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ORIGINAL PAPER

PHYSIOLOGICAL MARKERS, COMORBIDITIES, AND RISK SCORES IN RUPTURED ABDOMINAL AORTIC ANEURYSM: A PROSPECTIVE COHORT

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Abstract

Introduction. Ruptured abdominal aortic aneurysm (rAAA) is a life-threatening vascular emergency in which early prognosis reflects acute physiological derangement and pre-existing vascular reserve.
The objective of the study was to evaluate admission physiological markers and comorbidities associated with in-hospital mortality after open rAAA repair and to compare their discriminatory performance with the Hardman Index and Glasgow Aneurysm Score (GAS).
Material and methods. This prospective single-centre cohort included 131 consecutive patients with confirmed rAAA, who underwent open repair between 2019 and 2025. The primary outcome was in-hospital mortality. Univariable logistic regression examined admission variables. A parsimonious multivariable model was internally validated with 1,000 bootstrap resamples, and an exploratory Firth model assessed SpO_2, peripheral arterial disease (PAD), COPD, and symptom-to-admission time.
Results. In-hospital mortality was 24.4% (32/131). SpO_2 was lower in non-survivors (91.28% vs 93.53%; p=0.004) and was associated with mortality (OR 0.43 per 5-percentage-point increase; 95% CI 0.24–0.76). PAD (OR 3.47; 95% CI 1.36–8.82) and COPD (OR 2.53; 95% CI 1.06–6.05) were also associated with mortality. In the

Marqueurs physiologiques et scores pronostiques dans l'anévrisme rompu de l'aorte abdominale : cohorte prospective

Introduction. La rupture d'un anévrisme de l'aorte abdominale (rAAA) est une urgence vasculaire grave dont le pronostic dépend des perturbations physiologiques aiguës et de la réserve vasculaire.
L'objectif de l'étude a été d'évaluer les marqueurs physiologiques à l'admission et les comorbidités associés à la mortalité hospitalière après chirurgie ouverte d'un rAAA, et comparer leur discrimination à celle de l'indice de Hardman et du Glasgow Aneurysm Score (GAS).
Materiel et méthodes. Cette cohorte prospective monocentrique comprenait 131 patients consécutifs avec rAAA confirmé, traités par chirurgie ouverte entre 2019 et 2025. Le critère principal était la mortalité hospitalière. Une régression logistique univariée a évalué les variables d'admission. Un modèle multivarié parcimonieux a été validé par 1 000 rééchantillonnages bootstrap; un modèle exploratoire de Firth a évalué la SpO_2, l'artériopathie périphérique (PAD), la BPCO et le délai symptômes-admission.
Résultats. La mortalité hospitalière était de 24,4% (32/131). La SpO_2 était plus basse chez les non-survivants (91,28% vs 93,53%; p=0,004) et associée à la

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exploratory Firth model, SpO_2 (aOR 0.44; 95% CI 0.24–0.79) and PAD (aOR 3.00; 95% CI 1.14–7.91) remained associated with mortality. AUCs were 0.677 for SpO_2, 0.529 for Hardman Index, and 0.447 for GAS.

Conclusions. Admission SpO_2 and PAD were the strongest exploratory mortality signals, whereas conventional risk scores showed limited discrimination in this cohort.

Keywords: oxygen saturation, peripheral arterial disease, hospital mortality, open repair, bootstrap validation.

List of abbreviations:
aOR = adjusted odds ratio
AUC = area under the receiver operating characteristic curve
COPD = chronic obstructive pulmonary disease
GAS = Glasgow Aneurysm Score
OR = odds ratio
PAD = peripheral arterial disease
rAAA = ruptured abdominal aortic aneurysm
ROC = receiver operating characteristic
SpO_2 = peripheral oxygen saturation

mortalité (OR 0,43 par augmentation de 5 points; IC95% 0,24–0,76). La PAD (OR 3,47; IC95% 1,36–8,82) et la BPCO (OR 2,53; IC95% 1,06–6,05) étaient également associées. Dans le modèle de Firth, la SpO_2 (aOR 0,44; IC95% 0,24–0,79) et la PAD (aOR 3,00; IC95% 1,14–7,91) restaient associées à la mortalité.

