Journal Pre-proof

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PII: S1078-5884(23)00946-2

DOI: https://doi.org/10.1016/j.ejvs.2023.11.018

Reference: YEJVS 9042

To appear in: *European Journal of Vascular & Endovascular Surgery*

Received Date: 23 August 2023

Revised Date: 8 October 2023

Accepted Date: 14 November 2023

Please cite this article as: Dabravolskaité V, Aweys MM, Venermo M, Hakovirta H, Mufty H, Zimmermann A, Makaloski V, Meuli L, External Validation of a Prognostic Model for Survival of Patients With Abdominal Aortic Aneurysms Treated With Endovascular Aneurysm Repair, *European Journal of Vascular & Endovascular Surgery* (2023), doi: https://doi.org/10.1016/j.ejvs.2023.11.018.

External Validation of a Prognostic Model for Survival of Patients With Abdominal Aortic Aneurysms Treated With Endovascular Aneurysm Repair

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Short title: External validation of a prognostic model for survival of patients with AAA treated with EVAR

Keywords: Aortic aneurysm, Abdominal/surgery, Endovascular procedures/mortality, Predictive model, Risk factors, Proportional hazards models, Survival analysis

WHAT THIS PAPER ADDS

The validated prognostic model identifies a high risk subgroup of patients with asymptomatic abdominal aortic aneurysm (AAA) and a survival rate of only 16% at 10 years. The benefit of

endovascular aneurysm repair in these patients must be questioned as long as the AAA does not carry a relevant risk of rupture.

Objective: Current guidelines recommend diameter monitoring of small and asymptomatic abdominal aortic aneurysms (AAAs) due to the low risk of rupture. Elective AAA repair is recommended for diameters ≥ 5.5 cm in men and ≥ 5.0 cm in women. However, data supporting the efficacy of elective treatment for all patients above these thresholds are diverging. For a subgroup of patients, life expectancy might be very short, and elective AAA repair at the current threshold may not be justified. This study aimed to externally validate a predictive model for survival of patients with asymptomatic AAA treated with endovascular aneurysm repair (EVAR).

Methods: This was a multicentre international retrospective observational cohort study. Data were collected from four European aortic centres treating patients between 2001 and 2021. The initial model included age, estimated glomerular filtration rate (eGFR), and chronic obstructive pulmonary disease (COPD) as independent predictors for survival. Model performance was measured by discrimination and calibration.

Results: The validation cohort included 1 500 patients with a median follow up of 65 months, during which 54.6% of the patients died. The external validation showed slightly decreased discrimination ability and signs of overfitting in model calibration. However, a high risk subgroup of patients with impaired survival rates was identified: octogenarians with eGFR < 60 OR COPD, septuagenarians with eGFR < 30, and septuagenarians with eGFR < 60 and COPD having survival rates of only 55.2% and 15.5% at five and 10 years, respectively.

Conclusion: EVAR is a valuable treatment option for AAA, especially for patients unsuitable for open repair. Nonetheless, not all these patients will benefit from EVAR, and an

individualised treatment recommendation should include considerations on life expectancy. This study provides a risk stratification to identify patients who may not benefit from EVAR under the present diameter threshold.

INTRODUCTION

Current treatment guidelines of the European Society for Vascular Surgery (ESVS) recommend diameter monitoring of small and asymptomatic abdominal aortic aneurysms (AAAs) because the risk of rupture is very low.[1] Elective AAA repair is recommended for asymptomatic AAA with diameters ≥ 5.5 cm in men and ≥ 5.0 cm in women (class I, level A for men; class IIb, level C for women).[1–3] However, robust data on the efficacy of elective treatment of all patients with asymptomatic AAA with a diameter above this threshold are lacking, and the current level of evidence classification in men has been questioned.[4]

As the burden of comorbidities increases, elective AAA treatment becomes less effective or even futile in improving overall survival.[4] Thus, personalised decision making in patients with asymptomatic AAA could avoid unnecessary AAA treatments and reduce the overall morbidity and costs associated with AAA. Nonetheless, this requires a reliable assessment of the impending risk of aneurysm rupture, the risks related to elective repair, and life expectancy. Only a complete picture of all competing risks will ultimately enable the benefit of endovascular aneurysm repair (EVAR) to be assessed individually.[8]

The aim of the current study was to evaluate the overall survival of patients treated with EVAR and thereby provide information on all cause mortality, the main adversary to the efficiency of EVAR in comorbid patients. The survival of patients with asymptomatic AAA varies greatly after elective aneurysm repair.[5–7] Several patient characteristics and comorbidities have been associated with patient survival after elective AAA repair. Still,

predicting survival after elective AAA surgery to support personalised decision making is not yet established due to the lack of robust and validated tools.

