ASTR

ORIGINAL ARTICLE
pISSN 2288-6575 • eISSN 2288-6796
https://doi.org/10.4174/astr.2024.107.6.315
Annals of Surgical Treatment and Research

Validation of the Vascular Study Group of New England (VSGNE) risk prediction model for abdominal aortic aneurysm repair in Korea: a single-center retrospective study

Hyo-Shin Kim, Da-Hyun Kim, Dong-Ik Kim, Joon-Kee Park, Shin-Seok Yang, Yang-Jin Park

Division of Vascular Surgery, Department of Surgery, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Korea

Purpose: The Vascular Study Group of New England (VSGNE) risk prediction model is a simple method for estimating risk for elective abdominal aortic aneurysm (AAA) repair. The model considers both treatment methods and the physical characteristics of the aneurysm type as well as comorbidities. This research aimed to validate its effectiveness by analyzing retrospective data on Korean patients.
Methods: Our single-center retrospective analysis included 1,227 patients who underwent elective open repair surgery (ORS) or endovascular aortic repair (EVAR) from 2005 to 2021. We assessed the discrimination of the risk score and the effects of several risk factors.
Results: Most patients (66.7%) were classified as low risk in the model, with only 5.6% considered high risk. The mean risk score was 2.81, significantly lower than reported in previous studies. The actual 30-day mortality was only 0.7%, less than the predicted 1.1%. The accuracy of the model in predicting 30-day mortality was statistically significant (area under the curve, 0.822). Patients with high scores were associated with significantly increased mortality (odds ratio, 3.9; $P < 0.001$). Factors such as advanced age, cerebrovascular disease, and elevated creatinine levels were influential in mortality outcomes. However, a significant difference was not found in short-term mortality between ORS and EVAR.
Conclusion: Although the VSGNE model is an objective tool for assessing death risk in elective AAA repair, the actual risk scores in our patient population were lower than predicted. To create a more representative tool for the Korean population, we suggest developing a novel model based on multicenter data collection.
[Ann Surg Treat Res 2024;107(6):315-326]

Key Words: Abdominal aortic aneurysms, Calibration, In hospital mortality, Risk score, ROC curve, Validation

INTRODUCTION

Elective open repair surgery (ORS) and endovascular aortic repair (EVAR) are management options for asymptomatic but huge abdominal aortic aneurysms (AAAs). Asymptomatic AAA incidence is increasing due to screening for high-risk patients and worldwide aging [1]. Therefore, it is essential to determine treatment by predicting rupture risk, evaluating independent surgery risk factors, and objectively assessing life expectancy while performing surveillance.

Received June 19, 2024, **Revised** August 19, 2024,
Accepted September 18, 2024

Corresponding Author: Yang-Jin Park
Division of Vascular Surgery, Department of Surgery, Samsung Medical Center, Sungkyunkwan University School of Medicine, 81 Irwon-ro, Gangnam-gu, Seoul 06351, Korea
Tel: +82-2-3410-1782, **Fax:** +82-2-3410-3799
E-mail: yjpark1974@gmail.com
ORCID: https://orcid.org/0000-0001-8433-2202

Risk prediction tools, such as the Glasgow Aneurysm Score (GAS) in which the primary focus is ruptured aneurysms, have been introduced [2]. Subsequent advancements led to the development of more accurate risk prediction models such as the V-POSSUM (Vascular-Physiological and Operative Severity Score for the enUmeration of Mortality and Morbidity), the Medicare model, and Vascular Governance North West (VGNW) [3]. However, a notable drawback of these models is their reliance on complicated calculations that require dedicated websites to facilitate the computation process. In addition, there is a limitation of external validation. Eslami et al. [4] created the Vascular Study Group of New England (VSGNE) cohort-based risk predictive model, a more accessible, simpler, and parsimonious model for predicting the mortality of elective operable AAA patients. Because the VSGNE reflects treatment options, anatomical features, and the underlying risk factors of patients, the model is useful for predicting short-term risk in actual clinical practice. In addition, internal validation showed that the VSGNE can be used to predict in-hospital mortality more accurately than other scoring models (GAS, Medicare, VGNW). The VSGNE was introduced in the Society for Vascular Surgery guidelines in 2018, recently was recognized as an external validation for the Vascular Quality Initiative (VQI) database [5], and currently is used for risk stratification [1,6].

Recently, the prevalence of AAAs has notably increased on a national scale in Korea. Furthermore, a significant increase in the percentage of unruptured elective AAA repair cases has been observed. In addition, the average count of total AAA cases has been high in major metropolitan areas, reflecting the influence of accessibility to treatment [7].

It is important to identify high-risk patients and to focus on this in experienced high-volume centers in metropolitan areas. This approach can provide insight into national trends. The primary objective of this study is to apply the VSGNE to actual patients to assess its ability to predict short-term risk. In addition to identifying and comparing risk factors incorporated in the scoring schema, we aim to evaluate factors that impact short-term mortality.

METHODS

Ethics Statement

This study was approved by the Institutional Review Board of Samsung Medical Center in Seoul, Korea (No. 2023-11-076). The need for informed consent was waived due to the retrospective nature of the study.

Study design and patient selection

A retrospective analysis was conducted on patients who underwent elective repair for AAA (EVAR or ORS) from January 2005 to December 2021 at Samsung Medical Center in Seoul, Korea. To ensure precision of the scoring system, comparable with the VSGNE prediction model, only degenerative AAAs were considered. Aneurysm locations were limited to the abdominal region, including suprarenal, juxtarenal, and infrarenal areas, and the isolated iliac artery.

Patients were excluded from the analysis if they underwent nonelective AAA repair due to a ruptured aneurysm or other etiology or previous aortic surgery or if they lacked essential information regarding age, sex, or type of procedure. Furthermore, those who had undergone supraceliac or thoracoabdominal AAA repairs were excluded.

Patient data collection and study endpoint

We collected comprehensive demographic and clinical data for each patient, including age, sex, body mass index (BMI), medical comorbidities, baseline serum creatinine level, family history of degenerative aortic aneurysm, and the maximal anterior-posterior (AP) diameter of the AAA sac measured in centimeters. Chronic obstructive pulmonary disease (COPD) was defined as a moderate obstructive pattern observed on preoperative pulmonary function tests or ongoing nebulizer treatment for COPD. We also included patients with diabetes mellitus and myocardial and cerebrovascular diseases. The presence of congestive heart failure was determined based on medical records and preoperative echocardiography.

The risk score was calculated using the criteria proposed by Eslami et al. [6]. Points were assigned as follows: treatment: EVAR (0 points), ORS (infrarenal, 2 points), ORS (suprarenal, 4 points); aneurysm size ≥65 mm (1 point); age ≥75 years (1 point); sex: male (0 points), female (1 point); comorbidities: myocardial disease (1 point), cerebrovascular disease (1 point), COPD (2 points); creatinine ≥1.5 mg/dL (1 point).

