Non-Culprit Lesion Plaque Morphology in Patients With ST-Segment Elevation Myocardial Infarction:

Results from the COMPLETE Trial Optical Coherence Tomography (OCT) Substudy

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Disclosures

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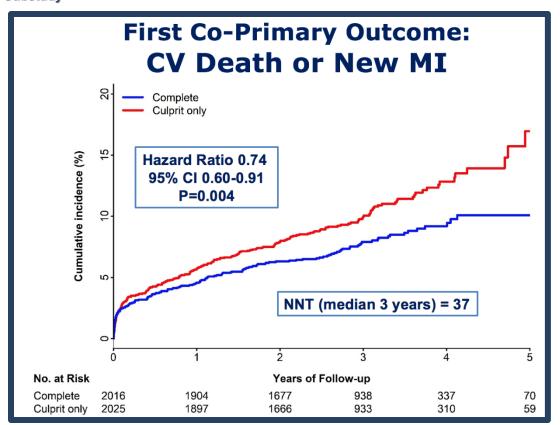


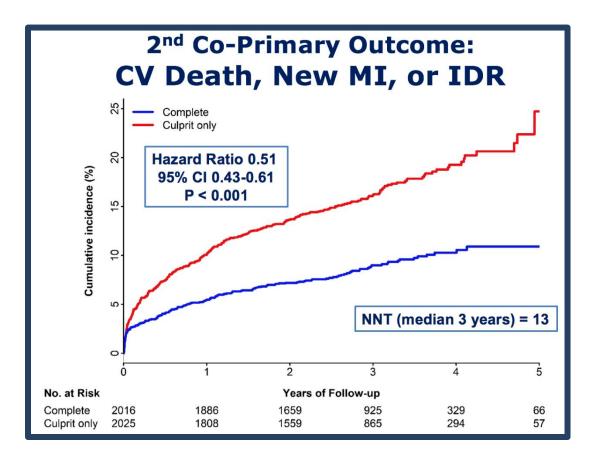






COMPLETE Trial – Primary outcomes





The COMPLETE trial demonstrated that routine angiography-guided staged PCI of non-culprit lesions reduced the composite of cardiovascular death or new myocardial infarction by 26% (p=0.004).





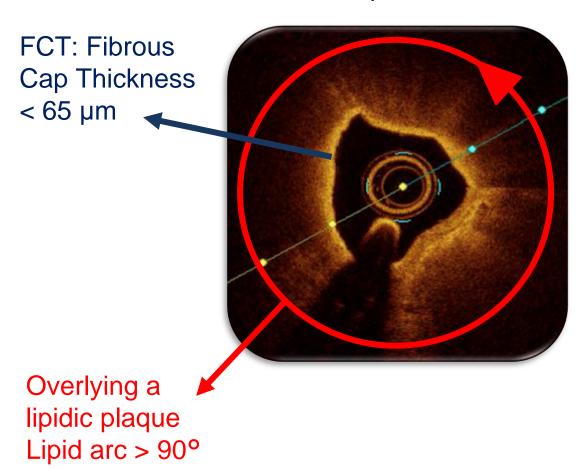




Background

TCFA: Thin Cap Fibro Atheroma

- Whether the benefit of routine non-culprit lesions PCI might be associated with underlying vulnerable plaque morphology is unclear.
- Thin-cap fibro atheroma (TCFA) is a well recognized feature of vulnerable plaque.
- Optical Coherence Tomography (OCT) is a high definition intracoronary imaging modality that can identify vulnerable plaque.







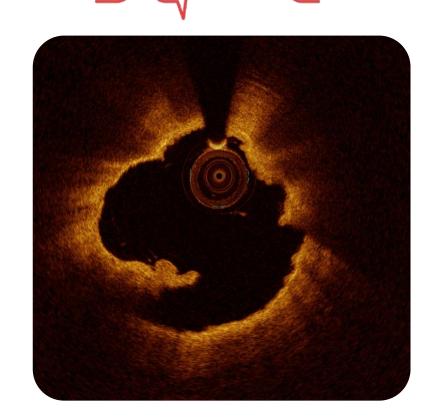


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Plaque rupture



unclear.







