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DEVELOPMENT AND VALIDATION OF A PROGNOSTIC SCORE FOR 30-DAY MORTALITY IN ACUTE CORONARY SYNDROME PATIENTS IN ORAN

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РАЗРАБОТВАНЕ И ВАЛИДИРАНЕ НА ПРОГНОСТИЧЕН РЕЗУЛТАТ ЗА 30-ДНЕВНА СМЪРТНОСТ ПРИ ПАЦИЕНТИ С ОСТЪР КОРОНАРЕН СИНДРОМ В ОРАН

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Abstract. Acute coronary syndrome (ACS) is a heterogeneous and severe acute cardiovascular event, with an early mortality rate estimated at 10.5% in western Algeria. Existing ischemic risk stratification scores for ACS vary in complexity and present certain limitations. The primary objective of this study is to develop and validate a prognostic score for ACS, based on a cohort of patients admitted to the three cardiology departments of Oran ; University Hospital Establishment (UHE), University Hospital Center (UHC) and Regional Military University Hospital (RMUH), with the aim of predicting 30-day mortality. This epidemiological, multicenter, observational study included all patients hospitalized for ACS between 2018 and 2020, with 30-day all-cause mortality as the primary endpoint. The final predictive model was derived using binary logistic regression in a cohort representing 71% of the initial population, while the remaining 29% was used for validation. Model calibration was assessed using the Hosmer-Lemeshow test, and discrimination was evaluated based on the area under the Receiver Operating Characteristic (ROC) curve and C-statistic. A total of 1692 eligible patients were recruited, with a predominance of males (75%) and a mean age of 61 ± 12 years. The developed score consists of seven independent dichotomous variables, yielding a maximum total of ten points. It demonstrated good calibration (p = 0.906) and a discrimination of 0.76 [95% CI: 0.73-0.78] in the derivation cohort, compared to 0.71 [95% CI: 0.66-0.75] in the validation cohort. A new, simple, reproducible, and validated decision-support tool is proposed for practitioners. This prognostic score, designed to be quickly and easily calculated, provides an effective means of predicting 30-day mortality in ACS patients and is well-suited to emergency department practices.

Key words: Acute coronary syndrome, Prognostic score, 30-day mortality, Oran

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Резюме. Острият коронарен синдром (ОКС) е хетерогенно и тежко остро сърдечно-съдово събитие с ранна смъртност, оценена на 10,5% в западен Алжир. Съществуващите резултати за стратификация на исхемичния риск за ОКС варират по сложност и имат определени ограничения. Основната цел на това проучване е да се разработи и валидира прогностичен резултат за ОКС, базиран на кохорта пациенти, приети в трите кардиологични отделения на Оран (EHU, CHU и HMRU), с цел прогнозиране на 30-дневна смъртност. Това епидемиологично, многоцентрово, наблюдателно проучване включва всички пациенти, хоспитализирани за ОКС между 2018 г. и 2020 г., с 30-дневна смъртност по всякаква причина като първична крайна точка. Окончателният прогнозен модел е получен с помощта на бинарна логистична регресия в кохорта, представляваща 71% от първоначалната популация, докато останалите 29% са използвани за валидиране. Калибрирането на модела беше оценено с помощта на теста на Hosmer-Lemeshow и дискриминацията беше оценена въз основа на площта под ROC кривата (C-статистика). Набрани са общо 1692 отговарящи на условията пациенти, с преобладаване на мъже (75%) и средна възраст 61 ± 12 години. Разработеният резултат се състои от 7 независими дихотомични променливи, даващи максимум 10 точки. Той демонстрира добро калибриране (p = 0,906) и дискриминация от 0,76 [95% CI: 0,73-0,78] в кохортата за деривация, в сравнение с 0,71 [95% CI: 0,66-0,75] в групата за валидиране. За практикуващите се предлага нов, прост, възпроизводим и валидиран инструмент за подпомагане на вземането на решения. Този прогностичен резултат, предназначен да бъде бързо и лесно изчислен, осигурява ефективно средство за прогнозиране на 30-дневна смъртност при пациенти с ОКС и е много подходящ за практиките в спешните отделения.

Ключови думи: остър коронарен синдром, прогностичен резултат, 30-дневна смъртност, Оран

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Introduction

Coronary pathologies, particularly acute coronary syndrome (ACS), remain a major cause of mortality and morbidity worldwide [1-3]. Early mortality rates (within 30 days) range from 5% to 10% [4]. Regardless of the ACS type, early mortality has significantly decreased in Europe and North America [5, 6]. However, in Africa, particularly Algeria, it continues to rise, with an in-hospital mortality rate of 10.5% in western Algeria [7-9].

Patients with ST-segment elevation Myocardial Infarction (STEMI) and non-ST-segment elevation Myocardial Infarction (NSTEMI) form a heterogeneous population with widely varying short- and long-term risks of death or ischemic recurrence [10]. Several risk stratification scores have been developed to integrate and weigh the main mortality markers based on parameters available at admission [2, 11]. However, these scores are often closely tied to the specific populations from which they were derived, limiting their generalizability [2].

