Acute Myocardial Infarction after Noncardiac Surgery

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Abstract

Over 230 million surgeries are performed annually worldwide, cardiac complications being the most common causes of postoperative morbidity and mortality. As life expectancy has extended worldwide, a growing number of patients with multiple comorbidities have undergone noncardiac surgeries. Consequently, cardiovascular complications associated with those procedures are expected to increase, and perioperative acute myocardial infarction (AMI) might become a frequent problem.

In Brazil, the number of noncardiac surgical procedures has also increased, and approximately three million surgeries are performed annually.

Despite advances in surgical and anesthetic techniques, mortality and costs related to those procedures have also increased, thus, requiring the development of strategies to reduce mortality[4]. The occurrence of perioperative AMI prolongs the need for intensive care and the hospital length of stay, increases the cost of hospitalization, and reduces long-term survival.

This literature review approaches the pathophysiology, incidence, diagnosis and treatment of perioperative AMI based on current evidence.

Introduction

Over 230 million surgeries are performed annually worldwide, cardiac complications being the most common causes of postoperative morbidity and mortality[1]. As life expectancy has extended worldwide, a growing number of patients with multiple comorbidities have undergone noncardiac surgeries. Consequently, cardiovascular complications associated with those procedures are expected to increase [2], and perioperative acute myocardial infarction (AMI) might become a frequent problem[1,3]. In Brazil, the number of noncardiac surgical procedures has also increased, and approximately three million surgeries are performed annually[4].

Keywords

Myocardial Infarction / surgery / complications; Perioperative Care

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Despite advances in surgical and anesthetic techniques, mortality and costs related to those procedures have also increased, thus, requiring the development of strategies to reduce mortality[4]. The occurrence of perioperative AMI prolongs the need for intensive care and the hospital length of stay, increases the cost of hospitalization, and reduces long-term survival[3,5-9].

Since the 1970s, several algorithms have been used to estimate the cardiovascular risk related to noncardiac surgical procedures, such as the Goldman multifactorial index[10], the American Society of Anesthesiologists' (ASA) Physical Status Classification[11], the modified Detsky index[12], and, more recently, Lee's Revised Cardiac Risk Index[13] and the Multicenter Study of Perioperative Evaluation (EMAPO)[14]. Based on those algorithms, flowcharts[15,16] have been created for perioperative assessment, in an attempt not only to establish cardiac risk, but also to guide the indication of the following: non-invasive tests to detect myocardial ischemia; methods to monitor cardiovascular events; and strategies to prevent complications with medications or myocardial revascularization.

Despite the increasing number of studies on perioperative medicine, most of them have focused on the following: risk stratification to predict the occurrence of cardiovascular events[17]; search for markers related to higher surgical risk[18-30]; monitoring for early detection of AMI[7,31-36]; and strategies to reduce the risk of AMI, such as medications (statins[37-44], beta-blockers[44-51], clonidine[52,53]) or even preoperative myocardial revascularization[54,55]. Evidence on the pathophysiology, diagnosis and treatment of perioperative AMI is scarce[56,57].

Pathophysiology

Two distinct mechanisms can lead to perioperative AMI: atherosclerotic plaque instability with rupture and thrombosis; and changes in the myocardial oxygen delivery/consumption ratio in patients with chronic coronary artery disease (CAD) and significant stenosis[1]. In spontaneous AMI, rupture of a vulnerable plaque is usually caused by an increase in luminal shear stress or by an inflammatory process inside the plaque. The vulnerable plaque is formed by a thin fibrous cap and a large amount of macrophages and lipids in the center. Rupture of the fibrous cap exposes the lipids, which leads to platelet aggregation, and, thus, local thrombosis with partial or total reduction in vascular lumen, triggering ischemia and myocardial infarction[56,58]. Emotional stress and extenuating exercise might cause plaque rupture.

