Transthoracic Echocardiography Provides Important Long-Term Prognostic Information in Selected Patients Undergoing Endovascular Abdominal Aortic Repair

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Background—The value of performing transthoracic echocardiography (TTE) as part of the clinical assessment of patients awaiting endovascular repair of the abdominal aorta 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 endovascular aneurysm repair.

Methods and Results—This was a retrospective cohort study of 273 consecutive patients selected for endovascular aneurysm repair. All patients included in the 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 (hazard ratio [HR] 5.6, 95% confidence interval [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 mellitus (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 endovascular aneurysm repair. 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 mellitus were independently associated with long-term mortality. (Circ Cardiovasc Imaging. 2016;9:e003557. DOI: 10.1161/CIRCIMAGING.115.003557.)

Key Words: abdominal aortic aneurysm ■ aneurysm ■ diabetes mellitus ■ endovascular repair ■ transthoracic echocardiography

Abdominal aortic aneurysms (AAA) remain a significant health challenge with a reported prevalence of 1.4% in the United States, 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

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See Clinical Perspective

Despite an established benefit in terms of perioperative 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- and sex-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 preoperative risk stratification of patients with AAA to optimize patient care and prevent complications, including mortality.10,11 Preoperative cardiac assessment appears to be of most importance because 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

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Consequently, physicians routinely request specialized 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. Periprocedural mortality for EV AR is very low, but these patients are usually elderly with other comorbidities with a 5-year survival of 52%. The long-term prognostic value of TTE is unclear in the EV AR population. Therefore, the aim of this study was to assess preoperative echocardiographic predictors of long-term all-cause mortality in patients undergoing EV AR.

Methods

Study Cohort

The study population consisted of 273 consecutive patients (73±10.7 years) undergoing elective EV AR between January 2008 and September 2010 from a single tertiary center. Patients with ruptured aneurysms and trauma-related cases were excluded. All patients had a TTE and ECG 4 weeks before their EV AR 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. Left ventricular (LV) end diastolic diameter, LV end systolic diameter, 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 was then calculated according to the American Society of Echocardiography recommendations. LV fractional shortening was calculated from LV end diastolic diameter and LV end systolic diameter. LV ejection fraction (LVEF) was determined by the modified biplane Simpson’s rule, with measurements averaged over 3 cardiac cycles. The LV endocardial border was traced continguously from one side of the mitral annulus to the other, excluding the papillary muscles and trabeculations. LV regional wall motion was analyzed 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 visualized segment. For patients with poor endocardial border definition in ≥2 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 4-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 4-chamber view, early diastolic (Ea) velocities were measured. LV filling pressure was estimated from the mitral E/Ea ratio.

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. Full quantitative and semi-quantitative assessment of valvular disease was performed. 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 before EV AR. All tracings were interpreted by a trained physician and coded on the basis of the Minnesota coding criteria. 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. Left ventricular hypertrophy was defined according to Sokolow and Lyon voltage criteria. The ECG was graded as normal or pathological.

Endovascular Aneurysm Repair

All patients in this cohort were elective admissions for EV AR. Endografting was performed via percutaneous or surgical approach to the femoral artery. The EV AR procedure was performed to an Institutional standard operating protocol and undertaken in a dedicated endovascular imaging suite under fluoroscopic guidance. The endografts used were proprietary endografts predominantly from Cook Medical or Medtronic. Postoperative 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 ≤5 years post-surgery. Patients were followed up from the date of their EV AR 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 sex 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 Table 1 and Figure 1 in the Data Supplement 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 3 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).

Results

Clinical Outcome

The primary end point 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 9 deaths within 30 days post surgery (see Figure 1).
During the study period, 273 consecutive patients were evaluated using TTE imaging 18±7 days before elective EVAR. Mean age was 73±10.7 years with a greater proportion of male patients (80%) in keeping with the recognized male-preponderance of AAA. The prevalence of hypertension, hypercholesterolaemia, and diabetes mellitus 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% of patients had a prior cerebrovascular accident and 36% had previous evidence of ischemic heart disease. Seventy-four (27%) patients were current smokers and 126 (46%) ex-smokers with a combined pack year of 32.8±24.2. Seventy-two percent of patients (196/273) were treated with ≥1 antilipid medication, 9% (25/273) were warfarinized, and 82% (229/273) were taking lipid-lowering therapies. However, use of other cardioprotective medication, such as β-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 interobserver agreement in reporting echo parameters between the 2 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 an 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 LV end diastolic diameter. Fifty-five patients (20%) had a mildly dilated left atrium, 12 (4%) moderately dilated, 15 (6%) severely dilated, and 191 (70%) had normal left atrium 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, 3 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, and 10 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 nonsurvivors. Previous medical history and use of medication was broadly similar across all groups.