The endpoints of this study were 30-day and 1-year mortality. The VSGNE model discrimination and calibration analysis were employed, and 1-year mortality was used for risk factor analysis of short-term mortality. The follow-up period for mortality analysis extended until March 31, 2023.

Statistical analysis

Baseline analysis

We compared the clinical characteristics of patients undergoing either EVAR or ORS and clamping site (infrarenal clamping *vs.* suprarenal clamping). Continuous variables were compared using the Student t-test and categorical variables with the chi-square test.

Discrimination and calibration analysis for the VSGNE risk prediction model

After the score distribution was identified, we compared it with that of the VSGNE cohort sample and other external

validation studies. Using 30-day and 1-year all-cause mortality as endpoints, we constructed the receiver operating characteristics curve (ROC) to obtain the area under the curve (AUC) for the VSGNE score with a 95% confidence interval. Utilizing Youden's index, we calculated the cutoff value for the VSGNE score associated with both 30-day and 1-year mortality. Furthermore, we corrected the reference value of the score risk group (low, medium, and high risk) based on data distribution.

To assess the performance of the model within the dataset, we stratified patients into 5 risk groups based on the VSGNE scoring system. Subsequently, we created a plot comparing predicted mortality with observed mortality, where perfect prediction aligned with the X = Y line.

Risk factor analysis

Multiple logistic regression analysis was utilized to assess the association between factors included in the VSGNE risk prediction model and short-term mortality. The significance of the regression model was determined using the Hosmer-Lemeshow goodness-of-fit test and a backward elimination procedure. The estimated coefficients from the logistic regression were exponentiated to obtain the odds ratios (ORs) for the predictor variables. Covariables such as diabetes mellitus, smoking history, congestive heart failure, and BMI were included as well as factors in the scoring model. The patients were stratified into low-, medium-, and high-risk score groups. The relevance of these risk stratifications was validated using univariate analysis, which minimized confounding by other variables included in the model. A multiple Cox regression model was used to further investigate the effects of these factors on mortality over time. Survival distributions were compared using the log-rank test. Survival rates across the risk-stratified groups were compared using Kaplan-Meier analysis, examining both 30-day and 1-year mortality.

R statistical software ver. 3.6.1 (The R Foundation) was used to perform all analyses, and significance was defined as a P-value of <0.05.

RESULTS

Baseline demographics

A total of 1,227 elective patients were treated for AAA over 17 years, 616 with EVAR and 611 with ORS. The mean age was 71.21 ± 7.78 years, 383 were >75 years of age (31.2%), and males were predominant (n = 1,061, 86.5%). Approximately 20% of patients had COPD; only 11 patients (0.9%) had congestive heart failure based on medical records and preoperative echocardiography. However, the proportion of patients with any angina event and coronary angiography or percutaneous coronary intervention history was 33.3%. Among subjects, 12.8% had cerebrovascular disease and 12.6% had baseline creatinine level >1.5 mg/dL. Regarding anatomical aneurysm features, the mean maximal sac size was 5.56 ± 1.08 cm; huge aneurysm (>6.5 cm) accounted for 17.2%.

Patients in the EVAR group were older, but a significant difference was not found in the group aged >75 years. In addition, more patients in the EVAR group were male and used statins preoperatively. Furthermore, basal creatinine level >1.5 mg/dL was more frequent in the ORS group than in the EVAR group (15.2% *vs.* 9.9%, P = 0.005), and the ORS group had more prevalent huge aneurysms (≥6.5 cm; 26.4% *vs.* 8.4%, P < 0.001). Significant differences were not observed in other underlying diseases or short-term mortality between EVAR and ORS groups (Table 1).

In total, the 30-day mortality was 0.7% (9 patients), the 1-year mortality was 3.7%, and other all-cause mortality was 33.5% in the follow-up period. The ORS group had a higher rate of all-cause mortality during the follow-up period (the mortality category in Table 1). However, the rate of basal creatinine >1.5 mg/dL was high in the group with suprarenal artery clamping, and 30-day mortality was higher in the suprarenal artery clamping group (3.4%); there was no significant difference in 1-year mortality based on clamping site (Table 2).

Discrimination and calibration of the VSGNE prediction model

The mean VSGNE model score among all patients was 2.81 (standard deviation, 2.08), lower than that of the VSGNE sample population of 3.18 (Fig. 1A). Notably, the distribution pattern was similar to that of the VSGNE sample population. Most patients (n = 980, 79.9%) scored within the range of 1–4, and only 3 patients (0.2%) were classified into the prohibitory high-risk group (Fig. 1B).

The ROC curve showed that the VSGNE model was effective at predicting 30-day mortality, with an AUC of 0.822. However, the performance of the model was lower for predicting 1-year mortality, with an AUC of 0.596. The cutoff value for predicting short-term mortality was 3.5 for all patients, while the EVAR and ORS groups had different cutoffs of 2.5 and 3.5, respectively, for 1-year mortality. Using the determined cutoff value and considering score distribution, the risk stages based on the score suitable for the study group were categorized as low (1–3 points), moderate (4–6 points), and high (≥7; Fig. 2).

Based on the VSGNE score for all patients, including the EVAR and ORS groups, the mean predicted mortality was overestimated compared with the actual 30-day mortality (1.1% *vs.* 0.7%) (Fig. 3A). There was a notable discrepancy in the ORS group; observed mortality was 1.10% while predicted mortality was 1.9%. Conversely, observed mortality in the EVAR group showed good agreement with the predicted value. In addition, when stratified based on the American Society of Anesthesiologists physical status classification, the predicted

