Frequency of Myocardial Infarction and Its Relationship to Angiographic Collateral Flow in Territories Supplied by Chronically Occluded Coronary Arteries

Running title: Choi et al.; CMR reveals hidden MI of CTO

Jin-Ho Choi, MD, PhD1,2; Sung-A Chang, MD, PhD1; Jin-Oh Choi, MD, PhD1; Young Bin Song MD, PhD1; Joo-Yong Hahn MD, PhD1; Seung Hyuk Choi MD, PhD1; Sang-Chol Lee, MD, PhD1; Sang-Hoon Lee, MD, PhD1; Jae K. Oh, MD1,4; YeonHyeon Choe, MD, PhD1,3; Hyeon-Cheol Gwon, MD, PhD1

1Dept of Medicine; 2Dept of Emergency Medicine; 3Dept of Radiology, Cardiovascular Imaging Center, Cardiac and Vascular Center, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Korea; 4Dept of Internal Medicine, Mayo Clinic College of Medicine, Rochester, MN

Address for Correspondence:
Hyeon-Cheol Gwon, M.D, PhD
Department of Medicine, Samsung Medical Center
Sungkyunkwan University School of Medicine
50 Irwon-dong, Gangnam-gu, Seoul, 135-710, Republic of Korea
Tel: 82-2-3410-3419
Fax: 82-2-3410-3849
E-mail: hcgwon62@gmail.com

Journal Subject Codes: [7] Chronic ischemic heart disease; [30] CT and MRI
Abstract:

Background—Despite complete interruption of antegrade coronary artery flow in the setting of a chronic total occlusion (CTO), clinical recognition of myocardial infarction (MI) is often challenging. Using cardiac magnetic resonance imaging (CMR), we investigated the frequency and extent of MI in patients with CTO, and assessed their relationship with regional systolic function and the extent of angiographic collateral flow.

Methods and Results—We included 170 consecutive patients (median age 62 years) with angiographically documented CTO. Regional late gadolinium enhancement (LGE) and wall motion score index (WMSI) were assessed by CMR using a 17-segment model. Angiographic collateral flow was assessed by the collateral connection grade and the Rentrop score. Evidence of prior MI was found in 25% of patients by ECG Q waves, in 69% by regional wall motion abnormality, and in 86% of patients by LGE. Increased angiographic collateral flow was associated with a lower frequency of Q waves on ECG, as well as a lower regional WMSI, LGE volume (%), and degree of LGE transmurality (all p<0.001).

Conclusions—The frequency of MI in territories subtended by CTO is significantly higher than previously recognized. The degree of myocardial injury downstream epicardial CTO is inversely correlated with the degree of angiographic collaterals.

Key words: cardiac magnetic resonance imaging; chronic total coronary occlusion; delayed enhancement MRI
Chronic total occlusion (CTO) of coronary arteries is defined by angiographic total occlusion of duration >3 months. CTO is not uncommon in patients with coronary artery disease and the catheterization-documented incidence of CTO has been reported to be as high as 20 to 30%. Despite the complete interruption of antegrade coronary artery blood flow caused by heavy atherosclerotic plaque burden, clinical recognition of myocardial infarction (MI) can be challenging. The latter can have important implications especially with respect to decisions regarding myocardial revascularization.

Contrast enhanced cardiac magnetic resonance imaging (CMR) enables direct visualization of infarcted myocardium and has become the gold standard for quantification of myocardial infarct size. We hypothesized that the prevalence of MI in patients with CTO is higher than previously known. We investigated the frequency and extent of MI measured by CMR in patients with CTO. The results were compared with ECG, left ventricular (LV) regional wall motion, and the extent of angiographic collateral flow.

Methods

Patients

From Jan 2007 to Dec 2011, we prospectively screened 217 consecutive patients who had undergone clinically indicated CAG and were found to have a CTO in at least one major epicardial coronary artery. Patients with more than 50% stenosis in the left main artery were excluded, as were those with acute coronary syndrome within 90 days of enrollment, decompensated heart failure, or contraindications to CMR. Patients with prior revascularization (N=38) were also excluded due to the potential confounding of peri-procedural myocardial injury. CMR was performed within 2 weeks of CAG (median interval period of 2 days).
Finally, patients with potential non-ischemic causes of myocardial injury (N=6) and poor CMR images (N=3) were also excluded. Thus, the remaining 170 patients comprised the study cohort (Figure 1). The institutional review board committee approved the study protocol and all patients provided written informed consent to participate in the study.

