Coronary liposuction during percutaneous coronary intervention: evidence by near-infrared spectroscopy that aspiration reduces culprit lesion lipid content prior to stent placement

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Aims
Intracoronary near-infrared spectroscopy (NIRS) in ST-segment elevation myocardial infarction (STEMI) has demonstrated substantial lipid in STEMI culprit plaques. Thrombus aspiration during primary PCI reduces distal embolization and improves reperfusion. This study was performed to examine if aspiration thrombectomy reduces the lipid content of acute coronary syndrome (ACS) culprit plaques.

Methods and results
NIRS-IVUS imaging was performed in patients with an ACS at two hospitals in the US and Sweden. After establishment of TIMI 3 flow with an undersized balloon, NIRS was performed, followed by aspiration thrombectomy, followed by repeated NIRS. The same culprit segment was identified on the post-aspiration chemogram. The culprit lipid content was quantified before and after thrombectomy as the lipid core burden index (LCBI). Aspirates were examined by histological staining for lipids, calcium, and macrophages. In 18 ACS patients (age 65 ± 11, 61% male), culprit lesions were characterized by high lipid content prior to aspiration thrombectomy. Thrombectomy resulted in a 28% reduction in culprit lesion lipid content (pre-aspiration LCBI 466 ±141 vs. post-aspiration 335 ±117, P = 0.0001). In addition to thrombus, histological analysis of aspirates demonstrated the presence of lipids, calcium, and macrophages, indicating that fragments of atherosclerotic plaques had been aspirated.

Conclusion
Thrombectomy aspirates both thrombus and lipid-rich fragments of the culprit atherosclerotic plaques, thereby reducing material that may embolize during stenting. Reduction of lipid content before stenting might contribute to the beneficial effects of thrombectomy and may be particularly useful if a large lipid core is present at the culprit site.

Keywords
Lipid-core plaque • Near-infrared spectroscopy • Thrombectomy

Introduction
The most common cause of acute coronary syndrome (ACS) is rupture and subsequent thrombosis of a thin-cap fibroatheroma (TCFA), a metabolically active lesion having an inflamed thin fibrous cap overlying a lipid-rich necrotic core containing cholesterol, cholesteryl esters, and necrotic cellular debris.1 One of the most important aspects of ACS management is establishment of normal coronary blood flow with percutaneous coronary intervention (PCI).

Failure to achieve normal coronary flow in a timely manner is associated with heart failure and death and is closely associated with reperfusion injury, which can lead to arrhythmias, contractile dysfunction, microvascular impairment, and irreversible myocardial damage.7 In the setting of ST-segment elevation myocardial infarction (STEMI), aspiration of luminal thrombus may improve coronary flow following PCI.3–7 Although aspiration thrombectomy is performed to reduce thrombus burden in the culprit segment, there are reports indicating that plaque fragments are also unintentionally
Thrombus aspiration reduces lipid core

extracted during this process, but the effect of aspiration on the lipid content of the culprit lesion has not been determined and aspirates have not been examined with lipid staining.

Intracoronary near-infrared spectroscopy (NIRS) has been developed for the explicit purpose of detecting lipid within the coronary arteries. There is considerable experience with the use of NIRS in stable patients. In patients with stable angina undergoing PCI for a stenotic lesion, NIRS-detected lipid is associated with myocardial infarction during the procedure. It has been documented that the NIRS signal of lipid decreases following balloon inflation and stenting, supporting the concept that some cases of periprocedural infarction and angiographic no-flow may be caused by embolization of friable, lipid-rich plaque contents. This has led to attempts to prevent no-reflow with the use of filter devices when lipid-rich stenotic lesions causing stable angina are dilated. While fewer studies have been performed in patients with ACS, NIRS-detected lipid has also been associated with unfavourable events in patients with acute events. NIRS-detected lipid has been associated with the procedural complication of acute stent thrombosis in a patient with an ACS. There is also evidence that NIRS-detected lipid is present at the culprit lesion site in over 80% of patients with STEMI.

There have been no reports of the use of NIRS or other imaging modalities to determine changes in the lipid content of ACS culprit plaques during aspiration thrombectomy. The reduction of exposed culprit lesion lipid by aspiration thrombectomy has the potential to decrease embolization during stenting and thereby add to the potential value of the aspiration procedure. This study was performed to: (i) determine if aspiration thrombectomy prior to stent placement reduces the lipid content of culprit lesions in ACS patients and (ii) to determine whether there is histological evidence of plaque lipid in the aspirated material.