Table 1. Baseline demographics of the EVAR *vs.* ORS groups

Characteristic	Overall	EVAR group	ORS group	p-value
No. of patients	1,227	616	611	
Age (yr)	71.21 ± 7.78	72.10 ± 7.1	70.31 ± 8.29	<0.001
<75	844 (68.8)	411 (66.7)	433 (70.9)	0.117
≥75	383 (31.2)	205 (33.3)	178 (29.1)	
Sex				
Male	1,061 (86.5)	554 (89.9)	507 (83.0)	<0.001
Female	166 (13.5)	62 (10.1)	104 (17.0)	
Body mass index (kg/m^2)	24.54 ± 3.34	24.72 ± 3.24	24.36 ± 3.44	0.03
<25	694 (56.5)	336 (54.5)	358 (58.6)	0.16
≥25	533 (43.5)	280 (45.5)	253 (41.4)	
Smoking				
Never	664 (54.1)	341 (55.4)	323 (52.9)	0.381
Current and prior	563 (45.9)	275 (44.6)	288 (47.1)	
Diabetes mellitus				
Yes	250 (20.4)	131 (21.3)	119 (19.5)	0.436
No	977 (79.6)	485 (78.7)	492 (80.5)	
Dyslipidemia (statin use)				
Yes	699 (57.0)	379 (61.5)	320 (52.4)	0.001
No	528 (43.0)	237 (38.5)	291 (47.6)	
COPD				
Yes	256 (20.9)	147 (23.9)	109 (17.8)	0.009
No	971 (79.1)	469 (76.1)	502 (82.2)	
Congestive heart failure				
Yes	11 (0.9)	6 (1.0)	5 (0.6)	0.774
No	1,216 (99.1)	610 (99.0)	606 (99.4)	
Myocardial disease				
Yes	408 (33.3)	193 (31.3)	215 (35.2)	0.152
No	819 (66.7)	423 (68.7)	396 (64.8)	
Cerebrovascular				
Yes	157 (12.8)	80 (13.0)	77 (12.6)	0.840
No	1,070 (87.2)	536 (87.0)	534 (87.4)	
Creatinine (mg/dL)	1.17 ± 0.83	1.14 ± 0.85	1.2 ± 0.81	0.120
0–1.5	1,072 (87.4)	554 (90.1)	518 (84.8)	0.005
>1.5	154 (12.6)	61 (9.9)	93 (15.2)	
Family history				
Yes	8 (0.7)	3 (0.5)	4 (0.7)	0.565
No	1,219 (99.3)	613 (99.6)	607 (99.3)	
Maximal AAA sac diameter (cm)	5.56 ± 1.08	5.41 ± 0.86	5.71 ± 1.25	<0.001
<6.5	1,014 (82.6)	564 (91.6)	450 (73.6)	<0.001
≥6.5	213 (17.4)	52 (8.4)	161 (26.4)	
Mortality				
30-day	8 (0.7)	2 (0.3)	7 (1.1)	0.092
1-year	45 (3.7)	24 (3.9)	21 (3.4)	0.669
All-cause	411 (33.5)	182 (29.5)	229 (37.5)	0.003

Values are presented as number only, mean ± standard deviation, or number (%).
EVAR, endovascular aortic repair; ORS, open repair surgery; COPD, chronic obstructive pulmonary disease; AAA, abdominal aortic aneurysm.

mortality was consistently overestimated compared with the actual mortality for all groups (Fig. 3B).

When total patient scores were stratified into quintiles for model calibration, observed mortality closely corresponded to predicted 30-day mortality. However, in cases of extremely high scores (≥9 points), observed mortality was lower than predicted mortality. In contrast, the actual 1-year mortality generally exceeded the predicted value, except for the group with the highest score, where observed mortality was lower than predicted (Fig. 4).

Table 2. Baseline characteristics of ORS group

Characteristic	ORS group (n = 611)	Clamping site		
		Infrarenal (n = 522)	Suprarenal (n = 89)	p-value
Age (yr)	70.31 ± 8.29	70.06 ± 8.46	71.8 ± 7.22	0.067
<75	433 (70.9)	376 (72.0)	57 (64.0)	0.125
≥75	178 (29.1)	146 (28.0)	32 (36.0)	
Sex				
Male	507 (83.0)	437 (83.7)	70 (78.7)	0.240
Female	104 (17.0)	85 (16.3)	19 (21.3)	
Body mass index (kg/m^2)	24.36 ± 3.44	24.27 ± 3.11	24.89 ± 4.95	0.113
<25	358 (58.6)	213 (40.8)	41 (45.5)	0.413
≥25	253 (41.4)	309 (59.2)	48 (54.5)	
Smoking				
Never	323 (52.9)	278 (53.3)	45 (50.6)	0.638
Current and prior	288 (47.1)	244 (46.7)	44 (49.4)	
Diabetes mellitus				
Yes	119 (19.5)	98 (18.8)	21 (23.6)	0.288
No	492 (80.5)	424 (81.2)	68 (76.4)	
Dyslipidemia (statin use)				
Yes	320 (52.4)	264 (50.6)	56 (62.9)	0.031
No	291 (47.6)	258 (49.4)	33 (37.1)	
COPD				
Yes	109 (17.8)	92 (17.6)	17 (19.1)	0.737
No	502 (82.2)	430 (82.4)	72 (80.9)	
Congestive heart failure				
Yes	5 (0.6)	5 (1.0)	0 (0)	0.353
No	606 (99.4)	516 (99.0)	89 (100)	
Myocardial disease				
Yes	215 (35.2)	184 (35.2)	31 (34.8)	0.939
No	396 (64.8)	338 (64.8)	58 (65.2)	
Cerebrovascular				
Yes	77 (12.6)	61 (11.7)	16 (18.0)	0.098
No	534 (87.4)	461 (88.3)	73 (82.0)	
Creatinine (mg/dL)	1.2 ± 0.81	1.18 ± 0.82	1.30 ± 0.76	0.196
0–1.5	518 (84.8)	449 (86.0)	69 (77.5)	0.049
>1.5	93 (15.2)	73 (14.0)	20 (22.5)	
Family history				
Yes	4 (0.7)	4 (0.8)	0 (0)	0.407
No	607 (99.3)	518 (99.2)	89 (100)	
Maximal AAA sac diameter (cm)	5.71 ± 1.25	5.69 ± 1.23	5.89 ± 1.31	0.158
<6.5	450 (73.6)	384 (73.6)	66 (74.2)	0.906
≥6.5	161 (26.4)	138 (26.4)	23 (25.8)	
Mortality				
30-day	7 (1.1)	4 (0.8)	3 (3.4)	0.067
1-year	21 (3.4)	16 (3.1)	5 (5.6)	0.212
All-cause	229 (37.5)	201 (38.5)	28 (31.5)	0.236

Values are presented as mean ± standard deviation or number (%).
ORS, open repair surgery; COPD, chronic obstructive pulmonary disease; AAA, abdominal aortic aneurysm.

Risk factor analysis

In the logistic regression analysis, age >75 years, cerebrovascular disease, and creatinine level ≥1.5 mg/dL were significant predictors of short-term mortality. The association between myocardial disease and mortality was not significant, with an observed OR of 0.54 (P = 0.26). Preoperative statin use was protective, reducing the risk of 1-year mortality (OR, 0.45, P = 0.017). In contrast, a BMI <25 kg/m^2 was associated with an increased risk of mortality (OR, 2.7, P = 0.015). These patterns persisted in the Cox regression model (Fig. 5A).

