Transthoracic echocardiography provides important long-term prognostic information in selected patients undergoing endovascular abdominal aortic repair.

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Abstract

Background The value of performing transthoracic echocardiography (TTE) as part of the clinical assessment of patients awaiting endovascular repair of the abdominal aorta (EVAR) is little evaluated. We aimed to estimate the prognostic importance of information derived from TTE on long-term all-cause mortality in a selected group of patients undergoing EVAR.

Methods and Results This was a retrospective cohort study of 273 consecutive patients selected for EVAR. All patients included in analysis underwent TTE before their procedure. Multivariable Cox regression analysis was used to estimate the effect of TTE measures on all-cause mortality. Over a mean follow up of 3.2±1.5 years there were 78 deaths with a mean time to death of 1.28±1.16 years. A greater tubular ascending aorta (HR 5.6, 95% CI 2.77-11.33), presence of mitral regurgitation (HR 8.13, 95% CI 4.09-12.16), lower left ventricular ejection fraction (HR 0.96, 95% CI 0.93-0.98), younger age (HR 0.97, 95% CI 0.95-0.99) and presence of diabetes (HR 1.46, 95% CI 1.24-1.89) were predictors of all-cause mortality.

Conclusions Echocardiography provides important long-term prognostic information in patients undergoing EVAR. These TTE indices were more important at predicting outcome than standard conventional risk factors in this patient group. A greater tubular ascending aorta, presence of mitral regurgitation, reduced left ventricular ejection fraction, younger age and diabetes were independently associated with long-term mortality.

Key words: Abdominal aortic aneurysm, Endovascular aneurysm repair, Transthoracic echocardiography.

Introduction

Abdominal aortic aneurysms (AAA) remain a significant health challenge with a reported prevalence of 1.4% in the US, increasing significantly in males over the age of 60. Traditionally, surgical repair for AAA has consisted of an open operative approach requiring laparotomy with extensive associated morbidity and mortality. Endovascular aneurysm repair (EVAR) has developed into a viable alternative to open surgery for AAA repair and is now the predominant method of aneurysm repair in most developed nations.[1]

Despite an established benefit in terms of peri-operative mortality for EVAR,[2, 3] the long-term outcome in patients with AAA, undergoing either open or endovascular surgery, remains poor. Pre-existing data suggest that long-term survival is worse in patients surviving AAA repair than in an age-gender matched population,[4-6] and that late mortality is predominantly attributable to cardiovascular causes.[7] There is a strong association between traditional cardiovascular disease risk factors and higher incidence of AAA.[8, 9]

Consensus guidelines recommend pre-operative risk stratification of patients with AAA to optimise patient care and prevent complications, including mortality.[10, 11] Pre-operative cardiac assessment appears to be of most importance, since the most frequent complications post EVAR are cardiac.[12] Risk prediction based on clinical risk factors alone have limitations[13] and existing models for elective AAA repair fail to predict outcome with sufficient accuracy to be widely adopted into routine clinical practice.[14] Consequently, physicians routinely request specialised cardiac tests to improve risk stratification. As such, a transthoracic echocardiogram (TTE) is often advised as part of the clinical assessment of patients awaiting aneurysm repair. This is despite the lack of evidence base. Peri-procedural

mortality for EVAR is very low but these patients are usually elderly with other comorbidities with a 5-year survival of 52%.[15] The long-term prognostic value of TTE is unclear in the EVAR population. Therefore, the aim of this study was to assess pre-operative echocardiographic predictors of long-term all-cause mortality in patients undergoing EVAR.

Methods

Study Cohort

The study population consisted of 273 consecutive patients (73±10.7 years) undergoing elective EVAR between January 2008 and September 2010 from a single tertiary centre. Patients with ruptured aneurysms and trauma-related cases were excluded. All patients had a TTE and electrocardiogram (ECG) 4-weeks prior to their EVAR in the outpatient setting, as part of a global risk stratification protocol. Clinical characteristics were recorded at the time of TTE. This investigation conformed to the Declaration of Helsinki principles. All patients provided informed consent before testing and the local research ethics committee approved the study.

