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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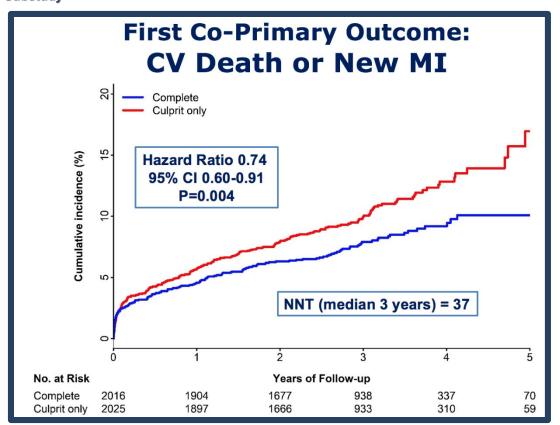


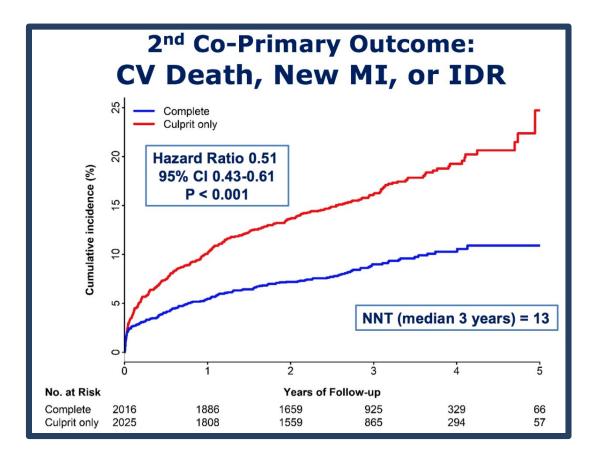






COMPLETE Trial – Primary outcomes





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).







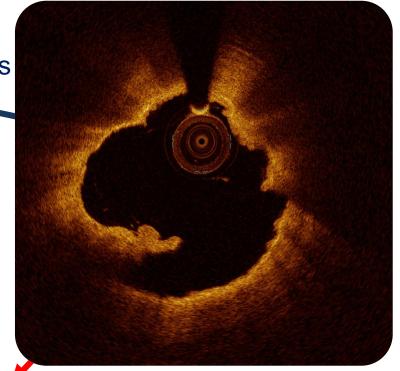


Background

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.

FCT: Fibrous
Cap Thickness
< 65 µm



Overlying a lipidic plaque Lipid arc > 90°

Plaque rupture









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 $\geq 70\%$ 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



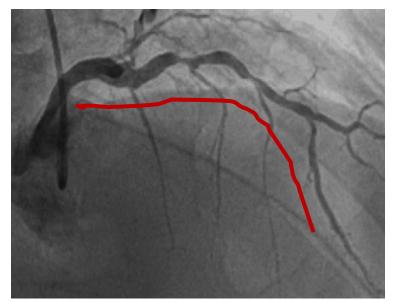






OCT COMPLETE: Imaging Protocol

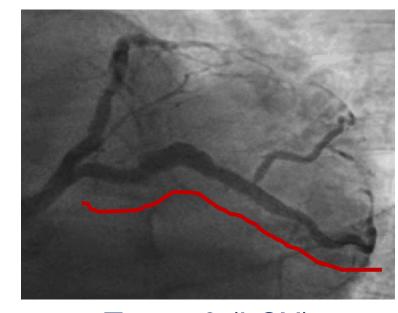
OCT imaged segment (staged non-culprit PCI procedure)



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

Case 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)

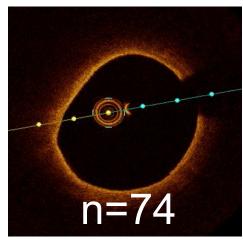
Yes No

Obstructive >70% DS

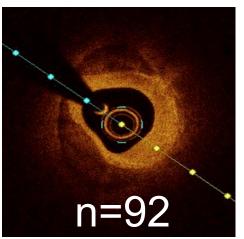
Yes

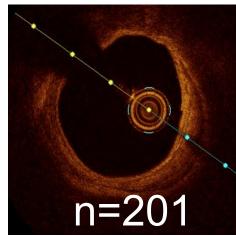
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n=58