**Clinical History and Electrocardiography**

The presence of prior MI on 12-lead ECG was defined as the presence of pathological Q waves involving ≥2 contiguous leads according to the 3rd universal definition of MI. Because ECG is known to underestimate the prevalence of MI in patients with CTO, we also evaluated a history of ischemic symptoms consistent with MI.

**Coronary Angiography**

CAG was performed using standard techniques. CTO was defined by the presence of a coronary artery stenosis causing complete interruption of antegrade flow in a major epicardial coronary artery or minimal contrast penetration though the lesion without distal vessel opacification (Thrombolysis in Myocardial Infarction [TIMI] grade 0-1 flow). Since patients with acute coronary syndromes within 90 days of enrollment were excluded, the presence of a total occlusion on CAG was assumed to be chronic (at least 3 months). Retrograde collateral filling of the vessel distal to a CTO was assessed by experienced interventional cardiologists blinded to other clinical and/or imaging data. The diameter and angiographic flow of collateral vessels was semi-quantitatively assessed using the collateral connection grade (CC0=no continuous connection, CC1=continuous thread-like connection, CC2=continuous, small branch-like connection) and the Rentrop classification (class 0=no visible filling of collaterals, class 1=filling of side branches, class 2=partial filling of epicardial segment of the occluded vessel, class 3=total filling of epicardial segment). The presence of well-developed angiographic
collaterals was defined as a collateral connection grade=2 and Rentrop score=3.

Cardiac Magnetic Resonance Imaging

A 1.5T scanner (Magnetom Avanto, Syngo MR; Siemens Medical Solutions, Erlangen, Germany) was used. Cine images of the LV myocardium were acquired using a steady-state free-precession sequence with 8 to 10 contiguous short-axis slices and a slice thickness of 6 mm and 4 mm gaps. Late gadolinium enhancement (LGE) was imaged by contiguous short-axis image acquisition of 10 to 12 slices. Phase-sensitive inversion recovery technique was performed 10 minutes after injection of 0.15 mmol/kg Gadovist (Bayer Schering Pharma, Berlin, Germany). Inversion delay time was 280 to 360 milliseconds.

CMR was analyzed by investigators blinded to other clinical and angiographic information. An independent workstation (CAAS MRV version 3.4, Pie medical imaging, Maastricht, The Netherlands) was used. Endocardial and epicardial borders were manually drawn on all LV short-axis images. LGE was defined by enhanced areas showing 6SD above the signal intensity of remote non-infarcted myocardium, which was measured automatically and manually corrected when needed. The infarct gray zone was defined as pixels with <50% of maximal signal intensity of highest signal intensity within LGE. The transmurality of LGE (0%, 1-24%, 25-49%, 50-74%, 75-100%) and regional wall motion abnormality (RWMA) score (normal=1, hypokinetic=2, akinetic=3, dyskinetic=4, aneurismal=5) were assessed in each segment of LV 17-segment model.

Assignment of LV segments to the vessel with CTO was defined based on the American Heart Association scientific statement. The global left ventricular LGE volume (%), as well as regional LGE transmurality and wall motion score index (WMSI) in vessels with or without angiographic CTO were determined and compared with the presence of Q wave and
angiographic extent of collateral flow.

**Statistical Analysis**

All analysis was done on per-patient basis if not indicated otherwise. Data were not normally distributed and non-parametric statistics were applied. Continuous variables are shown as the median with 1st and 3rd quartiles in parentheses. Continuous and categorical variables were compared by the Mann-Whitney U or chi-square test. Reclassification rate between diagnostic modalities was calculated using Pencina’s reclassification index. SPSS version 19.0 was used. A two-tailed p<0.05 was considered statistically significant.

The inter- and intra-observer agreement of CMR and angiographic scores was assessed from 21 randomly selected cases. Cohen’s kappa was 0.81 and 0.82 for collateral connection grade, 0.73 and 0.81 for Rentrop classification, 0.75 and 0.83 for wall motion score, and 0.80 and 0.87 for LGE transmurality. The limits of agreement of the LGE volume (%) were -2.1±7.4% and 2.6±6.4% by Bland-Altman analysis, respectively.