A single centre predictive model for survival after EVAR for AAA identified age, the estimated glomerular filtration rate (eGFR), and chronic obstructive pulmonary disease (COPD) as independent predictors for long term survival.[9] The previously published temporal validation demonstrated good discrimination ability for five year survival in four risk groups.[7] The five year survival probabilities were 89% in "low risk" patients, 83% in "low to moderate risk" patients, 68% in "moderate to high risk" patients, and only 40% in "high risk" patients. The current study aimed to validate externally this predictive model on an international multicentre clinical cohort.

MATERIALS AND METHODS

This retrospective observational cohort study includes all consecutive patients treated with standard EVAR for asymptomatic AAA at four different European aortic referral centres: the university hospital of Zurich, Switzerland (2003 – 2020), the university hospitals of Turku and Helsinki, Finland (2010 – 2021 and 2002 – 2016, respectively), and the university hospital of Leuven, Belgium (2001 – 2019). Patients with complex EVAR, including fenestrated, branched, or parallel grafts, were excluded from this study. Further, all patients treated for symptomatic or ruptured aneurysms and other indications like penetrating aortic ulcers were excluded.

This study was conducted according to the Principles of the Declaration of Helsinki and reported in adherence to the TRIPOD statement (Transparent Reporting of a multivariable prediction model for Individual Prognosis or Diagnosis).[10] The local ethics committees in Bern and Zurich Switzerland approved this study (BASEC-IDs: 2022-00489 and 2021-02311), whereas the local committees in Turku, Helsinki, and Leuven waived approval of the study due to its retrospective nature.

Data collection and definitions

Patient characteristics were obtained from local records and aggregated at an individual patient level. Treatment indications were according to available ESVS guidelines from 2011 and 2018.[1,11] Prior to that, a threshold of 55 mm (50 mm for females), rapid progress (≥ 5 mm in six months), or saccular anatomy was used as indication criterion. Diameter measurements were extracted from recordings without consulting the available images.

The baseline characteristics of the validation cohort were summarised and compared to the original cohort. COPD was defined as any diagnosis of COPD at the time of operation or any forced expiratory volume < 80% of the predicted capacity on pre-operative spirometry. eGFR was calculated using the modification of diet in renal disease study formula using the last pre-operative creatinine value within 30 days.

The primary outcome measure of this study was model performance measured by discrimination and calibration for overall survival in the validation cohort. For patients treated at the university hospitals of Leuven, Zurich, and Turku, survival information was obtained from local hospital databases. All patients without a documented date of death by the predefined study end date, 31 October 2022, were contacted during a cross-sectional telephone survey between November 2022 and March 2023. For patients treated at the University of Helsinki, survival information was provided by the Statistics Finland Cause of Death registry. Completeness of follow up information was reported using the Follow-up Index.[12] Survival information was trimmed at the study end date.

This overall dataset formed the “validation cohort”. The previously published patient cohort treated at the university hospital of Bern formed the “original cohort” and was used for comparison.

Statistical analysis

Predictor selection

The variable selection process for the predictive score based on the original cohort has been described in detail.[9] In summary, pre-selection of variables was conducted based on a literature review to avoid a complete data driven variable selection. Thereafter, a machine learning method (least absolute shrinkage and selection operator with 10 fold cross-validation in a Cox model) was used for the variable selection for the predictive model. The predictive model identified age, eGFR, and COPD (see Supplementary Table S1).

Predictive score

The beta-coefficients of the Cox model were used to create an easy to use risk score.[7] Age was grouped into quartiles and rounded to the next integer for practical reasons. eGFR was grouped into quartiles according to the KDIGO classification, but G4 and G5 were merged. COPD was available as a binary variable only and thus formed two groups.

The beta coefficients for each variable group were multiplied by 10 and rounded to the nearest integer to create the score. This resulted in the following scoring for age: < 70 years = 0 points; 70 – 74.9 years = 9 points; 75 – 79.9 years = 10 points; ≥ 80 years = 17 points; for eGFR: KDIGO G1 = 0 points; G2 = 1 point; G3a = 3 points; G3b = 6 points; G4/5 = 15 pts; for COPD: if present = 7 points.