### Table 1. Characteristics of Patients Survived Versus All-Cause Mortality

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Survived (n=195)</th>
<th>All-Cause Mortality (n=78)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Laboratory values</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemoglobin, g/dL</td>
<td>12.9±1.9</td>
<td>14.01±14.4</td>
</tr>
<tr>
<td>White blood cell count, K/μL</td>
<td>8.18±2.9</td>
<td>8.14±2.8</td>
</tr>
<tr>
<td>Platelet count, K/μL</td>
<td>253.8±97.3</td>
<td>262.7±114.8</td>
</tr>
<tr>
<td>Sodium, mmol/L</td>
<td>137.5±4.3</td>
<td>134±20</td>
</tr>
<tr>
<td>Potassium, mmol/L</td>
<td>4.28±0.4</td>
<td>6.52±15.6</td>
</tr>
<tr>
<td>Urea, mmol/L</td>
<td>7.35±3.1</td>
<td>8.89±5.3</td>
</tr>
<tr>
<td>Creatinine, μmol/L</td>
<td>101.9±54.1</td>
<td>124±98.4</td>
</tr>
<tr>
<td>eGFR, mL·min⁻¹·1.73 m²</td>
<td>66.4±23.8</td>
<td>62.2±26.3</td>
</tr>
<tr>
<td>eGFR &lt;45 mL·min⁻¹·1.73 m²</td>
<td>29 (14.9)</td>
<td>16 (20.5)</td>
</tr>
<tr>
<td>Cardiac troponin T, μg/L</td>
<td>0.08±0.02</td>
<td>2.29±5.57</td>
</tr>
<tr>
<td>C-reactive protein, mmol/L</td>
<td>50±58.5</td>
<td>67±87.6</td>
</tr>
<tr>
<td>Glucose, mmol/L</td>
<td>6.2±2.2</td>
<td>6.5±1.6</td>
</tr>
<tr>
<td>Total cholesterol, mmol/L</td>
<td>4.14±1.2</td>
<td>4.86±1.2</td>
</tr>
<tr>
<td>Low-density lipoprotein, mmol/L</td>
<td>2.33±0.9</td>
<td>2.98±1.3</td>
</tr>
<tr>
<td>High-density lipoprotein, mmol/L</td>
<td>1.21±0.4</td>
<td>1.27±0.5</td>
</tr>
</tbody>
</table>

eGFR indicates estimated glomerular filtration rate (calculated according to the Cockcroft-Gault formula); and PCI, percutaneous coronary intervention.
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 among 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 ($\chi^2=59.8; P<0.001$). 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 comorbidity index, there was a stepwise increase in the cumulative mortality attributable to comorbid disease ($\chi^2=44.7; P<0.001$).

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 mellitus (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 mellitus were independently associated with long-term all-cause mortality.

In keeping with previous studies,24 perioperative mortality was low in our study (1/273) with a 30-day mortality figure of 3%. As such, the use of TTE for perioperative 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.35

One important finding is that MR when graded according to published guidelines26 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 $>8\times$ that compared with 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. MR
However, Enriquez-Sarano et al. demonstrated that asymptomatic mitral valve disease (MV) is a significant predictor of mortality (odds ratio 1.85, 95% CI 1.04–3.15; P = 0.03) in a similar group of patients. A surprising finding in the current study was that younger age was independently associated with all-cause mortality. Patients with a greater tubular ascending aorta had an excess mortality that was >5× that compared with 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. At present, no recommendation exists based on aortic dilatation and MR severity. Future research is required to assess whether prophylactic intervention to correct aortic dilatation and MR valve disease before EVAR for AAA improves outcome.