N=425





TCFA: Thin Cap Fibro Atheroma FCT: Fibrous Cap Thickness

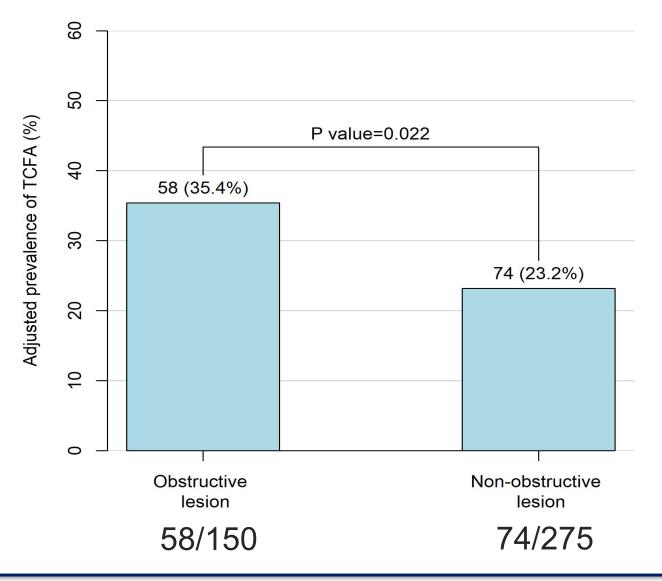








Primary Outcome: Prevalence of TCFA (per lesion)



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)

	Obstructive (N=58)	Non-obstructive (N=74)	P value
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
Minimum Lumen Area	1.9	4.8	<0.001
Macrophages	55	65	0.28
Microvessels	19	23	0.86
Cholesterol Crystals	48	29	<0.001
% of CALCIFIED quadrants Maximum Lipid Arc Mean Lipid Arc Mean FCT (μm) Minimum Lumen Area Macrophages Microvessels	4.1 342.2 203.8 54.5 1.9 55	7.0 304.0 191.8 54.5 4.8 65 23	0.39 0.06 0.34 0.98 <0.001 0.28 0.86

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

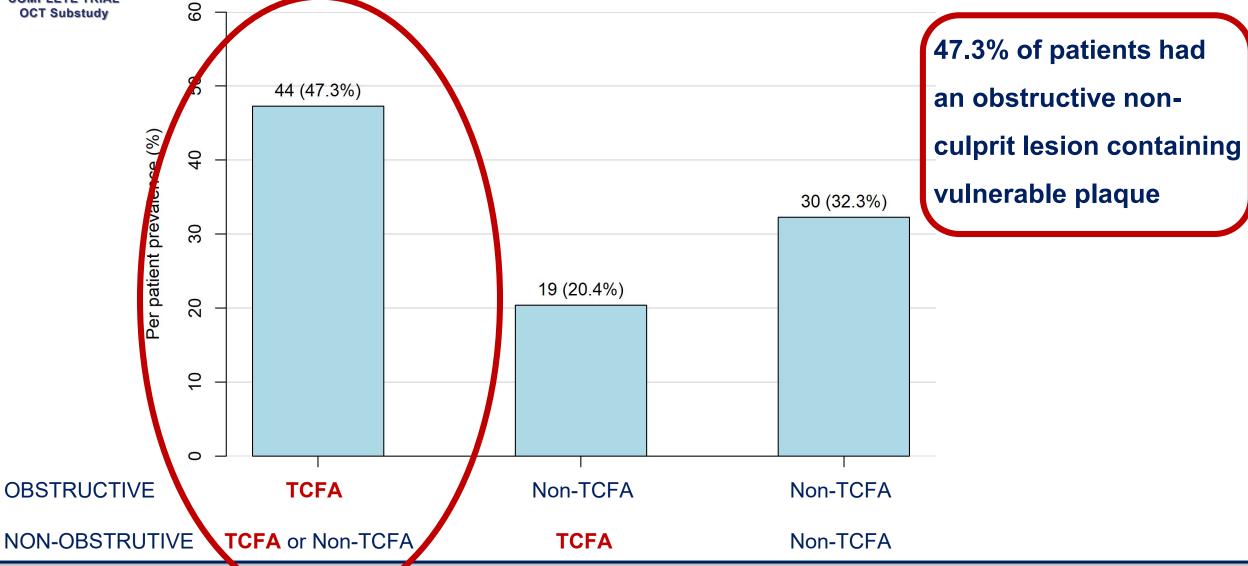








Results: Prevalence of TCFA (per patient)











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.