In the perioperative period, several factors can contribute to atherosclerotic plaque instability. The levels of catecholamines and cortisol increase after surgery and can remain high for days. Their increase result from pain, anemia, and hypothermia, and

can lead to coronary vasoconstriction and plaque instability. Tachycardia and hypertension are common in the postoperative period and can increase vascular shear stress, leading to rupture of vulnerable plaques. In addition, the following occur: increased levels of procoagulant substances (fibrinogen and von Willebrand factor); reduced levels of anticoagulant factors (protein C, antithrombin III and alpha-2-macroglobulin); and increased platelet aggregation[1]. The increases in surgery-induced procoagulant and antifibrinolytic activities can trigger coronary thrombosis in patients with CAD and reduced coronary flow velocity, even in the absence of plaque rupture[57].

On the other hand, tachycardia, arterial hypertension, hypotension (due to bleeding, hypovolemia or vasodilation), anemia and hypoxemia can lead to AMI due to an imbalance in the oxygen delivery/consumption ratio in patients with obstructive, but stable, coronary lesions[1]. The frequent combination of increased heart rate and depression of the ST segment, detected during Holter monitoring before the event, suggests that prolonged ischemia rather than thrombosis can be the cause of perioperative AMI[57].

However, evidence from clinical studies is scarce. In 1996, Dawood et als.[59] carried out the first study to determine the pathology of perioperative AMI. The study assessed the coronary arteries and the atherosclerotic plaque aspect of 42 patients who had died from AMI occurring in the intraoperative period or within 30 days from surgery. The presence of plaque rupture, plaque hemorrhage or thrombus was assessed. The findings were compared with those of patients who died within the first 21 days following AMI and had not undergone any surgical procedure in the last three months. Twenty-three patients (55%) with perioperative AMI and ten patients (40%) with spontaneous AMI had at least one of those characteristics ($p = 0.31$). The authors have concluded that the mechanism of perioperative fatal AMI would be the same of spontaneous AMI, that is, rupture of an atherosclerotic plaque leading to thrombosis and coronary artery obstruction[59].

In 1999, Cohen et als.[60] confirmed those findings in another anatomical-pathological study with 26 patients who had died due to perioperative AMI, and they reported evidence of plaque rupture in 12 patients (46%). The comparison of the clinical characteristics (age, sex, history of CAD, history of AMI, use of beta-blockers, sudden death, clinical AMI) between patients with and without plaque rupture revealed no difference between the groups. However, the time interval between surgery and death in patients with plaque rupture was longer (7.8 ± 4.4 days versus 4.4 ± 4.8 days; $p = 0.047$). Regarding the anatomical-pathological aspect, in 19% of the cases the AMI was circumferential, possibly related to changes in the oxygen delivery/consumption ratio. However, even in that situation, plaque rupture could not be excluded as a mechanism of AMI, which can be proved by the fact that one of those patients also had evidence of plaque rupture.

Those facts suggest that, to maximize the efficacy of the strategies to reduce the risk of AMI, both mechanisms should be considered as possible therapeutic targets[60]. Considering that most perioperative AMIs occur in the absence of ST-segment elevation on electrocardiogram (ECG), it seems contradictory that, according to the anatomical-pathological study, most AMIs are transmural[57].

In an interesting study, Poldermans et als.[61] have assessed the relationship between the AMI site (on autopsy) after vascular surgery and the ischemia site determined on preoperative stress dobutamine echocardiography. Patients undergoing stress dobutamine echocardiography prior to vascular surgery and dying in the first 30 days after the procedure were included in the study. Patients undergoing preoperative myocardial revascularization after echocardiography were excluded. Of the 32 patients meeting the inclusion criteria, 22 (66%) evidenced AMI on autopsy. Of that sample, five patients had a negative echocardiogram for ischemia, and, in 9 (56%) of the remaining 16, the pathological evidence of AMI was found in a coronary artery territory in which no ischemia had been demonstrated. The authors have concluded that a stress dobutamine echocardiogram positive for ischemia identified patients at higher risk for AMI, and have speculated that its failure in identifying the specific region at risk can be explained by the rupture and instability of less obstructive plaques that caused no flow reduction[61].