In the EVAR group, advanced age was not a risk factor, while sac size >6.5 cm had an OR for risk of 3.7 (P = 0.02). In the

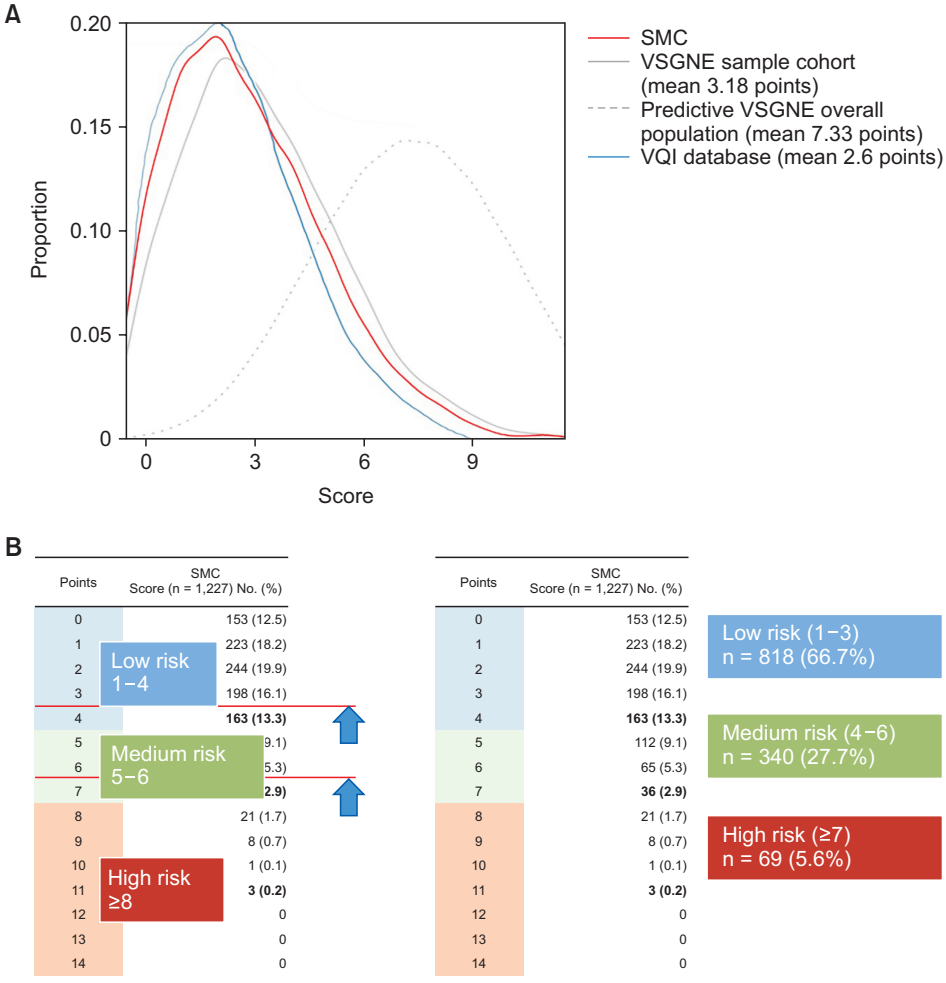


Fig. 1. (A) Score distributions of Samsung Medical Center (SMC, red line) and Vascular Study Group of New England (VSGNE) sample cohorts (gray line). The score distribution of the cohort sample used by Eslami et al. [6] in designing their risk prediction model and predictive distribution of the all-cohort population (dotted line). Distribution in the Vascular Quality Initiative (VQI) database for estimated external validation by Eslami et al. [5] (blue line). (B) Risk groups divided into low risk (1–3 points), medium risk (4–6 points), and high risk (≥7 points) based on score distribution and mortality cutoff (the cutoff value is shown in Fig. 3).

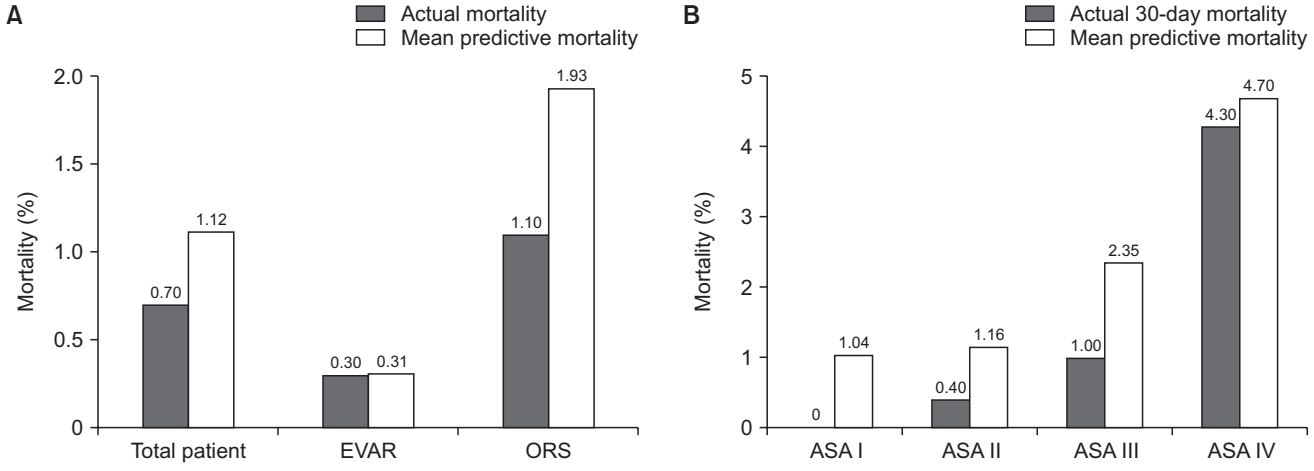
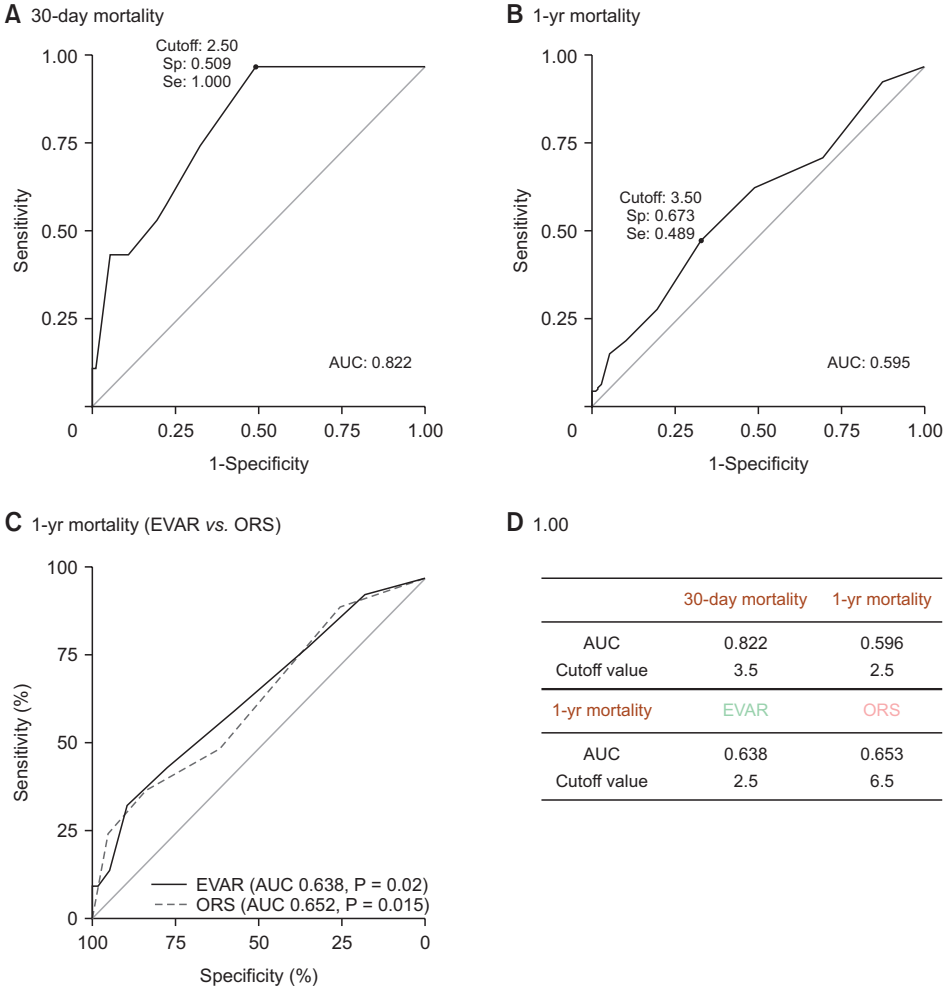


