Can strain rate imaging predict recovery of contraction after acute myocardial infarction?

Minna M. Kylmälä 1,2*, Margareta Antila 3, Sari M. Kivistö 3, Kirsi Lauerma 3, Lauri Toivonen 1, and Mika K. Laine 1

1Division of Cardiology, Helsinki University Central Hospital, Haartmaninkatu 4, P.O.Box 340, 00029 HUS, Helsinki, Finland; 2BioMag Laboratory, Hospital District of Helsinki and Uusimaa HUSLAB, Helsinki University Central Hospital, Helsinki, Finland; and 3Department of Radiology, Helsinki University Central Hospital, Haartmaninkatu 4, P.O. Box 340, 00029 HUS, Helsinki, Finland

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Aims

To assess whether strain rate imaging (SRI) can serve to evaluate myocardial viability in patients with acute coronary syndrome (ACS).

Methods and results

In 23 patients with ACS, we measured longitudinal tissue Doppler strain and strain rate values from left ventricular basal, mid, and apical segments (n = 414). These segments were grouped according to their acute end-systolic strain values (S_Es) into those with normocontraction (S_Es ≤ −13%), hypocontraction (S_Es between −13 and −7%), and severe contraction abnormality (S_Es > −7%). At 8 months, we evaluated the recovery of contraction: Segments with acutely severe contraction abnormality that improved their strain values to ≤ −7% were defined as viable, and those that failed to do so as non-viable. In the acute phase, S_Es, post-systolic strain, as well as systolic, early, and late diastolic strain rate values were significantly better in the viable than in the non-viable segments. Post-systolic strain had the best AUC 0.78, and a cut-off value of ≤ 3.8% predicted recovery from severe contraction abnormality with a sensitivity of 85% and specificity of 62%. The transmurality of the infarction, assessed by magnetic resonance imaging with delayed enhancement, was significantly larger in the non-viable than in the viable segments (P = 0.006). Acute global S_Es and systolic strain rate showed the best correlations with final global S_Es and global infarction percentage after recovery.

Conclusion

SRI can serve to evaluate myocardial viability in patients with ACS, and to assess the recovery of segmental as well as global left ventricular function.

Keywords

Acute coronary syndrome • Myocardial infarction • Strain rate imaging • Tissue Doppler • Viability

Introduction

In acute myocardial infarction (MI), it is clinically important to know whether myocardium with severely compromised function is permanently injured or reversibly dysfunctional because only viable myocardium can be salvaged by reperfusion therapy. Depending on the mechanisms that contribute to the ischaemia-induced myocardial dysfunction (stunning, hibernation, or subendocardial infarction), the time to recovery of myocardial function after reperfusion may vary from a few hours to several months. When viability assessment is necessary acutely, established methods for its evaluation [single photon emission computed tomography, cardiac magnetic resonance imaging with delayed enhancement (DE-CMR), and dobutamine stress echocardiography] are usually unsuitable or unavailable. However, in these settings, the echocardiographic tissue Doppler strain rate imaging (SRI) technique might prove a useful tool.

SRI has been validated for the assessment of segmental myocardial function.1,2 Systolic strain and especially strain rate correlate with contractility.3 Compared with visual wall-motion analysis, SRI is an objective and a more quantitative method for the assessment of myocardial contraction. Furthermore, SRI is more accurate in defining the presence and the transmurality of MI.4–7 In experimental and clinical studies, strain and strain rate parameters correlate with segmental infarct transmurality and with global extent of infarction in MI that is chronic8,9 as well as acute.5,6,10,11

* Corresponding author. Tel: +358 500 430632; fax: +358 9 5042412, Email: minna.kylmalan@hus.fi
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Clinical data concerning the use of SRI for the prediction of recovery of myocardial contraction after acute MI are scarce. Our aim was to investigate, in patients with acute coronary syndrome (ACS), the ability of SRI to distinguish segments with reversible dysfunction from those irreversibly damaged. The SRI was performed in patients with acute MI and was repeated after complete healing of the infarction scar to determine recovery of segmental myocardial contraction. DE-CMR was used as a validation method to evaluate the final transluminal infarction scar.

