Prediction of 6 months left ventricular dilatation after myocardial infarction in relation to cardiac morbidity and mortality

Application of a new dilatation model to GISSI-3 data

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Aims To predict the long-term left ventricular volume index early after myocardial infarction and to investigate the relationship between long-term left ventricular dilatation risk and clinical outcome.

Methods and Results By applying a previously developed dilatation model, we predicted the 6-month left ventricular volume index early after myocardial infarction (median 9 days) in 13 679 GISSI-3 patients, to identify patients at high risk of long-term left ventricular dilatation. The left ventricular systolic and diastolic volume indexes at 6 months were predicted with \( r = 0.72 \) and \( r = 0.68 \), respectively, in the subgroup of patients in whom a pre-discharge echo was available (\( n = 7842 \)). Patients predicted to be at risk for long-term left ventricular dilatation had an increased risk of mortality (RR 1.87, 95% CI: 1.48 to 2.36) and heart failure at 6 months (RR 2.59, 95% CI: 2.04 to 3.28), but no increased risk of reinfarction at 6 months (RR 1.12, 95% CI: 0.87 to 1.45) or of angina pectoris (RR 1.07, 95% CI: 0.95 to 1.20).

Conclusion Our prediction of long-term left ventricular dilatation, obtained by applying our new dilatation model in over 13 000 GISSI-3 patients, correlated well with mortality and heart failure after myocardial infarction. Therefore, our new dilatation model may contribute to more efficient risk stratification early after myocardial infarction.

Key Words: Prediction model, left ventricular dilatation, 2-D echocardiography, myocardial infarction, mortality, cardiac morbidity.

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Introduction

Survivors of an acute myocardial infarction are at increased risks of cardiac morbidity and mortality\(^{1-3}\). Mortality and morbidity are related to alterations in left ventricular structure after myocardial infarction, often referred to as left ventricular dilatation, or remodelling\(^{4-6}\). Therefore, early identification of patients at risk of long-term left ventricular dilatation might improve clinical outcome after myocardial infarction. We previously developed a dilatation model, which described 1 year left ventricular dilatation after myocardial infarction, using patient characteristics measured early after myocardial infarction\(^{7}\). The primary aim of the present study was to apply this early left ventricular dilatation prediction model to predict left ventricular volume indexes at 6 months for all 13 679 patients of the echocardiographic subpopulation of GISSI-3, a large
and less selected group of patients. The second aim of this study was to investigate whether patients at risk of long-term left ventricular dilatation were at increased risk of mortality and cardiac morbidity after myocardial infarction. Patients at risk of long-term left ventricular dilatation were identified using 6-month left ventricular volume indexes, as predicted by the early left ventricular dilatation prediction model.

**Methods**

**Study patients**

The present study used the data of all 13,679 patients of the echocardiographic subpopulation of GISSI-3 to predict the left ventricular volume index at 6 months, by applying our early left ventricular dilatation prediction model and to investigate the relationship between risk of long-term left ventricular dilatation and clinical outcome after myocardial infarction. The third Gruppo Italiano per lo Studio della Sopravvivenza nell’Infarto Miocardico (GISSI-3) study was a controlled multicentre, randomized mortality trial after myocardial infarction. Four treatment groups were assigned by randomization within 24 h of myocardial infarction to: lisinopril alone, transdermal glyceryl trinitrate alone, combined trial therapy and no trial therapy. The major inclusion criteria were a diagnosis of myocardial infarction, and admission to hospital within 24 h of myocardial infarction. In the GISSI-3 study, 19,394 patients were randomized to study treatment. Complete clinical data and 6 weeks follow-up were available for 18,895 (97.4%) patients of whom 17,825 were discharged alive. The echocardiographic subpopulation of GISSI-3 consisted of 13,679 patients discharged alive, with confirmed myocardial infarction and an ejection fraction available from at least one of three planned echocardiographic assessments during 6-months follow-up.

