NACB Guidelines - Chapter 6: Other Etiologies

1. Biomarkers in chronic renal failure

Background

Increases in the concentration of cardiac troponin T and I in the setting of end-stage renal disease (ESRD) likely represents ongoing cardiac damage in patients with suspected ACS and should be further investigated. These patients have a higher (long-term) risk for death than ESRD patients without an increase in troponin. However, the presence of ischemic injury cannot be inferred unless there is other supporting clinical or laboratory data. Increases in the concentration of B-type natriuretic peptide and NT-proBNP are also observed in patients with ESRD. High BNP and NTproBNP concentrations are likely due to impaired clearance of the peptide. There is insufficient evidence to make recommendations regarding the use of these biomarkers on the clinical management of these patients.

Recommendation

Routine troponin T/I measurement are not warranted in asymptomatic end-stage renal disease patients (Class III). Measurement of the biomarkers in *selected* EDSR patients may be warranted for risk assessment (Class IIB).

Routine BNP/NTproBNP measurement are not warranted in asymptomatic end-stage renal disease patients patients (Class III).

2. Biomarkers in other non-ischemic etiologies

Background

Increases in the concentration of cardiac troponin T and I, and BNP and NTproBNP can be observed in the clinical settings such as sepsis, myocarditis, blunt chest trauma, after cardioversion, and other clinical conditions, and represents ongoing cardiac damage. Several studies have shown that patients with high troponin in non-ischemic etiologies have increased risk of mobidity and mortalilty. Troponin can also be released in patients undergoing chemotherapy such as with the anthracyclines and in patients with congestive heart failure. The prognostic significance of these findings are not established. There is insufficient evidence to make recommendations regarding the impact of minor myocardial damage or left ventricular dysfunction on the clinical management of these patients.

Recommendation

Routine troponin T/I measurement are not warranted in patients with non-ischemic etiologies such as sepsis, myocarditis, after cardioversion (Class III). Troponin T/I measurements are not warranted in patients with CHF or in cancer patients undergoing chemotherapies that are toxic to the heart (Class III). However, increased cardiac telemetry may be warranted in patients who have high troponin following blunt chest trauma (Class IIB).

Routine BNP/NTproBNP measurement are not warranted in patients with non-ischemic etiologies such as sepsis, myocarditis, after cardioversion (Class III).

3. Biomarkers after non-cardiac surgery

Background

Ischemic myocardial damage can occur in patients undergoing surgery that does not involve the myocardium. Creatine kinase and CK-MB are not appropriate markers for ischemic myocardial complications because these enzymes are released from skeletal muscle injury. Cardiac troponin is specific to heart damage and is not normally released in non-cardiac surgery.

Recommendation

Cardiac troponin T and I is recommended for patients undergoing non-cardiac surgery if on an individual basis, there is a question of cardiac ischemia. Cutoff concentrations that are used for diagnosis of AMI are appropriate (Class IIB).

4. Biomarkers after PCI

Background

Cardiac troponin can be released after revascularization (PCI) and higher cutoff concentrations than for AMI should be used to indicate the presence of ischemic injury. There is clinical risk associated with increased concentration of biomarkers after PCI and CABG.

Recommendation

It is appropriate to measure cardiac troponin T/I before and after percutaneous coronary intervention (PCI) to determine the presence of ischemic cardiac injury. A higher cutoff concentration, defined as some multiple of the upper reference limit, is appropriate (Class IIB). There is insufficient evidence to recommend the specific cutoff at this time.