Most of these predictive models originate from randomized studies with selective samples, making their applicability to unselected populations controversial [10, 12]. Other scores, though less restrictive, remain complex and impractical for emergency use [13, 14].

To address these limitations, a predictive model was developed specifically for a North African, Algerian population. This multicenter study aims to establish and validate a 30-day prognostic score for ACS based on a cohort of patients admitted to the cardiology departments of three university hospitals in Oran, western Algeria.

Material and methods

1. Type of study. This is an epidemiological, multicenter, observational study including all patients hospitalized for ACS in the cardiology departments of the University Hospital Establishment, the University Hospital Center, and the Regional University Military Hospital in Oran (Western Algeria) between 2018 and 2020.

2. Study population. All patients over 18 years of age admitted for a first episode of ACS, diagnosed on Electrocardiogram (ECG) as either ST-segment elevation (STEMI) or non-ST-segment elevation (NSTEMI), regardless of Killip class, were included in the study. The following cases were excluded: ACS diagnosed during pregnancy, Infarctions identified more than 30 days after symptom onset and Silent infarctions where the symptom onset could not be precisely dated.

3. Definition. ACS was defined as any patient presenting with anginal pain lasting more than 20 minutes and an electrocardiogram showing one or more of the following criteria: ST-segment elevation, characterized by new ST-segment elevation at point J in two contiguous leads, with thresholds of at least 0.2 mV in men or 0.15 mV in women in precordial leads (V2-V3) and at least 0.1 mV in other leads; newly appearing left bundle branch block (LBBB); ST-segment depression or T-wave abnormalities, defined as new horizontal or descending ST-segment depression of at least 0.05 mV in two contiguous leads and/or T-wave inversion of at least 0.1 mV in two contiguous leads, accompanied by a prominent R-wave; and elevation of cardiac enzymes, primarily troponins T and I [15, 16].

4. Criteria for judgement. A single criterion for death was used, defined as mortality from any cause within 30 days of an acute ACS episode, occurring either during hospitalization or after discharge.

5. Predictive variables were selected based on data collected at patient admission. However, therapeutic and evolutionary variables were recorded during hospitalization, except for recurrence, which was monitored for up to one month after admission. Epidemiological variables included sex, age, smoking status, and medical history, such as overall obesity, arterial hypertension (AH), diabetes, dyslipidemia, chronic kidney disease (CKD), stroke, coronary history, and prior use of aspirin and statins. Clinical variables comprised data from the clinical examination at admission, including systolic (SBP) and diastolic blood pressure (DBP), heart rate (HR), respiratory rate (RR), presence of dyspnea, cardiac symptomatology, Killip classification, and time to hospitalization. Biological variables included blood glucose, cholesterol, triglycerides, serum creatinine ≥ 12 mg/L, and cardiac enzymes T and I. Electrocardiographic variables encompassed ACS type (STEMI or NSTEMI), anterior and inferior infarct topographies, mirror image, isolated ST sub-shift, left ventricular ejection fraction (LVEF) < 50%, and abnormal left ventricular (LV) kinetics. Therapeutic variables included thrombolysis and percutaneous coronary angioplasty. Progressive variables covered ACS complications such as cardiogenic shock (CS), left ventricular failure (LVF), ventricular tachycardia (VT), atrioventricular block (AVB), and recurrence.

6. Statistical analysis. A random resampling of the original cohort was performed, dividing it into two groups: a derivation cohort comprising 70.6% of the sample and a validation cohort representing 29.4%. Baseline characteristics of the two cohorts were compared using Pearson's Chi-square test for categorical variables and Student's t-test for continuous variables, with variance equality verified by Levene's test. The linearity of the relationship between each quantitative explanatory variable (X) and the binary dependent variable (Y = death at 30 days) was assessed using the fractional polynomial method. Variables displaying non-linear distributions were dichotomized in the logistic regression analysis, based on thresholds established in the literature or determined graphically.

To identify predictive factors for 30-day mortality, patients in the derivation cohort were categorized into two groups: those who died and a reference group of survivors. A binary logistic regression analysis was conducted to develop a predictive model for 30-day mortality. To enhance the model's interpretability, most categorical variables, as well as continuous variables with non-linear distributions, were dichotomized.

In the univariate analysis, associations between each explanatory variable and the dependent variable were assessed using Pearson's Chi-square test. Pairwise interactions between explanatory variables and the dependent variable were also examined. The initial logistic regression model included all variables statistically associated with 30-day mortality, with the final model obtained through a top-down, stepwise selection process. Significant factors were then used to construct the prognostic score.

Model calibration was assessed using the Hosmer-Lemeshow test, with a p-value > 0.05 indicating good calibration [17]. The prognostic score was calculated using the Hosmer-Lemeshow method, based on the β coefficients of the variables retained in the final model. Positive predictive values, representing the probability of 30-day mortality for each score level, were calculated and expressed as percentages [18, 19].

The optimal threshold of the prognostic score was determined graphically by identifying the intersection of the sensitivity and specificity curves. This threshold corresponded to the inflection point of the ROC curve in the derivation cohort [20]. The discriminative ability of the prognostic score was evaluated using the area under the ROC curve C-statistic, expressed as a percentage with a 95% confidence interval [21, 22]. The interpretation of the area under the curve (AUC) was based on the criteria established by Hosmer and Lemeshow [17].

