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Derivation and validation of a simple risk score for predicting 1-year mortality in stroke

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Abstract *Context* Numerous models have been presented for the prognosis in acute stroke; however they have been criticized for being difficult to use, and few have been validated in independent samples. *Objectives* To develop simple risk score models for 1-year mortality in acute stroke in patients > 60 years old and validate the models. *Design* From a cohort of 2321 consecutive patients > 60 years of age with acute stroke in one hospital, we randomly selected 800 patients for chart review. Among 737 patients with validated acute stroke, we randomly split the sample into (1) a derivation (60 %; n = 442) and (2) a validation sample (40 %; n = 295). We used logistic regression to develop three models with 2–4 covariates and a corresponding risk score from the derivation sample. The models were validated using area under the receiver operating curves. *Results* Three risk score models for 1-year mortality after stroke were developed using combinations of age, Canadian Neurological Scale score (CNS-score) (≤ 3.5 = 0, > 3.5 = 1), Charlson comorbidity index and stroke type (ischemic = 0, hemorrhagic = 1). Both 2-variable (Age – 60 + (30*CNSscore)), 3-variable (Age – 60 + (30*CNSscore) + 4*Charlson)) and 4-variable (Age – 60 + (25*CNSscore) + (5*Charlson) + (18*Stroke type)) models reliably predicted the outcome with an area under the receiver operating curve ranging 0.71 to 0.72. *Conclusions* Simple models incorporating two to four covariates reliably predicted 1-year mortality. Such models can be used to stratify prognosis in clinical practice, research or intervention trials.

Key words stroke · risk assessment · statistical models · mortality · epidemiology

Background

Prediction of prognosis in acute stroke is difficult, in particular in the early phases. Clinical prognostic models in acute stroke would be helpful in predicting outcome of future patients, informing patients and their relatives of reasons for treatment and clinical decisions, creating clinical risk groups and stratifying patients by disease severity in clinical trials.

A recent review identified 238 articles describing prognostic models in acute stroke [5]. After quality assessment, the authors reviewed 78 articles describing 68 different studies and 83 separate prognostic models. Most models predicted functional outcomes such as independent living, and only a few studies reported validated mortality models [9, 24–26, 28]. Further, most models had serious deficiencies in internal and statistical validity. Many had limited generalizability, none had been adequately validated and some of the older models

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are difficult to use in clinical practice [5, 24]. Therefore, there clearly is a need for new prognostic models for the mortality outcome of acute stroke.

In this study, we wanted to develop a new model for 1-year mortality in acute stroke with subsequent validation in an independent cohort of stroke patients. To increase the applicability of the models, we derived the models in a hospital with a geographical catchment area and focused on simple models with scores based on clinical variables that could be collected within the first 24 hours after hospital admission.

Methods

Subjects

The Akershus University Hospital is the only provider of hospital care for its catchment population of 300,000. From 1 January 1993, patients > 60 years old presenting with symptoms of acute stroke were admitted to a stroke unit in the Department of Neurology or 1 of 5 general medical wards in the Department of Medicine.

In this study, we included all 2321 consecutive patients > 60 years who were admitted between 1 January 1993 and 31 December 1998 with first or recurrent acute stroke, independent of the time between stroke onset and admission. Inclusion criteria were the principal discharge diagnosis codes 431, 432, 434.1, 434.9, 436, 437, and 437.9 of the International Classification of Diseases, 9th revision (ICD-9). Patients with transitory ischemic attacks (TIA), subarachnoidal hemorrhages and subdural hematomas were excluded. Based on reports of low probability of stroke among patients with several of these ICD-9 codes [1, 3, 7, 10, 13], to reduce problems with misclassification we excluded patients (n = 52) with the codes 432 (other or unspecified intracerebral hemorrhage), 433 (occlusion and stenosis of precerebral arteries) and 437 (other and ill-defined cerebrovascular disease).