Conclusions. La SpO_2 à l'admission et la PAD étaient les principaux signaux exploratoires de mortalité, tandis que les scores conventionnels présentaient une discrimination limitée.

Mots-clés: saturation en oxygène, artériopathie périphérique, mortalité hospitalière, chirurgie ouverte, validation bootstrap

Introduction

Ruptured abdominal aortic aneurysm (rAAA) combines catastrophic haemorrhage with major physiological stress in a population that is typically elderly and multimorbid. Contemporary management therefore requires rapid evaluation, not only of aneurysm anatomy, but also of haemodynamic reserve, metabolic derangement, cardiopulmonary status, and the burden of systemic vascular disease[1,2].

Several prognostic systems have been developed to support risk stratification. The Hardman Index and Glasgow Aneurysm Score (GAS) are among the best known, but both were derived in historical cohorts and may perform variably when applied to contemporary populations[3–7]. More recent scores incorporate severe hypotension, pH (???), renal dysfunction, and other early physiological variables, yet external validation remains essential.

Simple admission measurements may also carry prognostic information. Oxygen saturation is routinely available within minutes, while peripheral arterial disease (PAD) and chronic obstructive pulmonary disease (COPD) reflect systemic vascular and respiratory reserve. Their value relative to conventional rAAA scores is less well defined.

The objective of the study was to evaluate the association of admission physiological markers and comorbidities with in-hospital mortality in patients undergoing open rAAA repair, to compare the discriminatory performance of simple admission markers with the Hardman Index and GAS, and to assess whether a multivariable model was sufficiently stable to support clinical prognostication.

Material and methods

Study design and population

This prospective, single-centre observational cohort, included 131 consecutive patients with documented rAAA treated by open repair at the Vascular Surgery Service, University Hospital Centre "Mother Teresa", Tirana, Albania, during 2019–2025. The patients were enrolled prospectively and followed through the index hospitalization. The primary endpoint was all-cause in-hospital mortality.

Participants and case definition

Eligible patients were adults with confirmed ruptured abdominal aortic aneurysms, who underwent open surgical repair during the study period. Rupture was established by imaging evidence of haemorrhage outside the aneurysm sac and/or unequivocal intraoperative confirmation. Symptomatic but non-ruptured aneurysms were not included.

Prospective data collection and completeness

Demographic characteristics, comorbidities, admission physiological and laboratory variables, anatomical findings, operative information, and in-hospital outcomes were recorded prospectively using predefined study variables and verified against source clinical documentation. A standardized case-report form was used from enrolment through hospital discharge, and admission variables were recorded before surgery, whenever clinically feasible. The two principal timing variables were available for all 131 patients, and no reconstructed or deduced values were used for these timing variables.

Ethics and informed consent

The study protocol was reviewed and approved by the Ethics Committee of the Faculty of Medicine, University of Medicine of Tirana, Albania, on 8 January 2019 (protocol no. 291/3), before the start of patient enrolment. Written informed consent to participate was obtained from all patients; when a patient's clinical condition precluded direct consent, consent was obtained from a legally authorized representative, in accordance with the approved study protocol. The study was conducted in accordance with the principles of the Declaration of Helsinki.