Hypothesis

 TCFA will be more prevalent in obstructive compared with non-obstructive non-culprit lesions.







Primary Objective

In patients presenting with STEMI and multi-vessel coronary artery disease undergoing staged non-culprit lesion PCI after successful primary PCI, the objective is:

To determine the prevalence of vulnerable plaque (i.e., biologically active TCFA) in obstructive compared with non-obstructive non-culprit lesions.







Methods

STEMI patients after culprit lesion PCI AND at least one target non-culprit lesion with > 70% stenosis suitable for OCT imaging

Randomized to complete revascularization as part of the COMPLETE trial (N=66)
OR planned to undergo NCL PCI (N=38)

Multivessel OCT imaging (N=93)

- 1. Vessel with non-culprit lesion for PCI
- 2. Additional vessel with or without target non-culprit lesion for PCI
- 3. STEMI vessel if ≥50 mm of unstented segment



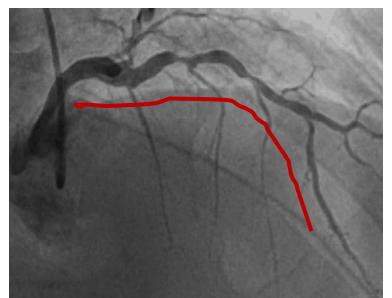






OCT COMPLETE: Imaging Protocol

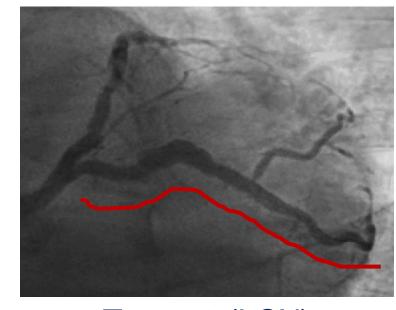
OCT imaged segment (staged non-culprit PCI procedure)



Target 1 (LAD) **Obstructive NCL**



Target 2 (RCA) Additional vessel Obstructive or Non-obstructive lesions If \geq 50 mm unstented segment



Target 3 (LCX) STEMI vessel

- Number of pullbacks / patient (mean): 2.82
- Imaged length / patient (mean): 152.5 mm

Example: Inferior STEMI Culprit lesion LCX, Non-culprit lesion LAD









Baseline and Procedure Characteristics

	OCT Complete N=93
Age (yrs)	61.2
Gender (% male)	82.8
Diabetes (%)	12.9
Chronic renal insuff. (%)	1.1
Prior MI (%)	8.6
Current smoker (%)	38.5
Hypertension (%)	41.9
Dyslipidemia (%)	43
Prior PCI (%)	7.5
Prior stroke (%)	1.1
Hemoglobin A1C	6.1
LDL (mmol/L)	2.9
Creatinine (µmol/L)	82

	OCT Complete N=93			
Residual diseased vessels				
1	64%			
≥2	36%			
NCL location				
Left main	0%			
LAD	41%			
Proximal LAD	10.4%			
Mid LAD	24.6%			
Circumflex	32.1%			
RCA	26.9%			
NCL stenosis (visual)				
70-79%	40.7%			
80-89%	31.7%			
90-99%	26.8%			
100%	0.8%			

No significant differences compared with the overall COMPLETE trial characteristics









Classification of non-culprit lesions

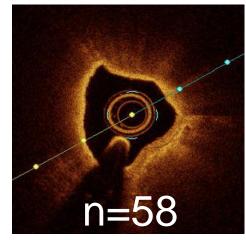
TCFA (FCT < 65 μm overlying a lipidic plaque)