Results

Flow chart of study population

In this multicenter study, 1692 eligible patients were selected from the initial global cohort. This cohort was then divided into two groups: two-thirds were assigned to the 30-day prognostic score derivation cohort (n = 1,195), while one-third was allocated to the prognostic score validation cohort (n = 497) (Fig. 1).

Characteristics of the study population

Among the 1692 patients recruited for the study, there was a clear male predominance (74.7% vs. 25.3%), resulting in a sex ratio of 3. The mean age was 60.8 ± 12.3 years, ranging from 27 to 97 years. An analysis of the various characteristics of patients admitted for ACS revealed no significant differences between the two cohorts, except for a history of chronic renal failure, which was more frequent in the bypass cohort ($p < 0.05$) (Table 1).

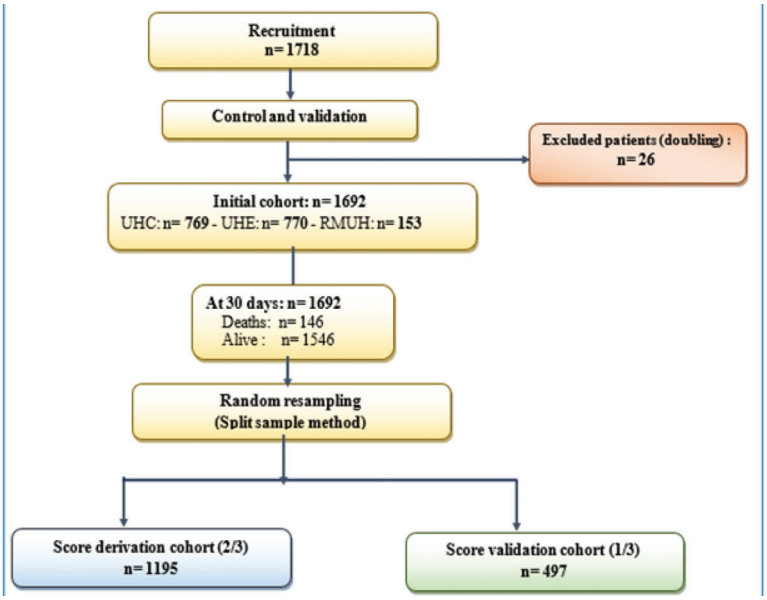


Fig. 1. Flow chart of study population

Table 1. Characteristics of patients admitted for ACS in the two cohorts

Characteristics	Derivation cohort of score (n = 1195)	Validation cohort of the score (n = 497)	p-value
Epidemiological characteristics			
Age (year)	60.82 ± 12.4	60.71 ± 12.02	0.86
Gender Male Female	 884 (74) 311 (26)	 380 (76.5) 117 (23.5)	0.27
Smoking status Non-smoker Former smoker Current smoker	 646 (54.1) 82 (6.9) 467 (39.1)	 269 (54.1) 39 (7.8) 189 (38)	0.74
Obesity	135 (11.3)	54 (10.9)	0.79
Arterial hypertension (AH)	477 (39.9)	193 (38.8)	0.67
Diabetes	425 (35.6)	176 (35.4)	0.95
Dyslipidemia	94 (7.9)	31 (6.2)	0.24
Chronic kidney disease (CKD)	15 (1.3)	13 (2.6)	0.04*
Stroke	26 (2.2)	10 (2)	0.83
Coronary history	66 (5.5)	37 (7.4)	0.13
Aspirine use	109 (9.1)	40 (8)	0.47
Statines use	59 (4.9)	22 (4.4)	0.65
Clinical characteristics			
SBP (mm Hg)	129.75 ± 25.45	128.46 ± 24.93	0.34
DBP (mm Hg)	75.85 ± 13.86	75.11 ± 14.19	0.32
RR (cycles/minutes)	18.38 ± 4.15	17.75 ± 3.76	0.46
HC (beats /minutes)	83.17 ± 20.55	82.27 ± 21.2	0.44
Dyspnea	158 (15.3)	56 (12.8)	0.21
Cardiac symptomatology Typical chest pain Atypical chest pain	 1051 (87.9) 144 (12.1)	 433 (87.1) 64 (12.9)	0.63
Killip classification Class I Class II. III and IV	 1067 (89.3) 128 (10.7)	 443 (89.1) 54 (10.9)	0.92
Time to hospital (hours) ≤ 6 h > 6 h	 544 (51) 522 (49)	 229 (50.6) 224 (49.4)	0.86