Clinical variables and risk scores

Admission variables included age, sex, systolic and diastolic blood pressure, heart rate, respiratory rate, peripheral oxygen saturation (SpO_2), shock index, loss of consciousness, electrocardiographic ischaemia, haemoglobin, creatinine, lactate, pH (blood pH?), aneurysm diameter, retroperitoneal haematoma, active contrast extravasation, and major pre-existing comorbidities. SpO_2 was defined as the earliest reliable peripheral oxygen saturation recorded during the initial preoperative assessment, before induction of anaesthesia; supplemental oxygen at the time of measurement was recorded when documented. PAD was defined by a pre-existing documented diagnosis and/or objective evidence of lower-extremity arterial disease, including an ankle-brachial index ≤0.90, abnormal arterial imaging, previous peripheral revascularization, or prior amputation attributable to atherosclerotic arterial disease. COPD was defined by a physician-documented diagnosis established before the index admission; when available, prior spirometry demonstrating persistent post-bronchodilator airflow obstruction and chronic respiratory therapy were used as supporting evidence. The comorbidity profile included hypertension, diabetes, dyslipidaemia, COPD, chronic kidney disease, coronary artery disease, prior myocardial infarction, atrial fibrillation, prior stroke/transient ischaemic attack, PAD, and obesity. The Hardman Index was calculated from the original five criteria: age >76 years, loss of consciousness, haemoglobin <9 g/dL, serum creatinine >190 µmol/L, and electrocardiographic evidence of myocardial ischaemia. One point was assigned for each criterion present. The Glasgow Aneurysm Score was calculated using the published formula: age in years +17 points for shock +7 for myocardial disease +10 for cerebrovascular disease +14 for renal disease. For both scores, the earliest available preoperative variables recorded at presentation were used. The scores were evaluated solely as prognostic comparators and were not used to determine eligibility for surgery.

Statistical analysis

Continuous variables were summarized as mean ± standard deviation or median (interquartile range), and categorical variables as n (%). Mortality groups were compared using Welch t, Mann–Whitney, Pearson chi-square, or Fisher exact tests as appropriate. Univariable logistic regression generated odds ratios (OR) with 95% confidence intervals. A parsimonious primary multivariable model included age, symptom-to-admission time, comorbidity count, shock index, and lactate; these variables represented demographic risk, delay, chronic disease burden, haemodynamic compromise, and metabolic hypoperfusion while limiting model complexity in the presence of only 32 deaths. Firth penalized logistic regression was used as a small-sample sensitivity analysis. Discrimination was assessed by ROC AUC, calibration by Brier score and calibration intercept/slope, and internal validation by 1,000 bootstrap resamples. A secondary post hoc Firth model included SpO_2 (per 5-percentage-point increase), PAD, COPD, and symptom-to-admission time. Because these covariates were selected after inspection of clinical and univariable signals, this model was explicitly considered exploratory and was not interpreted as a validated prediction model. Exploratory ROC thresholds were presented descriptively and were not intended as treatment cut-offs. The component variables required for the Hardman Index and Glasgow Aneurysm Score were complete for all patients included in the score analyses. No multiplicity adjustment was applied to exploratory analyses; therefore, secondary p values were interpreted together with effect sizes, confidence intervals, biological plausibility, and consistency across analyses. All statistical analyses were performed using IBM SPSS Statistics for Windows, version 28.0 (IBM Corp., Armonk, NY, USA).

The manuscript was prepared in accordance with STROBE principles for reporting observational cohort studies.

Table 1. Univariable associations with in-hospital mortality

Variable	*OR*	*95% CI*	*p-value*
Age, per 10 years	0.85	0.53–1.36	0.492
Symptom-to-admission, per 6 h	1.11	1.00–1.24	0.055
Shock index, per 0.5	1.13	0.77–1.66	0.532
SpO2, per +5 percentage points	0.43	0.24–0.76	0.004
PAD	3.47	1.36–8.82	0.009
COPD	2.53	1.06–6.05	0.037
Lactate, per 1 mmol/L	1.11	0.91–1.36	0.298
Maximum diameter, per 10 mm	1.36	0.91–2.03	0.130

Abbreviations: CI = confidence interval; COPD = chronic obstructive pulmonary disease; OR = odds ratio; PAD = peripheral arterial disease; SpO_2 = peripheral oxygen saturation.

