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Clinical Investigation

CLINICAL INVESTIGATION

# Incidence of thrombosis in perioperative and non-operative myocardial infarction

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## Abstract

**Background:** The contribution of thrombosis to the aetiology of perioperative myocardial infarction (MI) is uncertain. We used optical coherence tomography (OCT) to determine the presence of thrombus and plaque morphology in patients experiencing a perioperative MI and matched patients experiencing a non-operative MI using OCT.
**Methods:** We conducted a single-centre, prospective, cohort study. Thirty patients experiencing a perioperative MI and 30 matched patients experiencing a non-operative MI, without ST elevation, underwent OCT to determine the presence of thrombus and culprit lesion plaque morphology. Angiography and OCT were performed a mean of 1.93(1.09) days and 1.53(0.68) days after the onset of perioperative and non-operative MI, respectively. OCT images were evaluated by an independent core laboratory without knowledge of whether the patient had suffered a perioperative or non-operative MI.
**Results:** We identified thrombus at the culprit lesion in four of 30 patients (13.3%) who experienced a perioperative MI and in 20 of 30 patients (66.7%) who experienced a non-operative MI, $P<0.01$. The only non-culprit lesion with thrombus was in a perioperative MI patient who also had a culprit lesion thrombus. Perioperative and non-operative MI culprit lesions demonstrated fibroatheroma in 18 patients (60.0%) us 20 patients (66.7%), respectively ($P=0.52$) and thin cap fibroatheroma in one patient (3.3%) us five patients (16.7%), respectively ($P=0.11$). One perioperative MI patient (3.3%) suffered a cardiac death and no non-operative MI patient died during the 30-day follow-up.
**Conclusions:** Thrombosis was less common in perioperative than non-operative MI, despite similar underlying plaque morphology.

**Key words:** coronary artery disease; myocardial infarction; optical coherence tomography; perioperative; thrombosis

The pathophysiology of myocardial infarction (MI) is classified as type 1 if it results from atherosclerotic plaque rupture, erosion, or dissection with resulting intraluminal thrombosis in a coronary artery, or as type 2 if it results from an imbalance between myocardial oxygen supply and demand.[1] Patients undergoing noncardiac surgery are potentially at risk for both type 1 and type 2 MI. An improved understanding of the mechanism of perioperative MI may help to identify effective prevention and management interventions for this group of patients who have a poor prognosis.[2]

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### Editor's key points

- Surgical patients with coronary artery disease manifest their disease differently to that of non-operative setting.
- Intracoronary optical coherence tomography is an imaging technique to detect coronary arterial thrombus.
- This study found that thrombosis occurred less often in postoperative patients with myocardial infarction.
- Non-operative MI culprit lesions had greater stenosis, more angiographic complexity, and patients were more likely to undergo revascularization.

Studies of invasive coronary angiography in patients with perioperative MI suggest that both mechanisms may play a role;[3 4] however, these studies are limited by the low sensitivity of angiography to detect thrombus.[5] Intracoronary optical coherence tomography (OCT) is a highly accurate intravascular imaging technique for the detection of thrombus.[6 7] We conducted the OPtical coherence Tomographic IMaging of thrombUS (OPTIMUS) Study to determine the prevalence of culprit lesion thrombus and plaque morphology in patients experiencing a perioperative MI as compared with a matched cohort of patients experiencing a non-operative MI.

## Methods

### Study design

The OPTIMUS Study was a single-centre, prospective, cohort study. The study was approved by the Hamilton Integrated Research Ethics Board, and all patients provided written informed consent. Patients were eligible if they were age >18 yr, experienced a perioperative MI (within two weeks after noncardiac surgery) or non-operative MI that fulfilled the Universal Definition of MI and underwent cardiac catheterization within three days of the MI. All patients included in the study had an elevated troponin and fulfilled the universal definition of MI. Troponin concentrations were measured at local institutions using a variety of conventional and high-sensitivity troponin I and T assays.

