Mini Review

Interventional Cardiology

Clinical approach to myocardial injury after non-cardiac surgery

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Abstract

Myocardial Injury after Noncardiac Surgery (MINS) is the most frequent cardiovascular complication following noncardiac surgery and independently poses a significant risk of perioperative morbidity and mortality. MINS can be with or without myocardial infarction and is often driven by intraoperative factors such as hypotension, tachycardia, and anemia. Prevention of MINS centers on a thorough preoperative risk assessment and personalized risk-factor modification, including the individualized use of beta-blockers, antiplatelet agents, statins, and ACE inhibitors/ARB. Routine postoperative troponin surveillance for high-risk individuals can facilitate early detection, closer monitoring, and timely intervention. Despite an absence of consensus on a standardized treatment approach, recent evidence suggests that interventions, such as early cardiology consultation and judicious use of moderate-intensity anticoagulation, could improve outcomes. This brief review aims to enhance understanding of MINS and offer a potential framework for approaching these patients.

Keywords: Myocardial injury • Cardiovascular diseases • Myocardial infarction • Electrocardiographic

Introduction

Over 300 million major surgeries are performed every year, 85% of which are noncardiac [1]. Among American adults undergoing noncardiac surgery, over 70% have ≥ 1, and nearly half have ≥ 2 cardiovascular risk factors [2]. As annual surgical volume continues to rise in an increasingly aging population, perioperative Major Adverse Cardiovascular Events (MACE) present a major threat within 30 days of surgery [3].

Myocardial Injury after Noncardiac Surgery (MINS) is relatively common, with incidence>10%, and encompasses myocardial injury with and without Myocardial Infarction (MI). Even in the absence of ischemic symptoms or Electrocardiographic (ECG) changes, it still contributes significantly to both short- and long-term morbidity and mortality. Apart from its often silent presentation, which makes recognition more challenging, there also continues to be ambiguity about an optimal approach to the clinical management of MINS [4-9].

Literature Review

Clinical presentation

Cardiac biomarkers, such as Troponins (cTn), are extremely sensitive and have long been used to identify the presence of myocardial damage. With the advent of high-sensitivity Troponin (hsTn) assays, their use has gained popularity, although there is concern about specificity. These biomarkers are frequently elevated in the perioperative setting [10-12]. Increased inflammation, coagulopathy, and physiologic stress unique to the perioperative period can leave cardiac myocytes vulnerable to injury. This is particularly clear in the presence of pre-existing Atherosclerotic Cardiovascular

Disease (ASCVD), as the effects of hypotension, tachycardia, and vasoconstriction are exacerbated at the level of the myocardium [13-16].

MINS is defined as cardiac myocyte injury due to ischemia, indicated by elevated cTn or hsTn, within 30 days of noncardiac surgery. It excludes non-ischemic causes such as sepsis, renal failure, and pulmonary emboli. There are two main mechanisms for perioperative myocardial injury. Physiologic changes can exact shear stress and promote coronary thrombus formation at the site of a disrupted plaque. This is referred to as Type 1 injury. However, its Type 2 counterpart, which results from sustained myocardial oxygen supply-demand mismatch, is more common perioperatively [6,17-19].

Diagnostic and prognostic troponin thresholds for MINS were established using vascular events in noncardiac surgery patients cohort evaluation (VISION) study data [5,20]. In the absence of a clear non-ischemic etiology, the diagnosis of MINS can be made with a measured troponin exceeding the 99th percentile of the Upper Limit of Normal (ULN) for the assay being used within 30 days of noncardiac surgery. The presence of clinical signs of ischemia is not a prerequisite for diagnosis [6]. When MINS is accompanied by clinical evidence of myocardial cell death, e.g., angina/angina equivalent, ischemic ECG changes, regional wall motion abnormality on echocardiography/imaging, or evidence of coronary thrombosis on angiography, the diagnosis of Perioperative Myocardial Infarction (PMI) can be made (Table 1). The majority of PMI cases can be attributed to Type 2 MI from supply-demand mismatch, which contrasts with non-operative MI, where Type 1 MI is more prevalent [15,21,22].