The total score of the three variables was formed, and the cohort was divided into quartiles compiling the four risk groups: ≤ 8 points, “low risk”; 9 – 13 points, “low to moderate risk”; 15 – 18 points, “moderate to high risk”; ≥ 19 points, “high risk.” Of note, no combination resulted in a sum score of 14 points. The risk score is provided in Supplementary Table S1 and is available online.[7]

Model discrimination and calibration

The discrimination ability of the model was tested on the validation cohort using Harrell's concordance statistics *C* with 95% confidence interval (95% CI) using DeLong's method.[13] Calibration was visually inspected and quantified using the jack-knife pseudo-value method. Discrimination and calibration of the predictive score were tested separately at five and 10 years. The same analysis was performed for the predictive model using age and eGFR as continuous rather than categorical variables. Further, the observed survival for each of the risk groups was compared between the original cohort and the validation cohort using the likelihood ratio test.

Further descriptive analysis

Continuous variables (i.e., age, eGFR, creatinine, body mass index, AAA diameter) were visually inspected for normality and summarised using the median and quartiles (Q1, Q3) since they were skewed. Factor variables were compared by chi squared test and continuous variables by the Kruskal–Wallis rank test. A Cox proportional hazard model was calculated for the validation cohort, including all available variables previously identified to be associated with survival to allow the inclusion of these data in future studies. For this analysis, multiple imputations were performed for missing comorbidities using the "mice" package. Predictive mean matching was used for continuous variables, multinomial logistic regressions were used to impute factor variables. The number of imputed datasets was $m = 25$. The proportional hazards assumption was tested and verified using scaled Schoenfeld residuals for each Cox model.

All statistical analyses were performed using R Studio version 4.2.3 on MacOS version 12.5.1.

RESULTS

The four study sites treated a total of 1 616 consecutive patients with asymptomatic AAA using EVAR during their respective inclusion periods. Information on COPD was unavailable in 87 patients and eGFR was missing in 29 patients, resulting in a final validation cohort of 1 500 patients. Baseline characteristics of the aggregated validation cohort prior to EVAR are summarised in Table 1 and compared with the original cohort. Supplementary Table S2 summarises the baseline characteristics stratified by centre. The baseline characteristics of the validation cohort were comparable with the original cohort. However, significant differences were found for two variables of the predictive model. Patients in the validation cohort had a diagnosis of COPD more often (31.1% *vs.* 23.7%, $p < .001$). Further, the eGFR was significantly higher in the validation cohort than in the original cohort (77.8 *vs.* 66.1 ml/min/1.73^2, $p < .001$).

The median overall follow up of the validation cohort was 65 (Q1, Q3 37, 101) months with almost complete follow up information (Follow-up Index 0.97). The overall survival times were identical among the two cohorts: 71% (95% CI 67 – 75%) in the original and 71% (68 – 73%) in the validation cohort; 39% (32 – 48) in the original and 39% (36 – 42%) in the validation cohort ($p = .70$), at five and 10 years, respectively. During the follow up, 54.6% ($n = 819$) of the patients in the validation cohort died, whereas 35.7% ($n = 197$) of the patients died in the original cohort. Further details on follow up and survival at one, five, and 10 years are presented in Table 2.

External validation of the predictive model

The external validation of the predictive model showed a slightly decreased discrimination ability compared with the previously reported model performance: Harrell's *C* 0.62 (95% CI 0.60 – 0.65) compared with 0.70 (0.66 – 0.75) on the original cohort. However, calibration was

186 excellent: the predicted and observed overall survival was 69.5% and 70.3% after five years
187 and 37.0% and 38.3% after 10 years, respectively. The calibration curves at five and 10 years
188 (Fig. 1) are slightly S shaped but very close to the perfect calibration reflected by the diagonal
189 line.
190 Figure 2 and Table 2 show the survival at five and 10 years by risk group, comparing the original
191 and external validation cohorts. There was no statistically significant difference in the
192 observed survival between the original cohort and the validation cohort in the "low risk" group
193 (p = .052), the "low to moderate-risk" group (p = .33), and the "moderate to high risk"
194 (p = .31). However, the observed survival was significantly better in the validation cohort
195 compared to the original cohort for the "high risk" group (p = .046). The five year survival in
196 the original cohort for "high risk" patients was 40% (95% CI 32 – 50%) and 55% (49 – 62%) in
197 the validation cohort. This demonstrates a slight but significant overfitting of the model for
198 "high risk" patients with significantly better survival than predicted.
199 A multivariable Cox proportional hazard model for survival after EVAR, including all
200 available variables in the dataset, is available in Supplementary Table S3.
201 ***EVAR cohort over time***
202 Figure 3 and Supplementary Figure S1 show changes in the risk scores and age groups of
203 patients treated for EVAR in the validation cohort. A steady increase in the proportion of
204 octogenarians can be seen from 2001, where only 8.8% were 80+ years old, to 2019, where
205 39.7% of the treated patients were octogenarians. Of note, there was a change in this pattern
206 with a decrease in the proportion of octogenarians and a decrease in the proportion of < 70
207 years old in the years 2020 and 2021 (Supplementary Figure S1).
208 The same observations were made for the burden of risk score. Patients were
209 healthier, in terms of the risk score, with only 3.9% high risk patients in 2001, whereas 32%

high risk patients in 2020. A steep drop in the proportion of high risk patients was documented for 2021, when only 11.1% were in the high risk group (Fig. 3).

