Royal College of Surgeons of England
ADVANCING SURGICAL CARE

REVIEW ARTICLE

DOI: 10.1308/rcsann.2026.0070

Interpreting troponin elevation after surgery: diagnostic challenges and a pragmatic approach

Introduction

Cardiac troponin is a highly sensitive marker of myocardial injury, although its interpretation after non-cardiac surgery remains challenging. Postoperative troponin elevation is common, frequently asymptomatic and associated with a markedly increased risk of short-term mortality, yet it is often misinterpreted or overlooked in surgical practice.

Methods

We performed a narrative review of contemporary evidence and international guidelines addressing perioperative troponin measurement, myocardial injury after non-cardiac surgery (MINS), and perioperative myocardial infarction (MI), with a focus on their relevance to surgical teams. MEDLINE and Embase were searched for adult English-language studies, prioritising the past 10–15 years and key consensus statements. Studies relevant to postoperative outcomes, risk stratification and management were synthesised into a pragmatic stepwise approach for surgical teams.

Findings

Studies including VISION and POISE demonstrate that even modest postoperative troponin rises are independently associated with substantially increased 30-day mortality, most often in the absence of ischaemic symptoms. Differentiating type 1 MI, type 2 MI and non-ischaemic myocardial injury is difficult in the perioperative setting because clinical features, electrocardiography and baseline troponin values are frequently non-specific. Contemporary European and North American guidelines now support targeted perioperative troponin surveillance in high-risk patients to allow early detection and risk stratification.

Conclusions

Postoperative troponin elevation represents a powerful prognostic marker rather than a single disease entity. A structured, stepwise approach incorporating preoperative risk assessment, serial troponin measurement, clinical correlation and selective imaging can help surgical teams distinguish clinically significant myocardial injury from chronic or non-ischaemic elevation, enabling timely multidisciplinary management and potentially improving outcomes.

Keywords: Troponin; Myocardial injury; Perioperative care; Non-cardiac surgical procedures; Risk assessment

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Introduction

Cardiovascular complications remain a significant source of morbidity and mortality following surgery. An estimated 5% to 25% of patients who undergo non-cardiac surgery have an elevated postoperative troponin level.[1] Elevated troponin may indicate myocardial infarction (MI), demand ischaemia, or simply reflect physiological stress following surgery. However, interpreting elevated troponin in the absence of classical signs of MI presents a clinical dilemma, with few guidelines available addressing this. We aim to review the existing literature, highlight diagnostic challenges and outline considerations for interpreting troponin elevations in surgical patients.

Methods

We undertook a narrative review of the literature on perioperative cardiac troponin measurement, myocardial injury after non-cardiac surgery (MINS) and perioperative MI, with a focus on their implications for surgical teams. We searched MEDLINE and Embase via Ovid, using combinations of the terms “cardiac troponin”, “myocardial injury after non-cardiac surgery”, “MINS”, “perioperative myocardial infarction”, “non-cardiac surgery”, “high-risk surgery” and “perioperative risk stratification”. Searches were limited to adult human studies published in English, with an emphasis on articles from approximately the past 10–15 years to reflect current practice and

Accepted: 7 June 2026

high-sensitivity troponin assays, including key consensus statements where relevant. Particular attention was given to studies reporting postoperative outcomes (including 30-day mortality), differentiating high- and low-risk surgical settings, and describing therapeutic strategies for patients with postoperative troponin elevation. Evidence was then reviewed to develop a pragmatic, stepwise framework for risk stratification, troponin interpretation and early management in non-cardiac surgical patients.