Transthoracic Echocardiography Image Acquisition

A full cross-sectional study was performed using a General Electric Vingmed System 7. All image acquisitions and measurements were performed as recommended by the American Society of Echocardiography (ASE).[16] Left ventricular end diastolic diameter (LVEDD), LV end systolic diameter (LVESD), interventricular and LV posterior wall thickness at end diastole were measured from parasternal M mode recordings of the LV, with the cursor at the tips of the mitral valve leaflets. LV mass (LVM) was then calculated according to the ASE recommendations.[16] LV fractional shortening was calculated from LVEDD and LVESD. LV ejection fraction (LVEF) was determined by the modified biplane Simpson's rule, with measurements averaged over three cardiac cycles. The LV endocardial border was traced contiguously from one side of the mitral annulus to the other, excluding the papillary muscles and trabeculations. LV regional wall motion was analysed visually using the standard 17-segment model for qualitative analysis and wall motion was scored on a 4-point scale (1 =

normal wall motion, 2 = hypokinesis, 3 = akinesis, and 4 = dyskinesis). The wall motion score index was calculated as an average of the individual wall motion scores of each visualised segment. For patients with poor endocardial border definition in 2 or more contiguous LV segments, the intravenous LV contrast agent SonoVue® was used to ensure optimal LV border definition.

Transmitral inflow was recorded using pulsed wave Doppler recordings at the mitral valve leaflet tips in the apical four-chamber view. Peak velocity of early filling (E), peak velocity of atrial filling (A), the E/A ratio and E deceleration time were measured. From pulsed wave real-time tissue Doppler images obtained in the four-chamber view, early diastolic (Ea) velocities were measured. LV filling pressure was estimated from the mitral E/Ea ratio.[17] Echocardiographic evaluation of the aorta was performed in the parasternal long-axis and suprasternal view with measures recorded at the aortic annulus, sinuses of Valsalva, sinotubular junction, tubular ascending aorta, aortic arch and descending aorta as recommended.[18] Full quantitative and semi-quantitative assessment of valvular disease was performed.[19, 20] Valve disease was then graded as none, mild, moderate or severe. Two accredited TTE imaging specialists retrospectively examined all TTE data in an echocardiography core laboratory.

Electrocardiography

A 12-lead ECG was recorded in the supine position prior to EVAR. All tracings were interpreted by a trained physician and coded on the basis of the Minnesota coding criteria.[21] Evidence of ischemia on ECG was defined as pathological Q waves, ST-segment depression, T wave inversion of any degree or left bundle branch block.[22] Left ventricular hypertrophy

was defined according to Sokolow and Lyon voltage criteria.[23] The ECG was graded as normal or pathological.

Endovascular Aneurysm Repair

All patients in this cohort were elective admissions for EVAR. Endografting was performed via percutaneous or surgical approaches to the femoral artery. The EVAR procedure was performed to an Institutional standard operating protocol and undertaken in a dedicated endovascular imaging suite under fluoroscopic guidance. The endografts employed were proprietary endografts predominantly from Cook© Medical, or Medtronic. Post-operative follow-up consists of a 6-week, 12-week, 6-month and subsequent annual consultation with a vascular surgeon.

Primary Outcome Measure

The primary outcome measure was long-term mortality up to 5 years post-surgery. Patients were followed up from the date of their EVAR procedure through to December 2013 and censored at the time of death or at last known follow-up. Mortality data were established through interrogation of electronic hospital or general practitioner records, and through the national death registry.

Statistical Analysis

Continuous variables were expressed as mean±standard deviation and categorical variables as n (%). Multivariable adjusted Cox proportional hazard models were constructed to ascertain predictors of all-cause mortality. For model building, demographic, clinical history, medication, laboratory measures and echocardiographic parameters were evaluated for their association with mortality. Age and gender were included in all models. Forward stepwise

selection procedures were used to compare models for goodness-of-fit and a *P*-value <0.1 was used for retention in the final model. The final multivariate model consisted of 11 variables (see Supplementary Table 1 and Supplementary Figure 1 for model goodness of fit and discrimination selection). Hazard ratios (HR) and corresponding 95% confidence intervals (CI) are reported.

Kaplan-Meier survival curves were constructed and compared using the log-rank test and a *P* value <0.05 was used to report statistical significance. The survival curves were stratified first according to the presence or absence of mitral regurgitation (MR) and, second, by patient risk profile (low, intermediate, or high-risk), which was calculated from the multivariable Cox model by splitting patients into three tertiles of predicted risk. Event rates were calculated and expressed as percentages per annum. All analyses were conducted using the statistical package for social sciences (SPSS 21 release version of SPSS for Windows; SPSS Inc., Chicago IL, USA).

