Prevalence and pathophysiological mechanisms of elevated cardiac troponin I levels in a population-based sample of elderly subjects

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Aims
To evaluate the prevalence of cardiac troponin I (cTnI) elevation in an elderly community population and the association of cTnI levels with cardiovascular risk factors, vascular inflammation, atherosclerosis, cardiac performance, and areas indicative of infarcted myocardium identified by cardiac magnetic resonance imaging.

Methods and results
cTnI elevation defined as cTnI levels >0.01 µg/L (Access AccuTnI, Beckman Coulter) was found in 21.8% of the study participants (n = 1005). cTnI >0.01 µg/L was associated with cardiovascular high-risk features, the burden of atherosclerosis in the carotid arteries, left-ventricular mass, and impaired left-ventricular systolic function. No associations were found between cTnI and inflammatory activity, diastolic dysfunction, or myocardial scars. Male gender (OR 1.6; 95% CI 1.1–2.4), ischaemic ECG changes (OR 1.7; 95% CI 1.1–2.7), and NT-pro-brain natriuretic peptide levels (OR 1.4; 95% CI 1.1–1.7) independently predicted cTnI >0.01 µg/L. cTnI >0.01 µg/L correlated also to an increased cardiovascular risk according to the Framingham risk score.

Conclusion
cTnI >0.01 µg/L is relatively common in elderly subjects and is associated with cardiovascular high-risk features and impaired cardiac performance. Cardiac troponin determined by a highly sensitive assay might thus serve as an instrument for the identification of subjects at high cardiovascular risk in general populations.

Keywords
Cardiac troponin • Cardiovascular disease • Risk prediction

Introduction
In patients with acute coronary syndrome (ACS), any cardiac troponin elevation is indicative of myocardial damage. However, with the implementation of assays with improved sensitivity, minor troponin elevations have become reliably detectable in patients with non-ischaemic conditions and apparently healthy subjects. In the absence of unstable coronary artery disease, pathophysiological mechanisms as increased demand ischaemia, myocardial strain because of volume and pressure overload, or disturbance of cardiomyocyte cell membrane integrity due to systemic inflammatory response or apoptosis have been discussed as possible causes for troponin leakage. However, as the rupture of coronary plaques with overlying thrombus formation and micro-embolization might go clinically unrecognized and without any signs or symptoms of an ACS, subclinical myocardial micro-infarctions might be another explanation for troponin elevation in general populations.

We undertook this analysis in order to further evaluate the prevalence of troponin elevation in a population of 70-year-old subjects included in the PIVUS (Prospective Investigation of the Vasculature in Uppsala Seniors) study and to investigate its association with atherosclerosis, findings on echocardiography, and areas indicative of infarcted myocardium identified by cardiac magnetic resonance imaging (MRI) using late enhancement. Furthermore, we studied the association of cardiac troponin I (cTnI) elevation with cardiovascular risk factors including the Framingham risk score, a widely used scoring system to quantify the risk for
coronary heart disease,\textsuperscript{5} and biomarkers of cardiac performance [NT-pro-brain natriuretic peptide (NT-pro-BNP)], inflammation [C-reactive protein, interleukin-6 (IL-6)], and renal function (creatinine clearance, cystatin C).

**Methods**

**Study design**
In the PIVUS study, all subjects aged 70 years and living in the community of Uppsala, Sweden were eligible for participation.\textsuperscript{6} The subjects were chosen in a randomized way from the register of community inhabitants. Of the 2025 subjects invited, 1016 participated in the study between April 2001 and June 2005. cTnI results were available in 1005 study participants who formed the sample population for the present study. Written informed consent was obtained from all participants and the study protocol was approved by the local Ethics Committee and complies with the Declaration of Helsinki.

**Basic investigations**
All subjects underwent a physical examination, and their medical history, smoking habits, and regular medication were recorded. Hypertension was defined as antihypertensive treatment or blood pressure >140/90 mmHg at rest, diabetes as diabetic treatment including diet or fasting glucose >6.1 mmol/L, and hyperlipidaemia as hyperlipidaemic treatment, LDL-cholesterol >3.5 mmol/L, or serum triglycerides >1.7 mmol/L. Previous cardiovascular disease was defined as coronary heart disease (self-reported), heart failure (self-reported), previous stroke (self-reported), or hypertension.