Characteristics	Derivation cohort of score (n = 1195)	Validation cohort of the score (n = 497)	p-value
Biological characteristics			
Blood glucose (g/l)	1.72 ± 0.96	1.66 ± 0.88	0.25
Total cholesterol (g/l)	1.73 ± 0.48	1.79 ± 0.45	0.16
Triglycerides (g/l)	1.55 ± 0.92	1.48 ± 0.75	
Serum creatinine (mg/l)	11.73 ± 7.10	12.13 ± 8.56	0.37
Positive cardiac enzymes	1045 (87.4)	434 (87.3)	0.94
Electrocardiographic characteristics			
Type ACS ST+ ACS ST-	 938 (78.5) 257 (21.5)	 386 (77.7) 111 (22.3)	0.7
Topography Previous infarctions Inferior infarctions	 509 (57.2) 381 (42.8)	 207 (58.3) 148 (41.7)	0.71
Mirror image	280 (29.9)	102 (26.4)	0.21
Isolated ST offset	148 (57.6)	70 (63.1)	0.32
LVEF (%)	51.15 ± 12.7	49.85 ± 13.08	0.08
Normal LV kinetics	163 (13.6)	62 (12.5)	0.52
Therapeutic characteristics			
Thrombolysis	673 (71.7)	285 (73.8)	0.44
Coronary angioplasty	66 (5.5)	28 (5.6)	0.92
Evolutionary characteristics			
Complication (CS. LVF. VT or AVB)	292 (24.4)	109 (21.9)	0.27
Recurrence	53 (4.4)	17 (3.4)	0.34
Death within 30 days	109 (9.1)	37 (7.4)	0.26

Time to go to hospital was less than six hours for 56% STEMI patients versus 33% for NSTEMI patients in the score derivation cohort (Fig. 2 and Fig. 3).

Analysis of factors predicting death at 30 days

3-1 Modelling quantitative variables

The relationship between the different quantitative variables and the dependent variable was linear, except for three variables – systolic blood pressure, diastolic blood pressure, and serum creatinine where the scatter plot demonstrated a non-linear distribution (Fig. 4 and Fig. 5).

3-2 Determination of the 30-day predictive model

A total of 35 variables were considered as candidates for the prognostic score. At the 25% threshold, univariate analysis identified 27 significant variables, which are highlighted in bold in the two sections of Table 2.

In contrast, multivariate analysis retained seven significant variables in the final predictive model: female

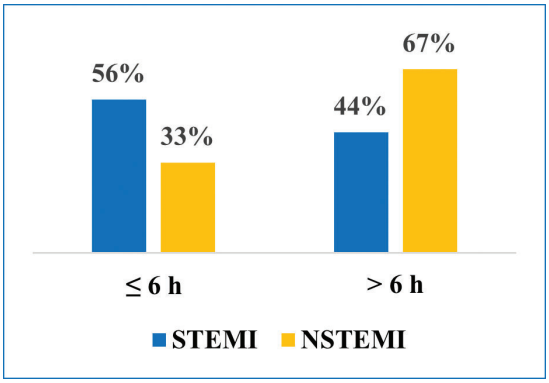
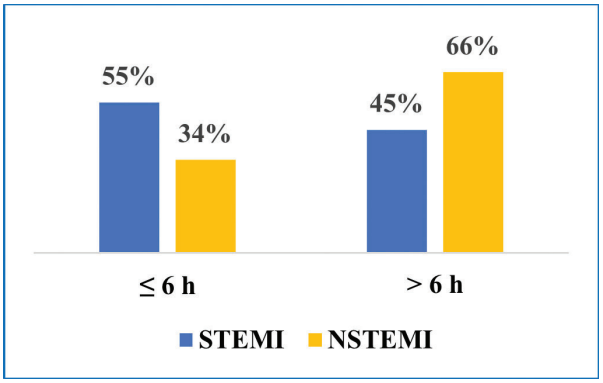


Fig. 2. Time to hospital (hours) by ACS type in the score derivation cohort



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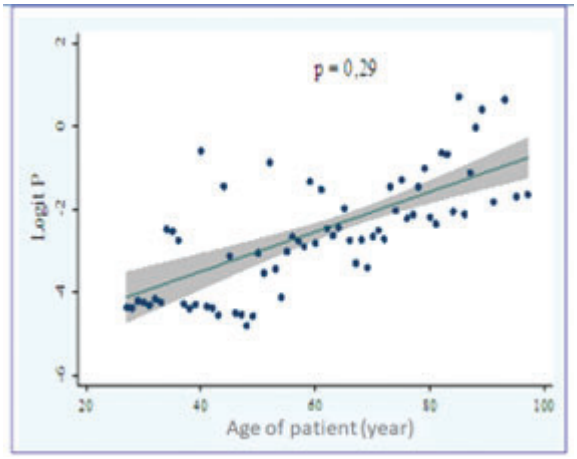

Fig. 4. Relationship between age (year) and death at 30 days, modelled by fractional polynomial

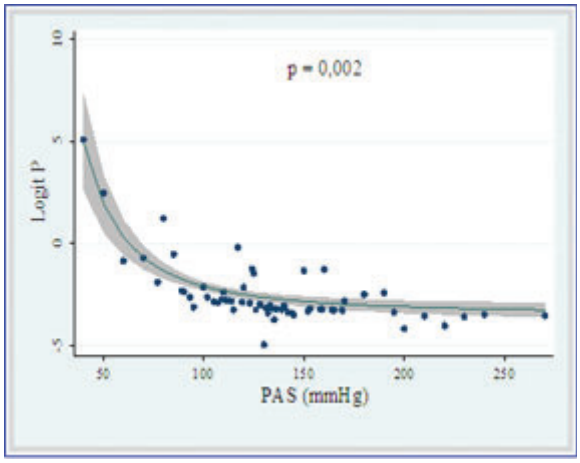

Fig. 5. Relationship between SBP (mm Hg) at admission and death at 30 days, modelled by fractional polynomial