Results

Clinical Outcome

The primary endpoint of all-cause mortality was observed in 78 (29%) patients during a mean follow-up period of 3.2±1.5 years, with a mean time to death of 1.28±1.16 years. The clinical characteristics of subjects survived versus all-cause mortality are shown in Table 1. There was one peri-operative death in a 91-year old patient who experienced a post-procedural lateral myocardial infarct, and nine deaths within 30-days post surgery (see Figure 1).

During the study period 273 consecutive patients were evaluated using TTE imaging 18±7 days prior to elective EVAR. Mean age was 73±10.7 years with a greater proportion of male patients (80%) in keeping with the recognised male-preponderance of AAA. The prevalence of hypertension, hypercholesterolaemia and diabetes were 77%, 53% and 13% respectively with 2% of patients having a prior history of coronary revascularisation and 5% a prior myocardial infarction. In addition, 13% patients had a prior cerebrovascular accident and 36% had previous evidence of ischaemic heart disease. Seventy-four (27%) patients were current smokers and 126 (46%) ex-smokers with a combined pack year of 32.8±24.2. 72% of patients (196/273) were treated with one or more anti-platelet medication, 9% (25/273) were warfarinised and 82% (229/273) were taking lipid-lowering therapies. However, use of other cardio-protective medication, such as beta-blockers (35%, 95/273) and angiotensin converting enzyme inhibitors (28%, 76/273) was low (Table 1).

Transthoracic Echocardiography

TTE was completed in all patients and the level of inter-observer agreement in reporting echo parameters between the two sonographers was K=0.89 (range 0.86-1.0). The TTE results for subjects survived versus all-cause mortality are shown in Table 2. Two hundred and thirteen patients (78%) had a LVEF reported within the normal range, 34 (12%) mildly impaired, 22 (8%) moderately impaired and 4 (2%) severely impaired.

Thirteen patients (5%) had a mildly dilated LV, 3 (1%) moderately dilated, 3 (1%) severely dilated and 254 (93%) had a normal LVEDD. Fifty-five patients (20%) had a mildly dilated left atrium (LA), 12 (4%) moderately dilated, 15 (6%) severely dilated and 191 (70%) had normal LA dimensions. Sixty-four patients (23%) had resting wall motion abnormalities, 86 (32%) had aortic valve disease, 137 (50%) had mitral valve disease (101 mild MR and 36 moderate/severe MR), 58 (21%) had tricuspid valve disease, and 22 (8%) had pulmonary valve disease. Four patients (1%) had a dilated aortic annulus, three patients (1%) had a dilated sinus of Valsalva, 36 patients (13%) had a dilated sinotubular junction, 80 patients (29%) had a dilated tubular ascending aorta, 26 patients (10%) had a dilated aortic arch, and 17 patients (6%) had a dilated descending aorta.

Electrocardiography

The resting ECG was performed 14±5 days before EVAR and was reported as pathological in 69 (25%) patients and signs of myocardial ischemia were found in 20 (7%) patients (Table 2).

Analyses

In unadjusted analysis, LV fractional shortening, LV ejection fraction, mitral E, pulmonary artery pressure, proportion of patients with a pulmonary artery pressure >35 mmHg, diameter of the aortic sinotubular junction, tubular ascending aorta, aortic arch, and presence of MR were parameters that significantly differed between survivors and non-survivors. Previous medical history and use of medication was broadly similar across all groups.

The unadjusted Kaplan-Meier curves for the cumulative incidence of long-term all-cause mortality, dichotomized according to the presence or absence of MR and low, intermediate and high-risk patients are presented in Figure 2 and Figure 3 respectively. The differences amongst these curves were significant (P<0.001). The all-cause mortality event rate for patients with no MR was 3% per year, increasing to 15% for those with mild MR, 16% with moderate MR and peaking at 26% in those with severe MR. With each increased degree of MR, there was a stepwise increase in the cumulative mortality (log rank χ^2 = 59.8; p<0.001). The all-cause mortality event rate for low risk patients was 1% per year, increasing to 8% for intermediate risk patients, and highest amongst those with high risk (16%). With each increased level of the co-morbidity index, there was a stepwise increase in the cumulative mortality attributable to co-morbid disease (log rank χ^2 = 44.7; p<0.001).