**Laboratory analysis**
cTnI was measured in frozen EDTA plasma samples using the current version of the AccuTnI assay (Beckman Coulter Inc., Fullerton, CA, USA). As a result of recent modifications, the sensitivity of this assay has been improved considerably with its previous version with 0.006 \(\mu\)g/L as its lower limit of detection and 0.014 \(\mu\)g/L as the lowest concentration measurable with a coefficient of variation (CV) of <10\%.\textsuperscript{7} According to a previous analysis, the 99th percentile among PIVUS participants is 0.044 \(\mu\)g/L,\textsuperscript{8} which is in the same range as for the prior version of the assay.\textsuperscript{10} cTnI levels >0.01 \(\mu\)g/L have shown to be prognostically useful in stabilized patients after an episode of ACS\textsuperscript{7} as to why this cut-off was chosen to define cTnI elevation.

NT-pro-BNP was measured using the Elecsys pro-BNP sandwich immunoassay on an Elecsys 1010 instrument (Roche Diagnostics, Mannheim, Germany). IL-6 was determined using an ELISA (Quantikine, R&D Systems, Minneapolis, MN, USA). Plasma creatinine, C-reactive protein, and cystatin C were analysed on an Architect ci8200 analyser (Abbott Laboratories, Abbott Park, IL, USA). Plasma creatinine measurements were performed by the means of the modified kinetic Jaffe reaction. Creatinine clearance was calculated according to the Cockcroft–Gault formula.\textsuperscript{11}

**Electrocardiography**
A conventional 12-lead ECG was recorded and analysed regarding the presence of ST-segment depression (Minnesota codes 4-1 or 4-2), T-wave inversion (Minnesota codes 5-1, 5-2, or 5-3), pathological Q-waves (Minnesota code 1-1), or left bundle branch block (Minnesota code 7-1).\textsuperscript{12} ECG changes indicative of ischaemic heart disease were considered when any of the aforementioned abnormalities was present.

**Carotid artery ultrasound examination**
The intima-media thickness (IMT) and the prevalence of atherosclerotic plaques in the common carotid artery, the bulb, and the internal carotid artery were assessed using an Acuson XP124 cardiac ultrasound unit (Acuson, Los Angeles, CA, USA) with a 10 MHz linear transducer. As described elsewhere, IMT and atherosclerotic plaques were quantified by an automated software program.\textsuperscript{13} A small plaque was considered to be present if the IMT was locally thickened >50\% compared with the surrounding IMT. A moderate plaque was present if the plaque area was >10 mm\(^2\). A flow-limiting plaque was defined by a decreased flow velocity distal of the plaque.

**Echocardiography and Doppler**
A two-dimensional Doppler echocardiography was performed in 944 study participants, with an Acuson XP124 cardiac ultrasound unit using a 2.5 MHz transducer for the majority of examinations. Cardiac dimensions were measured online with M-mode from parasternal positions, applying the leading-edge-to-leading-edge convention. Left ventricular (LV) volumes were calculated according to the Teichholz formula, and from that, ejection fraction (LV-EF) was determined. LV mass index was calculated according to the recommendations of the American Society of Echocardiography.\textsuperscript{14} LV hypertrophy was considered present if LV mass index was >116 g/m\(^2\) in men and >104 g/m\(^2\) in women.\textsuperscript{15}

The LV diastolic function was examined from the apical position by analysing the mitral inflow with the sample volume between the tips of the mitral leaflets during diastole. The peak velocities of the early rapid filling wave (E-wave) and the atrial filling wave (A-wave) were recorded and the E/A ratio was calculated. LV isovolumic relaxation time was measured as the time between aortic valve closure and start of mitral flow, using the Doppler signal from the area between the LV outflow tract and mitral flow.