Methods

Patients

A group of 32 patients admitted for ACS to the Helsinki University Central Hospital were prospectively recruited to this study, which includes only the 23 who also underwent follow-up measurements. Inclusion criteria were prolonged chest pain within 48 h of recruitment with associated ischaemic ECG changes or elevated cardiac enzymes (CK-MBm >7 μg/L and/or TnT >0.03 μg/L). Exclusion criteria were atrial fibrillation, pacemaker rhythm, bundle branch block, and need for ventilatory support.

Among these 23 patients, 16 had ST-elevation MI, 4 had non-ST-elevation MI, and 3 had unstable angina pectoris (CK-MBm <7 μg/L and/or TnT <0.03 μg/L). All patients received complete reperfusion treatment verified by coronary angiography, and the majority within 12 h of the onset of chest pain. Baseline characteristics are described in Table 1.

Baseline echocardiography with colour tissue Doppler imaging took place within 24 h after the onset of chest pain in the majority of patients (mean delay 16.6 ± 14.4 h). Seven patients were imaged before revascularization; the others were imaged 10.6 ± 8.2 (range: 3–30) hours after reperfusion. The follow-up echocardiography, together with DE-CMR, was performed 8 months (range: 7–12) after the index event. None of the patients suffered from new ischaemic events before the follow-up measurement. The patients gave their written informed consent, and the study was approved by the ethics committee of the Helsinki University Central Hospital and complies with the Declaration of Helsinki.

Echocardiography

Echocardiographical data were acquired by second-generation tissue harmonic imaging and tissue velocity imaging with a Vivid 7 ultrasound scanner (GE Medical Systems) and a 1.5–4.0 MHz phased array transducer (M3S). Images were analysed off-line on a personal computer using EchoPAC software provided by GE. Two-dimensional images of the heart were obtained in all standard apical views (2-chamber, 3-chamber, and 4-chamber views). For analysis of regional myocardial function with tissue Doppler SRI, the left ventricle was divided into 18 segments according to the model defined by the American Heart Association, modified by the addition of apical anteroseptal and inferolateral segments. For comparisons with DE-CMR results, the original 16-segment model was used.12

Colour tissue Doppler loops were recorded from the apical views by limiting the imaging sector to one myocardial wall at a time (sector angle: 30°). The tissue velocity scale was adjusted to avoid aliasing (pulse repetition frequency: 1.00–1.5 kHz). The frame rate was 52 ± 4/209 ± 17 frames per second. Patients were asked to hold their breath if needed in order to position the myocardial wall at the centre of the imaging sector during three consecutive heartbeats. Segments were excluded from the analysis if the angle between the ultrasound beam and the myocardial wall exceeded 30°. Segments with artefacts or bad quality imaging data were also excluded. Strain and strain rate curves were obtained by placing the sample volume in the middle of each segment. Manual adjustments were made in order to assure that the sample volume remained within the myocardium. We used a default strain length of 12 mm. The profiles of three consecutive heartbeats were averaged for analysis. End-systole was defined by aortic valve closure.

Strain and strain rate measurements

Strain describes local myocardial deformation. Longitudinal strain is a measure of percentage shortening or percentage lengthening of the myocardium. Strain rate is a measure of the speed of myocardial deformation, with the unit s⁻¹. Longitudinal strain and strain rate values are negative when the myocardium contracts and become positive when it lengthens. The principles and the technique of SRI are extensively described in a review article by d’Hooge et al.13 The strain values are reported here as Lagrangian strain.

In the acute phase, segmental end-systolic strain (Ses) and post-systolic strain (Sps) values were obtained from the strain curves. Sps was defined as the maximal negative strain value occurring after aortic valve closure. Peak strain rate values were obtained from the strain rate curves in systole (SRe), early diastole (SRA), and late diastole (SRA). Final Ses was measured after recovery. Figure 1 demonstrates representative segmental strain values in the acute phase and after MI healing.