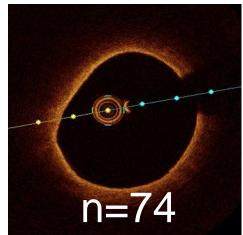
Yes No

Obstructive ≥70% DS

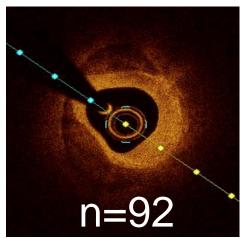
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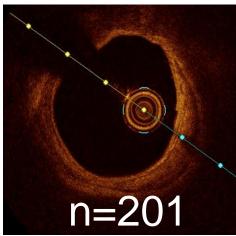
Yes





N=425





TCFA: Thin Cap Fibro Atheroma FCT: Fibrous Cap Thickness

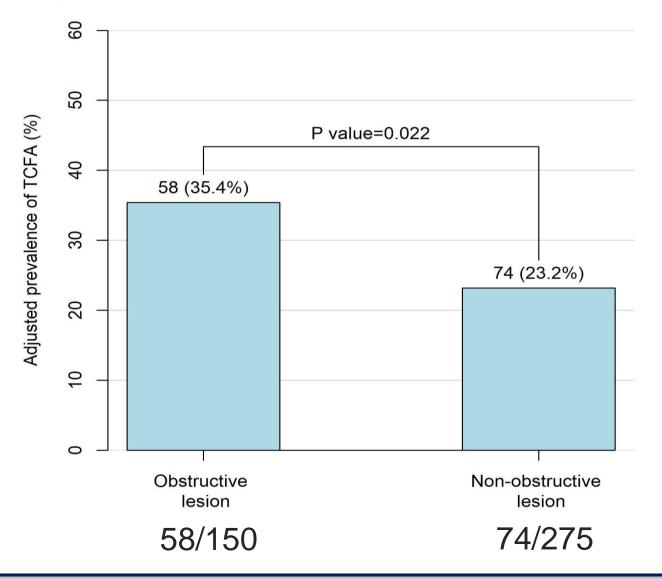








Primary Outcome: Prevalence of TCFA (per lesion)



Obstructive non-culprit lesions are most likely to be vulnerable

TCFA: Thin Cap Fibro Atheroma









Results: Features of TCFA vs Non-TCFA in Obstructive lesions (≥ 70% diameter stenosis)

	TCFA (N=58)	Non-TCFA (N=92)	P value
Lesion Length (mm)	23.1	20.8	0.16
Number of I LIPID quadrants	55.2	19.2	<0.001
% of LIPID quadrants	78.4	36.5	<0.001
Number of Fibrous quadrants	9.4	21.2	<0.001
% of Fibrous quadrants	16.9	43.7	<0.001
Number of Calcified quadrants	2.5	9.8	<0.001
% of Calcified quadrants	4.1	20.1	<0.001
Maximum Lipid Arc	342.2	212.5	<0.001
Mean Lipid Arc	203.8	84.5	<0.001
Mean FCT (μm)	54.5	152.2	<0.001
Minimum Lumen Area	1.9	1.7	0.52
Macrophages	55	48	<0.001
Microvessels	19	28	0.77
Cholesterol Crystals	48	42	<0.001

Obstructive TCFA lesions had significantly more lipid and more features of plaque vulnerability compared with non-obstructive TCFA **lesions**

TCFA: Thin Cap Fibro Atheroma









Results: TCFA (FCT < 65 µm overlying a lipidic plaque)

Lesion Length (mm) 23.1 16.7 <0.001 Number of LIPID quadrants 55.2 36.4 0.05 % of LIPID quadrants 78.4 76.8 0.73 Number of FIBROUS quadrants 9.4 7.1 0.10 % of FIBROUS quadrants 16.9 16.2 0.88 Number of CALCIFIED quadrants 2.5 1.7 0.26 % of CALCIFIED quadrants 4.1 7.0 0.39 Maximum Lipid Arc 342.2 304.0 0.06 Mean Lipid Arc 203.8 191.8 0.34 Mean FCT (μm) 54.5 54.5 0.98		Obstructive (N=58)	Non-obstructive (N=74)	P value
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Minimum Lumen Area 1.9 4.8 <0.001	Minimum Lumen Area	1.9	4.8	<0.001
Macrophages 55 65 0.28	Macrophages	55	65	0.28
Microvessels 19 23 0.86	Microvessels	19	23	0.86
Cholesterol Crystals 48 29 <0.001	Cholesterol Crystals	48	29	<0.001