**Cardiac magnetic resonance imaging**
Gadolinium contrast-enhanced cardiac MRI was performed in 259 subjects with a mean delay of 16 months (range 3–22 months) from the primary investigation. As described previously, a 1.5 T MRI system (Gyroscan Intera, Philips Medical Systems, Best, The Netherlands) was used.\textsuperscript{16} MRI images were analysed in a consensus reading by two observers blinded to further data on the medical history of the participating subjects. A previous myocardial infarction was validated in this subcohort by evaluation of the hospital records of the participants.\textsuperscript{16} Contrast enhancement had to involve the subendocardial layer as a criterion for the identification of a scar indicative of a myocardial infarction.\textsuperscript{17}

**Statistical analysis**
cTnI was entered into all analyses as dichotomized variable, using 0.01 \(\mu\)g/L as threshold, considering the analytical performance of the AccuTnI assay and previous findings from our study group.\textsuperscript{7} Continuous variables are described as medians (with 25th, 75th percentiles) or means (with standard deviation), as appropriate. Differences between continuous variables were evaluated using the Mann–Whitney \(U\) test. The relationship between continuous variables was investigated by calculating the Spearman rank correlation coefficients. Categoric variables are expressed as frequencies and percentages. Differences between categoric variables were analysed with the Pearson \(\chi^2\) test. Independent predictors for cTnI levels >0.01 \(\mu\)g/L were identified by multivariable logistic regression analysis adjusted for all baseline characteristics that were significant predictors in the univariate analysis including ischaemic ECG changes (model 1), with additional adjustment.
for biochemical markers significantly correlated to cTnI > 0.01 µg/L on univariate analysis (model 2) and LV-EF and LV mass index (model 3). Owing to highly skewed levels, NT-pro-BNP was log-transformed before being entered into the multivariable analysis. The association between cTnI and 10 year risk for cardiovascular events was evaluated by testing its relation to the Framingham risk score using categories with estimated risks of <10, 10–19.9, and ≥20%. For all comparisons, a two-sided P-value of <0.05 was considered statistically significant. All data analyses were performed using the Statistical Package for Social Sciences (SPSS 12.0.1 and 14.0) software program (SPSS Inc., Chicago, IL, USA).

Results

Prevalence of cardiac troponin I elevation and its relation to clinical characteristics

cTnI levels >0.01 µg/L were found in 219 subjects (21.8%), and 41 subjects (4.1%) had cTnI levels >0.02 µg/L. The relation of cTnI > 0.01 µg/L to self-reported history and further clinical data is shown in Tables 1 and 2. cTnI levels were weakly correlated to levels of NT-pro-BNP (r = 0.12; P < 0.001), creatinine clearance (r = −0.12; P < 0.001), and cystatin C (r = 0.11; P < 0.001) and showed no correlation to levels of C-reactive protein and IL-6 (r < 0.10). As shown in Figure 1, cTnI > 0.01 µg/L was also correlated to an increased risk for coronary heart disease as defined by the Framingham risk score.

Relation of cardiac troponin I elevation to atherosclerosis in the carotid arteries

Results regarding IMT and the presence of atherosclerotic plaques were available in 914 subjects. cTnI levels >0.01 µg/L were not more common in subjects in whom IMT or atherosclerotic plaques were not assessed. Although cTnI > 0.01 µg/L showed no association with the IMT in the carotid artery, significant correlations were found to different measures of atherosclerotic burden (Table 3).

Relation of cardiac troponin I elevation to echocardiographic findings

The frequency of high-quality echocardiographic recordings allowing the reliable evaluation of the studied variables ranged from 85 to 97%, leaving 762 subjects in whom results for all variables were available. The prevalence of cTnI elevation >0.01 µg/L in the remaining study participants did not differ significantly from that cohort. As given in Table 4, cTnI > 0.01 µg/L was highly correlated to systolic LV function and LV mass. cTnI > 0.01 µg/L was also more common