**Methods**

**Study population**

This study was conducted in patients with an ACS who were referred to the catheterization laboratory of two institutions (Lund University, Lund, Sweden and Frederik Meijer Heart & Vascular Institute, Spectrum Health, Grand Rapids, MI, USA) for PCI. Consecutive patients meeting the following criteria were included: (i) clinical presentation was ACS; (ii) invasive angiography revealed a culprit lesion responsible for the clinical presentation; (iii) prior to stent placement, combined NIRS-intravascular ultrasound (IVUS) was performed, followed by aspiration thrombectomy due to the presence of a filling defect, followed by repeat NIRS-IVUS. Patients were excluded if haemodynamic instability was present or NIRS spectral images within the culprit segment were uninterpretable. The decision to use NIRS-IVUS imaging and perform aspiration thrombectomy were made by the operator at the time of invasive angiography. The Ethics Committees of both participating institutions approved this study.

**Invasive coronary angiography**

The culprit lesion location was identified angiographically by the physician performing PCI. For patients presenting with STEMI, the culprit lesion was in a location consistent with the distribution of ST-segment elevation on the electrocardiogram. Initial flow within the culprit vessel was characterized according to Thrombolysis in Myocardial Infarction (TIMI) flow grade. For patients having TIMI flow < 3 on the initial angiogram, a 2 mm balloon was inflated briefly to nominal pressure to minimize alteration of the culprit plaque while still achieving vessel patency and TIMI 3 flow.

**NIRS-IVUS imaging and aspiration thrombectomy**

Once TIMI 3 flow was present, initial NIRS-IVUS imaging (True Vessel Characterization Imaging System, Infraredx, Burlington, MA, USA) was performed by advancing the NIRS-IVUS catheter (Insight Catheter, Infraredx, Inc. Burlington, MA, USA) beyond the culprit lesion and initiating a motorized pullback at 0.5 mm/s. In all patients, initial NIRS-IVUS imaging was immediately followed by aspiration thrombectomy, performed by continuous manual suction using a proximal-to-distal approach with one of the following catheters: Eliminate (Terumo crossing profile, 0.068 in.), Export Aspiration Catheter (Medtronic; crossing profile, 0.067 in.), or Pronto Extraction Catheter (Vascular Solutions, Minneapolis, MN, USA). The choice of aspiration catheter and the duration of aspiration were left to the discretion of the operator. Upon completion of aspiration thrombectomy NIRS-IVUS imaging was repeated in all patients prior to further intervention. Stent placement was then used to treat the culprit lesion in all cases.

**NIRS-IVUS analysis**

NIRS images were interpreted as previously described. NIRS data are displayed in a chemogram, a rectangular map in which the x-axis represents millimetre of pullback within the artery and the y-axis rotation from 0 to 360 degrees. A high likelihood of LCP is indicated by a yellow signal, whereas a red signal indicates LCP is not likely to be present. NIRS images were considered uninterpretable in a 2 mm segment and excluded from further analysis if the corresponding block chemogram (a summary of the NIRS values in a 2 mm length of pullback) was black in colour, indicating inadequate reliable data for the corresponding 2 mm segment of the chemogram.

Using anatomic landmarks present on angiography, the culprit lesion location was identified on the IVUS images. Since all IVUS images are automatically co-registered with the NIRS data when using the TCV Imaging System, the culprit lesion margins could be associated with its corresponding segments on the pre-aspiration NIRS chemogram. The culprit lesion margins were defined on the pre-aspiration chemogram by the margins of all confluent yellow present at the culprit site. Using fiducial anatomic landmarks, these exact lesion margins were marked on the post-aspiration NIRS chemogram ensuring that lesion length was identical on the pre- and post-aspiration chemograms. Culprit lesion lipid content was quantified before and after thrombectomy as the lipid core burden index (LCBI), defined as the fraction of pixels indicating lipid within a scanned region multiplied by 1000. Lipid content is also described as the maximum LCBI in any 4 mm segment (maxLCBI$_{4mm}$) within the culprit lesion.

On both the pre-thrombectomy and post-thrombectomy images, IVUS images within the margins of the culprit lesion were analysed quantitatively for minimal lumen area (MLA), minimal lumen diameter (MLD), and plaque burden according to expert consensus recommendations.

