

Artificial Intelligence Guided Evaluation of Atherosclerosis and Vessel Morphology in Non-ST Elevation Myocardial Infarction from Cardiac Computed Tomography (AI NSTEMI-CCTA)

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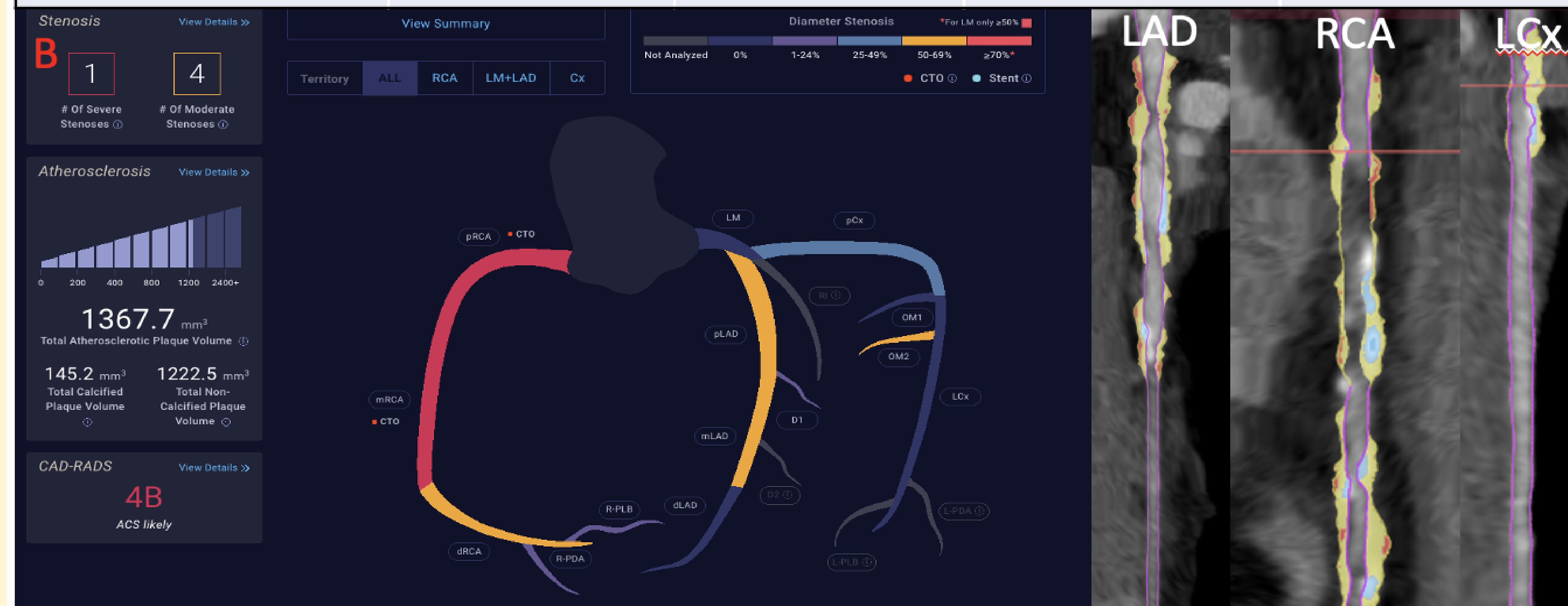
Background

- Invasive and pathologic evaluation of acute coronary syndrome/non-ST elevation myocardial infarction (NSTEMI) has established the presence of high plaque burden, thin-cap fibroatheroma and necrotic core.
- Artificial intelligence (AI) guided assessment of atherosclerosis from cardiac computed tomography angiography (CCTA) is a novel approach, has been rarely performed in NSTEMI, and may enable individualized understanding of plaque in these patients.

Methods

- Using Artificial Intelligence Aided CCTA analysis, (Cleerly, NY, NY; figure below), whole heart atherosclerosis burden was quantified in consecutive low-intermediate risk (GRACE score <140) NSTEMI patients (N=53) versus troponin negative acute chest pain (TN-ACP) patients (N=121) without known coronary artery disease undergoing CCTA.
- On a per-patient and per-vessel territory basis, atherosclerotic plaque characteristics (APC) studied included %diameter stenosis, plaque atheroma volume (PAV), non-calcified plaque (NCP) volume, calcified plaque (CP) volume, low-density noncalcified plaque (LD-NCP), positive remodeling (PR).