Following adjusted multivariable Cox regression, a greater tubular ascending aorta (HR 5.6, 95% CI 2.77-11.33, p<0.001), presence of MR (HR 8.13, 95% CI 4.09-12.16, p<0.001), lower left ventricular ejection fraction (HR 0.96, 95% CI 0.93-0.98, p<0.001), younger age (HR 0.97, 95% CI 0.95-0.99, p=0.005) and presence of diabetes (HR 1.46, 95% CI 1.24-1.89, p=0.021) were predictors of all-cause mortality (Table 3). When MR severity was added to

the multivariable model, mild MR (HR 4.84, 95% CI 2.8-13.92, p=0.002) and moderate/severe MR (HR 7, 95% CI 3.52-13.92, p<0.001) were predictors of mortality.

Discussion

This large observational study of selected patients undergoing elective EVAR has identified for the first time that parameters routinely measured on TTE are powerful predictors of long-term survival. In this study, the presence of MR, a greater tubular ascending aorta, a reduced LVEF, younger age, and presence of diabetes were independently associated with long-term all-cause mortality.

In keeping with previous studies,[24] peri-operative mortality was low in our study (1/273) with a 30-day mortality figure of 3%. As such, the use of TTE for peri-operative evaluation alone for patients undergoing elective EVAR is weak and this is supported by recent American Society of Echocardiography guidelines.[25] Long-term mortality within the current study was similar to previous research.[15]

One important finding is that MR when graded according to published guidelines[20] is a powerful predictor of clinical outcome. Indeed, the prognostic power superseded all other predictive parameters. Patients with any MR had an excess all-cause mortality that was more than eight times that compared to patients with no MR. In addition, increasing severity of MR had a progressively negative impact on survival with mild and moderate/severe MR being predictors of poor outcome. Mitral regurgitation is common and most patients are asymptomatic at diagnosis, which was previously believed to be a benign finding.[26] However, Enriquez-Sarano et al. (2005)[27] demonstrated that asymptomatic MR is a powerful predictor of death from any cause, death from cardiac causes and cardiac events. The management of asymptomatic patients with valve disease is an important medical problem[28] and the results of our study suggest that quantitative assessment of MR severity permits risk stratification of

patients undergoing EVAR. Recent guidelines on the management of valvular heart disease[29] suggest that mitral valve surgery is recommended in patients presenting with asymptomatic severe MR with decompensated left or right ventricular function and reasonable in patients presenting with asymptomatic severe MR with compensated ventricular function. However, in patients with MR present, regular surveillance is necessary since early surgery has been shown to have better outcomes compared to conservative approaches.[30] TTE is recommended for the initial evaluation of valvular heart disease[29] and due to its prognostic power should be included as part of the clinical decision making process for patients undergoing elective EVAR for AAA.

Disease of the aorta is an important cause of cardiovascular morbidity and mortality. Unless complications are life threatening, diseases of the aorta are asymptomatic and concealed on physical examination. As such, imaging tools are exclusively relied upon for diagnosis. There has been strong support in the literature for studying the abdominal aorta during conventional TTE,[31] with the prevalence of AAA during TTE ranging from 0.43% to 8.8%.[32-38] Our study demonstrated that dimensions of the proximal aorta on TTE, including the aortic sinotubular junction, tubular ascending aorta, and aortic arch were significantly different between alive versus deceased patients. In adjusted multivariable analysis, the tubular ascending aorta was an important independent discriminator of all-cause mortality. Patients with a greater tubular ascending aorta had an excess mortality that was more than five times that compared to patients with normal dimensions. Current guidelines on the management of patients with aortic dilatation have advised on intervention in combination with a bicuspid aortic valve or aortic regurgitation.[39] At present no recommendation exists based on aortic dilatation and MR severity. Future research is required to assess if prophylactic intervention to correct aortic dilatation and/or MR valve disease prior to EVAR for AAA improves outcome.