Table 2 (part 1). Factors Predictive of 30-Day Mortality in the Score Derivation Cohort: Univariate Analysis by Logistic Regression (n = 1195)

Variables (p-value < 25%)	OR brut	[IC 95%]	p- value
Epidemiological variables			
Age (year)	1.04	1.02-1.06	**< 10^{-3}**
Gender			
Male	1		
Female	1.9	1.26-2.86	**< 0.002**
Obesity			
No	1		
Yes	1.94	0.88-4.28	0.09
Smoking status			
Non-smoker	1.11	0.56-2.18	0.75
Former smoker	0.3	0.18-0.51	**< 10^{-3}**
Currentsmoker	1		
Arterial hypertension			
No	1		
Yes	1.81	1.21-2.68	**< 0.003**
Diabetes			
No	1		
Type I (DID)	2.42	0.88-6.61	**0.08**
Type II (DNID)	1.12	0.74-1.71	0.57
Dyslipidemia			
No	1		
Yes	0.92	0.43-1.95	0.83
Chronic kidney disease			
No	1		
Yes	1.54	0.34-6.92	0.57
Stroke			
No	1		
Yes	1.84	0.62-5.44	**0.24**
Coronaryhistory			
No	1		
Yes	1.62	0.78-3.37	**0.19**
Aspirine use			
No	1		
Yes	1.007	0.50-1.99	0.98
Statines use			
No	1		
Yes	1.08	0.42-2.78	0.86
Clinical variables			
SBP(mmHg)			
≥ 90	1		
< 90	5.8	3.26-10.31	**< 10^{-3}**

Table 2 (part 2): Factors predictive of 30-day mortality in the score derivation cohort: Univariate analysis by logistic regression (n = 1195).

Variables (p-value < 25%)	OR brut	[IC 95%]	p-value
Biological variables			
Blood glucose (g/l)	1.59	1.28-1.99	**< 10^{-3}**
Total cholesterol (g/l)	1.08	0.42-2.81	0.86
Triglycerides (g/l)	0.79	0.4-1.58	0.52
Serum creatinine (mg/l)			
≤ 12	1		
> 12	3.7	2.14-6.41	**< 10^{-3}**
Cardiac enzymes			
Negative	1		
Positive	3.2	1.28-7.99	**0.01**
Electrocardiographic variables			
Type			
ACS ST-	1		
ACS ST+	2.9	1.49-5.67	**< 0.002**
Anteriortopography of the infarct			
No	1		
Yes	2.12	1.31-3.43	**< 0.002**
Lower infarct topography			
No	1		
Yes	0.47	0.29-0.76	**< 0.002**
Mirror image			
No	1		
Yes	1.02	0.64-1.62	0.91
Isolated ST offset			
No	1		
Yes	4.01	0.09-0.68	**< 0.007**
LVEF (%)			
≥ 50	1		
< 50	1.79	1.16-2.76	**< 0.008**

Variables (p-value < 25%)	OR brut	[IC 95%]	p- value
DBP (mmHg)			
≥ 60	1		
< 60	2.7	1.75-4.16	**< 10^{-3}**
RR (cycles/minutes)	1.02	0.87-1.20	0.75
HC (beats /minutes)	1.01	1.006-1.02	**< 0.001**
Dyspnea			
No	1		
Yes	2.43	1.50-3.94	**< 10^{-3}**
Cardiac symptomatology			
Typical chest pain	1		
Atypicalchest pain	2.57	1.59-4.15	**< 10^{-3*}**
Killip classification			
Class I	1		
Class II	3.02	1.67-5.46	**< 10^{-3}**
Class III and IV	9.92	5.15-19.09	**< 10^{-3}**
Time to hospital (hours)			
0-6	1.85	0.81-4.20	**0.13**
6-12	1		
12-24	2.34	0.90-6.07	**0.07**
> 24	2.34	0.99-5.57	**0.05**

*CI – Confidence Interval; **IDD – Insulin Dependent Diabetes; ***NIDD – Non-Insulin Dependent Diabetes

Variables (p-value < 25%)	OR brut	[IC 95%]	p-value
Normal LV kinetics			
Yes	1		
No	6.1	1.91-19.46	**< 0.002**
Therapeutic variables			
Thrombolysis			
Yes	1		
No	2.309	1.539-3.465	**< 10^{-3}**
Coronaryangioplasty			
Yes	1		
No	6.8	0.94-50	**0.05**
Evolutionary variables			
Complication (CS. LVF. VT or AVB)			
No	1		
Yes	3.8	2.54-5.69	**< 10^{-3}**
Recurrence			
No	1		
Yes	1.7	0.52-5.56	0.37

gender, ACS type STEMI, SBP below 90 mmHg, serum creatinine > 12 mg/L, the presence of a complication (cardiogenic shock, left ventricular failure, ventricular tachycardia, or atrioventricular block), Killip class II or IV, and the absence of intravenous thrombolysis.

