Coronary plaque rupture in patients with myocardial infarction after noncardiac surgery: Frequent and dangerous

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A B S T R A C T

Purpose: The pathophysiology of acute coronary syndromes (ACS) after noncardiac surgery is not established yet. Thrombosis over a vulnerable plaque or decreased oxygen supply secondary to anemia or hypotension may be involved. The purpose of this study was to investigate the pathophysiology of ACS complicating noncardiac surgery.

Methods: Clinical and angiographic data were prospectively recorded into a database for 120 consecutive patients that had an ACS after noncardiac surgery (PACS), for 120 patients with spontaneous ACS (SACS), and 240 patients with stable coronary artery disease (CAD). Coronary lesions with obstructions greater than 50% were classified based on two criteria: Ambrose's classification and complex morphology. The presence of Ambrose's type II or complex lesions were compared between the three groups.

Results: We analyzed 1470 lesions in 480 patients. In PACS group, 45% of patients had Ambrose's type II lesions vs. 56.7% in SACS group and 16.4% in stable CAD group (P < 0.001). Both PACS and SACS patients had more complex lesions than patients in stable CAD group (56.7% vs. 79.2% vs. 31.8%, respectively; P < 0.001). Overall, the independent predictors of plaque rupture were being in the group PACS (P < 0.001, OR 2.86; CI, 1.82–4.52 for complex lesions and P < 0.001, OR 3.43; CI, 2.1–5.6 for Ambrose's type II lesions) or SACS (P < 0.001, OR 8.71; CI, 5.15–14.73 for complex lesions and P < 0.001, OR 5.99; CI, 3.66–9.81 for Ambrose's type II lesions).

Conclusions: Nearly 50% of patients with perioperative ACS have evidence of coronary plaque rupture, characterizing a type 1 myocardial infarction.

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1. Introduction

Annually, more than 230 million noncardiac surgeries are performed worldwide [1]. Despite improvements in surgical and anesthetic techniques, mortality and cost related to these procedures are raising [2]. Cardiac complications are a major cause of morbidity and mortality after noncardiac surgeries, and patients experiencing a perioperative myocardial infarction (MI) have a high mortality and prolonged hospital stay [3].

The etiology and pathophysiology of myocardial ischemia and infarction after noncardiac surgery is still subject of controversies [1,3–7]. In this setting, it may involve thrombosis over a vulnerable plaque or decreased oxygen supply secondary to anemia or hypotension, designated type 1 and type 2 by the universal definition of MI [8,9]. Depending on the predominant mechanism, prognosis and treatment may be different. Although two retrospective pathology studies reported that nearly 50% of patients with fatal perioperative MI have plaque disruption [10,11], it has been suggested that, in patients who survive a perioperative MI, the incidence of type 2 MI would be much higher than type 1 [1]. However, there are no studies designed to establish the pathophysiology in patients that survived a perioperative acute coronary syndrome (ACS).

The presence of coronary plaques with complex morphologic features in coronary angiography is the angiographic hallmark of unstable coronary syndromes and correlates with pathologic plaque rupture and thrombus, characterizing a type 1 MI [12–19]. Ambrose's type II eccentric lesions are strongly associated to disrupted plaques and their finding have 92% specificity [17–20].

In order to determine the pathophysiology of ACS complicating noncardiac surgery we compared the presence of plaque rupture as a marker of type 1 MI in patients with ACS after noncardiac surgery (PACS), patients in the emergency room with spontaneous ACS (SACS), and patients with stable coronary artery disease (CAD). The present study was performed at the biggest University Hospital in...
Brazil where, roughly, 40,000 non-cardiac surgeries are performed annually.

2. Methods

Between February 2006 and June 2010 clinical and angiographic data were prospectively recorded into a database for 120 consecutive patients that had PACS after noncardiac surgery, for 120 patients with SACS, and for 240 patients with stable CAD. The study protocol was approved by the hospital’s ethics committee.

2.1. Inclusion criteria

Consecutive patients submitted to noncardiac surgery who presented with ACS within 30 days after the procedure were included in the PACS group. Patients with suspected perioperative ACS were evaluated by a cardiologist and were included if they had unstable angina with electrocardiographic ischemic signs (ST segment depression or T wave abnormalities) or MI, defined as follows: detection of a typical rise and fall of biochemical markers of myocardial necrosis (troponin) with at least one value above the 99th percentile of the upper reference limit together with: ischemic symptoms or development of pathological Q waves on the electrocardiogram (ECG) or ECG changes indicative of ischemia (ST segment elevation or depression) [21].