Although those anatomical-pathological studies indicate that the major mechanism of perioperative fatal AMI is atherosclerotic plaque instability and rupture, they are retrospective and have included a small number of patients. Ellis et als.[62] have attempted to determine the cause of perioperative AMI in patients surviving those events in a retrospective study with 21 patients experiencing perioperative AMI and 42 controls undergoing vascular surgeries and preoperative coronary angiography. The angiographic characteristics (number, site and aspect of the lesions) were compared between patients with and without perioperative AMI in an attempt to establish which lesions caused AMI. The authors have concluded that most perioperative AMI occur due to insufficiency of collateral circulation to territories with occluded arteries, while a smaller number occurs with no significant obstruction[62]. However, that study has severe methodological limitations, because coronary angiography was performed in the preoperative period and not repeated after surgery. In addition, patients with important lesions might have undergone myocardial revascularization prior to vascular surgery, making the determination of a cause-effect relationship between the lesions found and perioperative AMI very difficult.

In the first clinical study assessing the pathophysiology of perioperative AMI in patients surviving those events, we compared the angiographic characteristics of 120 patients with perioperative acute coronary syndromes (ACS), 120 patients with spontaneous ACS, and 240 patients with stable CAD. Patients with perioperative ACS had fewer Ambrose's type II and complex lesions than patients with spontaneous ACS, but more lesions of that type than patients with stable CAD (Figure 1). Thus, we have shown that approximately 50% of the patients with perioperative ACS have signs of plaque instability as the pathophysiological mechanism, characterizing type 1 AMI. It is worth noting that, in patients with perioperative ACS, the time between the event and coronary angiography was longer than in patients with spontaneous ACS (5.5 ± 8.0 days versus 1.3 ± 1.4; $p < 0.001$, respectively), which might have underestimated the presence of plaque rupture in the perioperative group[63].