No patient recruitment was performed from the surgical wards. Patients were recruited at the catheterization laboratory after the decision to refer for invasive angiography was made on clinical grounds. For each included perioperative MI patient, we evaluated each consecutive patient referred for angiography who had suffered a non-operative MI to identify a patient who was a match based on gender, age ( plus/minus five yr), and the presence or absence of ischaemic electrocardiographic changes. Patients fulfilling any of the following criteria were excluded: ST segment elevation myocardial infarction (STEMI), cardiogenic shock, prior coronary artery bypass surgery, percutaneous coronary intervention with stent implantation in the prior six months, or estimated glomerular filtration rate<35 mL/min.

### OCT procedures

After angiography, the interventional cardiologist identified the culprit artery and one additional non-culprit artery for OCT imaging. Section 1 in the Supplementary Appendix describes the criteria for identifying the culprit artery and an additional non-culprit artery for OCT imaging. Section 2 in the Supplementary Appendix describes the angiography and OCT imaging procedures. Reasons for unsuccessful OCT imaging of a coronary artery included severe left main stenosis, severe coronary stenosis that could not be crossed, tortuosity, calcification, chronic total occlusion, absence of a ≥30% stenotic lesion, or inability to achieve blood clearance on the OCT image.

### Angiographic interpretation

All coronary angiograms were evaluated offline at an angiographic core laboratory (Hamilton Health Sciences). The coronary angiograms were analysed with QAngioXA software (Medis, Netherlands),[8] by an interventional cardiologist who was blinded to whether patients had suffered a perioperative or non-operative MI.

The SYNTAX score was determined to characterize the extent and complexity of coronary artery disease,[9] and the presence and locations of chronic total occlusions were identified. Left ventricular function and the presence of any wall motion abnormalities were determined. For all culprit and non-culprit lesions, quantitative coronary angiography was performed to assess the baseline TIMI (Thrombolysis in Myocardial Infarction) flow, reference vessel diameter, minimum lumen diameter, and diameter stenosis. TIMI flow is graded on a 4 point scale; 0 indicating no flow beyond the occlusion, 1 indicating contrast penetration beyond the occlusion but incomplete vessel opacification, 2 indicated complete vessel opacification beyond the occlusion but a reduced flow rate, and 3 indicating normal vessel opacification at a normal rate beyond the occlusion. Lesions were characterized as simple or complex (i.e. eccentric with narrow neck, irregular borders, abrupt shoulders, or ulcerations).[10] Evidence of thrombus (i.e. filling defect seen in multiple projections) was noted.

### OCT interpretation

OCT images were evaluated by a core laboratory (Cardiovascular Imaging Core Laboratory, Case Western Reserve University) using proprietary software (Off-line Review Software, version E.0.2; St Jude Medical, MN, USA).[11] The images were analysed without knowledge of whether the patient had suffered a perioperative or non-operative MI. Vessels were deemed non-assessable when residual blood obscured the underlying structures,[11] and non-assessable for the evaluation of plaque rupture, when pre-dilatation was performed before OCT imaging. Qualitative assessment was performed at every 0.2 mm interval to detect the presence of intraluminal thrombus, plaque rupture, fibrous cap, and to evaluate plaque components. The images were evaluated independently by two observers and any discrepancies were resolved by consensus. Section 3 in the Supplementary Appendix describes the OCT imaging outcome definitions (e.g. thrombus, plaque rupture).

### Outcome measures

Our primary outcome was the presence of thrombus at the culprit lesion based on OCT imaging. Secondary outcomes based on OCT imaging included: 1) fibroatheroma at the culprit lesion, 2) thin cap fibroatheroma at the culprit lesion, 3) plaque rupture at the culprit lesion, and 4) thrombus at the non-culprit lesion. Study personnel followed patients until 30 days after OCT imaging to determine if patients had a