The final predictive model demonstrated good calibration, as indicated by the Hosmer-Lemeshow test (p = 0.906), which is well above the 0.05 threshold (Table 3).

Table 3. Factors predictive of 30-day mortality in the score derivation cohort. Multivariate analysis by logistic regression (n = 1195)

Variables (p-value < 5%)	OR ajusté	[IC 95%]	p-value
Gender			
Male	1		
Female	2.11	1.12-3.98	**0.02**
Type			
NSTEMI	1		
STEMI	4.16	1.58-10.92	**< 0.004**
SBP (mm Hg)			
≥ 90	1		
< 90	2.82	1.086-7.34	**0.03**
Serumcreatinine(mg/l)			
≤ 12	1		
>12	2.14	1.15-3.97	**0.01**
Complication (CS. LVF. VT or AVB)			
Absent	1		
Present	3.33	1.75-6.32	**< 10-3**
Thrombolysis			
Practised	1		
Not practised	2.42	1.25-4.67	**< 0.008**
Killip classification			
Class I	1		
Class II	0.83	0.30-2.32	0.73*
Class III or IV	4.55	1.83-11.33	**< 0.001**

p-value of the Hosmer-Lemeshow test = 0.906 (p> 0.05)

* p-value not significant

Calculation of the 30-day prognostic score

The 30-day prognostic score was calculated using the Hosmer-Lemeshow method, based on the β coefficients of the seven variables retained in the final logistic regression model. The results were rounded to whole numbers, and the prognostic score was obtained by summing the assigned points for each variable. In total, the calculated prognostic score had a maximum of ten points (Table 4).

Validation of the 30-day prognostic score

The discrimination of the prognostic score demonstrated a C-statistic of 0.76 (95% CI [0.728-0.784]) in the derivation cohort and 0.71 (95% CI [0.664-0.754]) in the validation cohort.

The prognostic score effectively identified true positives (sensitivity) in 71% (95% CI [47.8-88.7]) of cases and true negatives (specificity) in 61% (95% CI [55.7-65.8]) of cases in the validation cohort (Fig. 6 and Fig. 7).

Calculation of the positive predictive values of the prognostic score

The estimation of the probability (or risk) of death when the score was positive revealed that, starting from a total of three points, the 30-day mortality risk for a patient hospitalized with ACS was 11%. This risk increased significantly, reaching 50% for a total score of eight points or more.

A prognostic score was thus developed based on a patient population from the Oran region, which was designated as the ASCO score (Acute Coronary Syndrome Score at Oran).

The ASCO score calculation method was applied to three different patients hospitalized for ACS (Table 6).

Table 4. Calculation of 30-day prognostic score points for each predictive factor

Gender	Points	SBP (mmHg)	Points	Killip classification	Points	Type SCA	Points
Male Female	0 1	≥ 90 < 90	0 1	Class I Class II Class III Class IV	0 0 2 2	NSTEMI STEMI	0 2
Serum creatinine (mg/l)	Points	Complications	Points	Thrombolysis	Points		
≤ 12 > 12	0 1	Absent Present*	0 2	Practised Not practised	0 1		
*Presence of one of the following complications: CS, LVF, VT or AVB							

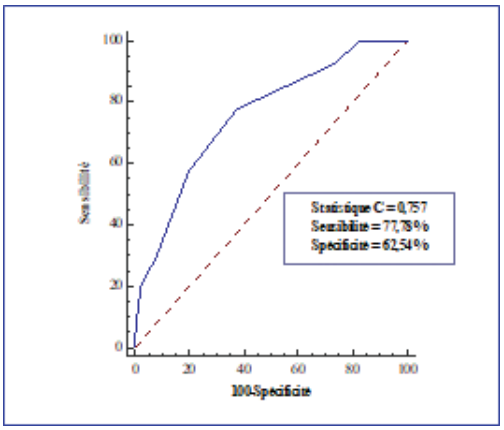

Fig. 6. Prognostic score discrimination in the derivationcohort (n = 1195)

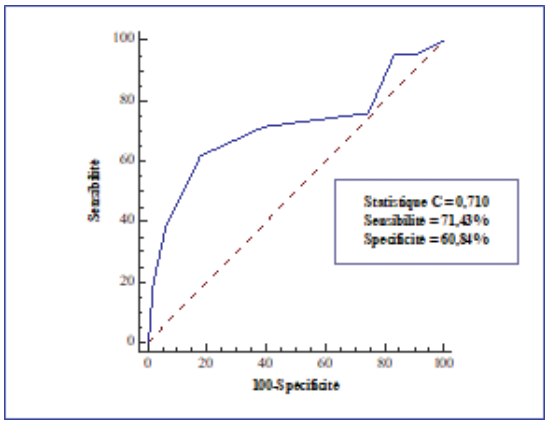

Fig. 7. Prognostic score discrimination in the validation cohort (n = 497)

Table 5. Estimated probability of death at 30 days based on total prognostic score points

Total points score	≤ 2	3	4	5	6	7	≥ 8
Probability of death at 30 days (%)	≤ 7	11	15	19	34	42	**≥ 50**