**Echocardiography**

According to the GISSI-3 study protocol, an echocardiographic examination was required at 6 weeks, and 6 months. A pre-discharge echocardiographic examination was recommended. All echocardiograms were stored on videotape and analysed at each participating centre. End-diastole was defined as the frame with the largest left ventricular cavity area closest to the onset of the QRS complex on the electrocardiogram, and end-systole as the subsequent frame with the smallest ventricular cavity area. Three orthogonal left ventricular endocardial axes were measured at end-diastole and end-systole (average of three cardiac cycles). From these axes, left ventricular systolic and diastolic volumes were calculated, and indexed by body surface area. Body surface area was not available for all patients, which resulted in a database of 7,883 patients with pre-discharge, 11,083 patients with 6 week and 9,778 patients with 6 month left ventricular systolic and diastolic volume indexes.

**Dilatation model and criterion**

Based on the two-dimensional echocardiographic data of a previous study, we developed a random-effects, or dilatation model, which described the progression of both left ventricular systolic and diastolic volume indexes for 1 year after myocardial infarction in 845 patients. The predictors of 1 year progression of both left ventricular diastolic and systolic volume indexes were selected from demographics, clinical history, haemodynamics, medication history, infarct characteristics (including a clinical definition of infarct artery patency) and baseline echocardiography (Table 1). The significant predictors, as included in the dilatation model, for both the left ventricular diastolic, and systolic volume indexes were: baseline left ventricular volume (median 2 days after myocardial infarction), gender, peak creatine phosphokinase (CPK) as a marker for infarct size, and the relationship between peak creatine phosphokinase and time after myocardial infarction on progression of left ventricular volume (interaction between peak creatine phosphokinase and time). A detailed description of the dilatation model is provided in the appendix. After applying the dilatation model to all 13,679 GISSI-3 patients, we identified those patients at high risk of long-term left ventricular dilatation by applying a previously reported dilatation criterion. This implied that the risk of long-term left ventricular dilatation was assumed if the patient’s predicted 6-month left ventricular diastolic volume index was larger than 63 ml . m$^{-2}$, or the predicted left ventricular systolic volume index was larger than 30 ml . m$^{-2}$, and the sum of the predicted left ventricular diastolic and systolic volume indexes were larger than 88 ml . m$^{-2}$.

**Mortality and cardiac morbidity**

The association between long-term left ventricular dilatation risk, mortality and cardiac morbidity was assessed by comparing the number of cardiac morbidity events occurring after discharge until 6 months after myocardial infarction, between patients at high and low risk of long-term left ventricular dilatation. The predefined cardiac morbidity events, heart failure, angina pectoris and reinfarction were collected on a separate data collection sheet, and examined by an ad hoc attribution committee (composed of three senior clinicians) prior to unblinding of the trial.

**Statistical methods**

Continuous, normally distributed patient characteristics are reported as mean ± standard deviation, and continuous skewed distributed variables are presented.
by median and inter-quartile range. Categorical patient characteristics are described by frequencies and percentages. The effect of left ventricular dilation on mortality and cardiac morbidity at 6 months was evaluated by multiple Cox regression analysis using backward selection, correcting for all clinical characteristics with \( P < 0.01 \). Continuous clinical characteristics were divided into quartiles. If the mortality or cardiac morbidity risk increased disproportionately from one quartile to the next, quartiles with comparable risk-ratios were combined; otherwise, the clinical characteristic was included continuously. Using this procedure, the calculated risk ratios (RR) for mortality and cardiac morbidity were corrected for all clinical characteristics with \( P < 0.01 \). To assess which echocardiographic measure was a better predictor of mortality, risk-ratios of ejection fraction and long-term left ventricular dilatation as predicted by the dilatation model, were calculated in two multiple Cox regression models, correcting for the same (dichotomous) clinical characteristics. All reported \( P \)-values were two-sided. SAS version 6.12 (Cary, North Carolina) was used for all statistical evaluations.

