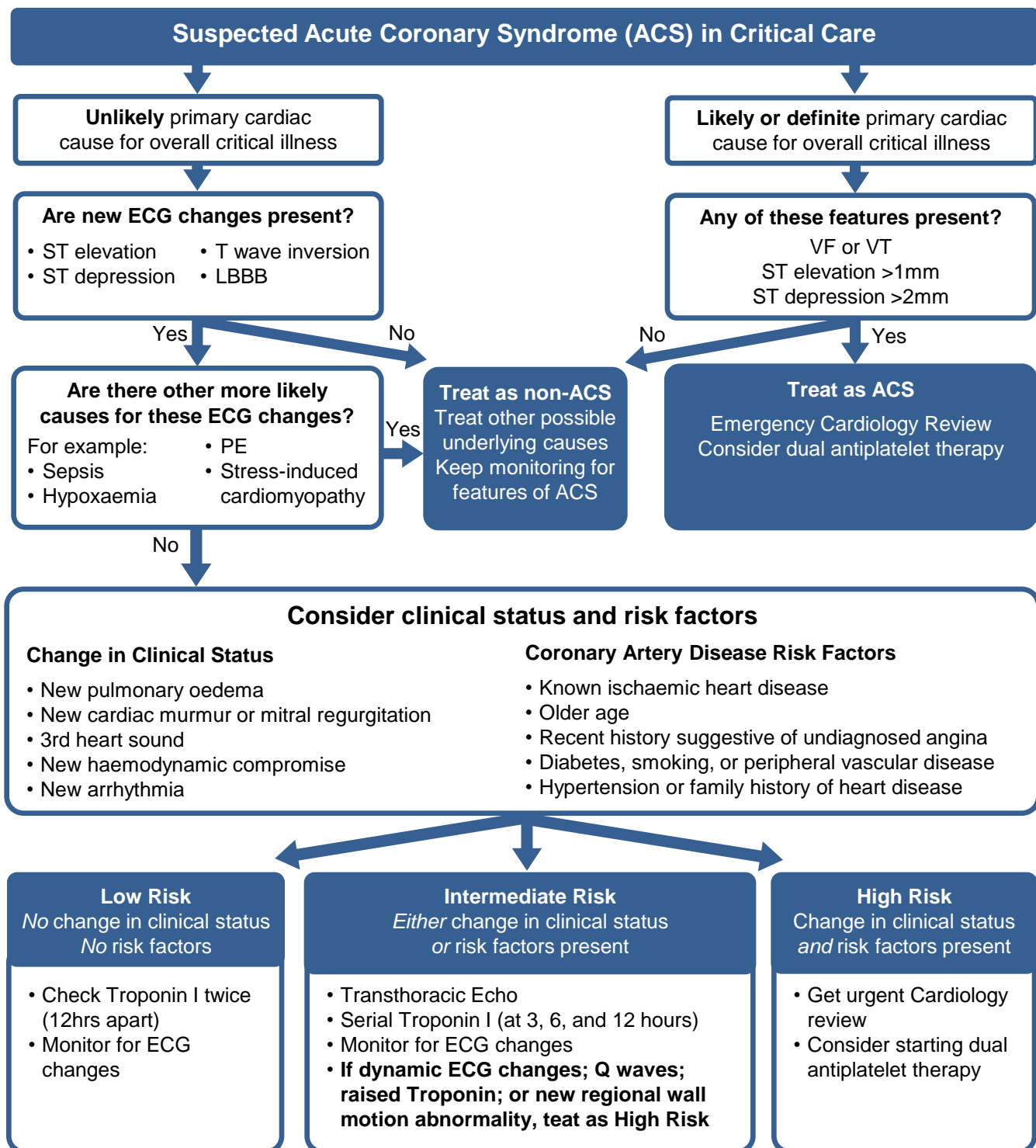


Acute Coronary Syndrome in Critical Care

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



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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

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