**Results**

**Clinical Characteristics**

Table 1 summarizes the clinical characteristics of the study cohort. The median age of our study population was 62 (IQR, 55 – 70) years, and 89% of patients were male. The majority of patients had angina (61%). Fifty eight percent of patients did not have a documented clinical history of ischemic symptoms consistent with prior MI.

**Frequency of Myocardial Infarction by Clinical History, ECG, and CMR**

The frequency of prior MI was strikingly different based on clinical and imaging criteria. It was found only in 25% of patients by pathological Q waves on ECG, in 42% by prior ischemic
symptom consistent with MI, and in 69% by the presence of RWMA. However, 86% of patients had evidence of LGE by CMR. Representative cases showing discrepancies between these diagnostic modalities are shown in Figure 2A-D.

**Relationship between Angiographic Collateral Flow, Presence of Q waves on ECG, Regional Wall Motion, and Transmurality of Late Gadolinium Enhancement**

We investigated whether LGE transmurality or RWMA are specific to the presence of Q waves or LV segments assigned to vessels with CTO. The degree of LGE transmurality, LGE volume (%), and WMSI were higher in patients with Q waves on ECG (all p<0.001; Figure 3A-C). Likewise, regional WMSI was higher in LV segments subtended by CTO than in vessels without CTO, as well as in LV segments with higher degrees of transmural scar on LGE (Figure 3C-D).

The presence of well-developed angiographic collaterals distal to a CTO was associated with a lower frequency of ECG with Q waves, as well as lower regional LGE transmurality and volume (%), and WMSI (all p<0.001; Figure 3E-H). The detailed results are listed in online-only materials (Supplementary Table).

**Discussion**

To the best of our knowledge, our study is the first to show that myocardial scar, as defined by contrast-enhanced CMR, in territories subtended by upstream CTO is much more common than previously known. Consistent with prior experimental and clinical data, we showed that the presence of well-developed angiographic collaterals distal to CTO was associated with a lower frequency and transmurality of prior MI, suggesting a protective role of timely developed collaterals distal to a CTO.

**Frequency of Late Gadolinium Enhancement and Regional Wall Motion Abnormality**
CTO is presumed to originate from the organization of thrombi developed in the context of a non-fatal MI. However, objective evidence of myocardial injury has been documented in up to fifty percent of patients in most clinical studies.\(^1\),\(^3\)-\(^6\),\(^9\)-\(^12\) The 25% frequency of abnormal ECGs displaying Q waves is consistent with this notion. However, the use of CMR imaging in our study revealed a significantly higher frequency of MI – 86% in our study patients. On the other hand, 14% of our patients did not show LGE, and all had normal ECGs. Nonetheless, LGE may not be detected in areas of very small myocardial injury.\(^7\),\(^26\)-\(^28\) Hence, our data suggest that most myocardial territories distal to a CTO contain varying degrees of myocardial scar.

Although some degree of LGE was found in 86% of patients, an associated RWMA was found only in 69% of them. However, most patients with a RWMA showed LGE transmurality \(>25\%\), which is consistent with the notion of a threshold phenomenon governing the relationship between scar burden and regional systolic function.\(^24\) The varying extent of transmural injury explains, at least in part, some of the apparently incongruent results between the frequency of LGE and regional function. In these patients, a RWMA may also represent viable but stunned and/or hibernating myocardium due to inadequate coronary flow reserve.\(^29\)-\(^31\)

**Coronary Collaterals and Transmurality of Myocardial Infarction**

We found that the presence of well-developed collateral vessels was inversely correlated with the degree transmural injury and was also associated with a lower frequency of abnormal ECG with Q waves. Indeed, the frequency of transmural infarct defined by LGE transmurality \(\geq 50\%\) was 19% in patients with well-developed collaterals whereas it was 49% among those with poorly developed collateral vessels (**Figure 3F**).