Table 6. Method for calculating the ASCO score nomogram, applied to three patients hospitalized for ACS

Predictive factors		**Patient n°1**	**ASCO**	**Patient n°2**	**ASCO**	**Patient n°3**	**ASCO**
Gender Male Female	 = 0 = 1	Male	**0**	Male	**0**	Female	**1**
SBP (mmHg) ≥ 90 = 0 < 90 = 1	 = 0 = 1	130	**0**	97	**0**	150	**0**
Killip Class I Class II Class III Class IV	 = 0 = 0 = 2 = 2	Class I	**0**	Class III	**2**	Class III	**2**
Type NSTEMI STEMI	 = 0 = 2	ACS ST+	**2**	ACS ST-	**0**	ACSST+	**2**
Serum creatinine (mg/l) ≤ 12 > 12	 = 0 = 1	09	**0**	13,7	**1**	10	**0**
Complication Absent Present	 = 0 = 2	Absent	**0**	LVF	**2**	VT	**2**
Thrombolysis Practised Not practised	 = 0 = 1	Yes	**0**	No	**1**	No	**1**
Total points			**2**		**6**		**8**
Probability of death at 30 days (%)			**≤ 7**		**34**		**≥ 50**

Discussion

To establish the ASCO score, we recruited a cohort of 1692 eligible patients from the Cardiology Departments of Oran (Northwester Algeria) over three consecutive years.

Our study population was predominantly male (75%), consistent with findings from the Acute Coronary Events – a Multinational Survey of Current Management Strategies (ACCESS) registry conducted in three Maghreb countries (76%) [23]. This proportion is comparable to those reported in the Thrombolysis in Myocardial Infarction (TIMI) [24], Euro Heart [25], and acute coronary syndrome in Brazil hospital (Hcor) [4] score population databases but differs from the Global Registry of Acuts Coronary Events (GRACE) registry (69%) [26]. The over representation of males may partly be attributed to the well-established predominance of coronary artery disease in men [27, 28].

Increasing age correlates with prolonged exposure to other risk factors. However, the role of age in atherosclerosis pathophysiology remains complex [29]. Our analysis identified a significant proportion (14%) of ACS cases occurring in patients under 47 years of age. According to S. Joussein-Remacle [30] and J. B. Estève [31], the incidence of premature coronary disease is rising, accounting for approximately 10% of all coronary patients.

In our study, patient inclusion criteria were broad, encompassing both ACS type STEMI and NSTEMI, regardless of Killip class. According to the literature, observational study-derived scores tend to be less restrictive, capturing ACS in its broadest spectrum [13, 26, 32-34]. Conversely, many classic scores originate from randomized studies, with selective samples and specialized centers [10, 11, 13, 32]. Similarly, contemporary scores have excluded patients who did not undergo percutaneous angioplasty [25, 35-38]. According to E. Puymirat [5], in-hospital complications are more frequent among patients treated conservatively, whereas invasive strategies are associated with reduced overall and cardiovascular mortality.

In the ASCO cohort, more than two-thirds of patients (78%) had STEMI, diagnosed via ECG and confirmed by enzymatic assays. This trend appears to be reversed in the populations analyzed for the Euro Heart [25] and Hcor [4] scores. The GRACE study [26] reported approximately equal proportions of STEMI and NSTEMI. These variations highlight the temporal and geographical variability in ACS epidemiology.

Our study reported an in-hospital mortality rate of 8% and a 30-day mortality rate of approximately 9%. This is higher than rates found in other scoring populations: 7% in TIMI [11], 5% in GRACE [26], and only 2% in Euro Heart [25]. Lower mortality in clinical studies can be attributed to patient selection (often younger) and adherence to guideline-recommended treatments during and after hospitalization [33, 11]. The higher early mortality observed in our study may be partially explained by admission logistics, 30% of deceased patients had been transferred from other health facilities, often from neighboring provinces, and delayed admission to intensive cardiac care, which exceeded 12 hours in 41% of deceased patients.

The primary outcome in the ASCO study was 30-day all-cause mortality following patient admission. Literature reviews reveal significant variability in evaluation criteria, with some studies combining multiple endpoints [39, 40]. M. E. Bertrand [41] emphasized that ACS complications, regardless of type, primarily arise within the first 30 days and are closely linked to atherothrombotic risk. Beyond this period, prognosis depends largely on the progression of atherosclerotic disease. The ASCO score is therefore calculated based on 30-day follow-up after admission. Notably, follow-up durations and assessment criteria vary widely among published studies [24, 34, 38, 40, 42-46].

At the conclusion of multivariate analysis (p-value < 0.05), seven variables were retained in the final predictive model: female gender, ST+ ACS, systolic blood pressure (SBP) < 90 mm Hg, serum creatinine > 12 mg/L, presence of complications (cardiogenic shock, left ventricular failure, ventricular tachycardia, or atrioventricular block), Killip class III or IV, and lack of thrombolysis.

Comparison of the predictive factors in the ASCO model with existing literature revealed that several variables overlap with other prognostic scores for early mortality, including ST+ ACS, low SBP, elevated serum creatinine, and advanced Killip class [4, 11, 24, 26, 36].