**Histological analysis of the thrombi aspirate**

When possible, aspirates were collected for histological analysis. Of the 18 NIRS-examined patients in this study, seven had their aspirates sent for further analysis. Thrombus aspirates from an additional 15 STEMI patients who did not undergo NIRS imaging were also collected to evaluate for lipid within the aspirated material. In all cases, aspirates were frozen (−80°C), embedded in optimal cutting temperature and compound embedding medium, and cryosectioned. The sections (8 μm) were then thawed for staining. To stain lipids, the sections were fixed with...
Histochoice (Amresco, OH, USA), dipped in 60% isopropanol and then in 0.4% Oil Red O in isopropanol (for 20 min). For macrophage assessment, primary antibody mouse anti-human CD68 (DakoCytomation, Glostrup, Denmark), diluted in 20% rabbit serum 1:100, and secondary antibody rabbit anti-mouse (DakoCytomation, Glostrup, Denmark), dilution 1:200 in 20% of rabbit serum, were used. To assess calcium, the sections were fixed with 99% ethanol, incubated first with 1% silver nitrate under ultraviolet for 20 min and afterwards with 5% sodium thiosulfate, according to the von Kossa method. All stained sections were scanned and photographed with ScanScope Console Version 8.2 (LRI imaging AB, Vista CA, USA).

### Statistical analysis

LCBI and maxLCBI 4mm were the primary measures of interest in this study. Descriptive statistics were used to summarize the NIRS and IVUS findings of ACS culprit segments before and after thrombectomy. NIRS and IVUS parameters for each ACS culprit segment were compared before and after thrombectomy using the paired t-test.

### Results

#### Study population

Between 5 February and 24 July 2013, 18 patients presenting with ACS met criteria for the study. The clinical presentation consisted of STEMI in 16 patients, non-STEMI in one, and unstable angina in one. The baseline characteristics of the study population are presented in Tables 1 and 2. The histology population consisted of seven patients from the NIRS population and 15 patients from a previous collection of thrombus aspirates.

#### Clinical 30 days outcomes

There were no deaths, MI, or stroke in any of the patients after 30 days.

#### Angiographic findings

A single culprit lesion responsible for the clinical presentation was identified by angiography in all 18 cases. The culprit lesion was located in the right coronary artery in 39%, the left-anterior descending artery in 56%, and the left circumflex artery in 6% of cases. One patient had a history of prior coronary bypass surgery and had a culprit lesion located in a saphenous vein graft to the RCA. In all cases, including the one case of non-STEMI and the one case of unstable angina, a filling defect was present on the angiogram which prompted the performance of thrombectomy. Initial flow within the culprit vessel was TIMI 0 in 56%, TIMI 1 in 22%, TIMI 2 in 6%, and TIMI 3 in 17% of cases. In most patients, balloon angioplasty with an undersized balloon was performed prior to initial NIRS-IVUS imaging.

### NIRS and IVUS findings

In all 18 patients, lipid core plaque was evident at the culprit lesion on the pre-aspiration NIRS chemogram (LCBI 466 ± 141 and maxLCBI 4mm 695 ± 200). Typical pre- and post-aspiration NIRS chemograms of two patients with STEMI are illustrated in Figure 1. Figure 2 shows the pre- and post-aspiration NIRS chemograms for all 18 patients. A reduction in lipid content at the culprit lesion, as measured by LCBI, was evident in 89% of patients after aspiration. Overall, aspiration resulted in a 28% decrease in lipid content by LCBI (pre-aspiration 466 ± 141; post-aspiration 335 ± 117; \(P = 0.0001\)) and in a 30% decrease in lipid content by maxLCBI 4mm (pre-aspiration 695 ± 200; post-aspiration 489 ± 240; \(P = 0.001\); Figure 3). Aspiration reduced the culprit lesion LCBI by >20% in 67% of cases and by >50% in 11% of cases. Aspiration reduced the culprit lesion