### Results

#### Patients

Of the 13,679 patients included in the echocardiographic subpopulation of GISSI-3, the dilatation model could predict 6 month left ventricular volume indexes for 13,555 patients (99%). For 7,842 patients, the predictions of 6 month left ventricular volume indexes were based on clinical and pre-discharge echocardiographic characteristics, and for 5,713 patients based on clinical characteristics only (since no pre-discharge left ventricular volume indexes were available). The dilatation model predicted 6 month left ventricular diastolic and systolic volume indexes, with respective \( r = 0.48 \) and \( r = 0.52 \), when a pre-discharge echo was available (Fig. 1). However, the 6 month left ventricular diastolic and systolic volume indexes were predicted with, respectively, \( r = 0.68 \) and \( r = 0.72 \), when a pre-discharge echo was available (based on clinical characteristics alone). Therefore, the dilatation criterion was only applied to the 7,842 GISSI-3 patients with a pre-discharge echo. Applying the dilatation criterion to the

### Table 1 Baseline characteristics of GISSI-3 patients for which long-term dilatation risk could be assessed with pre-discharge echo available

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>No long-term left ventricular dilatation risk (n=5234)</th>
<th>Long-term left ventricular dilatation risk (n=2608)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Demographics</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>61 (±12)</td>
<td>61 (±12)</td>
</tr>
<tr>
<td>Male gender*</td>
<td>4053 (77%)</td>
<td>2258 (87%)</td>
</tr>
<tr>
<td>Body surface area</td>
<td>1.82 (±0.17)</td>
<td>1.84 (±0.16)</td>
</tr>
<tr>
<td>Clinical history</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Myocardial infarction*</td>
<td>496 (10%)</td>
<td>485 (19%)</td>
</tr>
<tr>
<td>Angina pectoris</td>
<td>1774 (34%)</td>
<td>929 (36%)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1932 (38%)</td>
<td>958 (39%)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>736 (14%)</td>
<td>389 (15%)</td>
</tr>
<tr>
<td>Current smoker</td>
<td>2651 (56%)</td>
<td>1338 (58%)</td>
</tr>
<tr>
<td>Killip class I (%)*</td>
<td>4731 (90%)</td>
<td>2166 (83%)</td>
</tr>
<tr>
<td>Haemodynamics</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean arterial pressure</td>
<td>99 (±13)</td>
<td>99 (±14)</td>
</tr>
<tr>
<td>Heart rate (beats . min (^{-1}))*</td>
<td>74 (±15)</td>
<td>76 (±16)</td>
</tr>
<tr>
<td>Medication history</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beta-blockers*</td>
<td>1777 (34%)</td>
<td>770 (30%)</td>
</tr>
<tr>
<td>Calcium antagonists*</td>
<td>946 (18%)</td>
<td>353 (14%)</td>
</tr>
<tr>
<td>Diuretics*</td>
<td>517 (10%)</td>
<td>543 (21%)</td>
</tr>
<tr>
<td>Nitrates</td>
<td>697 (13%)</td>
<td>354 (14%)</td>
</tr>
<tr>
<td>Infarct characteristics</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterior infarction*</td>
<td>1372 (29%)</td>
<td>1097 (48%)</td>
</tr>
<tr>
<td>Peak creatine phosphokinase (IU/1000)*</td>
<td>1.2 (0.7–2.0)</td>
<td>2.2 (1.2–3.5)</td>
</tr>
<tr>
<td>Thrombolytic therapy</td>
<td>3831 (73%)</td>
<td>1965 (75%)</td>
</tr>
<tr>
<td>Pre-discharge echocardiographic characteristics</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic volume index (ml . m (^{-2}))*</td>
<td>20 (15–24)</td>
<td>36 (31–43)</td>
</tr>
<tr>
<td>Diastolic volume index (ml . m (^{-2}))*</td>
<td>43 (36–50)</td>
<td>67 (60–76)</td>
</tr>
<tr>
<td>Ejection fraction (%)*</td>
<td>54 (47–61)</td>
<td>45 (38–51)</td>
</tr>
</tbody>
</table>

*Significantly different between patients with and without long-term left ventricular dilatation risk \( (P<0.001) \).
Left ventricular dilatation, as predicted by the dilatation model, was a stronger predictor of mortality than the classical characteristics Killip class and ejection fraction (Table 2). Patients at risk for long-term left ventricular dilatation had an increased risk of developing heart failure at 6 months (RR 2.59 (95% CI: 2.04 to 3.28, \( P=0.0001 \)). Furthermore, patients with long-term left ventricular dilatation risk showed a 1.12 (95% CI: 0.87 to 1.45, \( P=0.28 \)) times increased risk of developing reinfarctions and a 1.07 (95% CI: 0.95 to 1.20, \( P=0.28 \)) times increased risk of angina pectoris within 6 months of a myocardial infarction (Fig. 2).