For the SACS group, patients that had arrived in the emergency room on random days and met the same criteria of ACS were included at admission.

For the stable CAD group, patients that were submitted to elective coronary angiography on random days were included before the procedure. Angiography was indicated by clinic’s physician based on symptoms of stable angina or evidence of CAD on complementary tests.

2.2. Exclusion criteria

Patients in the PACS and SACS group were excluded if coronary angiography was not performed. Patients in the stable CAD group were excluded if they had had an ACS diagnosis in the previous 2 months.

2.3. Clinical data

Clinical data such as age, gender, presence of diabetes, hypertension, smoking status, history of prior MI, stable angina, heart failure, prior myocardial revascularization procedures, and cardiovascular medication use were collected for the three study groups. Patients in the PACS and SACS groups were followed-up until hospital discharge, and information about recurrent unstable angina and myocardial infarction and death was obtained. The use of antiplatelet and anticoagulant agents prescribed for ACS treatment and bleeding episodes were also recorded. Bleeding episodes were classified as major or minor based on TIMI’s criteria [22].

2.4. Angiographic analysis

All angiographies were analyzed by a single experienced observer who was unaware of the patients’ clinical diagnosis. The number and location of coronary lesions with obstructions greater than 50% were recorded [17–19]. Each lesion was classified based on Ambrose’s classification [17–19]. This classification divides the lesions into 4 types: concentric (symmetric and smooth narrowing), type I eccentric (asymmetric stenosis with smooth borders and a broad neck), type II eccentric (asymmetric stenosis in the form of a convex intraluminal obstruction with a narrow neck due to one or more overhanging edges or irregular or scalloped borders, or both) and multiple irregularities (three or more serial, closely spaced narrowing or severe diffuse irregularities within a vessel) [17–19].

Lesions were also categorized as complex or not using a classification adapted from Goldstein et al. [12]. Lesions were considered complex if they caused at least 50% stenosis and had one or more of the following morphologic features:

- An intraluminal filling defect consistent with thrombus, defined as abrupt vessel cutoff with persistence of contrast, or an intraluminal filling defect in a vessel within or adjacent to a stenotic region with surrounding homogeneous contrast opacification;
- Plaque ulceration, defined by the presence of contrast and hazy contour beyond the vessel lumen;
- Plaque irregularity (haziness), defined by irregular margins or overhanging edges;
- Impaired flow (TIMI flow < 3, except lesions characteristic of chronic total occlusion, identified as tapering lesions with multiple fine collaterals).

The presence of at least one Ambrose’s type II lesion or a complex lesion per patient was compared between the three study groups.

2.5. Statistical analysis

Base-line demographic characteristics, clinical and angiographic variables were compared between the three groups. Frequencies and percentages are given for categorical variables. These variables were compared by chi-square test when applicable and otherwise by Fisher’s exact test. Numerical variables are reported as means ± standard deviation (SD). For continuous variables, statistical comparisons were made with use of Student’s t-test (normal distribution) or Mann–Whitney test (asymmetric distribution). Post hoc analysis for continuous variables was made by the Tukey-HSD (honest significant difference) test. Multivariate logistic regression analysis was performed to access independent clinical predictors for plaque rupture in patients from all three groups. All clinical variables with a value of P < 0.25 in univariate analysis were tested. Belonging to PACS or SACS group, presence of diabetes or anemia, age > 70 years old, and lack of use of aspirin, betablocker and statins were included in the multivariable model to test association with complex lesions. Belonging to PACS or SACS group, not having hypertension or history of prior MI, anemia, and lack of use of aspirin, betablocker, and statins were included in the multivariable model to test association with Ambrose’s type II lesions. Adjusted odds ratio (OR) is reported with corresponding 95% confidence intervals (CI). Model adequacy was measured by Hosmer & Lemeshow’s goodness of fit test. A P value of less than 0.05 was considered to indicate statistical significance. All analyses were performed using the SPSS 15.0 software.

3. Results

One hundred seventy patients with suspected PACS were evaluated. Nine patients were not included because they did not have ACS (eight had isolated troponin elevations and one had pulmonary thromboembolism). Forty patients were excluded because coronary angiography was not performed and one patient was excluded due to technical reasons in analyzing the angiography, leaving 120 patients that were included in the PACS group. In the SACS group, 145 patients were evaluated and 120 were included. Overall, 480 patients and 1470 lesions were analyzed.
Table 1
Baseline characteristics.