#### Table 1: Baseline demographics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>ACS patients (n = 18)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>65.4 ± 10.6</td>
</tr>
<tr>
<td>Male, n (%)</td>
<td>12 (67)</td>
</tr>
<tr>
<td>Ethnicity</td>
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<tr>
<td>Caucasian</td>
<td>18 (100.0)</td>
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<tr>
<td>African-American</td>
<td>0</td>
</tr>
<tr>
<td>Hispanic</td>
<td>0</td>
</tr>
<tr>
<td>Body mass index</td>
<td>26.3 ± 6.1</td>
</tr>
<tr>
<td>History of CAD, n (%)</td>
<td>2 (11)</td>
</tr>
<tr>
<td>Previous MI, n (%)</td>
<td>2 (11)</td>
</tr>
<tr>
<td>Hypertension, n (%)</td>
<td>11 (61)</td>
</tr>
<tr>
<td>Diabetes mellitus, n (%)</td>
<td>2 (11)</td>
</tr>
<tr>
<td>Previous or current smoking, n (%)</td>
<td>9 (50)</td>
</tr>
</tbody>
</table>

#### Table 2: Clinical characteristics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>ACS patients (n = 18)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medications, n (%)</td>
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</tr>
<tr>
<td>Statin</td>
<td>4 (22)</td>
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<tr>
<td>Aspirin</td>
<td>2 (11)</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>1 (6)</td>
</tr>
<tr>
<td>Beta-blocker</td>
<td>1 (6)</td>
</tr>
<tr>
<td>ACE-inhibitor</td>
<td>4 (22)</td>
</tr>
<tr>
<td>ARB</td>
<td>6 (33)</td>
</tr>
<tr>
<td>Lipids</td>
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<tr>
<td>Total cholesterol (mg/dL)</td>
<td>152 ± 39</td>
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<tr>
<td>Triglycerides (mg/dL)</td>
<td>117 ± 55</td>
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<tr>
<td>HDL (mg/dL)</td>
<td>39 ± 6</td>
</tr>
<tr>
<td>LDL (mg/dL)</td>
<td>92 ± 31</td>
</tr>
<tr>
<td>Culprit vessel, n (%)</td>
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<tr>
<td>LAD</td>
<td>10 (56)</td>
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<tr>
<td>Circumflex</td>
<td>1 (6)</td>
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<tr>
<td>RCA</td>
<td>7 (39)</td>
</tr>
<tr>
<td>Initial TIMI flow, n (%)</td>
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<tr>
<td>TIMI 0</td>
<td>10 (56)</td>
</tr>
<tr>
<td>TIMI 1</td>
<td>4 (22)</td>
</tr>
<tr>
<td>TIMI 2</td>
<td>1 (6)</td>
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<tr>
<td>Final TIMI flow, n (%)</td>
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</tr>
<tr>
<td>TIMI 0</td>
<td>0 (0)</td>
</tr>
<tr>
<td>TIMI 1</td>
<td>0 (0)</td>
</tr>
<tr>
<td>TIMI 2</td>
<td>2 (11)</td>
</tr>
<tr>
<td>TIMI 3</td>
<td>16 (89)</td>
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</table>
**Figure 1** Typical NIRS findings before and after manual thrombectomy in two patients presenting with an acute STEMI. (A) A 74-year-old male presented with acute chest pain. An initial electrocardiogram demonstrated an acute anterior STEMI and the patient was referred emergently for primary PCI. The initial angiogram of the left-anterior descending coronary artery revealed complete occlusion. Aspiration thrombectomy yielded a solid yellow material. The NIRS chemogram obtained after TIMI 3 flow was established but before thrombectomy revealed a prominent, nearly circumferential lipid-rich plaque at the culprit site. After thrombectomy lipid content by LCBI was reduced from 604 to 466. (B) A 63-year-old male presented with an acute anterior STEMI. Initial angiography revealed a culprit lesion in the proximal left-anterior descending artery and TIMI 1 flow. An undersized balloon was used to perform initial angioplasty and resulted in TIMI 3 flow. NIRS-IVUS was then performed. By NIRS imaging, the culprit lesion (shown between the blue bookmarks in top chemogram) was characterized by a large lipid core. Aspiration thrombectomy was then performed and NIRS-IVUS was repeated immediately afterwards. Post-aspiration NIRS revealed a significant decrease in yellow at the culprit site and a 63% decrease in lipid content by LCBI. The aspirated contents are shown. IVUS, intravascular ultrasound; LCBI, lipid core burden index; NIRS, near-infrared spectroscopy; PCI, percutaneous coronary intervention; STEMI, ST-segment elevation myocardial infarction; TIMI, Thrombolysis in Myocardial Infarction.
maxLCBI\textsubscript{4mm}, by $>20\%$ in 56\% of cases and by $>50\%$ in 28\% of cases. Similar results were found when only the STEMI patients were analysed and when the vein graft analysis was excluded.