Fig. 2. Comparison of observed and predicted mortality based on the Vascular Study Group of New England risk predictive model. (A) Observed 30-day mortality *vs.* predicted mortality of patients in the endovascular aortic repair (EVAR) and open repair surgery (ORS) groups. (B) Observed 30-day mortality *vs.* predicted mortality based on the American Society of Anesthesiologists (ASA) physical status classification.

ORS group, subjects aged >75 years had markedly increased mortality risk (OR, 5.78, $p < 0.01$); however, aneurysm size was not a significant factor in this group (Fig. 5B).

The univariable analysis confirmed the VSGNE risk score and categorization into risk stages as significant in 1-year mortality. The risk of death increased 1.4-fold with each additional VSGNE



	30-day mortality	1-yr mortality
AUC	0.822	0.596
Cutoff value	3.5	2.5
1-yr mortality	EVAR	ORS
AUC	0.638	0.653
Cutoff value	2.5	6.5

Fig. 3. The receiver operating characteristics curve (ROC) for the area under the curve (AUC) values for the Vascular Study Group of New England risk predictive score regarding short-term mortality. (A) The 30-day mortality ROC of all patients. (B) The 1-year mortality ROC of all patients. (C) The 1-year mortality ROC of the EVAR and ORS groups. (D) Calculated Delong-adjusted AUC for each curve and Youden's cutoff value.

point. The high-risk group had a mortality risk 4-fold higher than the low-risk group (Fig. 5C).

Kaplan-Meier analysis revealed a significant difference in mortality between the low-risk and high-risk groups over time, both at 30 days and 1 year. However, no significant difference was found in mortality between the low- and medium-risk groups. In 1-year mortality, a significant difference was observed in the EVAR group between the medium-risk group (18.1%) and the low-risk group (3.1%). Similarly, the ORS group exhibited notably higher mortality in the high-risk group (10.1%) compared with the low-risk group (2.1%) (Fig. 6).

DISCUSSION

In this analysis, the VSGNE risk model was applied to a single-center cohort to predict short-term mortality. The examination of risk score distribution compared with actual mortality confirmed the importance of the risk factors in the scoring system.

The distribution pattern of scores within the dataset exhibited similarities to the VSGNE sample cohort group. However, compared to the VSGNE cohort assessed by Eslami et al. [6], our mean score was slightly lower, measuring 2.81 in contrast to their 3.18. Notably, our mean score was similar to that of the VQI sample, in which a mean score of 2.6 was reported [5]. Furthermore, in comparison to the model's prediction for the total population (mean 8.33), our patient scores were underestimated.

Our study demonstrated a high accuracy in predicting short-term mortality, with an AUC of 0.822. In terms of accuracy, the model tended to overestimate observed mortality. This finding contradicts the assessment made by de Guerre et al. [8], who reported this risk prediction model tends to underestimate mortality in their analysis of large datasets such as National Inpatient Sample, VQI, and National Surgical Quality Improvement Program. When stratifying risk scores into quintiles, our analysis revealed notably lower observed mortality than predicted within the cohort exhibiting extremely high scores.

Notably, advanced age (>75 years), cardiovascular disease, and elevated creatinine level (components included in the risk score) were significant factors influencing short-term mortality.

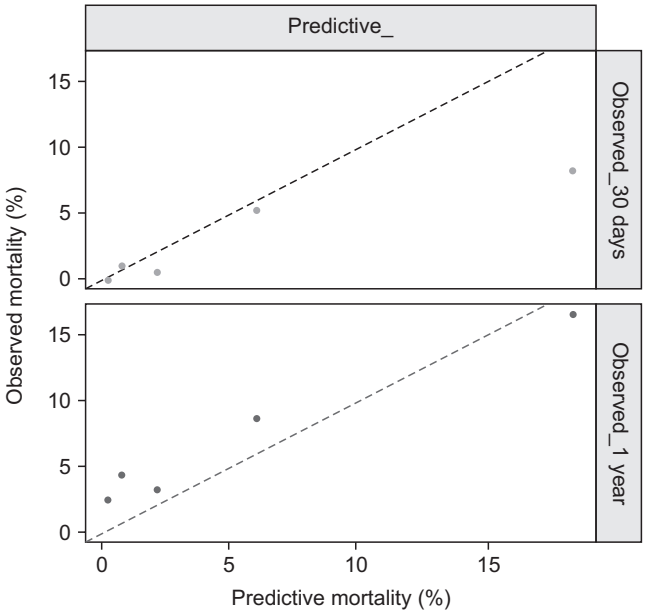


Fig. 4. Performance of the Vascular Study Group of New England risk prediction model (quantiles). The plot comparing actual and predicted mortality after stratifying patients based on score. From the 1st to the 4th quantiles, the difference in 30-day mortality was minimal and 1-year mortality tended to be underestimated. The 5th quantile (scores > 9 points) showed a tendency to overestimate both 30-day and 1-year mortality scores.

Application of the model demonstrated an approximately 4-fold higher mortality in the high-risk group compared with the low-risk group.

However, underlying myocardial disease or COPD did not show significant effects on short-term mortality in our analysis. Although these factors were expected to have a clinically significant effect, the presence of major cardiopulmonary complications did not correlate with short-term mortality in our dataset. The major complications leading to mortality may have been prevented by more frequent follow-up and close observation of underlying diseases. In addition, the beneficial effects of collaborative treatment in high-volume centers could contribute to the reduction of mortality. Myocardial disease significantly increased all-cause death. These findings indicate that long-term mortality outcomes may differ.