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Clinical characteristics</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>cTnI ≤ 0.01 µg/L (n = 786), n (%)</td>
</tr>
<tr>
<td>Males</td>
<td>369 (46.9)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>555 (70.6)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>90 (11.5)</td>
</tr>
<tr>
<td>Hyperlipidaemia</td>
<td>479 (60.9)</td>
</tr>
<tr>
<td>Current smoking</td>
<td>78 (9.9)</td>
</tr>
<tr>
<td>Previous smoking</td>
<td>326 (41.5)</td>
</tr>
<tr>
<td>Self-reported history</td>
<td></td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>45 (5.7)</td>
</tr>
<tr>
<td>Heart failure</td>
<td>21 (2.7)</td>
</tr>
<tr>
<td>PCI/CABG</td>
<td>33 (4.2)</td>
</tr>
<tr>
<td>Stroke</td>
<td>25 (3.2)</td>
</tr>
<tr>
<td>Medication</td>
<td></td>
</tr>
<tr>
<td>ACE-inhibitors/AII-ant</td>
<td>112 (14.2)</td>
</tr>
<tr>
<td>ASA</td>
<td>131 (16.7)</td>
</tr>
<tr>
<td>Beta-blockers</td>
<td>158 (20.1)</td>
</tr>
<tr>
<td>Calcium-antagonists</td>
<td>80 (10.2)</td>
</tr>
<tr>
<td>Diuretics</td>
<td>90 (11.5)</td>
</tr>
<tr>
<td>Lipid-lowering drugs</td>
<td>112 (14.2)</td>
</tr>
<tr>
<td>Nitrates</td>
<td>19 (2.4)</td>
</tr>
<tr>
<td>ECG</td>
<td></td>
</tr>
<tr>
<td>ST-segment depression</td>
<td>40 (5.1)</td>
</tr>
<tr>
<td>T-wave inversion</td>
<td>52 (6.6)</td>
</tr>
<tr>
<td>Left bundle branch block</td>
<td>7 (0.9)</td>
</tr>
<tr>
<td>Abnormal Q-wave</td>
<td>33 (4.2)</td>
</tr>
<tr>
<td>Ischaemic ECG changes</td>
<td>108 (13.7)</td>
</tr>
</tbody>
</table>
in the 182 study participants with LV hypertrophy [52 participants (28.6%) vs. 109 participants (18.8%); \(P = 0.007\)] but showed no associations with measures of diastolic LV function (Table 4).

**Relation of cardiac troponin I elevation to myocardial scars**

The presence of hyperenhanced myocardium according to cardiac MRI was evaluated in 248 subjects, as 11 examinations were not assessable because of poor image quality. Hyperenhancement indicative of infarcted myocardium was detected in 60 subjects. Among these, 49 did not have a previously recognized myocardial infarction. The prevalence of cTnI elevation \(>0.01\) \(\mu\)g/L in subjects with myocardial hyperenhancement compared with those without was not significantly increased [19 subjects (31.7%) vs. 44 subjects (23.4%); \(P = 0.23\)].

**Independent predictors of cardiac troponin I elevation**

As given in Table 5, male gender, ischaemic ECG changes, and NT-pro-BNP levels independently predicted cTnI levels \(>0.01\) \(\mu\)g/L. There was also a strong correlation to increased LV mass index which, however, did not reach levels of statistical significance. These associations remained unchanged when LV mass index was replaced by the number of carotid arteries with atherosclerotic plaques or the plaque size. The number of carotid arteries with plaques or the plaque size were not independently associated with cTnI \(>0.01\) \(\mu\)g/L.

**Discussion**

In this population-based sample of 70-year-old subjects, a high rate of cTnI levels \(>0.01\) \(\mu\)g/L was found. Consistent with other studies, cTnI elevation was more frequent in males and subjects without was not significantly increased [19 subjects (31.7%) vs. 44 subjects (23.4%); \(P = 0.23\)].
cTnI elevation >0.01 μg/L was further associated with impaired LV systolic function and increased LV mass identified by echocardiography and/or NT-pro-BNP levels. In subjects with impaired LV systolic function, troponin release has been related to decreased subendocardial perfusion because of increased myocardial wall tension or apoptotic processes resulting in myocardial damage. In case of LV hypertrophy, troponin leakage may be caused by a myocardial oxygen supply/demand mismatch owing to hypertrophied cardiomyocytes in combination with decreased coronary flow reserve because of remodelled circulation. An association between troponin elevation and impaired diastolic LV function, in contrast, could not be established by our results.