<table>
<thead>
<tr>
<th></th>
<th>PACS (n = 120)</th>
<th>SACS (n = 120)</th>
<th>CAD (n = 240)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male sex, n (%)</td>
<td>86 (71.7)</td>
<td>81 (67.5)</td>
<td>157 (65.7)</td>
<td>0.52</td>
</tr>
<tr>
<td>Age (years; mean ± SD)</td>
<td>67.82 ± 10.02</td>
<td>64.53 ± 12.41</td>
<td>61.95 ± 9.72</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>History of D. mellitus</td>
<td>51 (42.5)</td>
<td>42 (35)</td>
<td>106 (44.4)</td>
<td>0.23</td>
</tr>
<tr>
<td>Hypertension, n (%)</td>
<td>96 (82.5)</td>
<td>101 (84.2)</td>
<td>203 (84.9)</td>
<td>0.84</td>
</tr>
<tr>
<td>Prior MI, n (%)</td>
<td>22 (18.3)</td>
<td>50 (41.7)</td>
<td>84 (35.1)</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Stable angina, n (%)</td>
<td>20 (16.7)</td>
<td>19 (15.8)</td>
<td>197 (82.5)</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Heart failure, n (%)</td>
<td>18 (15)</td>
<td>7 (5.8)</td>
<td>59 (24.7)</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Smoking status, n (%)</td>
<td>41 (34.2)</td>
<td>52 (43.3)</td>
<td>122 (51)</td>
<td>0.01</td>
</tr>
<tr>
<td>No</td>
<td>27 (22.5)</td>
<td>26 (21.7)</td>
<td>30 (12.6)</td>
<td></td>
</tr>
<tr>
<td>Current</td>
<td>122 (31)</td>
<td>140 (36.4)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Previous</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prior revascularization procedure, n (%)</td>
<td>52 (43.3)</td>
<td>42 (35)</td>
<td>87 (36.4)</td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>98 (81.7)</td>
<td>69 (57.5)</td>
<td>160 (66.9)</td>
<td></td>
</tr>
<tr>
<td>PTCA</td>
<td>11 (9.2)</td>
<td>26 (21.7)</td>
<td>45 (18.8)</td>
<td>0.004</td>
</tr>
<tr>
<td>CABG</td>
<td>10 (8.3)</td>
<td>17 (14.2)</td>
<td>24 (10)</td>
<td></td>
</tr>
<tr>
<td>PTCA and CABG</td>
<td>1 (0.8)</td>
<td>8 (6.7)</td>
<td>10 (4.2)</td>
<td></td>
</tr>
<tr>
<td>Hemoglobin (g/dL; mean ± SD)</td>
<td>12.48 ± 2.21</td>
<td>13.73 ± 1.72</td>
<td>14.08 ± 1.57</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Cr (mg/dL; mean ± SD)</td>
<td>1.68 ± 1.93</td>
<td>1.2 ± 0.72</td>
<td>1.25 ± 1.43</td>
<td>0.43</td>
</tr>
</tbody>
</table>

PACS: Perioperative acute coronary syndrome; SACS: Spontaneous acute coronary syndrome; CAD: coronary artery disease; n: number; SD: standard deviation; PTCA: percutaneous transluminal coronary angioplasty; CABG: coronary artery bypass grafting; Cr: creatinine.

1 PACS vs. SACS (P=0.04); PACS vs. stable CAD (P<0.001); SACS vs. stable CAD (P=0.08).
2 PACS vs. SACS (P<0.001); PACS vs. stable CAD (P<0.001); SACS vs. stable CAD (P=0.21).

Table 2
Medication at admission.

<table>
<thead>
<tr>
<th></th>
<th>PACS (n = 120)</th>
<th>SACS (n = 120)</th>
<th>CAD (n = 240)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspirin, n (%)</td>
<td>60 (50)</td>
<td>68 (56.7)</td>
<td>229 (95.8)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>β-Blockers, n (%)</td>
<td>47 (39.2)</td>
<td>60 (50)</td>
<td>195 (81.6)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Statin, n (%)</td>
<td>36 (30)</td>
<td>59 (49.2)</td>
<td>201 (84.1)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>ACE inhibitor, n (%)</td>
<td>58 (48.3)</td>
<td>53 (44.2)</td>
<td>160 (70.5)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

PACS: perioperative acute coronary syndrome; SACS: spontaneous acute coronary syndrome; CAD: coronary artery disease; n: number; ACE: angiotensin-converting enzyme.