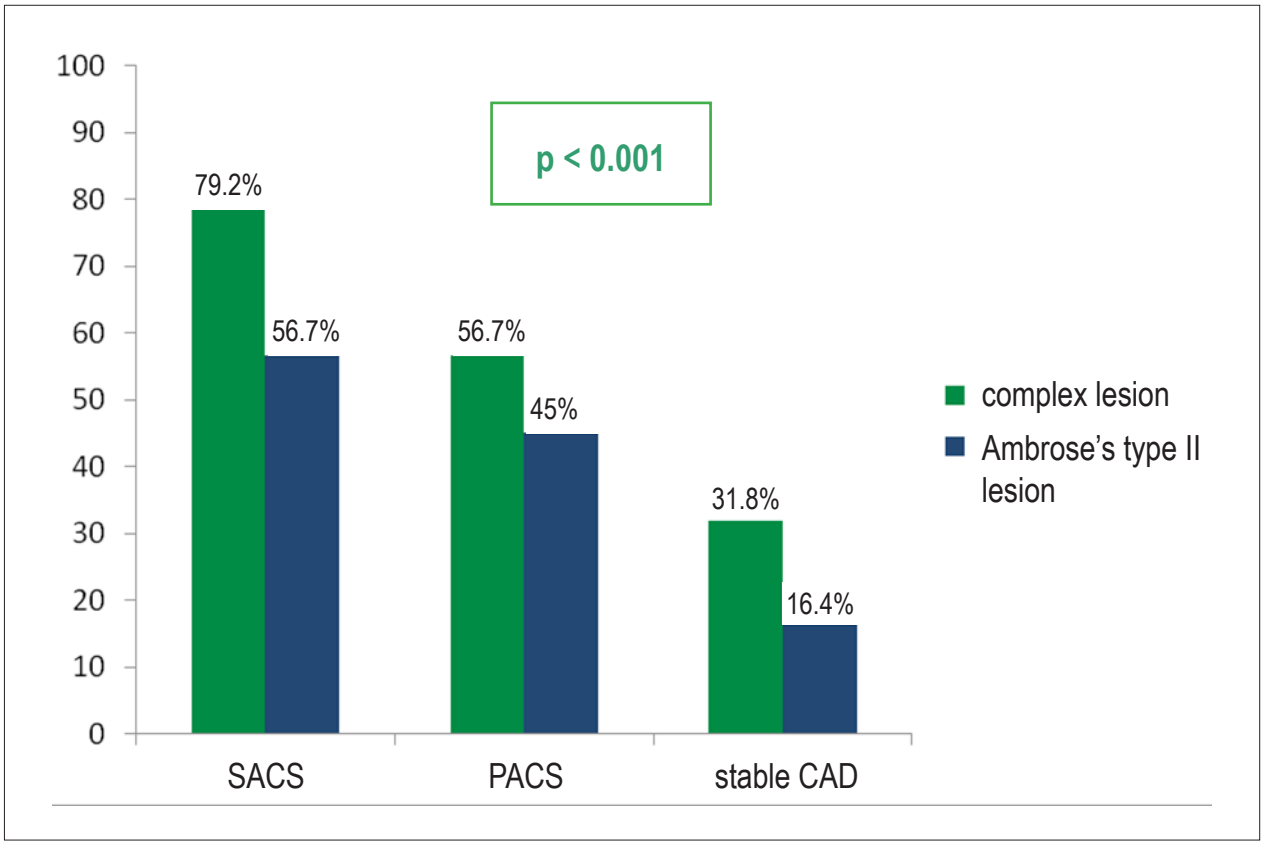

Figure 1 - *Presence of Ambrose's type II and complex lesions on coronary angiography 63; SACS: spontaneous acute coronary syndromes; PACS: Perioperative acute coronary syndromes; CAD: Coronary artery disease.*

Incidence

Data on the incidence, outcome and mortality of perioperative AMI are also controversial. The incidence of perioperative AMI in low-risk patients with no history of CAD ranges from 0.3% to 3%, but can reach 33% in high-risk patients with history of CAD[9,55,64]. That large variation in the incidence of perioperative AMI can be explained not only by the type of the population assessed and the surgery performed[65], but also by the lack of uniformity in the diagnostic criteria adopted by the studies. In addition, perioperative AMI can pass unnoticed if monitoring with serial electrocardiogram and postoperative measurement of troponin are not performed. Patients undergoing vascular surgeries are at higher risk for developing perioperative AMI, because they already have atherosclerosis[3]. In a study of the 1980s, in which coronary angiography was performed in thousand patients undergoing vascular surgeries, the authors reported that 91% of the patients had CAD[66].

In a study with 577 elderly patients undergoing vascular surgeries, the incidence of perioperative AMI was 9.7%[42]. In another study with 570 patients undergoing abdominal aortic aneurysm surgery, the incidence of perioperative AMI was 8.9%[43]. In Brazilian studies with patients undergoing arterial surgeries, the incidence of perioperative AMI has ranged from 9% to 11%[37,67]. Even endovascular procedures to repair abdominal aortic aneurysm have a 7% incidence of AMI[68]. On the other hand, when assessing non-selected populations, the incidence seems to be lower, ranging between 0.27% and 1.8%[5,69,70]. It is worth noting that such incidences might be underestimated due to the retrospective design of those studies, to the different diagnostic criteria used, and the lack of monitoring for the active search for events.

Prospective studies have also shown a variable incidence, although usually higher than that of retrospective studies.

In 1986, Detsky et als.[12], assessing 455 patients over 40 years of age, who had undergone noncardiac surgeries, reported a 3.1% incidence of AMI. In 1990, Shah et als.[71], assessing 275 patients with previous AMI who had undergone noncardiac surgeries, reported a 4.7% incidence of postoperative AMI, and that incidence was 10.9% in the subgroup undergoing vascular surgeries. The diagnostic criterion for AMI was based on the presence of chest pain or electrocardiographic abnormalities associated with elevations in the MB fraction of creatine phosphokinase (CK-MB). Kumar et als.[65], studying 1,121 patients with CAD and performing serial measurements of CK-MB and ECG in the first three postoperative days, have reported a 2.8% incidence of AMI. After the appearance of troponins, they became the myocardial necrosis marker of choice for the diagnosis of AMI; prospective studies monitoring ECG and troponin in the postoperative period have reported AMI incidences between 3.6% and 5.6%[44,50,72,73].

Clinical findings and diagnosis

The risk of AMI is higher in the first three postoperative days, and most events occur in the immediate postoperative period or in the following day[3,57,58]. In the study by Kikura et als.[69], most events (57.4%) have occurred on the day of surgery or on the first day after surgery. Regarding the classification of AMI, 91 patients (74.6%) had non-ST-segment elevation AMI, and 31 patients (25.4%) had ST-segment elevation AMI. Those authors have concluded that patients with perioperative AMI are usually men, who had undergone high-risk surgery, with history of CAD and heart failure, and higher Lee's Revised Cardiac Risk Index[69].