Global Ses, SRe, SRA, and SRI values were calculated by averaging each patient’s segmental values. The American Heart Association 16-segment model served for measuring global values.12

Defining contraction capacity and viability by strain

The segments were grouped in the acute phase based on their Ses. We regarded segments with Ses ≤–13% as normocontractile, between −7 and −13% as hypocontractile, and >−7% as having a severe contraction abnormality. We chose these cut-off values based on the

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Patient baseline characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients, n</td>
<td>23 (18 male)</td>
</tr>
<tr>
<td>Age, years</td>
<td>60 ± 10 (45–78)</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>27.6 ± 5.1</td>
</tr>
<tr>
<td>Previous myocardial infarction, n</td>
<td>3</td>
</tr>
<tr>
<td>Hypertension, n</td>
<td>10</td>
</tr>
<tr>
<td>Dyslipidaemia, n</td>
<td>5</td>
</tr>
<tr>
<td>Smokers, n</td>
<td>18</td>
</tr>
<tr>
<td>CK-MBm, μg/L</td>
<td>148 ± 145 (5–612)</td>
</tr>
<tr>
<td>EF, % in the acute phase</td>
<td>58 ± 9 (44–78)</td>
</tr>
<tr>
<td>EF, % after 8 ± 2 months</td>
<td>62 ± 9 (47–82)</td>
</tr>
<tr>
<td>Culprit artery</td>
<td></td>
</tr>
<tr>
<td>LAD, n</td>
<td>12</td>
</tr>
<tr>
<td>LCX, n</td>
<td>4</td>
</tr>
<tr>
<td>RCA, n</td>
<td>7</td>
</tr>
</tbody>
</table>

Data are presented as number of patients or mean ± SD (range). EF, ejection fraction measured by Simpson’s rule; LAD, left anterior descending coronary artery; LCX, left circumflex coronary artery; RCA, right coronary artery.
results of our previous study that defined strain values in segments with different degrees of infarct transmurality by DE-CMR in chronic MI. In the current study, the median acute $S_{ES}$ was $-18\%$ in segments with normocontraction, $-11\%$ in segments with hypocontraction, and $-1\%$ in segments with a severe contraction abnormality. We further divided the segments with an acutely severe contraction abnormality ($S_{ES} > -7\%$) into viable vs. non-viable, according to their final $S_{ES}$—values that revealed whether the segments had recovered (final $S_{ES} \leq -7\%$) or not (final $S_{ES} > -7\%$), thus yielding a total of four groups of segments (Table 2).

### Feasibility

In the acute phase, strain analysis was possible in 78% (321 of 414) of the segments. For the data analyses, we used the values from those 305 segments that allowed strain analysis also in the follow-up measurement. Analysis of the other acute phase parameters was possible as follows: $SR_S$ in 304, $SR_E$ in 304, and $SR_A$ in 287 segments. $S_{ES}$, not usually seen in healthy myocardium, was present in 143 segments.

### Cardiac magnetic resonance imaging

DE-CMR performed in a 1.5 T scanner (Sonata, Siemens Medical Solutions) served to determine the size and location of the MI at 7–12 months (mean 8) after the acute event in order to validate the SRI results. DE-CMR images were obtained as described previously. For DE-CMR analysis, we used the 16-segment model defined by the American Heart Association. The transmural extent of the infarction scar in each segment was assessed as the percentage of DE of the wall thickness. Global infarction percentage was calculated by averaging the scar extent in the same segments used for assessing global tissue Doppler strain and strain rate values.

Myocardial wall-motion was analysed from cine images independently by two experienced radiologists blinded to the DE analysis. The cine images served only to validate the DE analysis: 18 segments of 368 were excluded because of a discrepancy between the DE- and wall-motion analyses.

### Statistical analysis

Continuous parameters are expressed as mean ± SD or median and inter-quartile range (inter-quartile range equals the range of values falling between the 25th and the 75th percentiles). Comparisons between groups were done by the Mann–Whitney U-test, and correlation analyses by two-tailed Pearson correlation. A $P$-value of $<0.05$ was considered statistically significant. Sensitivity and specificity for prediction of recovery of those segments with a severe contraction abnormality was calculated by receiver operating characteristic curve analysis. The value yielding the largest sum of sensitivity and specificity became the cut-off value. Values better than the cut-off were defined as positive. The positive predictive value was calculated as the number of true positives (=recovered segments) divided by the sum of true and false positives. The negative predictive value was the number of true negatives (=segments not recovered) divided by the sum of true and false negatives. In all calculations, the values unavailable for analysis were treated as missing values. Intraclass repeatability was assessed from 100 randomly chosen segments by the intra-class correlation coefficient (ANOVA, two-way mixed model, single measures) for all strain and strain rate parameters, and by the coefficient of agreement ‘kappa’ for the strain-groups defining contraction. A ‘kappa’-value $<0.19$ means poor agreement, 0.20–0.39 fair agreement, 0.40–0.59 moderate agreement, 0.60–0.79 substantial agreement, and $>0.8$ almost perfect agreement. All statistical calculations were done by SPSS 14.0 statistical software.
**Results**