DISCUSSION

This international, multicenter, external validation of a predictive model for the survival of patients with AAA treated with EVAR showed a modest reduction in the discriminatory ability, but excellent model calibration. The prognostic model confirmed a high risk subgroup of patients with a survival rate of only 55% at five and 16% at 10 years, respectively: octogenarians with eGFR < 60 *or* COPD, septuagenarians with eGFR < 30, and septuagenarians with eGFR < 60 *and* COPD. This contrasts with the excellent median life expectancy of the general population aged 80 in Switzerland, which in 2022 was 8.8 years for men and 10.4 years for women.[14] The benefit of EVAR in high risk patients must therefore be questioned as long as the AAA does not carry a relevant risk of rupture.

Nonetheless, a complete picture of the risk–benefit balance of EVAR in this patient cohort should consider the following three points: (1) the median AAA diameter was 58 mm, about 1 cm smaller than in the patients who had participated in the EVAR-2 study.[4] The diameter of high risk patients was significantly larger ($p < .001$, Supplementary Figure S2). However, 50% of all patients in the high risk group had AAA diameters < 60 mm, and approximately 25% had AAA diameters < 55 mm. The risk for AAA rupture might be lower than historical data suggested, but understanding the natural progression and rupture rates remains limited.[15–17] (2) The 30 day mortality rates after EVAR decreased but for these electively treated asymptomatic patients were still 1.1%.[5] (3) Like previous studies, this study shows that patients treated electively for an AAA have a relatively poor long term survival of only about 40% after 10 years.[5,18–20] Given this context, a substantial proportion of patients treated in this validation cohort may not have lived sufficiently long to realise the advantages

of this preventative treatment. Strict adherence to the current diameter threshold in high risk patients or even expanding the treatment criteria for AAA patients beyond the binary threshold of 55 mm diameter would enhance the quality of patient care. Of note, an association between initial AAA diameter and survival after elective EVAR has been previously described.[21] However, AAA was eliminated in the variable selection process as the magnitude of this association was not strong enough.[7,9] Still, this association was confirmed in the multivariable analysis of this cohort, HR 1.01 per millimetre AAA diameter increase (95% CI 1.00 – 1.02, $p < .001$); see Supplementary Table S3.

The decision regarding preventive treatment for asymptomatic AAA in elderly and/or severely comorbid patients is challenging. The long term results of the EVAR-2 trial demonstrated no increase in overall life expectancy for the EVAR group *vs.* the non-treated group.[6] Of the originally included 404 patients in the EVAR-2 trial, only 17% (69/404) survived more than eight years, and these patients were younger, with higher body mass index, higher eGFR, and better forced expiratory volume in 1 second at the time of enrolment.[6] The 10 year survival rate of the high risk cohort in the current study is comparable to the overall survival rate of patients from the EVAR-2 study. In contrast improvements in the peri-operative mortality rates have been achieved: a recent analysis from the American College of Surgeons National Surgical Quality Improvement Program of almost 25 000 patients undergoing EVAR between 2005 and 2013 showed a substantially lower 30 day mortality rate of 1.9% for high risk patients compared with the 7.3% reported by the EVAR-2 trial.[6,22] The peri-operative mortality rate in the high risk cohort was 2.2% and was comparable to these US data. Adkar *et al*. identified the presence of at least one criterion of impairment (respiratory, cardiac, or renal) or their combination as risk factors for 30 day mortality.[22] These risk factors for increased peri-operative mortality were comparable to the risk factors for long term survival

identified in this study. Hence, individuals with elevated peri-operative risk have a reduced life expectancy, prompting a need to scrutinise the advantages of elective AAA treatment based on the existing diameter threshold. Clinicians automatically and intuitively weigh different risks based on their experience and thus are likely to withhold EVAR in some patients. Diameters were already significantly larger in high-risk patients compared to the other risk groups ($p < .001$; Supplementary Figure S2). This study provides a risk stratification to support and improve such decisions in the future.

EVAR was initially invented as a less invasive alternative for patients unfit for open repair.[23] Figures 2 and 3 depict the initial application of EVAR in relatively healthier patients, while its extensive utilisation among elderly and more critically afflicted patients became predominant only during the last decade. This might be caused by a more restrictive use of EVAR in younger and healthier patients following long term results showing advantages for open repair.[5,24] A sharp decline in the proportion of high risk patients was documented for 2021, where only 11.1% were in the high risk group. This sharp decline falls within the COVID 19 pandemic and could be caused by reduced elective surgery capacity, especially for elderly and comorbid patients who are likely to require intermediate care or intensive care after treatment. It will be interesting to see if this trend continues in the years after COVID or if there is even a catch up effect.