Mechanisms of troponin elevation

Cardiac troponin (cTn) is a biomarker released in the bloodstream following myocardial injury, the mechanisms of which are:

- Type 1 MI: plaque rupture with intraluminal thrombosis causes classical MI. This is relatively uncommon after non-cardiac surgery.[2]
- Type 2 MI: far more frequent perioperatively, this arises from an imbalance between myocardial oxygen supply and demand – triggered by anaemia, hypotension, hypoxia, tachycardia or fluid shifts. Surgery often exacerbates these stressors.[2,3]

Type 1 MI has well-established therapeutic strategies focusing on anti-thrombotic therapy as well as percutaneous coronary intervention, whereas management of type 2 MI relies on the treatment of the underlying cause and symptom control. However, postoperative troponin elevation can also reflect non-ischaemic myocardial injury including:

- Non-ischaemic causes: sepsis, pulmonary embolism or acute/chronic renal dysfunction may cause troponin release without coronary occlusion.
- Physiological stress: the general stress of surgery, including inflammatory and adrenergic responses, can transiently injure cardiomyocytes even in the absence of sustained ischaemia or necrosis.[3]
- Other triggers: mechanical myocardial stretch from fluid overload and pre-existing cardiovascular disease further predispose patients to postoperative troponin rise.

Table 1 Incidence of postoperative myocardial injury by type of non-cardiac surgery.[4,8,9]

Surgical category	Typical incidence of troponin elevation/MINS (%)*	Approximate 30-day mortality risk in those with MINS (%)*
Major vascular	20–30	10–20
Major thoracic	10–20	8–15
Major general surgery	10–20	5–10
Elective major orthopaedic	5–10	5–10
Major urological/gynaecological	3–8	3–8
Low-risk/ambulatory procedures	<3	<3

*Ranges are approximate and derived from large cohort studies and systematic reviews of postoperative troponin elevation and myocardial injury after non-cardiac surgery (MINS).

Therefore, not all troponin elevations indicate an acute coronary syndrome (ACS) – many result from cardiomyocyte stress or reversible injury. Recognising this diversity is crucial for proper clinical interpretation and management.

Incidence and clinical implications

Postoperative troponin elevation is common in patients undergoing non-cardiac surgery, with prevalence estimates ranging from 5% to 25%. The Vascular Events in Noncardiac Surgery Patients Cohort Evaluation (VISION) study, which included more than 7,800 patients with serial high-sensitivity troponin T (hsTnT) measurements, found that a postoperative rise of at least 5ng/l in hsTnT within 3 days was associated with nearly a threefold increase in 30-day mortality (adjusted hazard ratio 2.81). Higher troponin elevations (≥40ng/l) portended an even greater risk, with a 15-fold increase in mortality.[4]

Similarly, the POISE trial, which diagnosed perioperative MI based on troponin rise plus ischaemic criteria, revealed that more than 65% of perioperative MIs were asymptomatic. Both symptomatic and asymptomatic perioperative MIs were linked to markedly increased 30-day mortality risk (adjusted odds ratio ~4.8 and 4.0, respectively).[5] Because many myocardial injuries present without symptoms, the VISION investigators proposed MINS as a diagnostic entity relying on elevated troponin levels attributed to ischaemia, regardless of symptoms.

Risk factors for postoperative myocardial injury include advanced age, pre-existing cardiovascular disease, renal impairment, intraoperative anaemia, hypotension and emergency surgery. These findings underscore the prognostic importance of troponin monitoring in at-risk patients to identify silent myocardial injury and guide perioperative management.[6,7] Together, these studies indicate that even modest postoperative troponin rises identify a group of patients at substantially increased short-term mortality risk, providing a rationale for closer monitoring, multidisciplinary review and timely initiation of cardioprotective therapies where appropriate.

In addition to patient-level factors, the risk of postoperative myocardial injury is not uniform across surgical procedures. Major vascular, thoracic and emergency intra-abdominal procedures carry the highest incidence of postoperative troponin elevation and MINS, whereas low-risk ambulatory or minor procedures in otherwise low-risk patients are associated with much lower event rates.[2,6] Large cohort studies such as VISION and related work have consistently shown greater troponin release and higher 30-day mortality after major inpatient surgery compared with day-case or minor procedures.[4,8,9] This gradient of risk across different surgical settings is summarised in Table 1.