Previous research evaluating cardiovascular predictors of long-term mortality after EVAR for AAA[40] has failed to identify any TTE parameters as predictors of mortality. Instead, Ohrlander et al., (2011) demonstrated that ischemia on electrocardiography (HR 1.6, 95% CI 1.1-1.4, p=0.02) and anaemia (HR 1.5, 95% CI 1-2.1, p=0.05) were predictors of long-term mortality. More recently, any valve disease on TTE was a predictor of 1-year mortality (Odds Ratio 3.5, 95% CI 1.2-10.7; p=0.03) in a similar group of patients.[41] However, in this study only visual assessment of valvular heart disease was used, no measures of the aorta were noted, and not all patients underwent pre-operative TTE assessment. Other predictors of outcome following EVAR for AAA include pre-operative cardiac risk factors, length of hospital intensive care stay, ST-segment elevation myocardial infarction,[42] pre-operative aneurysm size,[43] renal disease,[41] and reduced statin use.[44, 45] In our study, these parameters were not significantly different between alive and deceased patients.

LVEF was a predictor of outcome. An impaired LV is an established risk factor for early cardiovascular morbidity and mortality. Interestingly, previous research has failed to show LVEF as a predictor of outcome in patients undergoing EVAR for AAA. However, in patients undergoing repair of the thoracic aorta, an impaired LV function was the strongest cardiac predictor of mortality (Odds Ratio 1.85, 95% CI 1.09-3.15; p=0.03).[46] A surprising finding in the current study was that younger age was independently associated with all-cause mortality. This finding reflects the fact that the youngest patients selected for EVAR are typically those with the greatest comorbidity, precluding them from open surgical repair. In addition, the presence of diabetes was independently associated with all-cause mortality. Diabetes is a recognised co-morbid disease that negatively influences outcome in patients with AAA.[11]

Our data support recent AAA guidelines,[11] which detail that patients considered medium to high-risk should be sent for cardiology review in order to examine cardiac risk with a view to optimise cardiac function, initiate cardio-protective medication before planned procedures and address behavioural CVD risk factors.[47] It is worth noting that although no significant differences existed between groups with regards to medication, use of cardio-protective medication was low and optimisation before surgery, such as beta-blockers and angiotensin converting enzyme inhibitors may improve outcome particularly in high-risk patients and those with heart failure.[48] In addition, it could be argued that high-risk patients also undergo counselling to ensure full comprehension of the risks of surgical intervention, bearing in mind that 43% of patients in the high-risk group were deceased at 2-years post-EVAR; it might be the case that there is an identifiably high-risk group of patients to whom surgical intervention should not be offered. By the same token, the intermediate risk group may warrant more aggressive optimisation and follow-up to ensure they experience the maximum survival benefit that aneurysm repair offers.

To our knowledge this is the first study that used systematic TTE in the clinical work up of patients prior to elective EVAR for AAA, incorporating quantitative measures of valvular heart disease and imaging of the aorta in conjunction with standard TTE parameters. This study has demonstrated that TTE provides prognostic information independent of traditional risk factors and likely serves as a useful test for guiding clinical treatment and management of patients.

Our study has limitations. This was a single centre study, which recruited selected patients for EVAR for AAA and there is the potential for referral bias. Due to difficulties in accurately determining the cause of death by reviewing death certificates or medical records,

all-cause mortality was selected as a more objective and unbiased end point. Patients recruited in our study had satisfactory pulmonary function and as such we do not have data to estimate the prognostic significance of pre-existing pulmonary disease. In addition, we did not record intensive care stay post EVAR and are therefore unable to estimate if this had an impact on patient outcome in this study. Notwithstanding these limitations, the present study extends our knowledge of risk stratification in patients undergoing EVAR for AAA, especially in the context of long-term survival. This is all the more important given that peri-operative and 30-day mortality has improved so significantly in the era of endovascular surgery.

Conclusion

Transthoracic echocardiography provides important long-term prognostic information in patients undergoing EVAR. These TTE indices were more important at predicting outcome than standard conventional risk factors in this patient group and may serve as a useful tool for guiding clinical management. A greater tubular ascending aorta, presence of MR, reduced left ventricular ejection fraction and younger age were independently associated with long-term mortality.