Table 1: Diagnostic criteria for myocardial injury after noncardiac surgery and perioperative myocardial infarction.

MINS [5,6,20]	Detection of a rise and/or fall of cTn of hsTn exceeding 99th percentile ULN
	Within 30 days of noncardiac surgery
	No evidence of non-ischemic etiology, e.g. sepsis, pulmonary embolism etc.
	Does not require additional ischemic features (symptoms, ECG, imaging, etc.)
PMI [8,15,21]	MINS that also satisfies the Fourth Universal Definition of Myocardial Infraction:
	Rise and/or fall of cTn of hsTn with ≥ 1 measurement exceeding 99th percentile ULN and ≥ 1 of the following:
	Angina or angina equivalent
	New ischemic ECG changes or pathologic Q waves
	New wall motion abnormalities on echocardiography/imaging
	Coronary thrombosis on angiography or autopsy

Note: MIN: Myocardial Injury After Noncardiac Surgery; PMI: Perioperative Myocardial Infarction; cT: cardiac Troponin; hsTn: high sensitivity Troponin; ULN: Upper Limit of Normal; ECG: Electrocardiogram.

Outcomes

MINS's clinical significance lies in its impact on patient outcomes. Patients diagnosed with MINS face an over 8-fold increase in in-hospital mortality compared to those without it [23]. Within 30 days of surgery, mortality risk is over five times higher for MINS [6,7,23,24]. This elevated risk persists long-term, with a fourfold increase in 1 year and a gradual reduction to just over double beyond that timeframe [23,24].

However, the repercussions extend beyond just mortality. Patients diagnosed with MINS face an increased risk of additional complications, including nonfatal cardiac arrest, congestive heart failure, and stroke [6,7,19,23,24]. Although the presence of ischemic features portends a worse prognosis, even in their absence, elevated postoperative cTn alone (MINS without infarction) is associated with poor outcomes [6].

A better understanding of the etiology of MINS is imperative, as the underlying origin can influence outcomes and impact the timing of potential interventions. A proposed classification for cardiac causes of MINS designates Acute Heart Failure (AHF) and tachyarrhythmia as additional subtypes alongside Type 1 and Type 2 injury [18]. Cases with clinical or laboratory evidence of congestion for which diuretics are being considered can be classified as AHF, and those proceeded by tachycardia with sustained rates>120 bpm can be designated as tachyarrhythmia-induced. Studies indicate that all etiologies are linked to increased rates of MACE and mortality at one year compared to no MINS [18,19]. However, type 2 injury is shown to have lower rates than its counterparts. Furthermore, MACE arising from AHF and Type 1 injury typically occurs within days of diagnosis, while those from tachyarrhythmia and Type 2 injury tend to manifest around two weeks later.

Myocardial injury fulfilling the universal definition of Perioperative Myocardial Infarction (PMI), as expected, also significantly impacts patient outcomes. Interestingly, however, the 30-day and 1-year mortality rates for MINS with or without infarction show no significant difference [24]. Nonetheless, PMI is associated with prolonged hospital stays, increased readmission rates, and higher risks of nonfatal cardiac arrest, congestive heart failure, and all-cause mortality [4,25,26]. PMI diagnosed within the initial week following surgery carries a greater risk for 30-day mortality than PMI occurring thereafter, highlighting the potential importance of closely monitoring high-risk patients during the immediate postoperative period. Mortality is highest early in the disease course, with the majority of PMI-related deaths occurring within two days of diagnosis [4,27]. Even when compared to non-operative myocardial infarction, PMI exhibits higher rates of both short- and long-term adverse outcomes, including increased acute kidney injury, venous thromboembolism, and mortality [27].