Elevated cTnI levels were also more common in study participants with ECG abnormalities indicative of ischaemic heart disease. This together with the association between cTnI levels and the degree of atherosclerosis in the carotid arteries raises the question whether cTnI not only does reflect the degree of atherosclerotic burden but also unstable coronary plaques causing subclinical microembolizations. However, cTnI levels were not correlated to C-reactive protein and IL-6, which have been linked to low-grade inflammatory processes in the coronary vessels causing a higher propensity of plaques to rupture. In addition, results from the cardiac MRI examination did not reveal a significant association between cTnI elevation and myocardial hyperenhancement indicative of myocardial scars. Even though this analysis may have been underpowered, our findings taken together do not provide evidence supporting a potential association between cTnI leakage and subclinical plaque instability.

cTnI elevation has been reported in patients with renal failure, probably depending on myocardial stretch owing to volume over-load and/or clinically silent myocardial micro-infarctions. Even in our study, there was a significant trend towards a higher prevalence of elevated cTnI levels in patients with impaired renal function, expressed as increased levels of cystatin C, an inhibitor of elastolytic proteases involved in vascular inflammation. No association was found between creatinine clearance and cTnI, which may depend on the narrow range of renal function results in our apparently healthy population but also illustrates that cystatin C is a more accurate estimate of kidney function. Cystatin C, however, was not independently associated with cTnI elevation. This indicates that, contrary to previous experiences with cardiac troponin T, cTnI elevation is not determined by impaired renal function per se.

Several authors have described minor troponin elevation in general populations. In a recent study by Zethelius et al., elevated cTnI levels >0.02 μg/L determined by the prior version of the Access AccuTnI assay were documented in 28.5% of apparently healthy elderly men. The prevalence of cTnI elevation in our study was lower, which probably depends on a lower prevalence of previous cardiovascular diseases and the inclusion of both men and women in our study. In the study by Zethelius et al., cTnI levels >0.02 μg/L predicted mortality in subgroups both with and without previously known cardiovascular disease. However, it is important to emphasize that owing to an improved sensitivity, the recently modified AccuTnI assay allowed the reliable identification of a subset of our study participants with minor cTnI elevation who would have remained undetected otherwise, i.e. subjects with cTnI between 0.01 μg/L and the 99th percentile of the assay. This minor cTnI elevation is below currently applied thresholds for decision making but has probably to be regarded as prognostically important given its relationship with the Framingham risk score and results from stabilized post-ACS patients. This is supported by a recent analysis in an emergency department population, demonstrating that any measurable cTnI level using a sensitive assay is predictive for adverse events. Thus, cardiac troponin results determined by highly sensitive assays might serve as an instrument for the identification of high-risk subjects from a general population in whom further investigation and intensive risk factor modification are mandatory. This, however, needs to be prospectively validated together with the specification of subgroups in whom screening for cardiac troponin might be particularly valuable.

### Table 5 Multivariable analysis (independent predictors of cardiac troponin I levels >0.01 μg/L)

<table>
<thead>
<tr>
<th>Model 1 (n = 1005)</th>
<th>Model 2 (n = 1002)</th>
<th>Model 3 (n = 825)</th>
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<tbody>
<tr>
<td><strong>OR</strong></td>
<td><strong>P-value</strong></td>
<td><strong>OR</strong></td>
</tr>
<tr>
<td>Male gender (y/n)</td>
<td>1.7 (1.2–2.3)</td>
<td>0.001</td>
</tr>
<tr>
<td>Hyperlipidaemia (y/n)</td>
<td>1.4 (1.0–2.0)</td>
<td>0.05</td>
</tr>
<tr>
<td>Previous AMI (y/n)</td>
<td>1.0 (0.5–2.0)</td>
<td>0.94</td>
</tr>
<tr>
<td>Congestive heart failure (y/n)</td>
<td>1.7 (0.8–3.6)</td>
<td>0.17</td>
</tr>
<tr>
<td>Previous PCI/CABG (y/n)</td>
<td>1.3 (0.7–2.7)</td>
<td>0.41</td>
</tr>
<tr>
<td>Ischaemic ECG (y/n)</td>
<td>2.2 (1.5–3.3)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>NT-pro-BNP (log)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Cystatin C (continuous)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>LV mass index (10 g/m²)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>LV-EF (continuous)</td>
<td>—</td>
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</table>