**Discussion**
Early identification of patients at risk of long-term left ventricular dilatation after myocardial infarction may facilitate the choice and timing of medical therapy and may enable a more efficient regulation of control visits, which in turn may improve clinical outcome. In the present study, we applied a previously developed dilatation model to 13,679 GISSI-3 patients, and identified those patients at risk of long-term (6 months) left ventricular dilatation in the subgroup of patients in whom pre-discharge left ventricular volume indexes were available (n=7842). After thus identifying patients at risk of long-term left ventricular dilatation, the model attained adequate risk stratification (Table 1). In fact, left ventricular dilatation, as predicted by our dilatation model, proved to be a better predictor of 6-month mortality than classical predictors of mortality, such as ejection fraction and Killip class (Table 2). Finally, patients at long-term left ventricular dilatation risk after myocardial infarction were at increased risk of mortality and heart failure (Fig. 2). In contrast, long-term left ventricular dilatation risk after myocardial infarction was not associated with the development of ischaemic events (reinfarction and angina pectoris).

**Left ventricular dilatation**
Left ventricular dilatation results from infarct expansion\(^{[12]}\), occurring between 3 days and 2 weeks after the onset of an acute myocardial infarction\(^{[13]}\). Patients showing infarct expansion 10 to 21 days after infarction, may continue to expand over 6 to 30 months\(^{[14]}\). By applying a previously established dilatation model\(^{[3]}\), we were able to predict left ventricular dilatation at 6 months early after myocardial infarction (median 9 days after myocardial infarction). The predicted risk of long-term left ventricular dilatation was established as high, if the patient’s predicted left ventricular volume indexes at

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**Table 2 The predictors of 6-months mortality after myocardial infarction (in descending order of importance)**

<table>
<thead>
<tr>
<th>Patient characteristic (n=7842)</th>
<th>Wald (\chi^2)</th>
<th>Adjusted risk-ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>112.0</td>
<td>1.06 (1.05–1.08)</td>
</tr>
<tr>
<td>Heart rate (beats (\text{min}^{-1}))</td>
<td>46.2</td>
<td>1.02 (1.02–1.03)</td>
</tr>
<tr>
<td>Left ventricular dilatation (dilatation model)</td>
<td>27.1</td>
<td>1.87 (1.48–2.36)</td>
</tr>
<tr>
<td>Pre-discharge ejection fraction (\leq0.44^*)</td>
<td>19.6</td>
<td>1.71 (1.35–2.17)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>17.4</td>
<td>1.73 (1.34–2.24)</td>
</tr>
<tr>
<td>History of myocardial infarction</td>
<td>16.9</td>
<td>1.75 (1.34–2.28)</td>
</tr>
<tr>
<td>History of angina pectoris</td>
<td>13.3</td>
<td>1.55 (1.23–1.96)</td>
</tr>
<tr>
<td>Killip class &gt; I</td>
<td>12.2</td>
<td>1.61 (1.23–2.11)</td>
</tr>
</tbody>
</table>

\(^*\)Fourth quartile value\(^{[10]}\).
6 months were large enough to fulfil the dilatation criterion. In the present study, 33% of the subjects demonstrated long-term left ventricular dilatation risk, which was similar to other studies, reporting that about 25% of all patients with acute myocardial infarction develop significant left ventricular dilatation in the long-term\cite{15,16}. Patients with a high predicted risk of long-term left ventricular dilatation more often had an anterior infarction, higher heart rate, history of myocardial infarction and Killip class II, which is in accordance with the results of previous studies\cite{17–19}.