The use of EVAR in low risk patients (< 70 years with eGFR ≥ 60, independent of COPD) who are expected to live longer is a separate topic of discussion. More than 60% of these patients will still be alive after 10 years and beyond, thus at risk for late complications.[5] Primary open repair may still be the preferred treatment option for these patients. The upcoming years will show if there will be a trend towards an open first strategy for young low risk patients.

Limitations

The model performance was validated and confirmed robust discrimination ability and excellent calibration to successfully identify a subset of high risk patients for impaired long term survival. The main limitations of this international multicentre external validation study is the retrospective extraction of routinely collected data which inherently carries a risk of bias: no routine pre-operative measurement of forced expiratory volume in 1 second was performed and COPD diagnosis was partly subjectively coded; furthermore, some heterogeneity and inconsistencies in AAA diameter measurement must be assumed over the two decades of the study period.

The calibration curve (Supplementary Figure S1a,b) and the survival plots show an S shaped model performance with better accuracy for the moderate risk groups. In contrast, the model slightly overestimates mortality in the high risk group and slightly underestimates mortality in the low risk group. This can be indicative for some degree of model overfitting or underfitting. Further validation of the model in cohorts with different case mix (i.e., higher or lower degree of comorbidities) is needed to better understand calibration in the extremes of the calibration curve.

In general, the clinical applicability of any predictive model in daily routine needs to be carefully assessed. The idea behind risk stratification with predictive tools is to support clinical decisions rather than drive them.

Conclusion

The role of EVAR as a valuable treatment option remains undisputed, especially for patients unsuitable for open repair. Nonetheless, not all these patients will benefit from EVAR, and an individualised treatment recommendation should include considerations on life expectancy.

This study provides a risk stratification to identify patients who may not benefit from EVAR under the present diameter threshold.

CONFLICTS OF INTEREST

None.

FUNDING

None.

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Table 1. **Baseline characteristics of the aggregated validation cohort prior to endovascular aneurysm repair compared with original cohort.**

Variable	**Original cohort** ***N* = 552**	**Validation cohort** ***N* = 1 500**	***p* value**
Male sex	503 (91.1)	1 370 (91.3)	.88
Age – *y*	76.0 (69.4, 80.6)	75.2 (69.3, 80.0)	.44
Arterial hypertension	458 (84.3)	1 194 (80.6)	.055
Missing	9	19	
Diabetes mellitus	108 (19.6)	252 (17.2)	.23
Missing	0	39	
Dyslipidaemia	467 (84.6)	1 061 (72.6)	<.001
Missing	0	38	
BMI – kg/m^2	27.0 (24.0, 30.0)	26.1 (24.0, 29.2)	.004
Missing	29	447	
Smoking	390 (73.3)	855 (60.6)	<.001
Missing	20	90	

COPD	131 (23.7)	470 (31.3)	<.001
eGFR – mL/min/1.73 m²	66.1 (51.0, 80.9)	77.8 (63.0, 87.5)	<.001
Creatinine – *mmol/L*	91 (77, 111)	93 (80, 112)	.036
PAD, Fontaine class			<.001
No PAD	436 (79.0)	496 (48.5)	
Fontaine I	49 (8.9)	486 (47.6)	
Fontaine II	46 (8.3)	38 (3.7)	
Fontaine III	21 (3.8)	0 (0.0)	
Fontaine IV	0 (0.0)	2 (0.2)	
Missing	0	478	
Coronary artery disease	308 (56.0)	746 (50.1)	.019
Missing	2	12	
Myocardial infarction	113 (20.6)	353 (31.7)	<.001
Missing	4	387	
Aneurysm diameter – *mm*	58.0 (54.0, 62.5)	58.0 (55.0, 65.0)	.014
Missing	25	7	

Continuous variables are presented by median (quartiles 1, 3). Counts are presented as *n* (%). Data were complete if not stated explicitly. Factor variables were compared by chi squared test, continuous variables by the Kruskal–Wallis rank test, respectively. BMI = body mass index; COPD = chronic obstructive pulmonary disease; eGFR = estimated glomerular filtration rate according to the "Modification of Diet in Renal Disease Study" (MDRD) in mL/min/1.73m^2; PAD = peripheral arterial disease as clinical stage according to the Fontaine classification.