In our study with 120 patients with postoperative ACS, we have shown that 71.7% of the patients experienced the event in the first 72 hours as follows: 19 (15.8%) patients had unstable angina; 94 (78.3%) had non-ST-segment elevation AMI; and 7 (5.8%) had ST-segment elevation AMI[63]. Devereaux et als.[73] have also shown that, in 415 patients with perioperative AMI, 74.1% of the events occurred in the first 48 hours and only 10.6% of the patients had ST-segment elevation AMI.

Regarding symptoms, in their study, Devereaux et als.[73] have reported symptoms of ischemia in only 34.7% of the patients. In our study, 40.7% of the patients complained of chest pain[63]. Thus, AMI occurs most often in the first postoperative days, is asymptomatic, and has no ST-segment elevation[56,57,63,73]. The in-hospital mortality rate of perioperative AMI can vary from 11% to 25%, being higher in patients with greater troponin elevation[1,5,8,63,73].

Diagnosing perioperative AMI is difficult, because in 50% to 61% of the cases the characteristic chest pain is either absent due to sedation and cognitive changes or is attributed to surgical wound[10]. In addition, unspecific changes in the ST segment related to electrolytic imbalance, hypothermia, pericarditis or chest trauma, hyperventilation, effect of drugs or changes in decubitus position can occur.

Regarding the markers of myocardial necrosis, sensitivity and specificity of CK-MB are low in the perioperative period, in which skeletal muscle injury and frequent elevations in creatine phosphokinase (CPK) occur. On the other hand, increased troponin levels indicate myocardial injury, but not always result from coronary artery lesions causing ischemia and necrosis. Troponin levels can be elevated in the following situations that can occur in the perioperative period: pulmonary thromboembolism; decompensated heart failure; sepsis; myocarditis; renal failure; shock; and pericarditis[3,74]. In addition, defining perioperative AMI is difficult because most AMIs occur without chest pain, the ECG changes can be transient and pass unnoticed, and several clinical situations can change the myocardial necrosis markers. Thus, AMI is usually recognized late, explaining its high morbidity and mortality.

In 2007, the universal definition of AMI was published, when diagnostic criteria of AMI were revised, and a clinical classification for AMI was proposed. In that classification, AMI was divided into five types, allowing the development of treatment strategies specific for each group, because there are different pathophysiological mechanisms for each type[75]. In our opinion, AMI occurring after a noncardiac operation was not contemplated in that classification[76]. The medical knowledge of the pathophysiology of AMI has evolved considerably in the last decades, but the same has not occurred with perioperative AMI. So far, the exact pathophysiology of AMI occurring after noncardiac surgery has not been completely clarified. Evidence suggests that, in approximately 50% of the cases, it falls into type 1 category[63].

Perioperative AMI should be diagnosed in the presence of an elevation and a decrease in myocardial necrosis markers (preferably troponin) with at least one measure over the 99th percentile of the upper reference value, associated with: symptoms of ischemia or ECG changes indicating ischemia (alterations of the ST segment or new left bundle-branch block); or development of Q wave on ECG; or new change in segmentary contractility on echocardiogram[75]. In future studies about perioperative AMI, we expect those criteria are used to provide a better estimate of the incidence and outcome of perioperative AMI.

In addition, AMI should be differentiated from the isolated elevation of troponin, which is an entity characterized by elevated troponin levels with no clinical manifestations and no ECG changes, in the absence of other clinical situations that explain that increase. Although the isolated elevation of troponin also has implications on long-term prognosis, the patient should not receive treatment for AMI, but undergo additional cardiological stratification before hospital discharge[32,36,77].

According to the algorithms of perioperative assessment aimed at the early detection of perioperative AMI[16], monitoring with ECG and daily troponin measurement up to the third postoperative day is indicated to patients at intermediate to high cardiovascular risk.

Treatment

There are no randomized studies specifically directed at perioperative AMI. Usually, the guidelines for the treatment of spontaneous ACS are used, observing some specific considerations of the perioperative setting[16]. Perioperative ACS should be divided into two types: with and without ST-segment elevation.