Human coronary arteries are not functionally end-arteries but rather interconnected by a rich network of collateral vessels.\(^32\) It is estimated that about one-fourth of individuals have
functional collateral vessels able to reduce or prevent myocardial ischemia induced by brief abrupt reduction of antegrade flow.\textsuperscript{28,33} The size and transmural extent of myocardial infarction is determined by coronary artery occlusion time, the extent of myocardium at risk, and the degree of collateral blood flow at the time of coronary occlusion.\textsuperscript{34} Therefore, increased collateral flow to a territory suddenly deprived of its natural antegrade flow should ameliorate the extent of transmural myocardial necrosis and attenuate LV dysfunction.\textsuperscript{35-37} Indeed, well-developed collateral circulation reduces the infarct size in ST-elevation MI as well as long-term mortality in patients with stable angina.\textsuperscript{38-45}

**Limitations**

Our findings represent a single center experience. The duration of chronic total occlusion was likely heterogeneous. Our definition of prior MI was retrospective and predicated by on the presence of ischemic symptoms and Q waves on ECG, which may not be completely accurate.\textsuperscript{3} Our result was derived from a population with high prevalence of multivessel disease and need of revascularization and may not be translated to populations with less severe disease. The physiological consequence of collateral vessels was not interrogated by assessment of stress testing. We followed standardized but empirical assignment of coronary arteries to specific myocardial segments, which may be discordant especially for non-LAD vessels.\textsuperscript{46} Patient-specific coronary artery and myocardial mapping would be required for more refined evaluation.\textsuperscript{47}

**Conclusions**

Most patients with chronic coronary artery occlusions show evidence of prior MI. The transmural extent of myocardial injury and presence of regional LV dysfunction are inversely
related with the degree of angiographic collaterals. Our results are consistent with a protective role of collateral blood flow against myocardial damage related to chronic total occlusion.

Conflict of Interest Disclosures: None.

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Table 1. Clinical Characteristics.

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<tr>
<th>Demographics and risk factors</th>
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<tr>
<td>Age (year)</td>
<td>62 (55 – 70)</td>
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<tr>
<td>Male gender</td>
<td>88.8 (151)</td>
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<td>Body mass index (kg/M$^2$)</td>
<td>24.9 (22.7 – 26.9)</td>
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<tr>
<td>Hypertension</td>
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<tr>
<td>Diabetes</td>
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<td>Hyperlipidemia</td>
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<td>Smoking</td>
<td>45.9 (78)</td>
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<td>Maintenance dialysis</td>
<td>0.6 (1)</td>
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<td>Stroke</td>
<td>10.0 (17)</td>
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<tr>
<td>Hemoglobin (g/dL)</td>
<td>13.7 (12.5 – 15.3)</td>
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<tr>
<td>Creatinine (g/dL)</td>
<td>0.96 (0.79 – 1.07)</td>
</tr>
<tr>
<td>C-reactive protein (mg/dL)</td>
<td>0.10 (0.05 – 0.34)</td>
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Previous history of myocardial infarction

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<tr>
<td>3 – 12 month</td>
<td>28.9 (49)</td>
</tr>
<tr>
<td>&gt;12 month</td>
<td>12.9 (22)</td>
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<tr>
<td>No documented prior myocardial infarction</td>
<td>58.2 (99)</td>
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Diagnosis

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<tbody>
<tr>
<td>Stable angina</td>
<td>50.0 (85)</td>
</tr>
<tr>
<td>Silent ischemia*</td>
<td>38.8 (66)</td>
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<tr>
<td>Unstable angina</td>
<td>11.2 (19)</td>
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Coronary angiography

<table>
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<th>Coronary angiography</th>
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<tr>
<td>1-vessel disease</td>
<td>33.5 (57)</td>
</tr>
<tr>
<td>2-vessel disease</td>
<td>41.8 (71)</td>
</tr>
<tr>
<td>3-vessel disease</td>
<td>24.7 (42)</td>
</tr>
</tbody>
</table>

Location of CTO lesion**

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<tr>
<th>Location</th>
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<tbody>
<tr>
<td>LAD</td>
<td>42.9 (73)</td>
</tr>
<tr>
<td>LCX</td>
<td>17.1 (29)</td>
</tr>
<tr>
<td>RCA</td>
<td>40.0 (68)</td>
</tr>
</tbody>
</table>

Results are shown as median (1st quartile – 3rd quartile) or frequency % (n). CTO, chronic total occlusion; LAD, left anterior descending artery; LCX, left circumflex artery; RCA, right coronary artery.