Table 2. **Survival Information.**

Variable	Original *N* = 552	Validation *N* = 1 500	*p* value
Follow up – mo (Q1, Q3)	56 (23, 77)	65 (37, 101)	<.001
Follow up index	0.95	0.97	<.001
Number of deaths	197 (35.7)	819 (54.6)	n.a.
Overall survival			
30 days	98.7 (97.8–99.7)	98.9 (98.3–99.4)	.70
5 years	70.8 (66.7–75.2)	70.8 (68.4–73.2)	.70
10 years	39.2 (32.3–47.6)	38.7 (35.7–42.0)	.70
1 year survival by risk group			
Low risk	98.6 (96.6–100)	95.7 (93.8–97.7)	.052
Low to moderate	98.6 (96.8–100)	94.6 (92.5–96.8)	.33
Moderate to high	91.8 (87.0–96.8)	92.7 (90.1–95.3)	.31
High risk	81.6 (75.3–88.6)	90.6 (87.2–94.1)	.046
5 year survival by risk group			
Low risk	89.1 (83.7–94.9)	86.2 (82.7–89.8)	.052
Low to moderate	83.7 (77.4–90.6)	74.0 (69.8–78.5)	.33
Moderate to high	68.4 (59.5–78.6)	61.8 (56.9–67.2)	.31
High risk	39.9 (31.7–50.2)	55.2 (49.3–61.8)	.046
10 year survival by risk group			
Low risk	74.8 (63.5–88.1)	61.2 (55.6–67.5)	.052
Low to moderate	43.2 (30.1–62.0)	43.2 (37.6–49.6)	.33
Moderate to high	23.4 (12.3–44.8)	24.6 (19.6–30.8)	.31

High risk	9.6 (3.0–31.2)	15.5 (10.3–23.1)	.046

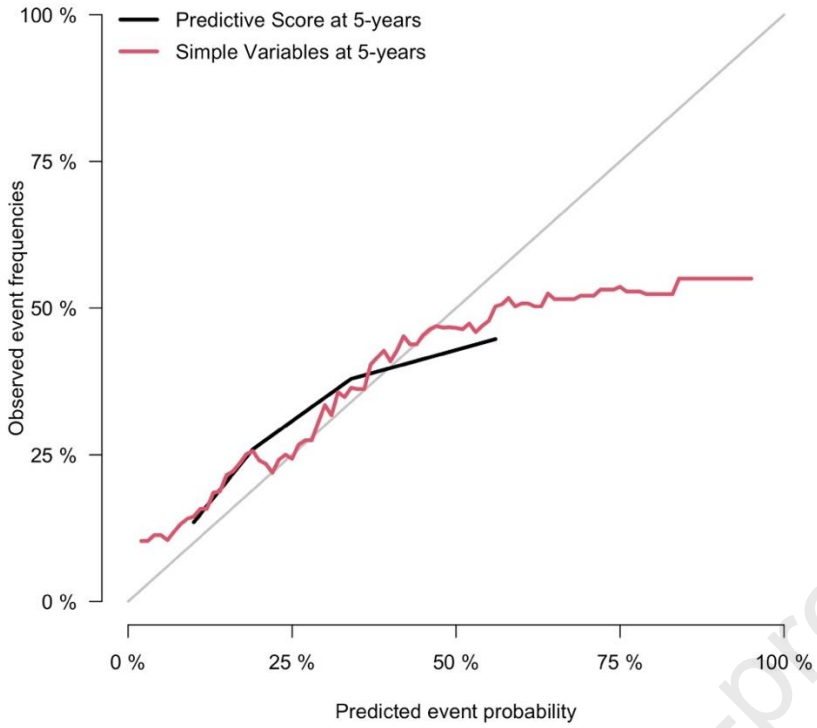
Data are presented as median (interquartile range), *n* (%), or % (95% CI), Median follow up time is presented with months and quartiles (Q1, Q3). Survival is presented overall and by risk group with percentages and 95% CI given by the Kaplan–Meier estimators. CI = confidence interval; n.a. = not available.

Figure 1. (A) Model calibration for survival at five years after endovascular aneurysm repair (EVAR). (B) Model calibration for survival at 10 years after endovascular aneurysm repair. Calibration curves of the Cox models at five years (Supplementary Figure S1a) and at 10 years (Supplementary Figure S1b). The black line reflects the performance of the risk score; the red line reflects the performance of a model using age and estimated glomerular filtration rate as continuous variables and chronic obstructive pulmonary disease as a binary variable.

Figure 2. (A) Survival at five years after endovascular aneurysm repair (EVAR). (B) Survival at 10 years after EVAR. Survival at five and 10 years after EVAR for the original and validation cohorts stratified by risk score groups and presented with a 95% confidence interval. A total of 819 deaths were observed in the validation cohort, which included 1 500 patients; a total of 197 deaths were observed in the original cohort, which included 552 patients.