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Clinical Perspective

Abdominal aortic aneurysms (AAA) remain a significant health challenge and its prevalence significantly increases with age. Endovascular aneurysm repair (EVAR) has developed into a viable alternative to open surgery for AAA repair. Despite an established benefit in terms of peri-operative mortality for EVAR the long-term outcome in patients with AAA remains poor. Due to a strong association between traditional cardiovascular disease risk factors and higher incidence of AAA, guidelines recommend pre-operative risk stratification of patients. As a consequence, a transthoracic echocardiogram (TTE) is often advised as part of the clinical assessment of patients awaiting aneurysm repair despite a lack of evidence base. This study highlights that within the EVAR population there is a high-risk cohort of patients with 43% mortality at 2-years post procedure despite a successful endovascular repair. TTE provides powerful parameters that can predict long-term outcome in this patient group. The results require further prospective study, but suggest a potential role for more aggressive pre-operative optimisation in higher risk populations. The results may also provide a framework for better selection of patients who may benefit long-term from EVAR.

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Figure Legends

Figure 1: Study flow diagram.

Figure 2: Kaplan-Meier curve for the cumulative survival according to the presence or absence of mitral regurgitation.

Figure 3: Kaplan-Meier curve for the cumulative survival according to low, intermediate and high-risk.

Table 1. Characteristics of Patients Survived versus All-Cause Mortality		
Parameters	Survived (n = 195)	All-Cause Mortality (n = 78)
Demographics		
Age, y	73 ± 10.3	73.1 ± 11.8
Men	159 (81.5)	59 (75.6)
History		
Angiogram	28 (14.4)	7 (9)
Current Smoker	49 (25.1)	25 (32.1)
Diabetes Mellitus	23 (11.8)	11 (14.1)
Family History of Cardiovascular Disease	14 (7.2)	5 (6.4)
Heart Failure	2 (1)	1 (1.3)
Hypercholesterolemia	107 (54.9)	38 (48.7)
Hypertension	149 (76.4)	61 (78.2)
Prior Cerebrovascular Accident	23 (11.8)	12 (15.4)
Prior Coronary Artery Bypass Graft Surgery	1 (0.5)	0 (0)
Prior Ischaemic Heart Disease	70 (35.9)	27 (34.6)
Prior Myocardial Infarction	8 (4.1)	5 (6.4)
Prior PCI	2 (1)	1 (1.3)
Prior Smoker	91 (46.7)	35 (44.9)
Long-term medication		
All Lipid Lowering Drugs	162 (83.1)	66 (84.6)
Angiotensin-Converting Enzyme Inhibitors	52 (26.7)	24 (30.8)
Angiotensin II Receptor Antagonist	27 (13.8)	9 (11.5)
Antiarrhythmic Agents	12 (6.2)	4 (5.1)
Aspirin	128 (65.6)	50 (64.1)
Beta Blockers	69 (35.4)	26 (33.3)
Calcium antagonists	75 (38.5)	25 (32.1)
Diuretic	57 (29.2)	15 (19.2)
Nitrates	22 (11.3)	10 (12.8)
Wafarin	18 (9.2)	7 (9)
Laboratory values		
Hemoglobin ($g \cdot dl^{-1}$)	12.98 ± 1.9	14.01 ± 14.4
White Blood Cell Count ($K \cdot ul^{-1}$)	8.18 ± 2.9	8.14 ± 2.8
Platelet Count ($K \cdot ul^{-1}$)	253. 8 ± 97.3	262.7 ± 114.8
Sodium ($mmol \cdot l^{-1}$)	137.5 ± 4.3	134.4 ± 20
Potassium ($mmol \cdot l^{-1}$)	4.28 ± 0.4	6.52 ± 15.6
Urea ($mmol \cdot l^{-1}$)	7.35 ± 3.1	8.89 ± 5.3
Creatinine ($\mu mol \cdot l^{-1}$)	101.9 ± 54.1	124 ± 98.4
eGFR ($ml \cdot min \cdot 1.73m^2$)	66.4 ± 23.8	62.2 ± 26.3
eGFR <45 $ml \cdot min \cdot 1.73m^2$	29 (14.9)	16 (20.5)
Cardiac Troponin T ($\mu g \cdot l^{-1}$)	0.08 ± 0.02	2.29 ± 5.57
C-Reactive Protein ($nmol \cdot l^{-1}$)	50 ± 58.5	67 ± 87.6
Glucose ($mmol \cdot l^{-1}$)	6.2 ± 2.2	6.5 ± 1.6
Total Cholesterol ($mmol \cdot l^{-1}$)	4.14 ± 1.2	4.86 ± 1
Low Density Lipoprotein ($mmol \cdot l^{-1}$)	2.33 ± 0.9	2.98 ± 1.3
High Density Lipoprotein ($mmol \cdot l^{-1}$)	1.21 ± 0.4	1.27 ± 0.5

Note: PCI = Percutaneous coronary intervention; eGFR = Estimated glomerular filtration rate (calculated according to the Cockroft-Gault formular).