* The causes of diagnostic coronary angiography in patients with silent ischemia (N=66) were followings; syncope (6.1%, N=4), evaluation of exertional dyspnea without chest pain (16.7%, N=11), preoperative cardiovascular screening (28.8%, N=19), cardiovascular screening of patients with stroke and multiple risk factors (6.1%, N=4), very severe coronary artery calcification (42.4%, N=28).

** The location of CTO lesion was defined as the CTO supplying the largest vascular territory if there were multiple CTO lesions, which was found in 18.8% (N=32).
Figure Legends:

**Figure 1.** Study Subjects. Schematic of selection of study cohort.

**Figure 2.** Representative Cases Showing Discrepancies between Diagnostic Modalities. Clinical cases showing discrepancies between ECG, RWMA, and LGE in patients with CTO. Coronary angiography of each case is shown in separate movie files. RWMA, regional wall motion abnormality; CMR, cardiac magnetic resonance imaging; LGE, late gadolinium enhancement. A. A case of proximal LAD total occlusion (blue arrow) showing no Q waves and no RWMA. Left ventricular ejection fraction (LVEF)=61%. No LGE was identified. B. A case of mid-LAD total occlusion (blue arrow) showing no Q waves and no RWMA. LVEF=64%. However, subendocardial LGE was identified (pink arrow). LGE mass=9.6 g. C. A case of mid-LAD total occlusion and distal LCX subtotal occlusion. There were no pathological Q waves on the ECG. LVEF=58%. CMR showed LGE involving the anteroseptal wall. D. A case of mid-LAD total occlusion (blue arrow) and proximal LCX stenosis. Q waves in anterior leads were identified. CMR showed mildly dilated LV cavity, systolic dysfunction (LVEF=45%), and hypokinesia of anterior to apical wall. Extensive LGE is seen from the mid-ventricular anterior to apical segments but not in basal inferolateral segments.

**Figure 3.** Relationship between Angiographic Collateral Flow, Presence of Q waves on ECG, Regional Wall Motion, and Transmurality of Late Gadolinium Enhancement. LGE, late gadolinium enhancement. *p<0.001 by Mann-Whitney U or chi-square rest. A. The frequency of transmural MI was significantly lower in patients without Q waves than patients with Q waves.
The frequency of LGE transmurality=0%, 1-25%, 25-49%, 50-74%, 75-100% was as followings: in patients without Q waves, 18.8% (N=24), 25.8% (N=33), 29.7% (N=38), 19.5% (N=25), 6.3% (N=8); in patients with Q waves, 0% (N=0), 4.8% (N=2), 16.7% (N=7), 40.5% (N=17), 38.1% (N=16) (p<0.001 by chi-square test). B. LGE volume was significantly lower in patients without Q waves than patients with Q waves, 5.7% (1.7 – 12.8%) versus 19.1% (13.4 – 26.1%) (p<0.001 by Mann-Whitney U test). In box plots, the top, middle line, and bottom of the box represent the 75th, 50th, and 25th percentile. The whiskers represent the highest and lowest values that are not outliers or extreme values, which are more than 1.5 times the interquartile range and shown as separate circles. C. Patients with Q waves in ECG showed significantly higher WMSI compared to patients without Q waves. WMSI of LV segments assigned to vessels with CTO; 2.00 (1.54 – 2.23) versus 1.11 (1.00 – 1.50); WMSI of LV segments assigned to vessels without CTO; 1.30 (1.13 – 1.51) versus 1.00 (1.00 – 1.10), p<0.001 by Mann-Whitney U test, both. D. Consistent correlation between LGE transmurality and regional WMSI is shown. Note the overall higher LGE transmurality and WMSI in LV segments assigned to vessels with CTO compared to the other LV segments. The WMSI of LV segments assigned to vessels with CTO according to the LGE transmurality=0%, 1-25%, 25-49%, 50-74%, 75-100% was as followings: 1.00 (1.00 – 1.00), 1.00 (1.00 – 1.20), 1.30 (1.08 – 1.57), 1.79 (1.40 – 2.14), 2.18 (1.85 – 2.54). The WMSI of the other LV segments according to the LGE transmurality: 1.00 (1.00 – 1.00), 1.00 (1.00 – 1.00), 1.04 (1.00 –1.25), 1.23 (1.10 – 1.46), 1.45 (1.14 – 1.87). p<0.001 by Kruskal-Wallis and Jonckheere-Terpstra test, all. E. Well-developed collaterals were defined by collateral connection grade=2 and Rentrop Class=3 collateral flow (N=82). Poorly developed collaterals were defined by collateral connection grade <2 and/or Rentrop Class <3 collateral flow (N=88). The frequency of Q waves was significantly lower in patients with well-developed collaterals compared to
patients with poorly developed collaterals: 12.2% (N=10) versus 36.4% (N=32) (p<0.001 by chi-square test). **F.** The frequency of transmural MI was significantly lower in patients with well-developed collaterals compared to patients with poorly developed collaterals. In patients with well-developed collaterals, LGE transmurality 0%=28.0% (N=23), 1-24%=23.2% (N=19), 25-49%=29.3% (N=24), 50-74%=12.2% (N=10), 75-100%=7.3% (N=6); in patients with poorly developed collaterals, it was 3.4% (N=3), 26.1% (N=23), 21.6% (N=19), 33.0% (N=29), and 15.9% (N=14), respectively (p<0.001 by chi-square test). **G.** LGE volume was significantly lower in patients with well-developed collaterals compared to patients with poorly developed collaterals, 5.8% (0.0 – 13.0%) versus 12.0% (4.9 – 21.7%) (p<0.001 by Mann-Whitney U test). **H.** The WMSI of LV segments assigned to vessels with CTO was significantly lower in patients with well-developed collaterals compared to patients with poorly developed collaterals, 1.077 (1.000 – 1.579) versus 1.414 (1.074 – 2.000) (p<0.001 by Mann-Whitney U test).
Figure 1.