After stenting maxLCBI\textsubscript{4mm} was measured in 12 out of the 18 patients. MaxLCBI\textsubscript{4mm} was reduced from 555 $\pm$ 283 to 321 $\pm$ 209; $P < 0.01$.

IVUS findings were also altered by thrombectomy. Aspiration thrombectomy resulted in a 38\% increase in MLA (pre-aspiration 3.35 $\pm$ 1.15; post-aspiration 4.61 $\pm$ 1.55 mm\textsuperscript{2}; $P < 0.0001$), a 12\% increase in MLD (pre-aspiration 1.84 $\pm$ 0.37; post-aspiration 2.07 $\pm$ 0.41 mm; $P = 0.001$), and in a 30\% decrease in plaque burden (pre-aspiration 79.3 $\pm$ 8.4; post-aspiration 70.8 $\pm$ 13.0; $P < 0.001$) at the culprit site (Figure 3).

MaxLCBI\textsubscript{4mm}, before thrombectomy correlated with pre-aspiration MLA ($r^2 = 0.27$, $P = 0.03$) and pre-aspiration plaque burden ($r^2 = 0.35$, $P = 0.01$), but not with pre-aspiration MLD. The percent change in maxLCBI\textsubscript{4mm}, from before to after thrombectomy correlated with percent change in MLA ($r^2 = 0.32$, $P = 0.02$), plaque burden ($r^2 = 0.48$, $P = 0.003$), and MLD ($r^2 = 0.32$, $P = 0.02$).

**Histology**

Histology was performed on 22 aspirates, seven of which were from the 18 patients for whom NIRS data were available. In the seven patients with paired NIRS and histological data, all showed reduction of lipid in the plaque by NIRS and the appearance of lipid in the aspirate by histology. Figure 4 demonstrates the presence of atherosclerotic plaque components in the thrombus aspirates. Overall, macrophages were observed in 21 of the 22 aspirates (95\%), lipids in 12 out of 22 (55\%), and calcium in 10 out of 22 (45\%).

**Discussion**

The primary finding of the present study is that aspiration thrombectomy significantly reduced the lipid content of culprit lesions in ACS, as demonstrated by NIRS and further supported by histological examination of the aspirates which documented the presence of dislodged lipid. By NIRS, 89\% of culprit lesions had evidence of reduced lipid content after aspiration, with a mean reduction in lipid content by LCBI of 28\%.

**Role of lipid-rich plaque in ACSs**

Consistent with the concept that rupture of a lipid-rich TCFA underlies most acute coronary events, NIRS has detected lipid-rich plaque (LRP) at the culprit site in $>80\%$ of patients with ACS. More recently, NIRS was performed in patients with STEMI and demonstrated that most STEMI culprit lesions are characterized by
a large, often circumferential LRP at the culprit site. These observations in STEMI patients have been recently validated, are consistent with post-mortem observations implicating rupture of LRP as the cause of acute myocardial infarction, and highlight the role of LRP in culprit lesions across the spectrum of acute coronary syndromes.

Figure 3  NIRS lipid core detection levels before and after thrombectomy expressed as LCBI and maxLCBI4mm. (A) LCBI, (B) maxLCBI4mm.

Figure 4  IVUS measurements of minimal lumenal area (MLA), plaque burden (PB), and minimum lumenal diameter (MLD) before and after thrombectomy. (A) MLA, (B) PB, (C) MLD.
Our finding that aspiration thrombectomy can reduce the lipid content at ACS culprit sites may have implications for PCI performance. High lipid content, as detected by NiRS prior to stent placement, has been associated with periprocedural myocardial infarction. Goldstein et al. demonstrated a 50% rate of periprocedural infarction when PCI is performed on culprit lesions having a maxLCBI \text{max} > 500. The concept that lipid-rich lesions are associated with an increased risk of acute PCI-related complications has

**Figure 5** Representative examples of histological findings in thrombectomy aspirates. (A) Lipid staining demonstrates lipid content in the aspirate, (B) calcium staining, (C) macrophage staining, (D) control, (E) typical examples of lipid containing aspirates.
been supported by numerous other studies employing advanced imaging techniques. A study using coronary CT angiography has identified low attenuation plaque (<30 Hounsfield units), which is believed to be a marker of lipid-rich lesions, to be associated with angiographic no-reflow during PCI. 