Obstructive and nonobstructive TCFA lesions have similar plaque composition

Obstructive TCFA
lesions were longer
and had a smaller
MLA

TCFA: Thin Cap Fibro Atheroma FCT: Fibrous Cap Thickness MLA: Minimum Lumen Area





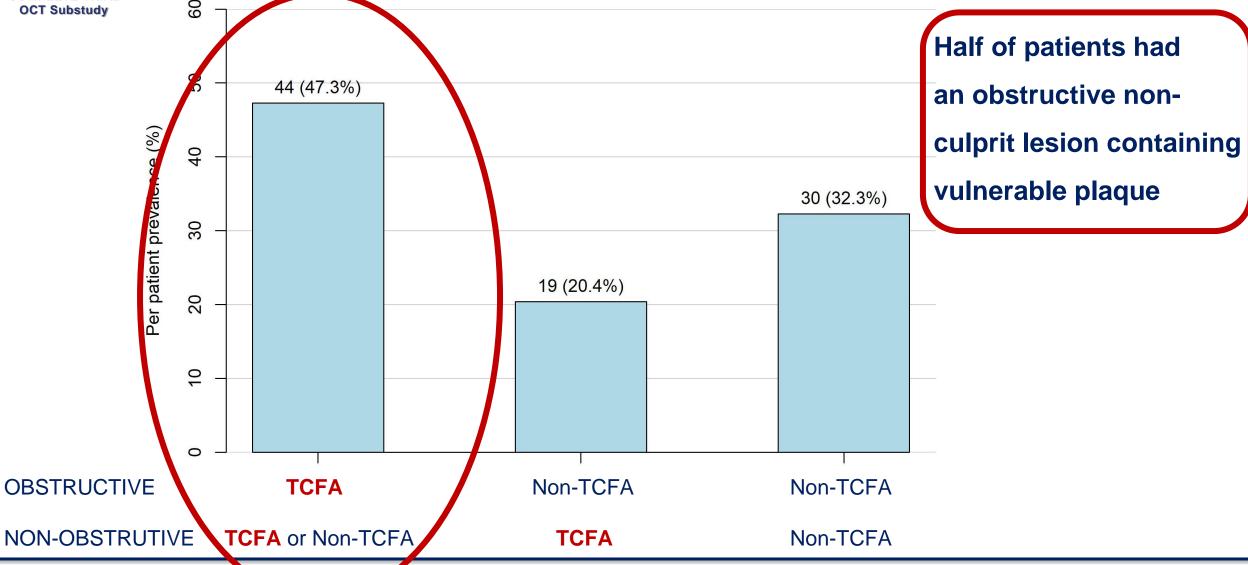




Population Health Research Institute

HEALTH THROUGH KNOWLEDGE

Results: Prevalence of TCFA (per patient)









Limitations

- The COMPLETE OCT substudy was observational and designed to better understand NCL plaque morphology. It was not powered to link clinical events to plaque morphology.
- The requirement for angiographically suitable arteries for OCT imaging may have excluded certain plaque types.
- Pre-dilatation was required in some severely-stenosed obstructive lesions before imaging (18.6%), the MLA may have been overestimated in these cases.







Conclusions

In patients with STEMI and multi-vessel coronary artery disease:

- Half of patients had a non-culprit lesion with vulnerable plaque morphology by OCT.
- Obstructive lesions (>70% visual diameter stenosis) more commonly harbor vulnerable plaque morphology than non-obstructive lesions.
- This may explain the benefit of routine PCI of obstructive non-culprit lesions in patients with STEMI and multivessel disease.