Although EVAR exhibited superior short-term outcomes regarding mortality compared with ORS in another study, this study found no significant difference in short-term mortality between the 2 groups in elective cases. In our study, the observed mortality (1.1%) in the ORS group was markedly lower than the predicted mortality (1.9%). These results indicated that, even in the short term, the risk of death associated with elective ORS was not higher compared with that of EVAR in our dataset. The low mortality associated with ORS observed in the data may have been influenced by hospital volume [9] and surgeon expertise [10]. Furthermore, corroborating findings from several randomized controlled trials and large cohort studies (EVAR-1, DREAM, OVER) indicate that EVAR reduced long-term survival and increased the need for secondary interventions [11]. Hwang et al. [12] emphasized the tendency to prefer EVAR due to patient vulnerability. They emphasized the importance of individual determination when considering delayed sac expansion, confirming the importance of the early survival advantage of EVAR when making decisions.

Our study identified a significant risk factor for short-term mortality in the EVAR group. In addressing issues associated with Instructions for Use compliance, it is crucial to consider sac size as well as neck length, angle, and anatomical severity as relevant factors. As these factors can affect mid-term mortality, including that due to endoleak, a comprehensive assessment of individual anatomical factors for risk stratification is warranted. Conversely, Min et al. [13] emphasized the importance of the ultrasound-based inner-to-inner maximum AP diameter, as recommended by the National Institute for Health and Care Excellence guidelines. However, they noted the potential for overtreatment due to the tendency of surgeons to measure the longest aortic diameter using CT [13], which could have led to the unnecessary inclusion of treated patients with small AAA (size < 5.5 cm), who do not meet the indications, especially because our dataset also relied on CT-based diameter measurements for screening.

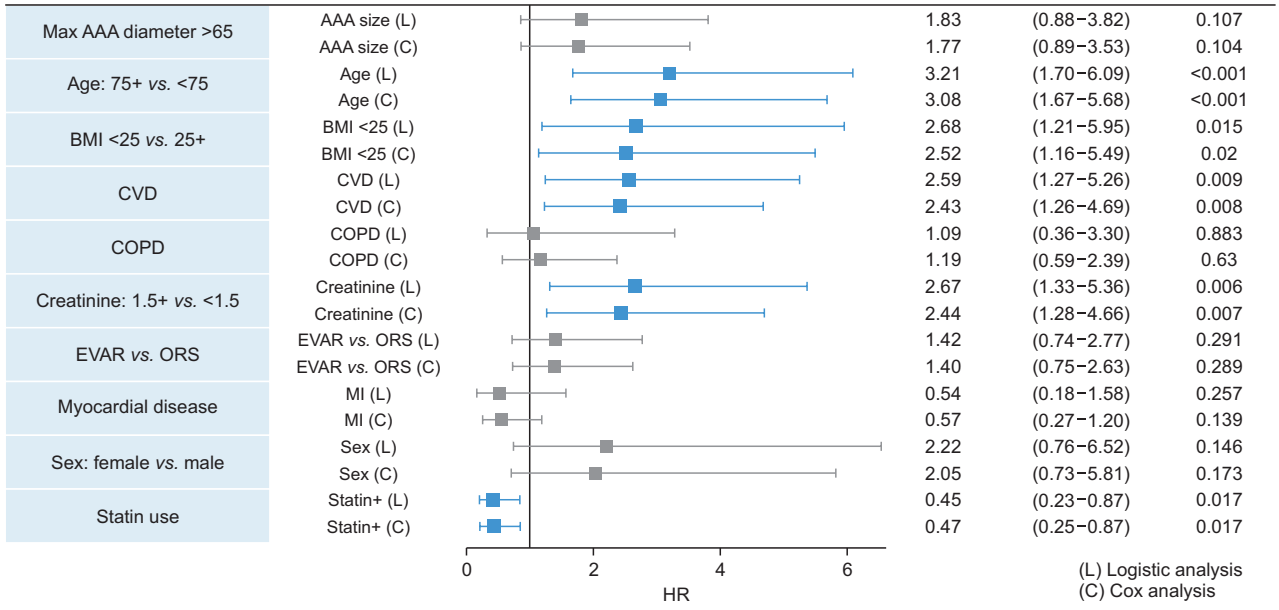
In a study by McNally et al. [14], the use of statins before surgery was associated with a significant reduction in both short-term and long-term mortality in patients undergoing AAA repair. It appears that patient adherence and compliance to prescribed medications or treatment plans reduced mortality and the pharmacological preventive effects of statins. In addition, preoperative BMI was a significant risk factor, possibly indicating preoperative malnutrition, in conjunction with established factors such as hypoalbuminemia [15] and psoas muscle volume [16].

The limitations of our study are as follows. Our study is limited by its retrospective analysis within a single medical center, potentially reducing the strength of causal relationships with risk factors. Due to a focus on patients who had already received treatment, a possibility of selection bias existed because notably high-risk patients may have been excluded. In addition, the single-center data may not be generalized nationally.

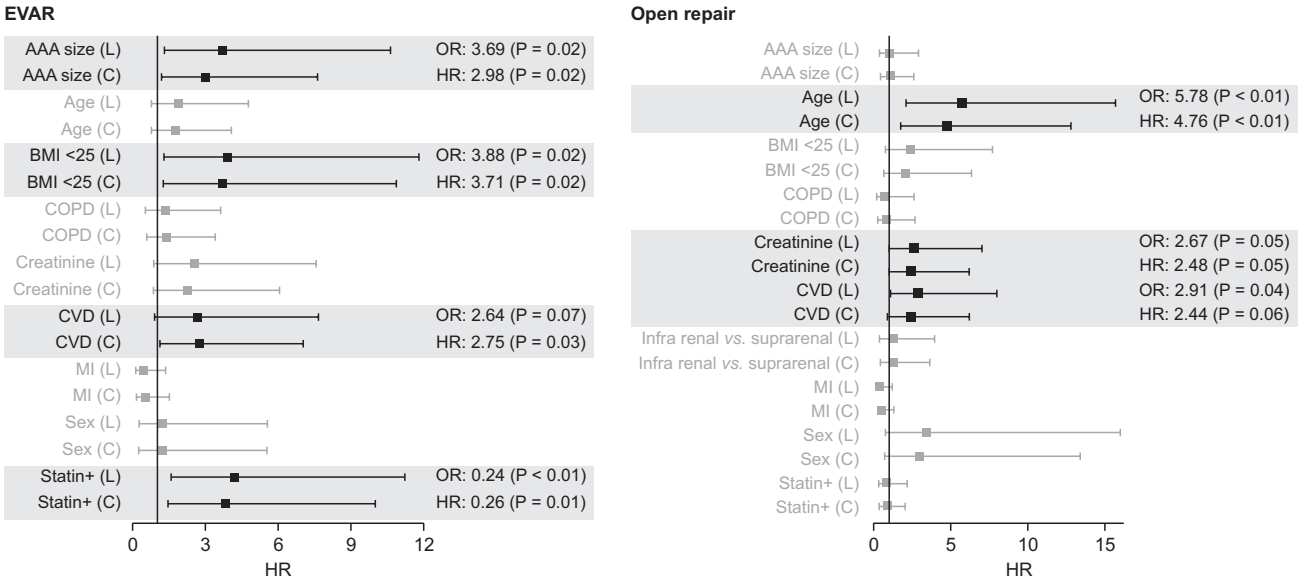
Data collection relied only on medical records even for intraoperative details, including suprarenal and infrarenal artery clamping, and the low rate of congestive heart failure was determined based on medical records or preoperative echocardiography, introducing limitations in data collection accuracy.