From this cohort of 2269 patients, we randomly selected 800 patients for medical record review, of whom we expected approximately 93 % to have a valid diagnosis of acute stroke [20]. After chart review, we excluded 63 patients because of diagnostic errors or unavailability of the chart. Hence, 737 patients had a validated stroke according to our criteria. After data collection, in accordance with the protocol, this sample was randomly split into two parts for further analysis: (1) a derivation sample of 442 patients (60 %) to derive a mortality model, and (2) a validation sample of 295 patients (40 %) devised by the protocol. This 60/40 split was considered a feasible compromise to have a satisfactory sample size both in the derivation and validation samples. To improve the prognostic reliability of a model, it seems reasonable that the derivation sample is larger than the validation sample and this approach has also been used by others [8].

Data collection and analysis

Predictors of mortality and definitions

Potential candidate predictors were identified in the literature and selected according to clinical relevance and availability [2, 5, 6, 14, 21]. The selected variables were vital signs at hospital presentation or data extractable within the first 24 hours, such as demographics, presenting clinical features, laboratory values, and pre-existing comorbidity. All charts were retrospectively reviewed. Details about systolic blood pressure in mmHg (categorized into tertiles), body temperature in °C, atrial fibrillation, hemoglobin (categorized into tertiles), serum glucose (categorized into tertiles) at presentation were recorded. Categorical variables, such as comorbidity, were coded as present or absent.

Comorbidity was recorded using the Charlson comorbidity index [4], including prior myocardial infarction, congestive heart failure, peripheral vascular disease, cerebrovascular disease, dementia, pulmonary disease, collagenoses, liver disease, peptic ulcer disease, diabetes mellitus, malignancies, HIV/AIDS, and renal disease. CT and MRI results were recorded when available.

Neurological status was assessed at hospital presentation using a retrospective algorithm of the Canadian Neurological Scale (CNS) [11], which evaluates level of consciousness, orientation, speech, facial weakness, and proximal and distal arm weakness. The CNS score can be reliably estimated retrospectively from medical records of routine neurological assessments. One of the physicians among the authors (OGS, MD, KS) reviewed each chart. Reliability of diagnosis and CNS assessments in a subset of our sample was high, as previously reported [19].

Definition of outcome

The study outcome was defined as death within 1 year after hospital admission. Information on death was collected through computer linkage to the Norwegian National Population Register. In Norway, every citizen has a unique personal ID number, and all deaths are reported to this register. This register is complete and is continuously updated; however, there might be a lag of a few weeks before it is updated. In theory, some patients might have migrated abroad during the 1-year follow-up and therefore lost to follow-up. We do not have detailed accounts of the number of people in the cohort who had moved abroad, though we think that in this cohort migration would be very rare. We included deaths until November 1, 1999 in our analyses [20].

Model derivation

Odds ratios (OR) for 1-year mortality were estimated in the derivation set (n = 442) using bivariate logistic regression analysis. Results of univariate and multivariable analysis are presented as OR with 95 % confidence intervals (CI) and p-values. OR for age is presented for an increase of 10 years.

In multivariable logistic regression analysis, we included independent variables with $P \leq 0.20$ from the univariate analysis. All variables were initially forced into the model. We then manually removed covariates one by one, examining changes in parameters and stability of the model. Spearman's rank correlation between the independent variables in the multivariable analysis was all < 0.7. We checked the models for multicollinearity, looked for interactions and assessed calibration using the Hosmer-Lemeshow chi square statistic ($P > 0.05$).

We aimed at developing models for clinical prediction with a small number of variables with clear and practical cut-offs. For several variables such as the CNS score, we assessed continuous scores, divisions into quartiles of the distribution in the derivation sample, and a dichotomy based on the 1–25th percentiles (Q1) vs. the 26–100th percentiles (Q2-Q4).