AI-CCTA Plaque Characteristics (Per Patient) of Acute Chest Pain Patients				
Variable, mean (SD)	All Patients (N=174)	NSTEMI (N=53)	Troponin Negative (N=121)	P-Value
Plaque Volume (PV), mm ³	162.3 (301.3)	311.6 (479.2)	99.9 (144.5)	0.0094
Low-Density Non-Calcified Plaque (LD-NCP), mm ³	7.4 (29.8)	19.2 (53.0)	2.5 (4.9)	0.0424
Non-Calcified Plaque (NCP), mm ³	110.4 (183.9)	192.9 (281.0)	76.0 (106.8)	0.0197
Calcified Plaque (CP), mm ³	44.5 (118.5)	99.6 (195.4)	21.5 (49.0)	0.0065
LD-NCP % Percent Atheroma Volume (PAV)	0.2 (0.7)	0.5 (1.2)	0.1 (0.2)	0.0178
NCP %PAV	3.4 (4.9)	5.4 (6.9)	2.5 (3.4)	0.0053
Remodeling Index	1.2 (0.2)	1.30 (0.29)	1.17 (0.15)	0.0029
Obstructive Stenosis (>50%), n(%)	16 (9%)	10 (19%)	6 (5%)	0.007
Obstructive Stenosis (>70%), n(%)	9 (5%)	6 (11%)	3 (2%)	0.0202



A. Plaque characteristics and obstructive stenosis by Artificial Intelligence Guided Cardiac Computed Tomography Angiography (AI-CCTA): Atherosclerotic plaque characteristics of non-ST elevation myocardial infarction (NSTEMI) and troponin negative acute chest pain patients on a per-patient basis. NSTEMI patients demonstrated significantly higher overall plaque burden as well as high risk plaque features including low-density non-calcified plaque and obstructive stenosis.
B. Case Example of AI-CCTA in NSTEMI: 42-year-old with hyperlipidemia presenting with acute chest pain found to have obstructive multi-vessel coronary artery disease identified clinically by cardiac computed Tomography Angiography (CCTA) and confirmed on invasive angiography. AI-guided CCTA found very high total atherosclerotic plaque burden including non-calcified and low attenuation plaque identified by AI-guided CCTA. Left panel shows graphical presentation of plaque burden. Right panel shows color-coded overlay guided of plaque burden in the coronary vessels as identified by AI-CCTA.

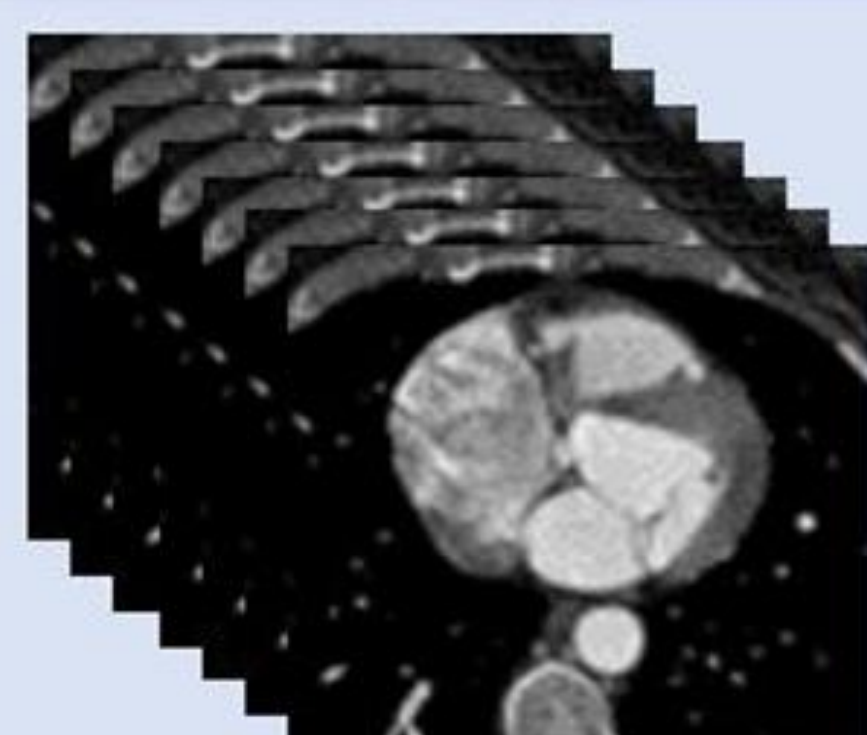
Results

- On a per-patient basis, NSTEMI patients demonstrated higher total PAV (312 mm³ vs 100mm³; p=0.0094), LD-NCP (19.2 mm³ vs 2.5mm³; p=0.0424), NCP (193 mm³ vs 76mm³; p=0.0197), CP (100 mm³ vs 22mm³; p=0.0065), and PR(1.30 vs 1.17; p=0.0029) (Figure)
- NSTEMI's, on a per-vessel basis, exhibited significantly higher PAV and lesion length in vessels with obstructive (≥ 50%) and non-obstructive (<50%) stenosis.
- In obstructive lesions, the NSTEMI group exhibited significantly higher LD-NCP

Disclosures:
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JPE – Equity & Employee, Cleerly
Other authors - none

Artificial Intelligence Aided CCTA Analysis

CCTA Data



Conclusions and Implications

Artificial Intelligence enabled precise quantification of adverse plaque characteristics including total plaque burden, LD-NCP and NCP plaque associated with NSTEMI

These findings may inform an **individualized approach** to prevention of future myocardial infarction guided by an enhanced understanding of plaque progression