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Editorial

Perioperative Cardiac Assessment for Non-cardiac Surgery: Black Hole of Cardiology Practice?

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Abbreviations

LVF: Left Ventricular Failure; STEMI: ST Elevation Myocardial Infarction; CK: Creatine Kinase; CK-MB: Creatine Kinase- Myocardial Band; CVD: Cardiovascular Disease; RCRI: Revised Cardiac Index Score; METs: Metabolic Equivalents; MINS: Myocardial Ischemia After Non-Cardiac Surgery; eGFR: Estimated Glomerular Filtration Rate; CAD: Coronary Artery Disease; PAD: Peripheral Artery Disease; CHF: Congestive Heart Failure; LBBB: Left Bundle Branch Block; BNP: Brain Natriuretic Peptide

When a cardiologist gets a call to assess cardiac status of a patient for non-cardiac surgery, what are the goals of assessment and how to assess? Do we really know the answer?

Till a decade back we used to think that aim is to prevent a cardiovascular event which mainly included various forms of acute coronary syndrome and acute left ventricular failure (LVF). The acute coronary syndromes mainly included ST elevation myocardial infarction (STEMI) and Non-STEMI. The definition of Non-STEMI was based upon ischemic symptoms and ECG changes with some help from cardiac markers like creatine kinase (CK) and creatine kinase- myocardial band (CK-MB). With the emergence of troponins as primary markers of cardiac injury, the definition and concept of acute coronary syndromes changed but we still used to look at clinical ischemic syndromes and LVF as primary events.

The evaluation of such patients used to begin with history of symptoms suggestive of cardiac disease and history of risk factor or presence of established cardiovascular disease (CVD). A number of scores were developed based upon the number of risk factors and established CVD like Revised Cardiac Index Score (RCRI). It was followed by a diligent examination which was supplemented by electrocardiogram and chest X-ray. In absence of any abnormality, further risk stratification was based upon the type of surgery. Patient undergoing low risk surgery required no further evaluation. Patients undergoing moderate and high risk surgeries were evaluated for their exercise capacity. Patients with exercise capacity of 10 metabolic equivalents (METs) or more were considered suitable for undergoing moderate as well as high risk surgeries. Patients with exercise capacity less than 4 METs were referred for stress testing but there was no clear consensus on this issue even between major guidelines (American versus European). Patients with exercise capacity between 4 - 10 METs were generally considered eligible for surgery though there was scant literature to support this recommendation [1,2].

The above mentioned approach had a number of conceptual and practical flaws. The recommendation for stress testing based upon three or more than three risk factors from RCRI is difficult to apply in current scenario, as one of the criteria namely "history of angina pectoris" is itself sufficient indication to perform further testing. Similarly, one 'standard exercise capacity limit' cannot be applied to different subjects with different age and gender. 10 METs of exercise for a 60 years old female is too much, however, for a 40 years old male

it is just adequate. In addition to these, literature does not consider exercise capacity to be a good marker of perioperative cardiac risk. No correlation between functional capacity as assessed in METs and perioperative adverse cardiac events was found in one of the studies [3]. Similarly, inability to climb two flights of stairs conferred worse prognosis for patient undergoing thoracic surgery but had no negative implications for non-thoracic surgeries [4]. Moreover, stress testing detects only flow limiting obstructive coronary artery disease while we know that severe pre-existing obstruction is not major contributor to myocardial ischemia/ infarction in majority of perioperative cases.

Therefore, despite rampant use of echocardiography and stress testing in preoperative patients, there was neither sufficient data to recommend their use nor there were strong conceptual basis for doing so.

With the increased use of troponins as major cardiac biomarkers, we tried to redefine perioperative myocardial ischemia and then came the landmark VISION study. In VISION trial, myocardial ischemia after non-cardiac surgery (MINS) was defined as troponin T level more than 0.03 ng/ml, within 3 days after the surgery. Non-cardiac causes of troponin elevation were excluded like pulmonary thromboembolism, stroke, sepsis, cardioversion etc. MINS was seen in 8% of patients. In these patients, 30-day mortality was 9.8% which was 9 times higher as compared to those without troponin T elevation. Out of these 8% patients with MINS, only 15.8% had ischemic symptoms and 58% did not fit into universal definition of myocardial infarction. Deaths related to MINS constituted one-third of all deaths in study. The predictors of MINS included age more than 75 years, current atrial fibrillation, eGFR < 60 ml/min, urgent or emergent surgery and history of cardiovascular risk factors (Diabetes mellitus, hypertension) or cardiovascular disease (CAD, PAD, stroke, CHF). Predictors of death in patients with MINS included age more than 75 years, ST elevation or LBBB and anterior ischemic electrographic findings [5]. In a larger study, Beattie., *et al*. found incidence of post-operative troponin I elevation to be 20.4% in a population of 51701 which incrementally predicted 30-days mortality [6]. In a more recent study, troponin elevation was seen in 16% of study subjects, though this study did not excluded extra-cardiac causes of troponin elevation. Typical chest pain was present in only 6% of those with troponin elevation and only 18% had any ischemic symptom. Those with troponin elevation had a 30-days mortality of 8.9% as compared to 1.5% in those without troponin elevation. In subgroup analysis, 30-days and 1 year mortality was more in extra-cardiac subgroup than cardiac subgroup of troponin elevated patients. Similar to VISION study, troponin elevation was commoner in non-elective surgeries, high risk surgeries and in those with higher number of cardiovascular risk factors [7].