Patients with angiographical total occlusion without history of recent myocardial infarction (<90 days) were consecutively enrolled (N=217)

Excluded due to potential peri-procedural myocardial injury (N=38)
  • Previous history of percutaneous coronary intervention (N=31)
  • Previous history of coronary bypass surgery (N=7)

Patients with angiographical chronic total occlusion were evaluated by cardiac magnetic resonance imaging (N=179)

Excluded due to potential non-coronary causes of myocardial injury or inadequate image (N=9)
  • Hypertrophic cardiomyopathy (N=1)
  • Dilated cardiomyopathy (N=2)
  • Cardiac amyloidosis (N=1)
  • Severe aortic valvular stenosis (N=1)
  • Kawasaki’s disease (N=1)
  • Poor magnetic resonance imaging quality (N=3)

Final study subjects enrolled into analyses (N=170)
Figure 2.

A. Q wave (-), RWMA (-), and CMR LGE (-)

B. Q wave (-), RWMA (-), and CMR LGE (+)

C. Q wave (-), RWMA (+), and CMR LGE (+)

D. Q wave (+), RWMA (+), and CMR LGE (+)
Figure 3.

A
Frequency of LGE transmurality (%) * p<0.001

B
LGE volume (%) * p<0.001

C
WMSI

D
WMSI

E
Frequency of ECG Q wave (%) * p<0.001

F
Frequency of LGE transmurality (%) * p<0.001

G
LGE volume (%) * p<0.001

H
WMSI of CTO vessel segments * p<0.001
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### Table I. Relationship between Angiographic Collateral Flow, Q waves on ECG, Regional Wall Motion, and Transmurality of Late Gadolinium Enhancement according to Coronary Artery Location of CTO