Preoperative risk assessment and reduction

Identifying at-risk patients in the perioperative period is crucial for clinical decision-making. Factors contributing to MINS risk include patient age, gender, comorbidities, preoperative biomarker levels (e.g., creatinine), and surgery-specific risk (Table 2) [4,6,19,28-34] The Revised Cardiac Risk Index (RCRI) and Gupta Preoperative Risk Score are two of the many well-validated prognostic models for predicting perioperative MACE [35,36]. The RCRI, however, is the only one that has shown an explicit correlation for MINS development [19,37]. For this reason, it is helpful for risk stratification but is most effectively used as a supplementary tool to comprehensive clinical management.

Table 2: Common factors that increase risk for myocardial injury after noncardiac surgery.

Patient characteristics [4,6,29,33]	Advanced age
	Male
	Poor functional capacity (DASI ≤ 34)
Comorbidities [4,6,30,34]	ASCVD
	Diabetes mellitus
	Hypertension
	Heart failure
	Atrial fibrillation
	Chronic kidney disease ≥ stage III
	Uncontrolled Obstructive Sleep Apnea
Elevated biomarkers [4,6,31,32]	B-natriuretic peptide
	Glucose
	Creatinine
Surgery-specific [4,6,19,28]	Emergent surgery
	Higher risk surgery

Note: DASI: Duke Activity Status Index; ASCVD: Atherosclerotic Cardiovascular Disease.

The American College of Cardiology (ACC) and American Heart Association (AHA) have outlined systematic guidelines, including appropriate testing, for preoperative assessment of coronary disease before noncardiac surgery [38-40]. In asymptomatic patients slated for low-risk surgery, all routine testing, including ECG, is unnecessary. However, for patients scheduled for intermediate- or high-risk surgeries, particularly those with pre-existing cardiac disease, a comprehensive clinical evaluation is required. Routine cardiac testing, including echocardiography and stress testing, should be avoided unless clinically indicated with potential to change management. Stress testing can be considered for risk stratification in intermediate-risk patients with poor functional capacity planned for high-risk noncardiac surgery.

Routine preoperative invasive angiography and Percutaneous Coronary Intervention (PCI) should also be avoided, regardless of patient risk, unless clinically indicated with shared decision-making with the patient and surgeon, as this may delay surgery and increase bleeding and perioperative stent thrombosis risk. Troponin surveillance for MINS is potentially cost-effective [41], and it should be considered before and up to 72 hours after surgery for patients at increased risk (e.g., RCRI>2).

MINS Prevention

In addition to thorough preoperative evaluation, the hallmark of MINS prevention lies in averting intraoperative factors that can promote myocardial injury. More specifically, strategies that mitigate physiologic changes, such as hypotension (mean and systolic pressures less than 65 and 100 mmHg, respectively), tachycardia (Increase of 10 bpm from baseline or absolute heart rate>100 bpm), and anemia (hemoglobin<13 g/dl) should be considered [4,42-44]. However, selecting an optimal approach remains challenging, particularly in light of recent findings from POISE-3, which indicate no significant difference in major outcomes between hypotension avoidance and hypertension avoidance strategies [45].

The preoperative use of anti-ischemic medications for MINS prevention is an area of ongoing discussion [38,39]. While beta-blockers can reasonably prevent intraoperative tachycardia, their introduction is associated with increased rates of clinically significant hypotension and risk of postoperative stroke in beta-blocker-naïve patients. However, in patients on chronic therapy for weeks to months, it is reasonable to continue since rates of all-cause mortality and myocardial infarctions are reduced [46,47]. Statin initiation for patients with risk factors is reasonable-particularly in patients with an indication for statin-due to their safety profile, proven efficacy for prevention and treatment of atherosclerotic disease, and promising trend toward MINS and PMI risk reduction [48-50].

Antiplatelet therapy with aspirin, commonly used for secondary prevention of ASCVD, should not be initiated perioperatively solely for MINS prevention as it does not portend an improved prognosis and only serves to increase bleeding risk. Ideally, it should be interrupted three days before noncardiac surgery and should only be continued where there is a history of PCI and a bleeding risk low enough to allow for it [48,51,52].

