Quantitative Coronary Artery Motion Analysis Predicts the Location of Future ST Segment Elevation Myocardial Infarctions

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Abstract
Background: Coronary artery motion may contribute to the development of plaques that rupture and cause acute myocardial infarctions. This study evaluates whether a quantitative measure of the compression type of coronary artery motion obtained from analysis of coronary angiograms can predict the location of culprit lesions in patients who have subsequent myocardial infarction.

Method: 28 patients were identified with coronary angiography performed on at least two occasions: related to primary or rescue percutaneous coronary intervention for a STEMI and coronary angiography before this that was available for review. These angiograms were used to determine a quantitative index of coronary artery motion (QCAM) (the ratio of the section lengths i.e. systolic length/diastolic length). The culprit section was subsequently identified and QCAM of this section was compared to non-culprit sections. Results: The two sample t-test comparing QCAM for the non-culprit and culprit sections was highly statistically significant with a p-value of 0.0004. The generalized linear mixed model with culprit section as the dependent variable and QCAM as the independent variable also showed a statistically significant result with a p-value of 0.026. Conclusion: QCAM is a predictor of the location of culprit lesions causing future ST segment elevation myocardial infarctions. Predicting the location of future culprit lesions using coronary angiography may allow targeted therapy to prevent myocardial infarctions.

Keywords Coronary Artery Motion, Coronary Artery Disease, Myocardial Infarction

1. Introduction

Acute myocardial infarctions are usually caused by rupture of atherosclerotic plaques. Plaques form in the coronary arteries as a response to injury [1]. The location of plaques within the coronary arteries is asymmetrical [2], and their distribution is likely attributable to local biomechanical factors relating to fluid dynamics and wall mechanics. Coronary artery motion contributes to these biomechanical factors [3] and has been suggested to have an important role in the mechanisms of local injury [4].

Qualitative evaluation of coronary artery motion for artery segments has previously shown that the compression type of motion correlates with the location of disease and the degree of stenosis within coronary artery segments [5] and is independently predictive of the location of future culprit lesions responsible for ST segment elevation myocardial infarctions [6,7]. The compression type of coronary motion is defined as occurring when the length of the arterial segment is shortened without vertical deviation of the artery [5].

These findings have been extended to a quantitative method using multislice computed tomography coronary angiography [8]. A significant correlation was found between a quantitative measure of vessel centerline shortening and the location of coronary plaque and the degree of stenosis.

2. Objective

This study aims to see if a quantitative measure of the compression type of coronary artery motion (QCAM) predicts the location of culprit lesions in patients who have subsequent ST segment elevation myocardial infarction.

3. Methods
3.1. Patients

Patients were identified using the angiography databases at Westmead and Liverpool hospitals in Sydney with coronary angiography performed on at least two occasions:
1. related to primary or rescue percutaneous coronary intervention for an STEMI; and
2. coronary angiography before this that was available for review.

STEMI was defined as chest pain characteristic of ischaemia with ST segment elevation of ≥1mm in two contiguous limb leads or ≥2mm in two contiguous chest leads. Patients were excluded if they had previous coronary artery bypass surgery or acute stent thrombosis. The patients identified had coronary angiography at the hospitals between March 1998 and June 2010.

3.2. Calculation of QCAM

On the angiography performed prior to subsequent representation with acute myocardial infarction (2 above), all three of the main coronary arteries were examined in two angiographic views using ezDICOM, a digital imaging and communications in medicine software program [9]. Single frames were selected that best represented the coronary arteries at end-diastole and end-systole. End-diastole was defined as the largest cardiac silhouette within the cardiac cycle close to the peak of the R-wave in the ECG (when available), and end-systole was defined as the smallest silhouette close to the end of the T-wave. Each artery was divided into sections based on unique identifying points and corresponding points were identified in both angiographic views.

The length of each section was measured from the single frames selected at end-diastole and end-systole using the software program Image J [10] as demonstrated in Figure 1. The quantitative measure of coronary artery motion (QCAM) was defined as the ratio of the section lengths i.e. systolic length/diastolic length.

Figure 1, Part A. Right Coronary Artery in Diastole from View 1 (Right anterior oblique 3.6°, Cranial 18.9°)

Figure 1, Part B. Right Coronary Artery in Systole from View 1

Figure 1, Part C. Right Coronary Artery in Diastole from View 2 (Right anterior oblique 28.9°, Caudal 1.2°)

Figure 1, Part D. Right Coronary Artery in Systole from View 2

These analyses were performed blinded to the location of the culprit lesion responsible for the subsequent presentation.
with ST segment elevation myocardial infarction (1 above). An experienced interventional cardiologist subsequently identified the location of the culprit plaque and vessel.