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<th>RCA (N=68)</th>
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<td><strong>Frequency of LGE transmurality</strong></td>
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<td>No Q wave</td>
<td>Q wave</td>
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<td>0%</td>
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<td>11.5 (3)</td>
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<td>1-24%</td>
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<td>50-74%</td>
<td>15.7 (8)</td>
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<td>15.4 (4)</td>
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<td>75-100%</td>
<td>5.9 (3)</td>
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<tr>
<td><strong>p-value</strong></td>
<td>p&lt;0.001</td>
<td>p=0.54</td>
<td>p=0.002</td>
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|                  |            |            |            |
| **LGE volume%**  | No Q wave | Q wave     | No Q wave | Q wave     | No Q wave | Q wave     |
|                  | 5.6 (0.0 – 13.3) | 21.4 (14.3 – 24.8) | 4.7 (2.0 – 11.9) | 17.7 (12.9 – 18.4) | 5.9 (2.2 – 13.8) | 18.0 (6.7 – 32.9) |
| **p-value**      | p<0.001   | p=0.045    | p=0.001   |            |            |            |

|                  |            |            |            |
| **Frequency of ECG Q wave** | W/D collaterals | P/D collaterals | W/D collaterals | P/D collaterals | W/D collaterals | P/D collaterals |
| Q wave           | 7.9 (3)   | 54.3 (19)  | 15.4 (2)   | 6.3 (1)      | 16.1 (5)     | 32.4 (12)    |
| No Q wave        | 92.1 (35) | 45.7 (16)  | 84.6 (11)  | 93.8 (15)   | 83.9 (26)   | 67.6 (25)   |
| **p-value**      | p<0.001   | p=0.42     | p=0.12     |            |            |            |

|                  |            |            |            |
| **Frequency of LGE transmurality** | W/D collaterals | P/D collaterals | W/D collaterals | P/D collaterals | W/D collaterals | P/D collaterals |
| 0%               | 31.6 (12) | 5.7 (2)    | 23.1 (3)   | 0.0 (0)      | 25.8 (8)     | 2.7 (1)      |
| 1-24%            | 23.7 (9)  | 17.1 (6)   | 15.4 (2)   | 43.8 (7)     | 25.8 (8)     | 27.0 (10)    |
| 25-49%           | 21.1 (8)  | 20.0 (7)   | 46.2 (6)   | 25.0 (4)     | 32.3 (10)    | 21.6 (8)     |
| 50-74%           | 13.2 (5)  | 34.3 (12)  | 7.7 (1)    | 25.0 (4)     | 12.9 (4)     | 35.1 (13)    |
| 75-100%          | 10.5 (4)  | 22.9 (8)   | 7.7 (1)    | 6.3 (1)      | 3.2 (1)      | 13.5 (5)     |
| **p-value**      | p=0.018   | p=0.10     | p=0.012    |            |            |            |

|                  |            |            |            |
|                  | 5.3 (0.0 – 14.9) | 14.3 (6.2 – 23.6) | 8.9 (0.7 – 15.3) | 4.7 (2.3 – 11.6) | 5.0 (7.2 – 11.0) | 11.8 (5.3 – 23.6) |
| **p-value**      | p=0.002   | p=0.98     | p=0.002    |            |            |            |

|                  |            |            |            |
| **WMSI of LV assigned to CTO** | W/D collaterals | P/D collaterals | W/D collaterals | P/D collaterals | W/D collaterals | P/D collaterals |
|                  | 1.000 (1.000 – 1.1589) | 1.571 (1.000 – 2.143) | 1.200 (1.000 – 1.500) | 1.250 (1.025 – 1.950) | 1.200 (1.000 – 1.600) | 1.400 (1.100 – 2.000) |
| **p-value**      | p=0.007   | p=0.423    | p=0.037    |            |            |            |

Results are shown as median (1st quartile – 3rd quartile) or frequency % (n). W/D, well-developed; P/D, poorly developed. p-value between No Q wave and Q wave groups, or W/D collaterals and P/D collaterals are shown. * p-value by Chi-square test, ** p-value by Mann-Whitney U test.
Legend for movie files

Each movie is coronary angiography of representative cases shown in Figure 2

CAG_A_LAD_xvid.avi: Case A, left coronary artery
CAG_A_RCA_xvid.avi: Case A, right coronary artery
CAG_B_LAD_xvid.avi: Case B, left coronary artery
CAG_B_RCA_xvid.avi: Case B, right coronary artery
CAG_C_LAD_xvid.avi: Case C, left coronary artery
CAG_C_RCA_xvid.avi: Case C, right coronary artery
CAG_D_LAD_xvid.avi: Case D, left coronary artery
CAG_D_RCA_xvid.avi: Case D, right coronary artery