Diagnostic challenges

Diagnosing MINS is complex because a considerable number of patients exhibit asymptomatic or atypical presentations, and perioperative sedation, analgesia or altered mental status can mask classic ischaemic symptoms such as chest pain.[8] Furthermore, clinical signs like tachycardia, hypotension and pain overlap with normal postoperative responses or complications, mimicking and masking signs of myocardial ischaemia.

Electrocardiography (ECG), although routinely used, has limited sensitivity and specificity in this context. Transient and subtle ischaemic changes are common but often overlooked, and postoperative ECG alterations may be non-specific.[10]

There is a lack of clear diagnostic criteria for perioperative MI, particularly for type 2 MI, which is primarily driven by a supply–demand mismatch rather than coronary thrombosis. This ambiguity challenges clinicians in applying the universal definition of MI perioperatively.[11]

Interpretation is further complicated by several confounding variables. For example, chronic kidney disease can cause persistently elevated baseline troponin level, and preoperative troponin levels may not be available for comparison. As such, the diagnosis requires a careful interpretation of troponin result in conjunction with the broader clinical picture.

Approach to interpretation

Elevated troponin in the perioperative period requires careful interpretation within the broader clinical context. This rise reflects myocardial injury and is an important prognostic tool in the postoperative period for cardiovascular complications and mortality. The 2022 European Society of Cardiology guidelines on cardiovascular assessment and management of patients undergoing non-cardiac surgery have recommended use of cTn T and I, to help improve perioperative outcomes.[12] Recognising troponin elevation early facilitates closer haemodynamic monitoring, further investigation or intervention that may improve perioperative outcomes.

Owing to interpretation being nuanced, a structured, stepwise approach is essential for interpreting postoperative troponin elevation.

Preoperative risk assessment and baseline troponin

In patients at greater cardiovascular risk (eg those with established heart disease, age >65 years or undergoing high-risk surgery such as major vascular, thoracic or emergency intra-abdominal surgery), preoperative baseline troponin assessment can help distinguish chronic elevation from acute injury and serves as a meaningful reference point postoperatively.[13]

Timing and trigger for postoperative troponin measurement

Serial troponin measurements are recommended within the first 48–72h following surgery, especially in high-risk individuals or when triggered by clinical suspicion (eg haemodynamic instability, arrhythmias).[14]

Serial measurements and kinetic assessment

Evaluating the rise and/or fall of troponin – ideally compared with a baseline – helps differentiate acute injury from chronic elevations. A perioperative hsTnT rise of ≥14ng/l above preoperative values, or any postoperative hsTnT value ≥65ng/l, is considered diagnostic for MINS in major studies.[4,15]

Correlation with symptoms, ECG findings and clinical context

Many perioperative myocardial injuries are asymptomatic, so an absence of chest pain does not exclude myocardial ischaemia. ECG may show relevant ischaemic changes but can also be non-specific.[8] Therefore, correlating all available findings, including troponin, may provide a clearer interpretation than the troponin, ECG or clinical assessment alone.

Use of imaging when diagnosis is unclear

If uncertainty persists, ECG can assess for new regional wall-motion abnormalities, signs of heart failure or other causes (eg pulmonary embolism, severe valvular disease, pericardial effusion). Additional cardiac or systemic causes must be ruled out to avoid overlooking serious but treatable conditions such as sepsis and acute decompensated heart failure.[8]

Management considerations and recommendations

The consistent association between postoperative troponin elevation, MINS and increased 30-day mortality across multiple large cohort studies underlines that troponin should be viewed as a powerful prognostic marker. Identifying higher risk groups postoperatively allows for real-time stratification of the level of care and management of patients.[11] However, without systematic hsTnT monitoring, MINS – an often asymptomatic condition – remains largely undetected. Implementing such monitoring therefore needs to be cost-effective and beneficial to patient outcomes. One Canadian study found that implementation of such a system was cost neutral and did not increase length of stay.[16]

Although an optimal treatment for MINS remains unclear, implementing existing evidence-based interventions for ACS has demonstrated a mortality advantage.[5,17] This suggests that outcomes in patients with MINS may be modifiable, even if a disease-specific management protocol is not yet available. However, the true extent of impact of specific medical therapies in patients with MINS remains incompletely defined as most evidence for medical therapy is derived largely from observational studies.