Risk scores for 1-year mortality were developed from the best-fitting 4-, 3- and 2-variable logistic regression models by using a regression coefficient-based scoring method [15, 16]. We divided the regression coefficients for the various risk factors by the age coefficient, multiplied by 10 and rounded the resulting coefficients to the nearest integer. The overall risk score was calculated by adding the components.

Model validation

Discriminative capacity of the models was validated in the validation set (n = 295) using the area under the receiver operating characteristic (ROC) curves with non-parametric 95 % CIs. For comparison, we also present the discriminative capacity in the derivation cohort.

We evaluated the predictive capacity of the risk scores to assess 1-year mortality in the derivation and validation sets through stratification according to risk score quintiles for each of the three models.

Mortality rates in the derivation and validation cohorts within risk score groups and overall were compared using the chi-square test. Finally, we illustrated survival over 1 year in the validation cohort with a Kaplan-Meier plot stratified according to risk class.

We estimated risk class-specific mortality rates in the total sample with non-parametric 95 % CIs. All analyses were conducted using the Stata version 8.2 (Stata Corp, College Station, TX, USA) statistical software.

Results

The derivation set comprised 442 patients and the validation set 295. The distributions of age, sex, clinical characteristics, comorbidity and time from stroke until admission were comparable in the two sets (Table 1).

Predictors of mortality

The univariable logistic regression analysis for all potential candidate variables is shown in Table 2.

Multivariable models for 1-year mortality are shown in Table 3. In the multivariable analysis age, CNS score (dichotomized), Charlson comorbidity index and stroke type (dichotomized) were significant independent predictors of 1-year mortality. Other significant variables in univariable analysis, such as atrial fibrillation, body temperature, systolic blood pressure and glucose, had little influence on the regression coefficients or led to poorly fitting models.

In addition to the 4-variable model, we also derived 3- and 2-variable models with satisfactory fit (Table 3). Using the CNS score as a continuous variable or categorical variable based on quartiles resulted in poorly fitting models (Hosmer-Lemeshow chi-square, HL(p) < 0.05), hence all models use the dichotomy Q1 (score ≤ 3.5) vs. Q2-Q4 (> 3.5).

In the derivation set, calibration of the logistic regression models was good and discrimination acceptable with area under the ROC curve ≥ 0.75 (Table 4).

Risk scores

In the derivation set, our analyses resulted in simplified scoring rules using 2 to 4 of the following variables: Age (age at admission, maximum 100); CNSscore (Canadian neurological scale score dichotomized: ≤ 3.5 (= 0), > 3.5

Table 1 Descriptive statistics at the index admission (n = 737)

	Derivation set (n = 435–442)	Validation set (n = 291–295)	P
Age in years, mean (SD)	77.3 (7.6)	76.7 (8.7)	0.34
Male sex, number (%)	212 (48)	150 (51)	0.44
Comorbidity, number (%)			
Previous myocardial infarction	90 (21)	53 (18)	0.42
Previous stroke/TIA	144 (33)	84 (29)	0.24
Ever cancer	56 (13)	29 (10)	0.22
Known diabetes	78 (18)	44 (15)	0.32
Charlson comorbidity index, number (%)			0.24
0	124 (28)	102 (35)	
1	141 (32)	84 (29)	
2	86 (20)	61 (21)	
3	57 (13)	27 (9)	
≥ 4	32 (7)	20 (6)	
At hospital admission			
Systolic BP in mmHg, mean (SD)	173 (33)	174 (32)	0.53
Body temperature, mean (SD)	36.8 (0.8)	36.9 (0.7)	0.56
Atrial fibrillation, number (%)	117 (26.5)	61 (21.2)	0.80
CNSscore, mean (SD)	7.1 (3.6)	7.5 (3.4)	0.11
Hemoglobin in g/100 mL, mean (SD)	13.7 (1.8)*	13.9 (1.6)[a]	0.20
Serum glucose in mmol/L, mean (SD)	7.1 (2.7)[b]	7.2 (2.7)[c]	0.64
Time from stroke to admission, number (%)			0.22
< 6 h	201 (46)	114 (39)	
6–12 h	107 (25)	91 (31)	
13–24 h	45 (10)	33 (11)	
25–48 h	30 (7)	22 (8)	
> 48 h	54 (12)	30 (10)	