Model 1: adjusted for male gender, hyperlipidaemia, previous AMI, congestive heart failure, previous coronary revascularization, ischaemic ECG changes; model 2: adjusted for the same variables as in model 1, with the addition of NT-pro-BNP and C-reactive protein levels; model 3: adjusted for the same variables as in model 2, with the addition of LV mass index (continuous) and LV-EF (continuous). AMI, acute myocardial infarction.
In the light of our and other authors results, the use of the term 'troponin elevation' also needs to be addressed. Current concepts suggest that circulating troponin should not be detected in the blood stream of normal subjects. However, considering the unexpected high rate of elevated cTnI levels in general populations, it appears that, as for the natriuretic peptides, increasing cTnI levels mirror a continuously increasing degree of impaired cardiac integrity, even in the absence of acute myocardial necrosis of ischaemic cause. This emphasizes that clinical cut-offs using highly sensitive assays should rather be based on analytical issues (i.e. CV levels) than on distribution characteristics (i.e. percentiles) in order to identify any 'true' troponin elevation. Also, the importance of dynamic changes in troponin levels and of complementary diagnostic criteria for the discrimination of acute causes for elevated troponin levels (e.g. myocardial infarction) from chronic causes (e.g. stable heart failure) needs to be reinforced as pointed out in a recent editorial by Jaffe.

Limitations
The present study is limited to Caucasians aged 70 years. Caution should therefore be made to draw conclusions to other ethnic or age groups. Information on cardiovascular diseases was mainly based on the study participants' self-reported history. However, elevated cTnI levels were significantly more common in subjects with self-reported cardiovascular disease which corresponds to findings from previous studies. Only relative few subjects had a depressed LV-EF. Thus, despite significant results, some caution is required regarding the relation between cTnI and systolic LV function. Owing to a relative small number of patients examined with MRI, this portion of the trial may have been not sufficiently powered to show a convincing positive or negative association between myocardial scars and elevation of cTnI. Owing to the relative short follow-up time of our study population, we did not present data on cardiovascular events. As a substitute, we applied the Framingham risk score, which has been shown to be highly predictive for myocardial infarction in another elderly general population from Uppsala, Sweden.

Conclusion
cTnI levels >0.01 µg/L can frequently be detected in a general population of 70-year-old subjects. Apart from cardiovascular high-risk features, cTnI > 0.01 µg/L was associated with the burden of atherosclerosis, LV hypertrophy, and impaired LV systolic function. No associations were found between cTnI elevation and increased inflammatory activity. The high prevalence of cTnI elevation in the population studied, without indications of acute ischaemic myocardial damage, reinforces the need for complementary criteria for the identification of acute causes for troponin leakage.

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Conflict of interest: B.L. is member of the scientific advisory board of Beckman Coulter Inc., and P.V. has received honoraria from that company. The other authors had no conflicts to report.

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CLINICAL VIGNETTE

Thrombus in left ventricular aneurysm: a change in morphology during echocardiographic follow-up

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A 72-year-old male was admitted with a history of subacute myocardial infarction. On coronary angiogram there was total occlusion of the proximal segment of the left anterior descending artery and non-obstructive disease of the other coronary arteries. An echocardiogram showed a large akinesia of anteroseptal segments with apical dyskinesis. It demonstrated an apical aneurysm with a mural thrombus inside the aneurysm. The size of the thrombus was 47 × 15 mm (Panel A).

The patient was started on full-dose low-molecular weight heparin and aspirin. Unfortunately, 1 week later he suffered a minor cerebral stroke which clinically resolved within another week. Follow-up echocardiograms performed during the following 4 weeks documented a change in thrombus morphology: from mural to highly mobile thrombus with a high risk of systemic embolization (Panels A–C). As conservative management seemed to be unsuccessful, the patient finally underwent urgent cardiac surgery—thrombus extraction (Panel D) with resection of the aneurysm and single coronary artery bypass grafting. The postoperative course was uneventful.

Panel A. Apical four-chamber view demonstrating mural thrombus (size 47 × 15 mm) on admission echocardiogram.
Panel B. Echocardiogram 2 weeks later with early separation of the thrombus.
Panel C. Mobile thrombus on a 4-week follow-up echocardiogram.
Panel D. Older ovoid thrombus sized 40 × 20 mm and small fresh thrombus after removing from the left ventricle.