**Mortality and heart failure**

Mortality after myocardial infarction is influenced by multiple patient characteristics such as age, sex, clinical history and infarct characteristics. However, the main determinants of prognosis are left ventricular function\cite{20,21} and size\cite{4,22}. Prediction of high risk on left ventricular dilatation provided the dissociation of clinical characteristics which are associated with mortality (Table 1). In addition, development of heart failure is one of the major morbidity events in the large clinical studies employing risk stratification after myocardial infarction\cite{23,24}. Also other echocardiographic studies\cite{5,6,25} clearly demonstrated that left ventricular dilatation after myocardial infarction is an independent risk factor of mortality and heart failure. In addition, the GISSI-3 investigators reported that the quartiles of pre-discharge left ventricular diastolic volume, systolic volume and ejection fraction significantly predicted both mortality and heart failure at 6 months\cite{10}. In the present study, we used a different approach to investigate the relationship between left ventricular dilatation, mortality and heart failure, by identifying GISSI-3 patients at risk of long-term left ventricular dilatation. Our results also showed increased mortality and heart failure rates for patients at risk of long-term left ventricular dilatation. Furthermore, prediction of left ventricular dilatation proved to be a stronger predictor of mortality than the classical predictors ejection fraction and Killip class.

**Reinfarction and angina pectoris**

Reinfarction occurs less frequently than angina pectoris after myocardial infarction. However, the prognosis after reinfarction is poor\cite{26}, while there is little information on the clinical importance of angina pectoris. In accordance with the results of our study, previous studies indicated that depressed ventricular function was neither a significant predictor for reinfarction\cite{27,28} nor for angina pectoris\cite{29,30}. Therefore, other progressive processes (e.g. endothelial dysfunction resulting in progression of atherosclerotic disease and ischaemia) rather than left ventricular dilatation may contribute to the adverse prognosis of patients with reinfarction and post-infarction angina pectoris.

**Study limitations**

Despite correlation coefficients of about 0.70 for the prediction of left ventricular systolic and diastolic volume indexes at 6 months, based on early clinical and echocardiographic characteristics using our new dilatation model, about half of the variation remains unexplained. Therefore, although early identification of patients at high risk of dilatation by our dilatation model may facilitate individual patient management, the
assessment of other patient-specific clinical parameters remains important for adequate risk stratification. Furthermore, the general population of the GISSI-3 study was a very low risk population. We speculate that the dilatation model may yield even more efficient risk stratification in a general post-myocardial infarction population, which would include high risk patients. Finally, since left ventricular dilatation is most extensive in the first days after myocardial infarction, we speculate that additional benefit would have been obtained from the dilatation model if the first echocardiographic assessment in GISSI-3 had been obtained earlier after myocardial infarction than at pre-discharge.

Conclusion

The present study proposes a dilatation model which can be used to identify, at an early stage, those patients at risk of long-term left ventricular dilatation, which, in addition to assessment of other clinical parameters, may contribute to a more efficient risk stratification early after myocardial infarction. In order to identify those patients at risk of long-term left ventricular dilatation at an early stage, early assessment of LV volume after MI is essential.

We showed that the subset of patients predicted to be at risk for long-term left ventricular dilatation have a two- to threefold increased risk of developing heart failure and a twofold increased risk of dying. Consequently, these high-risk patients may benefit from better choice and timing of medical therapy, and an increased frequency of control visits. Therefore, if attention can be focused on these high-risk patients at an early stage (i.e. by applying the dilatation model early after myocardial infarction), the clinical outcome of these patients may improve. Furthermore, we demonstrated that extensive left ventricular dilatation appeared to be independent of the development of angina pectoris and reinfarction after myocardial infarction. Accordingly, the results of the present study suggest that the development of cardiac events after myocardial infarction may be caused by at least two independent progressive processes. Firstly, progression of left ventricular dilatation, which is associated with the development of heart failure. Secondly, progression of atherosclerotic disease, which may be involved in the development of ischaemic events after myocardial infarction.