Figure 3. Change in risk group over the study period. This figure shows the proportion of patients treated in four risk groups for the validation cohort.

Figure 1.



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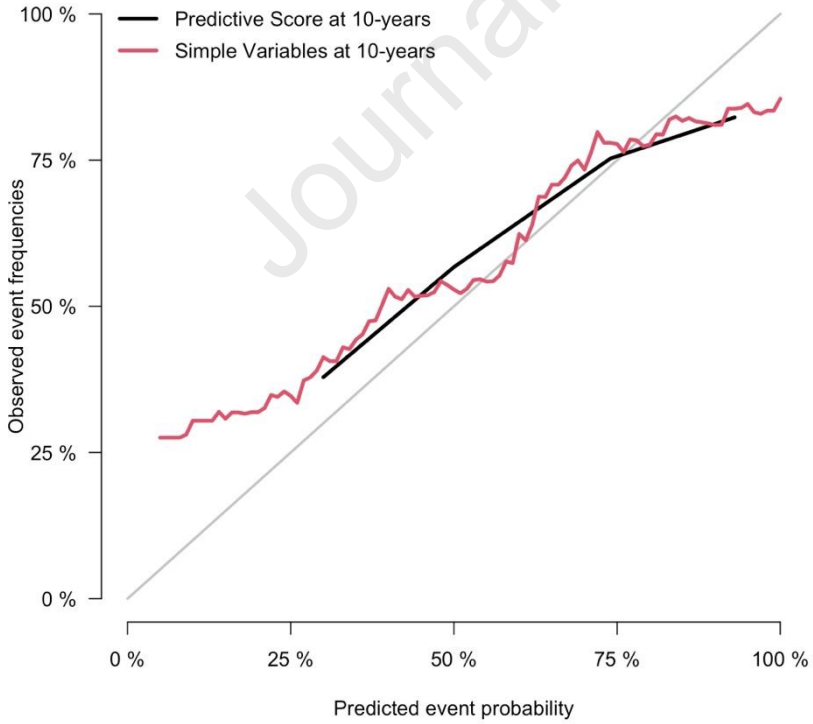
411 **Figure 1b**



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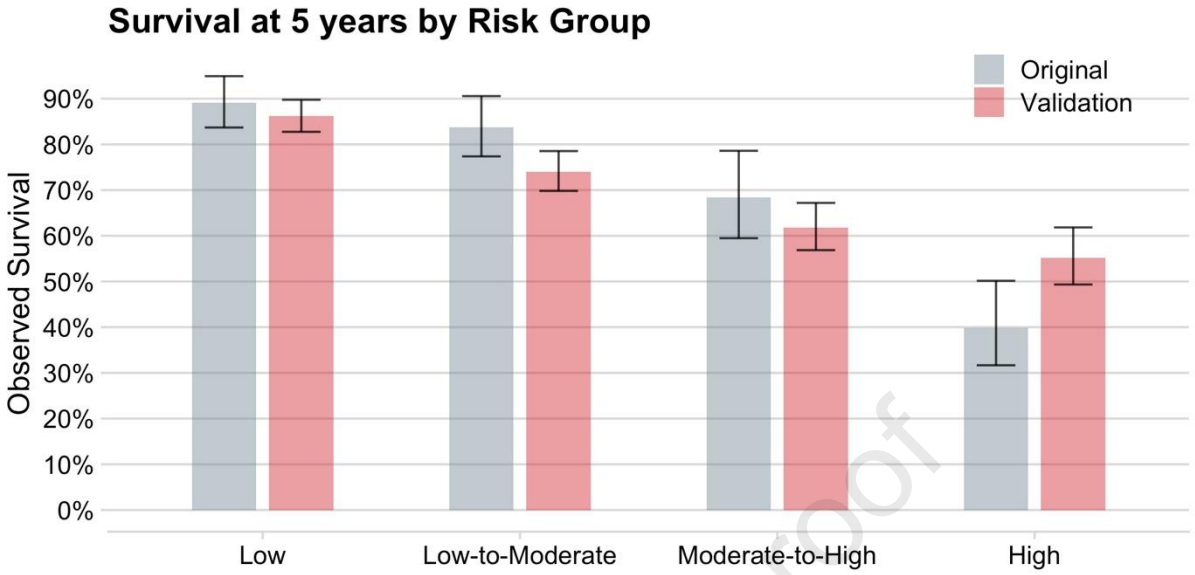
414 **Figure 2**. A.