Attenuated plaque by IVUS, which by histological comparisons is a marker of lipid-rich lesions, and necrotic core plaque by virtual histology-IVUS have similarly been linked to the occurrence of periprocedural myocardial infarction and angiographic no-reflow during PCI. Studies using optical coherence tomography have also linked lipid-rich lesions with the risk of periprocedural infarction and angiographic no-reflow. Importantly, many of these studies have demonstrated that it is not merely the presence of lipid, but rather the quantity of lipid at the culprit site that is an important determinant of acute PCI-related complications. Further study is required to determine if targeted removal of lipid content at the culprit site prior to stent placement reduces the likelihood of periprocedural myocardial infarction in stable patients and angiographic no-reflow in patients with a myocardial infarction in progress.

**Aspiration thrombectomy**

There are reports indicating that plaque fragments are unintentionally extracted during manual coronary thrombectomy, but aspirates have not been examined with lipid staining. We demonstrate that many of the aspirates are of plaque origin by demonstrating presence of macrophages and calcium, neither of which are present in thrombus or in the circulating blood (Figure 5). Importantly, we stain for lipid and show that thrombectomy aspirates lipid-rich plaque material (Figure 5). This supports the in vivo findings of reduced lipid content measured with NIRS. Even if we did not find lipid content in all aspirates, lipid content could have embolized distally during thrombectomy.

Our finding that aspiration thrombectomy reduces culprit lesion lipid content, which is prone to embolize during stenting, advances understanding of the mechanistic effects of aspiration thrombectomy. Thrombectomy may reduce lipid content both by aspirating plaque material, but also by embolizing lipid content distally. It has been hypothesized that removal of intracoronary thrombus, which triggers thrombotic, inflammatory, vasoconstrictor and other pathways, may be beneficial for the patient. A recent meta-analysis pointed towards a beneficial effect of thrombectomy aspiration on TIMI flow. However, in the INFUSE-AMI trial aspiration thrombectomy failed to improve myocardial reperfusion, TIMI flow, ST-segment resolution, or 30-day clinical event rates. Furthermore, thrombus aspiration may not be a risk-free procedure; systemic and local embolization can occur and in a recent meta-analysis thrombus aspiration was associated with a borderline significantly higher rate of stroke.

Thrombectomy of the culprit site in patients with STEMI has been shown to improve coronary flow following PCI, and even to reduce mortality. TIMI flow post-PCI was reported in recent meta-analyses and pooled data point towards a beneficial effect of thrombus aspiration on TIMI flow. Recently, in the large TASTE-trial, no mortality benefit was found for thrombectomy-treated patients. However, thrombectomy reduced the need for predilatation and there was a trend towards reduced stent thrombosis and non-fatal reinfarction.

**Limitations**

There are several important limitations to consider when interpreting the results of the present study. First, the present observations were generated from a small number of patients at two centres. These findings require validation in a larger series of patients. Secondly, the technique of aspiration, including aspiration device, negative pressure generated, and aspiration duration was not standardized in this study. Further study is needed to determine the effect of these procedural variables on lipid core reduction. Thirdly, it remains unknown whether reduction in lipid core size translates into improved clinical outcomes. Finally, although the majority of the patients in this study presented with STEMI, aspiration resulted in a reduction in lipid content by NIRS in one patient with unstable angina, one patient with non-STEMI, and in one saphenous vein graft culprit lesion. Further study is required to evaluate the potential benefits of aspiration in such patients.

**Conclusion**

Intracoronary thrombectomy in patients with ACS, aspirates both thrombus and parts of the culprit atherosclerotic plaque, thereby reducing lipid-rich material which may embolize during stenting. Reduction of lipid content before stenting might contribute to the beneficial effects of thrombectomy and may be particularly useful if a large lipid core is present at the culprit site.

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**Conflicts of interest**

D.E. has received speaker honoraria from Infraredx; J.H. received consulting honoraria from Boston Scientific and EPS Vascular; I.G. and M.G. have received consulting honoraria from Medtronic and Volcano; J.E.M. is an employee of Infraredx; R.D.M. has received research support and speaker honoraria from Infraredx and has served as a consultant for St Jude Medical.

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**References**