We did not quantify the calibration between actual

A



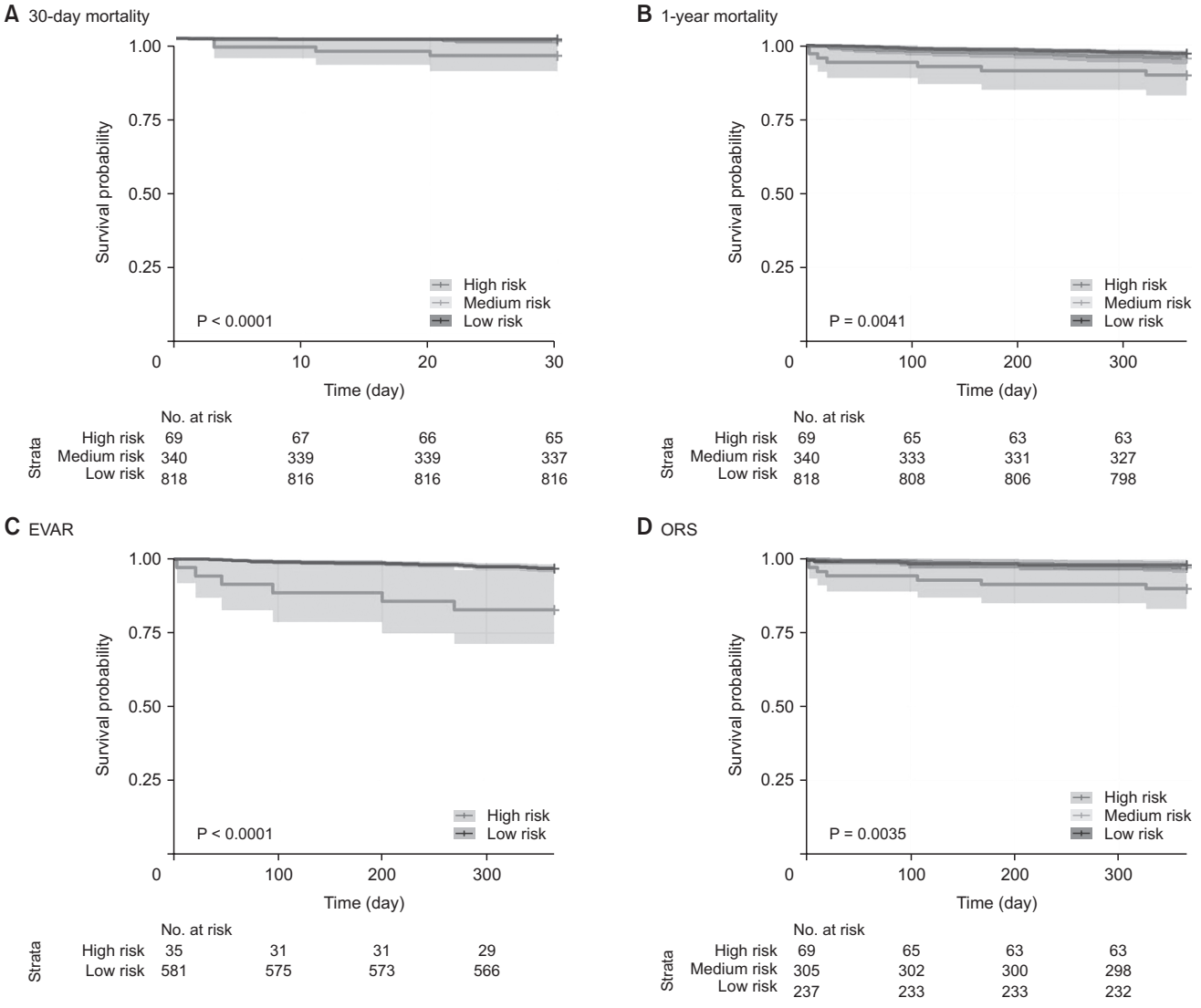
B



C

	OR or HR	95% CI	P-value
Total (low *vs.* high)	3.90	(1.61–9.45)	0.003
	3.82	(1.64–8.90)	0.002
EVAR (low *vs.* medium)	6.47	(2.39–17.53)	<0.001
	6.19	(2.45–15.59)	<0.001
ORS (low *vs.* high)	5.24	(1.61–17.07)	0.006
	5.04	(1.60–15.89)	0.006

Fig. 5. Logistic regression and Cox regression analyses. (A) Odds ratio (OR) and hazard ratio (HR) plot of multiple analyses (1-year mortality). (B) Subgroup multiple analysis for 1-year mortality. (C) Univariable analysis for Vascular Study Group of New England risk score groups: low (1–3 points), medium (4–6 points), and high (≥7 points). AAA, abdominal aortic aneurysm; BMI, body mass index; CVD, cardiovascular disease; COPD, chronic obstructive pulmonary disease; EVAR, endovascular aortic repair; ORS, open repair surgery; CI, confidence interval.



E Observed mortality

Risk group	Mortality rate			
	30-day mortality	1-year mortality	1-year EVAR	1-year ORS
Low risk	0.2	2.8	3.1	2.1
Medium	0.9	4.4	18.1	3.0
High	5.8	10.1	-	10.1

Fig. 6. Kaplan-Meier analysis. Survival curve based on stratification with Vascular Study Group of New England risk score groups: Risk group 3: low (1–3 points), group 2: medium (4–6 points), and group 1: high (≥7 points). (A) The 30-day survival curve for all patients. (B) The 1-year survival curve for all patients. (C) The 1-year survival curve for the endovascular aortic repair (EVAR) group. There was no high-risk group in EVAR. (D) The 1-year survival curve for the open repair surgery (ORS) group. (E) Mortality rate(%) based on stratified risk score.

mortality and expected mortality according to risk score, and comparisons with other model systems were not feasible. Therefore, the superiority of the VSGNE risk prediction model compared with other models could not be verified within the study dataset. In terms of the small number of deaths in our study, 1-year mortality was evaluated instead of the more common in-hospital mortality. This difference may have affected the accuracy of our analysis. However, in elective AAA repair, traditional risk assessment methods have transitioned to the use of artificial intelligence and machine learning [17,18]. This shift prompts a reevaluation of existing guidelines. In our study, we applied the modern VSGNE score, considered one of the best tools available, to assess patient risk in a real clinical setting. We aimed to determine whether the VSGNE score could

effectively predict risks and identify factors affecting mortality.