### 3.3. Statistical Analysis

A two sample t-test was performed to compare QCAM for the non-culprit and culprit sections within the coronary arteries that contained a culprit lesion. A generalized linear mixed model was then used with culprit section as the dependent variable and QCAM as the independent variable. Grouping was by angiographic view and by patient.

### 3.4. Ethics Approval of the Study Protocol

This study complied with the Declaration of Helsinki. The human research ethics committee of Royal Prince Alfred Hospital approved the study.

### 4. Results

Twenty-eight patients were identified. Two hundred and eighty seven sections in the coronary arteries containing the culprit sections were identified and analyzed as shown in Table 1. The mean QCAM (ratio of end systolic length: end diastolic length) of the non-culprit sections was 103.51 compared to 93.43 for the culprit sections.

<table>
<thead>
<tr>
<th>Section Type</th>
<th>Frequency</th>
<th>Mean</th>
<th>Standard Error of the Mean</th>
<th>Standard Deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-culprit</td>
<td>233</td>
<td>103.51</td>
<td>2.14</td>
<td>32.70</td>
</tr>
<tr>
<td>Culprit</td>
<td>54</td>
<td>93.43</td>
<td>1.83</td>
<td>13.41</td>
</tr>
</tbody>
</table>

Table 1. Summary statistics for QCAM by non-culprit and culprit section types

Figure 2. QCAM for each section for each of the two views for each patient. Non-culprit sections are blue (circles) and culprit sections are red (squares).

The two samples t-test comparing QCAM for the non-culprit and culprit sections was highly statistically significant with a p-value of 0.0004. The generalized linear mixed model with culprit section as the dependent variable and QCAM as the independent variable also showed a statistically significant result with a p-value of 0.026.

### 5. Discussion

These results demonstrate a significant association between QCAM and the location of a subsequent culprit lesion. Hypotheses regarding the mechanism of this link include the compression type of CAM being a mechanical stress on vascular tissue causing injury to the endothelium. This could not only allow atherogenesis but repetitive stress during the cardiac cycle could weaken plaques already formed and increase the risk of rupture [5].

Despite the results showing a link with differing mean values of QCAM for culprit and non-culprit sections, Figure 2 demonstrates that the QCAM of a number of non-culprit sections lie within the distribution seen for culprit sections. This may suggest that these non-culprit sections are under the same biomechanical stress due to coronary artery motion as the culprit sections and could be potential culprit sections in the future. One alternative explanation is the measurement error introduced by the foreshortening artifact and out of plane magnification error inherent in measuring a moving three-dimensional artery from a two dimensional image. Three-dimensional reconstructions of coronary arteries may overcome this limitation.

There is no current direct clinical application for this research. Further development of this line of research does, however, lead to a number of conceivable future clinical applications. Better understanding of the biomechanical effects of current generation stent technologies may lead to stent strategies that result in lower rates of major adverse cardiac events. Previous reports have shown that longitudinal straightening effect of stents to be a statistically significant predictor of major adverse cardiac events [11]. Modeling of a double stent versus a single stent strategy for a curved artery has suggested higher flexibility, more conformity and lower recoil [12]. Whether the effect of different stent strategies on QCAM in the vessel sections around the stented segments will result in different clinical outcomes is an untested hypothesis.

Bioresorbable vascular scaffolds, which have been termed the ‘fourth revolution’ in interventional cardiology [13], have been shown to result in less coronary artery straightening when compared to metallic platform stents in a retrospective analysis of 102 patients [14]. The progressive disappearance of the polymeric scaffold will likely result in a different effect on QCAM compared to that of current generation stent technologies. If this generation of coronary devices is to be used for current stent indications and also for potential new indications such as the treatment of vulnerable plaques, as has already been reported in the literature [15], then their effect of treatment on coronary biomechanics, including QCAM, may have clinical significance.
6. Conclusions

This study identifies that QCAM is a predictor of the location of culprit lesions responsible for subsequent ST segment elevation myocardial infarctions. Predicting the location of future culprit lesions using coronary angiography may allow targeted therapy to prevent future myocardial infarctions.

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Competing Interests

The authors declare they have no competing interests.

REFERENCES