**Assessment of myocardial viability by strain rate imaging**

Normo- and hypocontractile segments are viable by definition, but segments with a severe contraction abnormality can be either viable or non-viable. Of the segments with a severe contraction abnormality, 60% (34 of 57) recovered (17 to hypocontraction and 17 to normocontraction). Segments with a severe contraction abnormality that recovered (=viable segments) had a significantly better strain and strain rate acutely than did those not recovering (=non-viable segments) (Figure 2). Of the hypocontractile segments, 72% (49 of 68) recovered to normocontraction. Significant correlation appeared between the acute $S_{ES}$, $S_{PSS}$, $S_{RS}$, $S_{RE}$, $S_{RA}$, and the final $S_{ES}$ after recovery. The best correlations are shown in Figure 3.

As expected, the transmurality of infarction at 8 months by DE-CMR was significantly smaller in the viable than in the non-viable segments: The median infarction percentage was 19% in the viable vs. 60% in the non-viable group ($P = 0.006$), thus validating the SRI results. The correlation between the final segmental infarction transmurality and acute strains and strain rates was significant. The best correlations were for $S_{ES}$ ($r = 0.439$, $P < 0.001$), $S_{PSS}$ ($r = 0.362$, $P < 0.001$), and $S_{RSYS}$ ($r = 0.318$, $P < 0.001$).

**Predicting recovery of segments with a severe contraction abnormality**

All acute strain and strain rate parameters could predict recovery of segments with a severe contraction abnormality (Table 3, Figure 4). The most accurate parameter was $S_{PSS}$, which had the largest area under the curve (0.783, $P = 0.001$). With a cut-off value of $-3.8\%$, $S_{PSS}$ predicted recovery with a sensitivity of 85%, specificity of 62%, positive predictive value of 74%, and negative predictive value of 76%. $S_{ES}$ and $S_{RS}$ showed a higher specificity but lower sensitivity, and $S_{RE}$ and $S_{RA}$ showed a higher sensitivity but lower specificity than did $S_{PSS}$ for their respective cut-off values. All strain and strain rate parameters had good positive predictive values $>70\%$, but the negative predictive value was $<70\%$ for all parameters other than $S_{PSS}$.

**Predicting recovery of global left ventricular function**

In the acute phase, we measured global values for all strain and strain rate parameters except $S_{PSS}$. The acute global $S_{ES}$, $S_{RS}$, and $S_{RE}$ correlated with the final global $S_{ES}$. The global systolic parameters were shown to be the best at predicting global recovery (Table 4).

Comparison of global left ventricular infarction percentage defined by DE-CMR with the acute global strain and strain rate

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**Figure 2** Acute strain and strain rate values in non-viable vs. viable segments with a severe contraction abnormality. End-systolic strain (A), post-systolic strain (B), systolic strain rate (C), and early diastolic strain rate (D) in segments with a severe contraction abnormality (strain $< -7\%$) in ACS. Segments are grouped according to recovery 7–12 months after the ACS showing non-viable segments that do not recover, and viable segments that do recover. The strains and strain rates are significantly better in the viable segments. Each box shows the median and inter-quartile range, and the whiskers show the extreme values.
values showed that only acute global systolic strain and strain rate significantly correlated with the global infarction percentage (Table 4).

**Repeatability**

Intraobserver variability of all acute strain and strain rate parameters was assessed by the intra-class correlation coefficients: $S_{ES} 0.87$, $S_{PSS} 0.93$, $SR_e 0.77$, $SR_{re} 0.67$, and $SR_{ra} 0.53$. The coefficient of agreement ‘kappa’ was 0.65 (meaning substantial agreement) for grouping segments according to their acute $S_{ES}$ into segments with normocontraction, hypocontraction, or severe contraction abnormality.