In conclusion, our study evaluated the efficacy of the VSGNE risk prediction model in guiding preoperative risk assessment and treatment for patients with elective AAA. The application of this model facilitates the provision of objective risk information to patients, enhancing informed clinical decision-making.

Notably, our findings indicated lower than anticipated actual risk scores of patients who underwent treatment. Therefore, it is necessary to develop another risk prediction model suitable for the clinical characteristics of the Korean population. Further research should include multicenter data, ensuring a more comprehensive and representative analysis. Such research would be a significant step forward in the individual care of patients with AAA.

ACKNOWLEDGEMENTS

Fund/Grant Support

None.

Conflicts of Interest

No potential conflict of interest relevant to this article was reported.

ORCID iD

Hyo-Shin Kim: https://orcid.org/0009-0001-2570-9046
Da-Hyun Kim: https://orcid.org/0009-0006-9997-0649
Dong-Ik Kim: https://orcid.org/0000-0001-6984-5982
Joon-Kee Park: https://orcid.org/0000-0003-4908-3129
Shin-Seok Yang: https://orcid.org/0000-0003-4957-3080
Yang-Jin Park: https://orcid.org/0000-0001-8433-2202

Author Contribution

Conceptualization: YJP, SSY
Formal Analysis: HSK, YJP, DHK, DIK
Investigation: HSK, DHK
Methodology: YJP, SSY, JKP
Project Administration: YJP
Writing – Original Draft: HSK, YJP
Writing – Review & Editing: All authors

REFERENCES

1. Chaikof EL, Dalman RL, Eskandari MK, Jackson BM, Lee WA, Mansour MA, et al. The Society for Vascular Surgery practice guidelines on the care of patients with an abdominal aortic aneurysm. J Vasc Surg 2018;67:2-77.
2. Al Shakarchi J, Fairhead J, Rajagopalan S, Pherwani A, Jaipersad A. Impact of frailty on outcomes in patients undergoing open abdominal aortic aneurysm repair. Ann Vasc Surg 2020;67:100-4.
3. Grant SW, Grayson AD, Mitchell DC, McCollum CN. Evaluation of five risk prediction models for elective abdominal aortic aneurysm repair using the UK National Vascular Database. Br J Surg 2012;99:673-9.
4. Eslami MH, Rybin DV, Doros G, Siracuse JJ, Farber A. External validation of Vascular Study Group of New England risk predictive model of mortality after elective abdominal aorta aneurysm repair in the Vascular Quality Initiative and comparison against established models. J Vasc Surg 2018;67:143-50.
5. Cronenwett JL, Likosky DS, Russell MT, Eldrup-Jorgensen J, Stanley AC, Nolan BW, et al. A regional registry for quality assurance and improvement: the Vascular Study Group of Northern New England (VSGNNE). J Vasc Surg 2007;46:1093-102.
6. Eslami MH, Rybin D, Doros G, Kalish JA, Farber A; Vascular Study Group of New England. Comparison of a Vascular Study Group of New England risk prediction model with established risk prediction models of in-hospital mortality after elective abdominal aortic aneurysm repair. J Vasc Surg 2015;62:1125-33.
7. Choi C, Ahn S, Min SI, Ahn M, Ha J, Yoon HJ, et al. Nationwide epidemiologic study of abdominal aortic aneurysms in Korea: a cross-sectional study using national health insurance review and assessment service data. Vasc Specialist Int 2019;35:193-201.
8. de Guerre LE, Dansey KD, Patel PB, O'Donnell TF, Zettervall SL, van Herwaarden JA, et al. Not all risk scores are created equal: a comparison of risk scores for abdominal aortic aneurysm repair in administrative data and quality improvement registries. J Vasc Surg 2021;74:1874-84.
9. Giles KA, Stone DH, Beck AW, Huber TS, Upchurch GR Jr, Arnaoutakis DJ, et al. Association of hospital volume with patient selection, risk of complications, and mortality from failure to rescue after open abdominal aortic aneurysm repair. J Vasc Surg 2020;72:1681-90.
10. Arnaoutakis DJ, Scali ST, Neal D, Giles KA, Huber TS, Powell RJ, et al. Surgeon experience association with patient selection and outcomes after open abdominal aortic aneurysm repair. J Vasc Surg 2020;72:1325-36.
11. Powell JT, Sweeting MJ, Ulug P, Blankensteijn JD, Lederle FA, Becquemin JP, et al. Meta-analysis of individual-patient data from EVAR-1, DREAM, OVER and ACE trials comparing outcomes of endovascular or open repair for abdominal aortic aneurysm over 5 years. Br J Surg 2017;104:166-78.

12. Hwang D, Kim HK, Huh S. Incidence and risk factors for sac expansion after endovascular aneurysm repair of abdominal aortic aneurysms. Vasc Specialist Int 2021;37:34.
13. Min SK. Controversies in the Treatment of Abdominal Aortic Aneurysm (I): Is the NICE Guideline Nice to Patients?. Vasc Specialist Int 2021;37:39.
14. McNally MM, Agle SC, Parker FM, Bogey WM, Powell CS, Stoner MC. Preoperative statin therapy is associated with improved outcomes and resource utilization in patients undergoing aortic aneurysm repair. J Vasc Surg 2010;51:1390-6.
15. Wohlauer M, Brier C, Kuramochi Y, Eagleton M. Preoperative hypoalbuminemia is a risk factor for early and late mortality in patients undergoing endovascular juxtarenal and thoracoabdominal aortic aneurysm repair. Ann Vasc Surg 2017;42:198-204.
16. Lindström I, Khan N, Vänttinen T, Peltokangas M, Sillanpää N, Oksala N. Psoas muscle area and quality are independent predictors of survival in patients treated for abdominal aortic aneurysms. Ann Vasc Surg 2019;56:183-93.
17. Li B, Aljabri B, Verma R, Beaton D, Eisenberg N, Lee DS, et al. Using machine learning to predict outcomes following open abdominal aortic aneurysm repair. J Vasc Surg 2023;78:1426-38.
18. Li B, Aljabri B, Verma R, Beaton D, Eisenberg N, Lee DS, et al. Machine learning to predict outcomes following endovascular abdominal aortic aneurysm repair. Br J Surg 2023;110:1840-9.