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416 **B**

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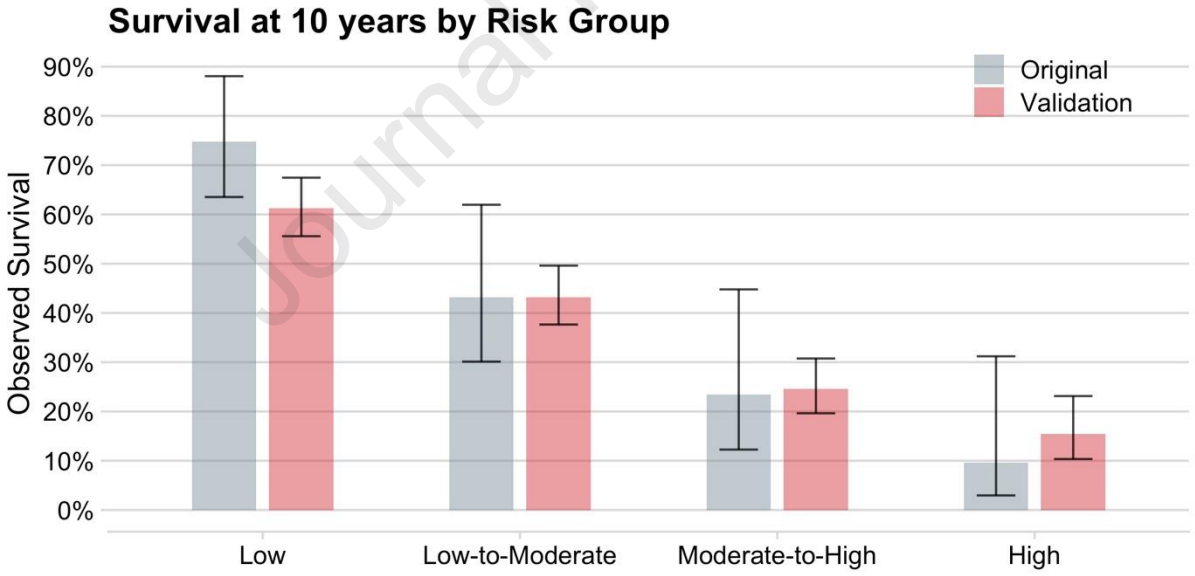

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420 **Figure 3**.

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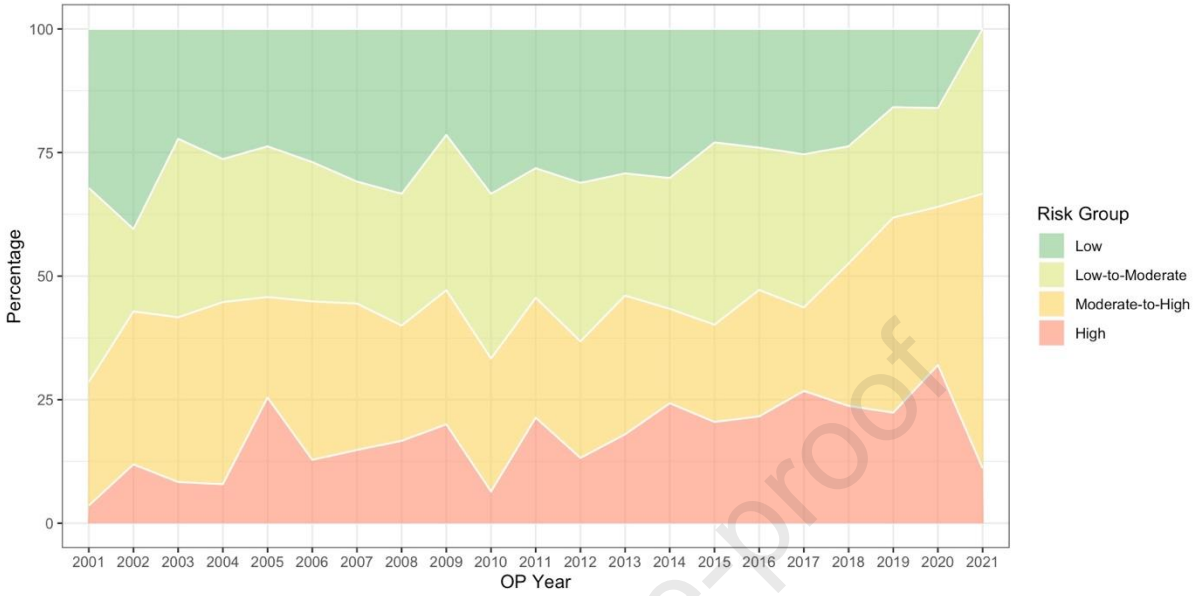

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