From a practical standpoint, therapeutic strategies should be tailored to the presumed mechanism of injury. In patients with features suggestive of type 1 MI (for example new ST-segment elevation or convincing plaque-rupture syndrome), early cardiology input, guideline-directed antiplatelet and anticoagulant therapy, and consideration of urgent coronary angiography and revascularisation are appropriate, balanced against bleeding and surgical risks.[8,12]

Evidence for routine invasive coronary angiography and revascularisation in patients with MINS or perioperative type 2 MI remains limited and largely observational. Invasive coronary angiography may be considered in selected high-risk patients, especially those with markedly elevated troponin, ischaemic symptoms, persistent ECG changes or new wall-motion abnormalities, but routine invasive testing is unlikely to help most patients and the optimal timing remains uncertain.[8,18]

In the more common scenario of type 2 MI or MINS, management focuses on correcting precipitating factors (optimising haemodynamics, treating sepsis, correcting anaemia and hypoxia, controlling tachyarrhythmias) and initiating or intensifying secondary prevention where tolerated, including statins, beta-blockers, angiotensin-converting enzyme inhibitors or angiotensin receptor blockers, and low-dose antiplatelet therapy. For patients in whom troponin elevation is judged to be non-ischaemic (eg isolated sepsis or pulmonary embolism), treatment should target the underlying condition while still prompting cardiovascular risk assessment and optimisation, given the strong association between postoperative troponin elevation and adverse outcomes.[8,12]

Given the prevalence and prognostic value of postoperative troponin elevation, there is a growing need for hospital-specific guidelines to streamline screening, diagnosis and management. Routine perioperative troponin screening – particularly in patients with elevated cardiovascular risks such as those aged >65 years, with pre-existing heart disease, or undergoing major procedures – enables earlier diagnosis and timely intervention.