Table 2. Echocardiography and Electrocardiography Results of Patients Survived versus All-Cause Mortality		
Parameters	Survived (n=195)	All-Cause Mortality (n=78)
Transthoracic Echocardiography		
LV End Systolic Diameter (cm)	3.12±0.7	3.42±0.8
LV End Diastolic Diameter (cm)	4.85±0.6	5.04±0.7
LV Fractional Shortening (%)	36±9.6	32±8.7
LV Ejection Fraction (%)	62±10.9	53±9
Left Atrial Size (cm)	3.8±0.7	4±0.8
IVSd Diastole (cm)	1.06±0.2	1.06±0.2
LVPWd Diastole (cm)	1.04±0.2	1.01±0.2
LVM (g)	193.5±57.9	201.7±60.1
E ($m \cdot s^{-1}$)	0.65±0.2	0.71±0.3
A ($m \cdot s^{-1}$)	0.81±0.2	0.82±0.2
E/A	0.86±0.5	0.9±0.3
Pulmonary Artery Pressure (mmHg)	30.3±14.1	36.4±15.1
Pulmonary Artery Pressure >35 mmHg	50 (26)	34 (44)
Aortic Annulus (cm)	2.39±0.3	2.4±0.3
Aortic Sinus of Valsalva (cm)	3.59±0.4	3.59±0.4
Aortic Sinotubular Junction (cm)	3.18±0.4	3.4±0.5
Tubular Ascending Aorta (cm)	3.35±0.4	3.74±0.6
Aortic Arch (cm)	3.09±0.4	3.38±0.5
Descending Aorta (cm)	2.55±0.4	2.56±0.4
Resting Wall Motion Abnormality	40 (21)	24 (31)
Aortic Regurgitation	55 (28)	19 (24)
Aortic Stenosis	13 (7)	7 (9)
Mitral Regurgitation	70 (36)	67 (86)
Mitral Stenosis	4 (2)	1 (1)
Tricuspid Regurgitation	41 (21)	17 (22)
Pulmonary Regurgitation	16 (8)	6 (8)
Electrocardiography (ECG)		
Pathological ECG	46 (24)	23 (30)
Ischemic ECG	16 (8)	4 (5)

Note: LV = Left ventricular; IVSd = Interventricular septal diameter; LVPWd Left ventricular posterior wall diameter; LVM = Left ventricular mass;

Table 3. Multivariable Predictors of Long-term Mortality

Predictor		Hazard Ratio (95% Confidence Interval)	P Value
Demographics			
	Age, y	0.97 (0.95-0.99)	0.005
	Men	1.03 (0.55-1.92)	0.925
History			
	Diabetes Mellitus	1.46 (1.24-1.89)	0.021
	Prior Cerebrovascular Accident	2.11 (0.6-7.4)	0.245
Transthoracic Echocardiography			
	LV Ejection Fraction (%)	0.96 (0.93-0.98)	<0.001
	Pulmonary Artery Pressure >35 mmHg	1.38 (0.81-2.33)	0.234
	Aortic Sinotubular Junction (cm)	2.18 (0.86-5.54)	0.1
	Tubular Ascending Aorta (cm)	5.6 (2.77-11.33)	<0.001
	Aortic Arch (cm)	1.1 (0.97-1.14)	0.337
	Mitral Regurgitation	8.13 (4.09-12.16)	<0.001
Laboratory values			
	eGFR ($ml \cdot min \cdot 1.73m^2$)	1.63 (0.62-4.28)	0.319

Note: Models were selected using forward stepwise selection. Age and gender were forced into all models. LV = Left ventricular; eGFR = Estimated glomerular filtration rate (calculated according to the Cockroft-Gault formula).