Angiotensin-Converting-Enzyme Inhibitors (ACEI) and Angiotensin-Receptor Blockers (ARB) are often held perioperatively to reduce the risk of perioperative hypotension and associated complications. More recently, the POISE-3 trial showed that while withholding ACEI/ARB does reduce rates of intraoperative hypotension, it does not affect the incidence of major cardiovascular complications, including MINS [23,45,53]. For ACEI/ARB therapy, an individualized approach should be employed, wherein the indication for treatment (e.g., resistant hypertension, heart failure) and surgery-specific risks (e.g., blood loss, hemodynamic shifts) are considered.

MINS Treatment

While there is no consensus on how to optimally manage MINS after diagnosis, a multidisciplinary, co-management approach, with prompt referral to cardiology, can improve perioperative outcomes and is strongly advised [54-57]. Early introduction or intensification of anti-ischemic medications after MINS or PMI can improve survival [58]; however, specific directions for MINS not fulfilling PMI criteria remain unclear and rely primarily on observational studies, illuminating the need for randomized trials. Nonetheless, treatment with aspirin and statins has been shown to reduce mortality in both MINS and PMI. This, coupled with their well-established benefit among patients with known ASCVD, makes it reasonable to initiate them after diagnosis, as reflected in Canadian Cardiovascular Society guidelines [4,40,59-61]. The benefit of non-aspirin antithrombotic therapy for Acute Coronary Syndrome (ACS) also seems to extend to MINS. The results from MANAGE, the first international, randomized, placebo-controlled trial designed to investigate the effect of Dabigatran on the prognosis of patients who experience MINS, are encouraging. Notably, Dabigatran 110 mg twice daily was associated with significant risk reduction for composite major vascular complications and non-hemorrhagic stroke, all achieved without a significant increase in major bleeding events. Further studies are certainly warranted, and professional societies are yet to recommend dabigatran use. Still, early moderate-intensity anticoagulation might offer a potential, cost-neutral direction for non-invasive MINS treatment [62,63].

Invasive strategies should be reserved for patients with high-risk features, including markedly elevated biomarkers, persistent ischemic changes, and, mainly, PMI. Evidence suggests that invasive angiography with potential PCI reduces in-hospital mortality among patients with PMI, although at the risk of increased postoperative bleeding [8,64]. Thus, it is essential to distinguish this subset of patients from those with MINS alone without PMI, especially since guidelines for the management of ACS, including potential early angiography and therapy with antiplatelet agents, statins, beta-blockers, and ACE inhibitors/ARBs, can better be applied to them [48,65]. Regardless, trends in clinical practice indicate a preference for a noninvasive approach to PMI, especially when compared to non-operative MI. This conservative management may be attributed to this cohort's relatively silent presentation and the lower proportion of ST-elevation myocardial infarction. Further studies evaluating the role of invasive angiography in MINS and PMI are desired [8,27].

Conclusion

MINS represents the most prevalent postoperative cardiovascular complication, affecting over 10% of all patients undergoing noncardiac surgery. It is diagnosed when a troponin elevation within 30 days of surgery is attributed to myocardial ischemia, and it encompasses a spectrum of disease severity, including perioperative myocardial infarction. MINS significantly heightens the risk of all-cause mortality and cardiovascular complications, even when compared to similar non-operative myocardial injury.

Recognition of risk factors and mitigating perioperative changes affecting myocardial oxygen supply and demand is important for MINS prevention. Given its often silent clinical presentation, early disease recognition poses challenges, emphasizing the importance of a high index of suspicion and attentive monitoring of high-risk patients postoperatively, potentially through surveillance with serial troponins. While an optimal treatment strategy has yet to be defined, adopting an individualized, patient-specific approach is essential, seeking expert opinions where appropriate.

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