* n = 413
[a] n = 273; [b] n = 366; [c] n = 252

Table 2 Mortality risk in univariable logistic regression analysis in the derivation set

	Odds ratio	95 % CI	P
Female sex	1.31	0.88–1.93	0.18
Age, increase of 10 years	1.97	1.49–2.60	0.0001
Cancer (ever)	1.2	0.67–2.13	0.54
Diabetes	1.32	0.80–2.18	0.27
Previous stroke/TIA	0.85	0.56–1.30	0.46
Previous myocardial infarction	1.18	0.73–1.91	0.49
Atrial fibrillation	1.57	1.02–2.43	0.04
Temperature, increase of 1 °C	0.77	0.58–1.02	0.06
Systolic blood pressure, mmHg			
Highest tertile (≥ 161)	0.31	0.14–0.70	0.004
Mid tertile (121–160)	0.37	0.16–0.86	0.02
Lowest tertile (≤ 120)	1		
Hemoglobin, g/100 mL			
Highest tertile (males > 17, females > 16)	0.67	0.21–2.11	0.49
Mid tertile (males 13–17; females (11–16)	0.46	0.27–0.81	0.007
Lowest tertile (males < 13; females < 11)	1		
Glucose, mmol/L			
Highest tertile (> 11)	2.12	1.00–4.49	0.05
Mid tertile (7–11)	1.77	1.10–2.86	0.02
Lowest tertile (< 7)	1		
CNSscore, ≤ 3.5 vs. > 3.5	6.1	3.86–9.64	0.0001
Charlson comorbidity index, increase of 1	1.24	1.08–1.43	0.003

TIA transitory ischemic attack; *CI* confidence interval

Table 3 Mortality risk in multivariable logistic regression analysis in the derivation set

2-variable model	Odds ratio	95 % CI	P
Age, increase of 10 years	1.79	1.36–2.48	< 0.001
CNS (≤ 3.5 vs. > 3.5)	5.57	3.19–8.20	< 0.001
3-variable model	**Odds ratio**	**95 % CI**	**P**
Age, increase of 10 years	1.82	1.35–2.46	< 0.001
CNS (≤ 3.5 vs. > 3.5)	5.62	3.50–9.04	< 0.001
Charlson comorbidity index, increase of 1	1.28	1.10–1.50	0.002
4-variable model	**Odds ratio**	**95 % CI**	**P**
Age, increase of 10 years	1.90	1.40–2.57	< 0.001
CNS (≤ 3.5 vs. > 3.5)	5.06	3.12–8.20	< 0.001
Charlson comorbidity index, increase of 1	1.33	1.13–1.56	< 0.001
Stroke type, hemorrhagic vs. ischemic or unknown	3.19	1.46–7.01	0.004

(= 1)); Stroke type (ischemic = 0, hemorrhagic = 1); Charlson (Charlson comorbidity index, integer (0, 1, 2, 3, 4, ≥ 5). The models were:

2-variables (range 0–70):

Risk score = Age – 60 + (30*CNSscore)

3-variables (range 0–90):

Risk score = Age – 60 + (30*CNSscore) + (4*Charlson)

4-variables (range 0–108):

Risk score = Age – 60 + (25*CNSscore) + (5*Charlson) + (18*Stroke type)

Model validation

In the derivation set (n = 295), the three logistic regression models retained good calibration (HL(p) > 0.5) and good discrimination with an area under the ROC curve ranging 0.71 to 0.72, although slightly lower than in the derivation set (Table 4). There was little difference in discrimination between the models.