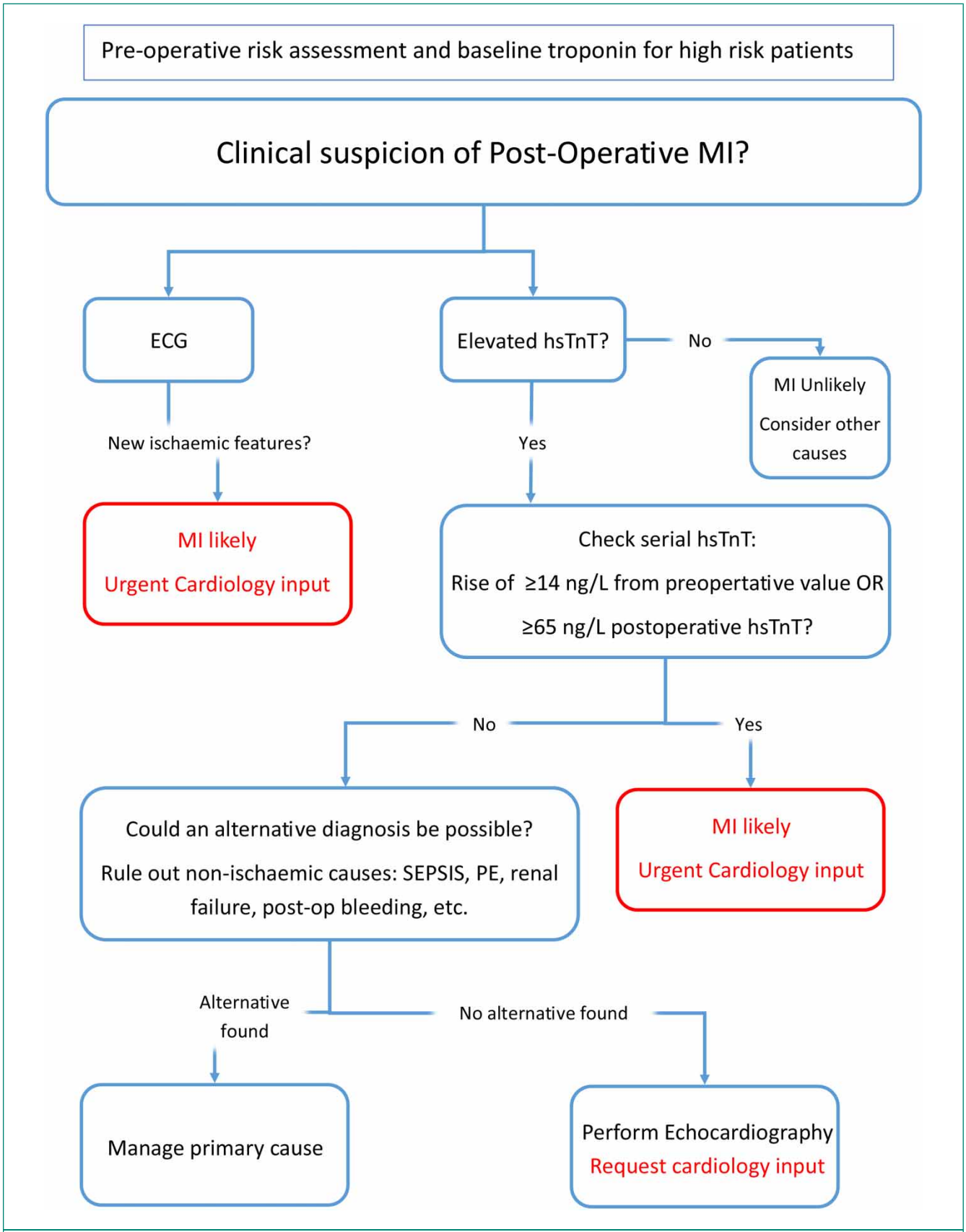

Figure 1 Flow chart for the diagnosis and management of suspected postoperative myocardial infarction with raised troponin level

Although the absence of symptoms has historically led to missed opportunities for management, applying ACS treatment strategies to those with postoperative myocardial injury (even if asymptomatic) can reduce mortality and guide escalation of care.

A stepwise, multidisciplinary approach integrating preoperative risk assessment, baseline troponin measurement, postoperative surveillance, and input from anaesthetics, cardiology and the surgical team optimises detection and

management, ensuring at-risk patients receive appropriate evaluation and therapy.[19]

Conclusion

Myocardial injury is a common and significant complication after non-cardiac surgery and is associated with an increase in postoperative mortality. The interpretation of troponin in the perioperative period requires a nuanced understanding of its pathophysiology and broader clinical picture along with set cut-off criteria. A pragmatic, stepwise approach to management – incorporating preoperative risk evaluation, serial troponin measurements, clinical correlation and appropriate imaging – can assist clinicians in differentiating acute myocardial injury from chronic elevation or non-ischaemic causes and guide management. A flow chart for this is set out in Figure 1.

Competing interests

The author/s declare no competing interests.

Funding

The author/s received no financial support for the research, authorship and/or publication of this article.

Ethics approval and consent to participate

Not applicable.

Author contributions

M Dave: Conceptualisation, Data curation, Investigation, Methodology, Project administration, Writing – original draft, Writing – review & editing. **CM Bailey:** Conceptualisation, Supervision, Writing – review & editing. **K Mokrzycki:** Data curation, Formal analysis, Writing – original draft.

Artificial Intelligence

The author/s declare that no AI was used to conduct the study or prepare the manuscript.

Acknowledgements

The authors would like to thank Dr Bet Mishra, consultant cardiologist, for expert cardiology input, and Mr Shoaib Saeed, FRCS, surgical specialist trainee, for reviewing the manuscript and providing valuable feedback.

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