The inclusion of ST+ ACS in our final multivariate analysis is expected, as it is widely recognized as a major predictor of early mortality. Retaining hypotension (low SBP) in our model is also consistent, given its predictive value for ischemic risk in nearly all classic scores [11, 26, 34, 42, 47, 48].

According to A. Bellemain-Appaix et al., heart failure (Killip class ≥ II) increases one-month coronary morbidity and mortality by a factor of four [23]. Similarly, Christopher B. et al. [11, 26] were the first to demonstrate that elevated serum creatinine at admission is an integrative marker for severe ischemic events (death or infarction) in the GRACE registry. This factor is also incorporated into more recent scores such as Hcor [4] and Acute Catheterization and Urgent Intervention Triage Strategy-Percutaneous Coronary Intervention (ACUITY-PCI) [36].

The prognosis of ACS, particularly in its early phase, remains unpredictable. Among complications occurring in the acute phase, heart failure and cardio-

genic shock have been widely documented as predictive factors in literature. Heart failure is a component of early mortality risk stratification in the National Cardiovascular Data Registry for Catheterization Percutaneous Coronary Intervention (NCDR-CathPCI) risk score [37]. Cardiogenic shock, present in 5-10% of ACS cases, typically occurs within the first 24 hours [49] and is included in the Euro Heart score [25]. Atrioventricular blocks, which complicate 25% of ACS cases, predominantly emerge within the first hours [50]. Ventricular tachycardia is the most severe rhythm disorder, posing a high risk of sudden cardiac arrest, especially after 48 hours [51].

A key and novel feature of the ASCO score is the inclusion of "female gender" as a predictive factor. Coronary artery disease remains a largely underrecognized epidemic in women, ranking as the leading cause of premature death across all age groups [52]. This contradicts the misconception that coronary artery disease is predominantly a male condition.

One possible explanation for this paradox is women's longer life expectancy [27]. In our cohort, women were older than men (mean age 65 vs. 59, $p < 0.001$). Additionally, 90% of female patients were postmenopausal. Early natural or surgical menopause is itself a cardiovascular risk factor [29]. According to Madika A.L. [53], several factors contribute to excess mortality in women: older age at diagnosis, higher prevalence of comorbidities (hypertension, diabetes), more severe or atypical presentation, increased iatrogenic risk, and disparities in early management.

Time to hospital in the derivation cohort was greater than six hours in 49% of patients with ACS. When stratified by type of ACS, this time was greater than six hours in 44% of STEMI versus 67% in NSTEMI. The time to hospital remains long compared to developed countries, this is partly explained by the remarkable diversity of the residence' places of ACS patients admitted to the UHC and UHE and the distance of certain provinces, such as Relizane, Tiaret, Saida and Naama located respectively 107, 181, 121 and 273 km from Oran. However, this delay is better than the one found in the ACCESS study in the Maghreb countries, which was greater than six hours in 50% of STEMI, and that found in a study carried out in Chad, which found an admission delay exceeding twelve hours in 79.17% of cases [6, 54].

Our findings confirm that the final ASCO predictive model is well-calibrated, with superior accuracy in calibration ($p = 0.906$) compared to other scores [4, 25, 36].

The ASCO, C-statistic was 0.76 [95% CI (0.73-0.78)] in the derivation cohort and 0.71 [95% CI (0.66-0.75)] in the validation cohort. According to Hosmer and Lemeshow's interpretation, ASCO demonstrated reasonable discrimination ($0.7 \leq AUC < 0.8$) in both cohorts. This performance is comparable to the Hcor score ($C = 0.71$) and superior to the ACUITY-PCI score ($C = 0.70$).

Furthermore, ASCO demonstrated a sensitivity of 71% and a specificity of 61% in the validation cohort.

Despite its strengths, our study has limitations. Due to missing data constraints, some explanatory variables were subjective. The highest percentage of missing data involved weight and height at admission, although overall obesity was assessed by the attending physician and corrected using Body Mass Index (BMI) when available. Similarly, left ventricular ejection fraction measurement was not standardized, being assessed via either the Simpson Biplane method or, when unavailable, the Teichholz method.

At one month of follow-up, among the patients who received intravenous thrombolysis (673), 34 (5%) underwent secondary coronary angioplasty and 10 (1.5%) required rescue coronary angioplasty. It should be noted that beyond one month, access to information was a challenge for us, given that patients are monitored at local healthcare facilities.

Conclusion

The ASCO score is composed of seven independent dichotomous variables for a maximum of ten points. It includes traditional haemodynamic and clinical variables (ACS type STEMI, low SBP, high serum creatinine, and advanced Killip class), unusual variables (presence of a complication and non-use of thrombolysis), and an innovative element represented by the variable "female sex". The ASCO score showed good intrinsic and extrinsic performance, with good calibration ($p = 0.906$) and reasonable discrimination ($C = 0.71$) in the validation cohort.

A larger-scale study, involving a new population and different hospital settings, is needed to test the transportability of ASCO, particularly its geographical validity. Thus, we propose to practitioners a decision-support tool that is simple to calculate, reproducible, capable of predicting 30-day mortality, validated, time-efficient, and adapted to emergency medical practices.

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