Table 2. Discrimination of individual markers, conventional risk scores, and the primary multivariable model

Marker / model	*AUC*	*95% CI*	*Exploratory threshold*
SpO2	0.677	0.572–0.782	≤94%
Comorbidity count	0.589	0.479–0.700	≥4
Lactate	0.573	0.460–0.685	≥1.9 mmol/L
Shock index	0.527	0.406–0.647	≥1.42
Hardman Index	0.529	0.415–0.643	≥3
Glasgow Aneurysm Score	0.447	0.322–0.573	≥108
Primary multivariable model	0.651	0.537–0.764	Predicted risk ≥0.281

Abbreviations: AUC = area under the receiver operating characteristic curve; CI = confidence interval; GAS = Glasgow Aneurysm Score; SpO_2 = peripheral oxygen saturation.

Table 3. Exploratory Firth penalized logistic regression model for in-hospital **mortality**

Predictor	*Firth aOR*	*95% CI*	*p-value*
SpO2, per +5 percentage points	0.44	0.24–0.79	0.005
Peripheral arterial disease	3.00	1.14–7.91	0.027
COPD	2.54	0.96–6.78	0.061
Symptom-to-admission, per 6 h	1.07	0.96–1.21	0.225

Abbreviations: aOR = adjusted odds ratio; CI = confidence interval; COPD = chronic obstructive pulmonary disease; PAD = peripheral arterial disease; SpO_2 = peripheral oxygen saturation.

Results

Mortality and admission physiology

Thirty-two of 131 patients died in hospital (24.4%). The mean age was 73.2±8.5 years. SpO_2 was the only continuous variable with a statistically significant difference between survivors and non-survivors: 93.53% versus 91.28% (p=0.004). Lactate was higher among non-survivors (2.95 vs 2.60 mmol/L), but the difference was not significant (p=0.218). The maximum aneurysm diameter was also greater among non-survivors (78.31 vs 75.18 mm; p=0.163).

Univariable mortality signals

The univariable associations with in-hospital mortality are summarized in Table 1. Admission SpO_2, PAD, and COPD showed the strongest individual associations with mortality.

Conventional scores and discrimination

SpO_2 had the highest AUC among the individual markers and was numerically higher than the primary multivariable model, although the difference was not significant (DeLong p=0.757). The model was significantly better than GAS (DeLong p=0.019). Discrimination estimates for individual markers and risk scores are presented in Table 2.

Exploratory Firth model

In the exploratory Firth model, higher SpO_2 remained associated with lower mortality and PAD with higher mortality. COPD retained a clinically substantial effect estimate but did not reach conventional statistical significance. The apparent AUC of this post hoc model was 0.749 and the Brier score 0.152; these values were not interpreted as validated performance. Model coefficients are presented in Table 3.

Internal validation of the primary model

The primary multivariable model had apparent AUC 0.651 and Brier score 0.175. After 1,000 bootstrap resamples, optimism-corrected AUC fell to 0.574 and the corrected Brier score worsened to 0.194. The mean bootstrap calibration slope was 0.57 (95% CI 0.19–1.03), indicating substantial overfitting. The corrected Brier score was worse than the prevalence-only benchmark (0.185).

Discussion

This study identified lower admission SpO_2 and PAD as the clearest mortality signals in a cohort of patients undergoing open rAAA repair. COPD also showed a clinically important association but remained borderline after penalized adjustment. In contrast, age, overall comorbidity count, shock index, lactate, and the conventional Hardman and Glasgow scores did not provide strong discrimination.

The association between oxygen saturation and mortality is clinically plausible. Oxygen delivery depends on arterial oxygenation, haemoglobin concentration, and cardiac output; during rAAA, these components are simultaneously threatened by blood loss, hypoperfusion, pulmonary disease, and perioperative physiological stress. A lower SpO_2 may therefore capture an integrated loss of cardiopulmonary reserve that is not fully represented by blood pressure or shock index alone.