Table 3
Acute coronary syndrome treatment medication.

<table>
<thead>
<tr>
<th>Medication</th>
<th>PACS, n (%)</th>
<th>SACS, n (%)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspirin</td>
<td>119 (99.2)</td>
<td>118 (98.3)</td>
<td>1.00</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>78 (65)</td>
<td>82 (70.1)</td>
<td>0.40</td>
</tr>
<tr>
<td>Heparin</td>
<td>104 (86.7)</td>
<td>120 (100)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Ticlopidine</td>
<td>11 (9.2)</td>
<td>90 (75)</td>
<td>-0.001</td>
</tr>
<tr>
<td>β-Blockers</td>
<td>102 (85)</td>
<td>110 (91.7)</td>
<td>0.10</td>
</tr>
<tr>
<td>Statin</td>
<td>118 (98.3)</td>
<td>120 (100)</td>
<td>0.50</td>
</tr>
<tr>
<td>ACE inhibitor</td>
<td>94 (78.3)</td>
<td>108 (90)</td>
<td>0.01</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>23 (19.2)</td>
<td>3 (2.5)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

PACS: perioperative acute coronary syndrome; SACS: spontaneous acute coronary syndrome; CAD: coronary artery disease; ACE: angiotensin-converting enzyme; n: number.

3.1. Baseline characteristics

Clinical and laboratory characteristics of the 480 patients and medications at the time of admission are listed in Tables 1 and 2, respectively. There were no differences between the three groups in the prevalence of male gender, hypertension or diabetes. Patients in PACS group were older and had lower hemoglobin levels than patients in SACS or stable CAD groups. Patients in PACS group also had less prior history of known CAD (history of MI or prior myocardial revascularization procedures) than patients of the other two groups.

3.2. Clinical data and outcome

In PACS group, the mean time between the procedure and the ACS was 2.2 ± 3.3 days and 71.7% of patients had an ACS within the first 72 h after surgery. Regarding the type of operation, 46 patients (38.3%) were submitted to vascular surgery, 25 (20.8%) to general abdominal surgery, 12 (10%) to urologic surgery, 10 (8.3%) to orthopedic surgery, 7 (5.8%) to head and neck surgery, 7 (5.8%) to neurosurgery, 4 (3.3%) to kidney transplantation, and 9 (7.7%) to other procedures. Regarding anesthesia, 67 (60.4%) patients received general anesthesia, 20 (18%) regional anesthesia and 24 (21.6%) combined general plus regional anesthesia. Mean anesthesi duration was 363 ± 212 min (range from 60 to 1425 min). Only 48 (40.7%) patients presented with chest pain as the clinical manifestation of ACS. As for ACS classification, 19 (15.8%) patients had unstable angina, 94 (78.3%) had non-ST elevation MI and 7 (5.8%) had ST elevation MI. In SACS group, 19 (5.8%) patients had unstable angina, 76 (65%) had non-ST elevation MI and 23 (19.2%) had ST elevation MI. Patients in the PACS group had a longer time from the ACS to angiography than patients in the SACS group (5.5 ± 8 days vs. 1.3 ± 1.4 days, respectively; P=0.001).

During follow-up there was no difference between the PACS and SACS groups regarding recurrent angina (12.5% vs. 10%, respectively; P=0.54) or myocardial infarction (10% vs. 5%, respectively; P=0.14), but patients in PACS group were more frequently on Killip’s Classification III and IV than patients in SACS group (35% vs.12.5%, respectively; P<0.001), and had higher mortality (15% vs. 4.2%; P=0.02).

The use of antiplatelet agents and anticoagulant therapy in both groups are shown in Table 3. Eleven (9.2%) patients in the PACS group had a bleeding episode (6 major bleeding, including 2 fatal, and 5 minor bleeding) whereas 10 (8.3%) patients in the SACS group presented with bleeding (6 major and 4 minor but none fatal; P=0.09). Interestingly, only 3 patients on the PACS group presented with bleeding from the operative site and 5 patients had gastrointestinal bleeding despite the use of ulcer prophylaxis.
3.3. Angiographic results

Twenty-eight patients did not have obstructions above 50%: 7 (5.8%) in PACS group, 3 (2.5%) in SACS group and 18 (7.5%) in stable CAD group. Of the 1471 lesions analyzed, 349 were in patients of the PACS group (mean 2.86 ± 1.71 lesions per patient), 404 were in patients of the SACS group (mean 3.31 ± 1.71 lesions per patient), and 717 were in patients of the stable CAD group (mean 2.94 ± 1.86 lesions per patient; P = 0.10). There was no difference between the three groups regarding the location of the lesions.