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Appendix
The dilatation model was constructed based on the
information available from repetitive echocardiographic
data and clinical characteristics of 845 patients. However,
to make it clinically useful and to gain a better
understanding of the effect of both the clinical and
echocardiographic explanatory variables on the predic-
tion of left ventricular systolic and diastolic volume
index at any point in time after myocardial infarction,
the following two formulas can be applied:

(1) \( \text{LVESVI}_t = \text{EXP}(a_1 \times \text{intercept} + a_2 \times \text{LVESVI}_0 + a_3 \times \text{male gender} + a_4 \times \text{peak CPK} + a_5 \times \text{peak CPK}^t) \)

(2) \( \text{LVEDVI}_t = \text{EXP}(a_1 \times \text{intercept} + a_2 \times \text{LVEDVI}_0 + a_4 \times \text{male gender} + a_5 \times \text{peak CPK} + a_6 \times \text{peak CPK}^t) \)

Where:
\( t \) the natural logarithm (ln) of any point in time (days) for 1 year after myocardial infarction;
\( \text{LVESVI}_t / \text{LVEDVI}_t = \) predicted left ventricular end-
systolic/diastolic volume index at time \( t \);
\( \text{LVESVI}_0 / \text{LVEDVI}_0 = \) measured left ventricular end-
systolic/diastolic volume index at baseline (if possible
measured within 3 days after myocardial infarction);
peak CPK = peak creatine phosphokinase value (IU)
divided by 1000;
peak CPK\( ^t \) = the interaction between peak creatine
phosphokinase and time after myocardial infarction;
a_1 \text{ to } a_6 = \) the regression coefficients as presented in
Table 1a.

Example*:
The 6 month LVESVI/LVEDVI of a male patient with
baseline LVESVI of 30 ml.m\(^{-2}\) and a baseline
LVEDVI of 50 ml.m\(^{-2}\) and a relatively large infarction
(pink CPK value of 3000 IU) would be predicted as
follows: \( t = \ln(\text{6 months} \approx \ln(365/2 \text{ days}) \approx 5.21; \text{ peak CPK}/1000 = 3.0; \)

\( \text{LVESVI}_{5.21} = \text{EXP}(2.39 + 0.0262 \times 30 + 0.0293 \times 3.0 + 0.0097 \times 3.0 \times 5.21) \approx \text{EXP}(3.84); \approx 32.6 \text{ ml.m}^{-2} \)

\( \text{LVEDVI}_{5.21} = \text{EXP}(3.23 + 0.0125 \times 30 + 0.0094 \times 3.0 + 0.0075 \times 3.0 \times 5.21) \approx \text{EXP}(4.06) \approx 58.0 \text{ ml.m}^{-2} \)

Since the predicted 6 month left ventricular diastolic
volume index was not larger than the required
36 ml.m\(^{-2}\), according to the dilatation criterion this
patient was not identified as being at high risk of
long-term left ventricular dilatation (although the pre-
dicted left ventricular systolic volume index was larger
than 30 ml.m\(^{-2}\), and the sum of the predicted left
ventricular diastolic and systolic volume index was
larger than 88 ml.m\(^{-2}\)).

*Of note, for individual prediction, although we
obtained a correlation coefficient of 0.68 for a diastolic
volume index and 0.72 for a systolic volume index
using GISSI-3 data, about half of the variation remains
unexplained.

<table>
<thead>
<tr>
<th>Table 1a</th>
<th>Regression coefficients and 95% confidence intervals for models 1 and 2 presented above, to predict left ventricular end-systolic and end-diastolic volume indexes at any point in time for 1 year after myocardial infarction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model term</td>
<td>Regression coefficient (95% CI) for prediction of LVESVI(_t)</td>
</tr>
<tr>
<td>Intercept ( (a_1) )</td>
<td>2.39 (2.33–2.45)</td>
</tr>
<tr>
<td>LVESVI(_0) ( (a_2) )</td>
<td>0.0262 (0.0242–0.0281)</td>
</tr>
<tr>
<td>LVEDVI(_0) ( (a_2) )</td>
<td>0.0681 (0.0196–0.1117)</td>
</tr>
<tr>
<td>Male gender ( (a_4) )</td>
<td>0.0293 (0.0142–0.0443)</td>
</tr>
<tr>
<td>Peak CPK ( (a_5) )</td>
<td>0.0097 (0.0063–0.0131)</td>
</tr>
</tbody>
</table>

*In cases of female gender, this regression coefficient will be zero.