The association between admission SpO_2 and mortality should be interpreted cautiously. Pulse oximetry can be influenced by peripheral hypoperfusion, vasoconstriction, hypotension, motion artefact, and supplemental oxygen, all of which are relevant in haemorrhagic shock[14]. Accordingly, SpO_2 was interpreted as a pragmatic marker of early physiological status rather than as an isolated measure of pulmonary function or a causal determinant of outcome. The observed association is hypothesis-generating and does not support a stand-alone treatment threshold.

PAD retained an approximately threefold association with mortality in the exploratory penalized model. PAD is a marker of systemic atherosclerotic burden and may identify patients with reduced coronary, cerebrovascular, and peripheral reserve. Its effect may also reflect more advanced multisite vascular disease than is captured by a simple comorbidity count.

COPD was associated with mortality in univariable analysis and remained close to statistical significance after Firth adjustment. This is consistent with the physiological vulnerability of patients with limited ventilatory reserve undergoing emergency laparotomy, massive transfusion, mechanical ventilation, and ischaemia-reperfusion stress.

The poor discrimination of the Hardman Index and GAS is clinically important. Both scores were developed in historical cohorts and external studies have shown variable transportability across treatment eras and centres[3-7]. In the present cohort, their AUCs were close to chance. These findings do not invalidate the original scores; rather, they indicate limited discriminatory performance in this specific contemporary open-repair cohort and reinforce that such scores should complement, not replace, clinical judgement.

Equally important is what the modelling did not support. The primary multivariable model appeared moderately discriminative in the development data but lost substantial performance after bootstrap correction. A calibration slope of 0.57 indicates overfitting, and the corrected Brier score was worse than a prevalence-only benchmark. With only 32 deaths, deriving a new clinical prediction rule from this cohort would therefore be statistically unjustified.

The study has several strengths, including prospective data collection, a clinically detailed dataset, penalized regression, direct comparison of simple admission markers with conventional scores, and bootstrap internal validation. Several limitations should be considered. First, this was a single-centre cohort restricted to patients who survived to undergo open repair; the findings therefore cannot be generalized to all patients with rAAA, including those who died before surgery or were managed with an endovascular strategy. Second, only 32 deaths occurred, limiting the effective sample size for multivariable modelling and increasing the risk of overfitting despite penalization and bootstrap validation. Third, admission physiological measurements, particularly SpO_2, may be influenced by resuscitation, supplemental oxygen, peripheral perfusion, and timing of measurement. Fourth, the secondary Firth model was post hoc and must be considered hypothesis-generating. Finally, no external validation cohort was available.

Conclusions

In this prospective cohort of patients undergoing open repair for rAAA, lower admission SpO_2 and the presence of PAD were associated with in-hospital mortality in exploratory penalized analyses, while COPD showed a borderline association. The Hardman Index and GAS demonstrated limited discrimination in this cohort. Bootstrap validation showed substantial optimism in the primary multivariable model, precluding development of a new clinical prediction rule from these data. Larger

multicentre cohorts with standardized physiological measurements and external validation are required to determine the reproducibility and clinical utility of these findings.

Authors' contributions

M.S.: Conceptualization, methodology, investigation, data curation, formal analysis, interpretation of results, project administration, and writing – original draft. H.K.: Methodology, investigation, validation, supervision, and writing – review and editing. Both authors approved the final version of the manuscript and agree to be accountable for all aspects of the work.

Data availability

The de-identified data supporting the findings of this study are available from the corresponding author on reasonable request, subject to institutional and ethical requirements.

Compliance with Ethics Requirements:

"The authors declare no conflict of interest regarding this article"

"The authors declare that all the procedures and experiments of this study respect the ethical standards in the Helsinki Declaration of 1975, as revised in 2008(5), as well as the national law. Informed consent was obtained from all the patients included in the study"

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