Though these studies did not investigate coronary angiographic profile of their patients, several other studies found that troponin positivity in perioperative phase was associated with low incidence of significant obstructive CAD. In one such study, out of 1093 patients with myocardial infarction, only 281 were found to have significant obstructive CAD. All these subjects had a non-cardiac surgery within last 7 days of angiogram. Importantly myocardial infarction was defined as rise of troponin T more than five times the upper limit of normal [8]. It is very much evident that incidence of significant obstructive CAD is low even in those with relatively higher troponin levels. Therefore, likelihood of significant obstructive CAD will go down further if we consider everyone having troponin above the normal limit of test.

In fact, studies examining pathophysiology of perioperative myocardial infarction (PMI) also points towards this. More than 80% of perioperative myocardial infarction occur early after surgery, are asymptomatic, preceded by ST depression than elevation and of Non-Q wave type. Prolonged duration of ischemia causing ST depression plays a rather important role in this scenario. The etiology of perioperative myocardial infarction is multifactorial. The perioperative stress induces unpredictable and unphysiological changes in sympathetic tone, cardiovascular performance, coagulation and inflammatory response. These changes induce erratic alterations in plaque morphology, function and progression. Simultaneous perioperative alterations in homeostasis and coronary plaque characteristics may cause a mismatch of myocardial oxygen supply and demand by numerous mechanisms [9]. In one of the observational studies on PMI after vascular surgery, all incidences of PMI were preceeded by increased in heart rate and two-third of them occurred at end of surgery and emergence of anesthesia [10].

Further on, not only significant coronary events cause rise in troponins but various other etiologies are associated with rise of troponins including sepsis, pulmonary embolism, renal dysfunction, gastro-intestinal bleeding, acute respiratory failure, stroke, structural heart disease or a combination of these. Lower ranges of post-operative troponin elevation have been proposed to be associated with non-cardiac complications while the higher ranges of elevation are predictive of cardiac complications, though all these elevations are associated with increased mortality [11].

These observations explain that why an approach based on just identifying obstructive coronary artery disease during preoperative evaluation was a failure and why echocardiography and stress testing was not the answer to perioperative risk assessment. In fact most recent Canadian guidelines removed echocardiography and stress testing from their recommendations for perioperative evaluation as they found brain natriuretic peptide (BNP) as stronger risk predictor of perioperative cardiac events as compared to echocardiographic variables. Canadian guidelines recommended testing for BNP or NT-proBNP in those with age more than 65 years, significant CVD or RCRI of 1 or more. It also recommended measuring troponin post-operatively, from day 1 to 3, in those with raised BNP or NT-pro BNP. Those subjects who were candidates for BNP measurement but could not undergo testing prior to surgery should also have troponins tested postoperatively [12]. Therefore, not only the preoperative evaluation has changed, the outcome at which we were looking also changed. Rather than a clinical ischemic event we are now looking at troponin elevation, as most of these events are asymptomatic. But we do not have any answer for how to prevent or manage these troponin elevations and eventually prevent mortality. We only have clues that if we can manage emergence from anesthesia in a better way so as to prevent abnormal rise in heart and blood pressure, we may be able to reduce the incidence of troponin elevation. Similarly, incidences leading to spurt in blood pressure and heart rate should be minimized by diligent pain control, prevention of sepsis and pulmonary embolism.

Although, we are now in a better position to detect perioperative cardiac events and know their risk factors, we still do not have any evidence based approach to decrease their incidence and treat such events to prevent mortality. We have thrown almost everything available to us including clinical risk scores, troponins, echocardiography, stress testing and angiograms in this black hole with no fruitful outcome as yet.

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