Previous research evaluating cardiovascular predictors of long-term mortality after EVAR for AAA 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 anemia (HR 1.5, 95% CI 1.2–2.1; 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. 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 preoperative TTE assessment. Other predictors of outcome after EVAR for AAA include preoperative cardiac risk factors, length of hospital intensive care stay, ST-segment elevation myocardial infarction, preoperative aneurysm size, renal disease, and reduced statin use. 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). 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 mellitus was independently associated with all-cause mortality. Diabetes mellitus is common, and most patients are asymptomatic at diagnosis, which was previously believed to be a benign finding. However, Enriquez-Sarano et al. 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, 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 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 because early surgery has been shown to have better outcomes compared with conservative approaches. TTE is recommended for the initial evaluation of valvular heart disease and because of 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 on for diagnosis. There has been strong support in the literature for studying the abdominal aorta during conventional TTE, with the prevalence of AAA during TTE ranging from 0.43% to 8.8%. 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 >5× that compared with 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. At present, no recommendation exists based on aortic dilatation and MR severity. Future research is required to assess whether prophylactic intervention to correct aortic dilatation and MR valve disease before EVAR for AAA improves outcome.

### Table 2. Echocardiography and Electrocardiography Results of Patients Survived Versus All-Cause Mortality

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Survived (n=195)</th>
<th>All-Cause Mortality (n=78)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV end systolic diameter, cm</td>
<td>3.12±0.7</td>
<td>3.42±0.8</td>
</tr>
<tr>
<td>LV end diastolic diameter, cm</td>
<td>4.85±0.6</td>
<td>5.04±0.7</td>
</tr>
<tr>
<td>LV fractional shortening, %</td>
<td>36±9.6</td>
<td>32±8.7</td>
</tr>
<tr>
<td>LV ejection fraction, %</td>
<td>62±10.9</td>
<td>53±8.9</td>
</tr>
<tr>
<td>Left atrial size, cm</td>
<td>3.8±0.7</td>
<td>4±0.8</td>
</tr>
<tr>
<td>IVSD diastole, cm</td>
<td>1.06±0.2</td>
<td>1.06±0.2</td>
</tr>
<tr>
<td>LVPWd diastole, cm</td>
<td>1.04±0.2</td>
<td>1.01±0.2</td>
</tr>
<tr>
<td>LVM, g</td>
<td>193.5±57.9</td>
<td>201.7±60.1</td>
</tr>
<tr>
<td>E, m/s</td>
<td>0.65±0.2</td>
<td>0.71±0.3</td>
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<tr>
<td>A, m/s</td>
<td>0.81±0.2</td>
<td>0.82±0.2</td>
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<tr>
<td>E/A</td>
<td>0.86±0.5</td>
<td>0.9±0.3</td>
</tr>
<tr>
<td>Pulmonary artery pressure, mm Hg</td>
<td>30.3±14.1</td>
<td>36.4±15.1</td>
</tr>
<tr>
<td>Pulmonary artery pressure &gt;35 mm Hg</td>
<td>50 (26)</td>
<td>34 (44)</td>
</tr>
<tr>
<td>Aortic annulus, cm</td>
<td>2.39±0.3</td>
<td>2.4±0.3</td>
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<tr>
<td>Aortic sinus of valsalva, cm</td>
<td>3.59±0.4</td>
<td>3.59±0.4</td>
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<tr>
<td>Aortic sinotubular junction, cm</td>
<td>3.18±0.4</td>
<td>3.4±0.5</td>
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<tr>
<td>Tubular ascending aorta, cm</td>
<td>3.35±0.4</td>
<td>3.74±0.6</td>
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<tr>
<td>Aortic arch, cm</td>
<td>3.09±0.4</td>
<td>3.38±0.5</td>
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<tr>
<td>Descending aorta, cm</td>
<td>2.55±0.4</td>
<td>2.56±0.4</td>
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<tr>
<td>Resting wall motion abnormality</td>
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<td>24 (31)</td>
</tr>
<tr>
<td>Aortic regurgitation</td>
<td>55 (28)</td>
<td>19 (24)</td>
</tr>
<tr>
<td>Aortic stenosis</td>
<td>13 (7)</td>
<td>7 (9)</td>
</tr>
<tr>
<td>Mitral regurgitation</td>
<td>70 (36)</td>
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<td>Mitral stenosis</td>
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<td>Tricuspid regurgitation</td>
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<td>Pulmonary regurgitation</td>
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<tr>
<td>Electrocardiography (ECG)</td>
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</tr>
<tr>
<td>Pathological ECG</td>
<td>46 (24)</td>
<td>23 (30)</td>
</tr>
<tr>
<td>Ischemic ECG</td>
<td>16 (8)</td>
<td>4 (5)</td>
</tr>
</tbody>
</table>