**Discussion**

We showed that in ACS, strain and strain rate parameters correlate with myocardial contraction after healing, and can differentiate segments with reversible dysfunction from segments permanently injured. We also found that SRI can predict recovery of global systolic function.

**Evaluation of myocardial viability in ACS by strain rate imaging**

In patients with ACS, it is useful to determine whether the myocardium supplied by the infarct-related artery is viable, as only viable
myocardium benefits from revascularization, and unnecessary revascularization may do more harm than good.

The ideal measure of viability is an objective estimation of myocardial capacity to contract. Echocardiographic SRI offers a sensitive and objective means of assessing myocardial contraction. It is a clinically interesting method because of the potential of performing it bedside, at a short notice at any hour.

We have shown earlier that in chronic MI, the mean $S_{ES}$ is $-14$, $-12$, $-7$, and $-4\%$ in segments with an infarction transmurality of $0\%$–$25\%$, $26\%$–$50\%$, $51\%$–$75\%$, and $75\%$, respectively. We can therefore assume that myocardial segments with $S_{ES} > -7\%$ have severely impaired contraction and are non-viable; segments with $S_{ES}$ between $-7\%$ and $-13\%$ have slightly/moderately reduced contraction; and most segments with $S_{ES} \leq -13\%$ are capable of normal contraction.

In the present study, we found that in ACS, $S_{ES}$, $S_{PSS}$, $S_{RS}$, $S_{RE}$, and $S_{RA}$ are significantly better in viable than in non-viable segments and correlate with infarct transmurality. These findings are supported by the results of an experimental study showing that in pigs, radial $S_{PSS}$, $S_{RS}$, $S_{RE}$ and $S_{RA}$ differentiate stunned from transmurally infarcted segments. Weidemann et al. found in a clinical study that longitudinal $S_{ES}$ and $S_{RS}$ show excellent accuracy in detecting segments with transmural infarction ($>75\%$ by DE-CMR) early after acute MI. Our main finding was that both systolic and diastolic strain and strain rate parameters can assess explicitly the recovery of segmental myocardial contraction in patients with acute MI. This issue has

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**Table 4** Correlation of acute phase global strain and strain rate values with final global end-systolic strain and left ventricular infarction percentage determined by DE-CMR

<table>
<thead>
<tr>
<th>SRI parameter</th>
<th>Final global $S_{ES}$ by SRI</th>
<th>Infarction percentage by DE-CMR</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$r$</td>
<td>$P$-value</td>
</tr>
<tr>
<td>Acute global $S_{ES}$</td>
<td>0.760</td>
<td>$&lt;0.001$</td>
</tr>
<tr>
<td>Acute global $S_{RS}$</td>
<td>0.732</td>
<td>$&lt;0.001$</td>
</tr>
<tr>
<td>Acute global $S_{RE}$</td>
<td>$-0.523$</td>
<td>0.01</td>
</tr>
<tr>
<td>Acute global $S_{RA}$</td>
<td>$-0.238$</td>
<td>NS</td>
</tr>
</tbody>
</table>

DE-CMR, cardiac magnetic resonance imaging with delayed enhancement; SRI, strain rate imaging; $S_{ES}$, end-systolic strain; $S_{PSS}$, post-systolic strain; $S_{RS}$, systolic strain rate; $S_{RE}$, early diastolic strain rate; $S_{RA}$, late systolic strain rate.
been addressed also in a study by Park et al.,18 in which only SR_e was significantly better in segments with functional recovery. Unlike in our study, visual wall-motion analysis was their reference method for the assessment of segmental contraction, which may explain—in part—why SRI in our study was more sensitive in detecting viability.

Interestingly, SPSS was the most accurate parameter in predicting myocardial recovery in patients with ACS. SPSS could correctly identify the majority of the viable segments, but with lower specificity. Importantly, we defined SPSS as the maximal negative strain occurring after aortic valve closure. Several studies suggest that post-systolic shortening (PSS) in acute MI may act as a marker of viability. In a swine model of acute MI, PSS determined from M-mode recordings shows a negative correlation with infarct transmurality and indicates viability. PSS did not appear in segments with transmural (>70%) infarcts.17 In their experimental study, Skulstad et al.,19 demonstrated that during acute ischaemia, PSS (measured by sonomicrometry and tissue Doppler strain) develops by active contraction in hypo- and akinetic segments, whereas in dyskinetic segments, PSS may be the result of passive recoil, with or without a component of active contraction. Hosokawa et al.20 found previously that in patients with acute MI, PSS (measured with the centreline method from contrast ventriculograms before reperfusion) predicts the recovery of even dyskinetic segments. The same finding was made in an experimental study by Takayama et al.,21 who also showed that myocardial recovery correlated better with PSS, as determined by sonomicrometry, than with myocardial blood flow or the histological extent of necrosis.