In PACS group, 45% of patients had Ambrose’s type II lesions vs. 56.7% in SACS group and 16.4% in stable CAD group (P < 0.001). Both PACS and SACS patients had more complex lesions than patients in stable CAD group (56.7% vs. 79.2% vs. 31.8%, respectively; P < 0.001). Comparison between angiographic characteristics of patients is shown in Table 4.

After univariate analysis, applied to the entire cohort of 480 patients, the following variables were associated to the presence of complex lesions, and were included in the multivariable model: belonging to PACS or SACS group (P < 0.001), presence of diabetes (P = 0.153), presence of anemia (P = 0.002), age > 70 years old (P = 0.002), and lack of medication use: aspirin (P < 0.001), beta-blocker (P < 0.001) and statins (P = 0.001). The independent predictors of complex lesions were being in the group PACS (P < 0.001; OR, 2.86; 95% CI, 1.82–4.52) or SACS (P < 0.001; OR, 8.71; 95% CI, 5.15–14.73) and the presence of diabetes (P = 0.025; OR, 1.58; 95% CI, 1.06–2.36). The variables associated to Ambrose’s type II lesions in the univariate model, that were included in the multivariable model, were: belonging to PACS or SACS group (P < 0.001), not having hypertension (P = 0.221) or history of prior MI (P = 0.148), anemia (P = 0.004), and lack of medication use: aspirin (P < 0.001), beta-blocker (P = 0.004), and statins (P = 0.002). The independent predictors of Ambrose’s type II lesions were being in the group PACS (P < 0.001; OR, 3.43; 95% CI, 2.1–5.6) or SACS (P < 0.001; OR, 5.99; 95% CI, 3.66–9.81) (Table 5).

4. Discussion

This was the first prospective study that evaluated the presence of plaque rupture in consecutive patients with ACS after noncardiac surgery. Regarding the clinical and outcome characteristics of patients with periperaoperative ACS, our study confirmed previous findings that periperaoperative ACS occurs mainly within the first 72 h of the procedure, most events are non ST-elevation MI and only 40% of patients have thoracic pain. As expected, inhospital mortality was higher in patients with perioperative ACS than spontaneous ACS. This finding could be attributed to baseline diseases (co-morbidities) that motivated surgery (malignant disease, vascular disease, trauma, etc.).

Our findings suggest that nearly 50% of patients with periperaoperative ACS have markers of plaque disruption, suggesting a type 1 MI. Our data is in line with the two retrospective autopsy studies that investigated coronary anatomy in patients with fatal periperaoperative MI. Dawood et al. [10] studied 42 patients with fatal periperaoperative MI and found out that 55% of patients had evidence of unstable plaques with disruption. Cohen et al. [11] also studied 26 patients with fatal periperaoperative MI and detected plaque rupture on autopsy in 45% of them. In a retrospective study that used a catheterization laboratory database, Berger et al. [27] identified 48 patients referred for emergency coronary angiography for acute MI within 7 days of noncardiac surgery. Only critically ill patients with postoperative MI were included: 33 patients (68.8%) had ST-segment elevation, and 21 patients had cardiogenic shock. Although the purpose of their study was to determine the clinical course and outcome of patients undergoing immediate angiography for periperaoperative MI and not to study the angiographic characteristics, they reported the presence of thrombus in 30 patients (62.5%).