IVSD indicates interventricular septal diameter; LV, left ventricular; LVM, left ventricular mass; and LVPWd, left ventricular posterior wall diameter.
is a recognized comorbid disease that negatively influences outcome in patients with AAA.\textsuperscript{11}

Our data support recent AAA guidelines,\textsuperscript{11} which detail that patients considered medium- to high-risk should be sent for cardiology review to examine cardiac risk with a view to optimize cardiac function, initiate cardioprotective medication before planned procedures, and address behavioral CVD risk factors.\textsuperscript{47} It is worth noting that although no significant differences existed between groups with regards to medication, use of cardioprotective medication was low and optimization before surgery, such as β-blockers and angiotensin-converting enzyme inhibitors, may improve outcome particularly in high-risk patients and those with heart failure.\textsuperscript{48} In addition, it could be argued that high-risk patients also undergo counseling 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 optimization 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 before 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. Because of 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 whether 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 perioperative and 30-day mortality has improved so significantly in the era of endovascular surgery.

Conclusions

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.

Disclosures

None.

References

Society of Cardiovascular Computed Tomography, Society for Association, American Society of Nuclear Cardiology, Heart Failure Task Force, American Society of Echocardiography, American Heart Association Task Force on宜

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cal aortic aneurysm repair do not predict postopera-


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Abdominal aortic aneurysms remain a significant health challenge, and its prevalence significantly increases with age. Endovascular aneurysm repair has developed into a viable alternative to open surgery for abdominal aortic aneurysms repair. Despite an established benefit in terms of perioperative mortality for endovascular aneurysm repair, the long-term outcome in patients with abdominal aortic aneurysms remains poor. Because of a strong association between traditional cardiovascular disease risk factors and higher incidence of abdominal aortic aneurysms, guidelines recommend preoperative risk stratification of patients. As a consequence, a transthoracic echocardiogram 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 endovascular aneurysm repair population, there is a high-risk cohort of patients with 43% mortality at 2 years post procedure, despite a successful endovascular repair. Transthoracic echocardiography 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 preoperative optimization in higher risk populations. The results may also provide a framework for better selection of patients who may benefit long term from endovascular aneurysm repair.
Transthoracic Echocardiography Provides Important Long-Term Prognostic Information in Selected Patients Undergoing Endovascular Abdominal Aortic Repair
Jamie M. O’Driscoll, Sandeep S. Bahia, Angela Gravina, Sara Di Fino, Matthew M. Thompson, Alan Karthikesalingam, Peter J.E. Holt and Rajan Sharma

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Supplementary Table 1: Multivariate model goodness of fit and discrimination

<table>
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<tr>
<th>Number of Variables</th>
<th>Akaike Information Criterion</th>
<th>C-Statistic</th>
<th>Hosmer-Lemeshow Test</th>
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</thead>
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<tr>
<td>10</td>
<td>221</td>
<td>0.882 (0.84-0.92)</td>
<td>5.6 (p=0.694)</td>
</tr>
<tr>
<td>11</td>
<td>221</td>
<td>0.882 (0.84-0.92)</td>
<td>3.1 (p=0.927)</td>
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<tr>
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<td>6.2 (p=0.627)</td>
</tr>
<tr>
<td>13</td>
<td>225</td>
<td>0.88 (0.84-0.92)</td>
<td>6.6 (p=0.58)</td>
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<tr>
<td>14</td>
<td>227</td>
<td>0.88 (0.84-0.92)</td>
<td>5.9 (p=0.658)</td>
</tr>
<tr>
<td>15</td>
<td>228</td>
<td>0.88 (0.84-0.92)</td>
<td>7.2 (p=0.521)</td>
</tr>
</tbody>
</table>

Supplementary Figure 1: Receiver operating characteristic curve for model discrimination comparing 10, 11, 12, 13, 14, and 15 variables.