**Strain rate imaging in assessment of global left ventricular function**

Our results suggest that global SSS, SR_e, and SR_c can predict recovery of global left ventricular function. To our knowledge, this finding has not been reported previously with the SRI method. Interestingly, Vartdal et al.11 showed that global systolic strain is actually superior to ejection fraction in predicting the size of the infarction scar in patients with acute MI. Furthermore, global systolic strain may prove better than ejection fraction or wall-motion score index for patient-risk stratification.

**Study limitations**

Most of the patients were imaged after re-establishment of coronary flow, but not all; we recognize that this inhomogeneity might confound our results. However, this reflects the real-life situation in cardiac care units. The cut-off SSS values we used for grading myocardial contraction in the acute phase were mainly based on SSS values associated with different degrees of infarction transmurality in chronic infarction.7 In addition, we considered studies that have defined SSS values in healthy subjects, during acute ischaemia, and in acute MI.2,11,22 On the basis of those studies, our cut-off values should classify most segments correctly into groups showing normocontraction, hypococontraction, or severe contraction abnormality—a fact also supported by the DE-CMR results here. Even so, some overlapping probably exists between groups in SSS values.

**Conclusions**

Strain rate imaging can serve to assess myocardial viability in patients with ACS and to predict functional recovery of myocardial segments as well as global left ventricular function in response to revascularization.

**Conflict of interest:** none declared.

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**References**

A giant right coronary artery aneurysm as an incidental finding

Enrique Ríos Gómez1, María Martín1*, Inigo Lozano1, and Luis H. Luyando2

1Cardiology Department, Hospital Universitario Central de Asturias, Oviedo, Asturias, Spain and 2Radiology Department, Hospital Universitario Central de Asturias, Oviedo, Asturias, Spain

* Corresponding author: Área del Corazón, Hospital Universitario Central de Asturias, Calle Julián Clavería s/n 33006, Oviedo, España, Spain. Tel: +34 985 108 000-36173, Email: mmartinf7@hotmail.com

Image focus

We present the case of a 63-year-old asymptomatic man, with a diastolic murmur who was submitted for echocardiographic assessment. He was hypertensive and smoker. Two-dimensional transthoracic echocardiogram revealed a moderate aortic insufficiency and an abnormal extra-cardiac shadow on the right cardiac border (Figures A and B, arrows). There were no other abnormalities. In order to complete the diagnosis, he was referred for non-invasive coronariography, which was performed with a 64-multidetector computed tomography. It revealed a giant atherosclerotic right coronary artery (RCA) aneurysm with a maximal diameter of 45 mm, which was compressing the right chambers (Figures D and E), distal RCA was also aneurysmatic. A giant intramural thrombus was present surrounding the coronary lumen. In order to complete diagnosis and thinking in a possible surgical treatment, invasive coronariography was practised confirming the finding (Figures C and F). Afterwards, the patient refused any invasive treatment.

Aneurysmal coronary artery disease is an uncommon disease (1.5–5%) defined as a coronary dilatation that exceeds the diameter of normal adjacent segments or the diameter of the patient’s largest coronary vessel by 1.5 times. The RCA seems to be the most commonly involved.

The most frequent aetiology is atherosclerosis followed by Kawasaki disease. Other less common causes include connective tissue diseases, Takayasu arteritis, trauma, or dissection.

In asymptomatic patients, diagnosis is usually incidental; cardiac computed tomography, cardiac-magnetic resonance imaging and cardiac catheterization are useful to make a definitive diagnosis. Ischaemia detection tests should also be performed to determine the significance of the aneurysm or stenoses and the best therapeutic options.

Conflict of interest: none declared.

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