Differently from Berger et al. [27], Dawood et al. (28%) [10] and Cohen et al. (35%) [11] we found a low percentage of thrombus in angiography of patients in PACS group (7.5%). This result may be related to the long time between MI and angiography in this group (5.5 days in average), consequently prolonged time under antiplatelet and anticoagulant agents, spontaneous lysis of some thrombi, and the small number of patients with ST-elevation MI (more prone to exhibit thrombus over culprit lesion). Indeed, patients with periperaoperative non-ST-elevation ACS usually are more severely ill than patients with spontaneous SCA, and before being referred to coronary angiography, physicians had to be sure that the patient could receive antiplatelet and anticoagulant therapy (considering the risk of bleeding) and that infections were under control. Reinforcing the presence of unstable coronary plaques in periperaoperative ACS, we found similar frequencies of haziness and ulceration on angiography in PACS and SACS groups, an unlikely finding in patients with stable CAD. Indeed, multivariate analysis indicated that belonging to PACS or SACS groups was associated to an increased risk of angiographic markers of plaque disruption. Out of the periperaoperative setting and using intravascular ultrasound, Hong et al. also found that the only independent predictor of coronary plaque disruption among patients with stable angina and myocardial infarction was having the diagnosis of acute MI [28]. Conversely, previous authors have suggested that postoperative tachycardia, hypotension, hypertension, anemia, and hypoxemia are common causes of prolonged ST-depression and type 2 infarction in patients with stable CAD undergoing noncardiac surgery [1]. The cornerstone of this hypothesis is the finding of prolonged

Table 4
Angiographic characteristics.

<table>
<thead>
<tr>
<th>Complex lesion</th>
<th>PACS, n (%)</th>
<th>SACS, n (%)</th>
<th>Stable CAD, n (%)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ambrose’s type II lesion</td>
<td>68(56.7)</td>
<td>95(79.2)</td>
<td>76(31.8)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Thrombus</td>
<td>54(45)</td>
<td>68(56.7)</td>
<td>44(16.4)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Ulceration</td>
<td>15(12.5)</td>
<td>18(15)</td>
<td>16(6.7)</td>
<td>0.03</td>
</tr>
<tr>
<td>Haziness</td>
<td>45(37.5)</td>
<td>54(45)</td>
<td>39(16.3)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>TIMI flow &lt; 3</td>
<td>27(22.5)</td>
<td>61(50.8)</td>
<td>48(20.1)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

PACS: perioperative acute coronary syndrome; SACS: spontaneous acute coronary syndrome; CAD: coronary artery disease; n: number; TIMI: thrombolysis in myocardial infarction.

Table 5
Independent predictors of plaque rupture in the multivariable logistic regression model.

<table>
<thead>
<tr>
<th>Predictors of complex lesions'</th>
<th>Adjusted OR</th>
<th>95% CI</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>PACS group</td>
<td>2.86</td>
<td>1.82–4.52</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>SACS group</td>
<td>8.71</td>
<td>5.15–14.73</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Diabetes</td>
<td>1.58</td>
<td>1.06–2.36</td>
<td>0.025</td>
</tr>
<tr>
<td>Predictors of Ambrose’s type II lesions'</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PACS group</td>
<td>3.43</td>
<td>1.20–5.60</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>SACS group</td>
<td>7.99</td>
<td>3.66–9.81</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

PACS: perioperative acute coronary syndrome; SACS: spontaneous acute coronary syndrome.

' Hosmer & Lemeshow goodness of fit: P = 0.647.

1 Hosmer & Lemeshow goodness of fit: P = 0.270.
ST depression in perioperative Holter monitoring preceding the ischemic event in previous studies [29]. In addition, the rare occurrence of ST elevation MI in PACS reinforces the theory that the incidence of type 2 MI could be higher than type 1 MI [1,29]. In spite of its theoretical biological plausibility, until now there was no clinical evidence about the true incidence of type 1 MI. The present study provided this missing evidence.

Our study has some limitations. Among the excluded patients, 18 (45%) died before the angiography could be done, reflecting their critical clinical status, and we missed their angiographic characteristics. We used coronary angiography for classifying lesions and determine the presence of plaque disruption. Although it is not the gold standard to diagnose plaque rupture, previous authors showed that complex angiographic lesion morphology and Ambrose’s type II lesions are strongly correlated with plaque rupture [20,16].

In conclusion, nearly 50% of patients with perioperative myocardial infarction have evidence of coronary plaque rupture, characterizing a type 1 MI.

5. Clinical implications

The present study indicates that, as well as in spontaneous ACS, plaque rupture plays an important role in the pathophysiology of perioperative ACS. At the bed side, this information is very useful, as antplatelet and anticoagulant therapies and invasive evaluation should be strongly considered. On the other hand, type 1 and type 2 MI mechanism are not mutually exclusive in the pathophysiology of perioperative ACS. In consequence, preventing hypotension, tachycardia, anemia and hypertension remain important in the care of patients with perioperative ACS. Therefore, prevention and treatment measures that act in both mechanisms are essential for reducing the occurrence and mortality of perioperative ACS.

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Conflict of interest

None declared.

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