To patients with ST-segment elevation, the use of thrombolytic agents is formally contraindicated because of the risk of bleeding[78]. The treatment of choice in such cases is primary angioplasty, as long as the patient has no active bleeding that contraindicates the use of clopidogrel[1,3,16]. Berger et als.[79] have specifically studied the clinical outcome and prognosis of 48 patients undergoing coronary angiography immediately after AMI occurring up to the seventh postoperative day, obtained from the database of the catheterization laboratory. Thirty-three patients (68.8%) had ST-segment elevation, four patients had new bundle-branch block, eight had ST-segment depression, and three had other alterations. In 32 patients (66.7%), the culprit artery was completely occluded, and 30 patients (62.5%) showed evidence of thrombus. The mean time between symptom onset and coronary angiography was four hours. Forty-one patients (85.4%) underwent angioplasty and three patients underwent coronary bypass graft surgery (6.25%). Two patients (4.2%) showed normal coronary arteries. Thirty-five patients (65%) survived until hospital discharge. The mean hospital length of stay was 13 days. It is worth noting that, in that study, only severely ill patients were assessed as follows: 21 patients had had cardiogenic shock and 12 patients had had cardiorespiratory arrest prior to coronary

angiography. Those authors have concluded that an immediate invasive strategy with angioplasty is safe and can reduce the mortality of selected patients with perioperative AMI[79].

In patients with perioperative ACS without ST-segment elevation, extrapolating the recommendations for the treatment of spontaneous ACS[80], antiplatelet therapy with acetylsalicylic acid (ASA) and clopidogrel and anticoagulation with low molecular weight **or** unfractionated heparin are used. However, the benefit of anticoagulation should be always weighed against the risk of bleeding.

Despite the lack of randomized studies in the perioperative setting, when the risk of bleeding is high, unfractionated heparin should be preferred because its effect can be rapidly reverted in cases of bleeding. The use of glycoprotein IIb IIIa inhibitors is not recommended due to the lack of studies on the safety of their use in the perioperative period.

In our study, bleedings occurred as follows: of the 120 patients with postoperative ACS, 11 (9.2%) bled (six major bleedings according to the TIMI classification and including two fatal, and five minor bleedings); and of the 120 patients with spontaneous ACS, 10 (8.3%) bled (six major bleedings and four minor bleedings, none fatal; p = 0.09). It is worth noting that only three patients had bleedings related to the surgical site, and five patients had them related to the gastrointestinal tract.

Although the groups did not differ significantly, the primary objective of this study was not the assessment of bleeding, and the sample size was small to reach definitive conclusions. Another aspect is that anticoagulation with heparin was initiated only when the clinical and surgical teams agreed upon it; 99.2% of the patients with perioperative AMI received ASA and 65% received clopidogrel, but only 86.7% of the patients received some type of full heparinization[63]. Thus, platelet antiaggregation and anticoagulation in perioperative AMI should be performed carefully and after an interdisciplinary discussion of the risk/benefit ratio.

In addition, the correction of secondary factors, such as anemia, pain, tachycardia and hypertension, should be considered[3,16]. The use of beta-blockers, statins, angiotensin-converting-enzyme inhibitors and aldosterone blockers should respect the same indications for spontaneous AMI[80]. To hemodynamically stable patients, a conservative strategy can be attempted at first, with correction of all secondary factors and medications, and invasive stratification before hospital discharge. However, patients with a poor prognosis should undergo invasive strategy as early as possible[3,16].

There is anatomical-pathological and clinical evidence that a significant number of perioperative AMIs would be type 1. Thus, those patients could benefit from platelet antiaggregation, anticoagulation and early invasive strategy, just as patients with spontaneous AMI[59,60,63]. However, in some patients, plaque rupture was not detected. Thus, in addition to including the use of platelet antiaggregant drugs and anticoagulants, and performing invasive strategy for myocardial revascularization, the treatment of perioperative AMI should be complemented with the correction of the secondary factors, such as anemia, hypertension and tachycardia. Based on this, we hope to reduce the occurrence of perioperative AMI and its high morbidity and mortality.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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