Using the 2-variable risk score, 1-year mortality in the derivation cohort ranged from 16 % in the lowest risk group (class 1) to 70 % in the highest risk group

Table 4 Discrimination and calibration performance of the logistic regression models in the derivation set

Set/model	ROC curve				
	N	AUC	95 %CI	HL chi square	HL(p)
Derivation set					
2-variable	442	0.747	0.696 to 0.796	5.52	0.701
3-variable	440	0.758	0.710 to 0.806	9.3	0.318
4-variable	440	0.777	0.732 to 0.822	9.39	0.310
Validation set					
2-variable	295	0.709	0.647 to 0.770	6.49	0.593
3-variable	294	0.708	0.646 to 0.769	4.86	0.773
4-variable	294	0.721	0.660 to 0.782	4.86	0.772

ROC receiver operating characteristic; *AUC* area under the curve; *HL* Hosmer-Lemeshow; *HL(p)* p-value for HL chi square; *CI* confidence interval

(class 5). In the validation cohort the mortality ranged from 20 % (class 1) to 73 % (class 5). There was no difference in mortality between the derivation and validation sets within each risk class across the entire spectrum of risk with this model or in the overall mortality (Table 5).

Similarly, there was no difference in risk class-specific mortality between the derivation and validation cohorts in the 3-variable model. In contrast, when applying the 4-variable model there was a significant difference in risk class-specific mortality in the two lowest risk classes (Table 5).

The Kaplan-Meier plot demonstrates that there was a difference in mortality between the three highest risk classes that started early and was retained throughout the year (Fig. 1). There was little difference between the two lowest risk classes.

Finally, we rounded the cut-offs to integer scores in the 2-variable model to ease its clinical application. In the total sample (n = 737), 1-year survival in risk class 1 (score 0 to 11, n = 161) was 18 % (95 % CI 12 to 25 %); class 2 (score 12 to 17, n = 145) 21 % (14 to 28 %); class 3 (score 18 to 24, n = 169) 33 % (26 to 40 %); class 4 (score 25 to 45, n = 138) 51 % (42 to 59 %); class 5 (score 46 to 70, n = 124) 72 % (63 to 79 %).

Discussion

We have demonstrated that simple models using data available during the initial hours of hospital presentation predicted 1-year mortality in a cohort of unselected stroke patients > 60 years. The models included acute physiological parameters, chronic disease comorbidities and an index of neurological deficit at admission. Simple models with the variables age, CNSscore, Charlson

Table 5 Comparison of risk class-specific mortality rates in the derivation and validation cohorts (risk class based on quintile of the derivation cohort)

	Risk class	Risk score	Derivation set		Validation set		P*
			N	% who died	N	% who died	
2-variable model	1	0 to 11.70	88	16	66	20	0.61
	2	11.71 to 17.40	88	19	54	26	0.46
	3	17.41 to 24.21	89	27	72	38	0.31
	4	24.22 to 45.20	88	47	59	49	0.86
	5	45.21 to 70	89	70	44	73	0.88
ROC curve area (95 % CI)			0.747 (0.698 to 0.796)		0.709 (0.647 to 0.770)		
3-variable model	1	0 to 16.12	88	15	62	21	0.41
	2	16.13 to 23.01	88	16	68	28	0.14
	3	23.02 to 30.70	88	27	57	35	0.47
	4	30.71 to 49.54	88	51	63	51	0.98
	5	49.55 to 90	88	69	44	68	0.95
ROC curve area (95 % CI)			0.758 (0.711 to 0.806)		0.697 (0.635 to 0.759)		
4-variable model	1	0 to 18.12	88	9	79	23	0.04
	2	18.13 to 25.17	88	17	74	31	0.04
	3	25.18 to 33.75	88	35	51	33	0.87
	4	33.76 to 49.08	88	48	43	58	0.53
	5	49.08 to 108	88	69	47	66	0.86
ROC curve area (95 % CI)			0.777 (0.732 to 0.822)		0.711 (0.650 to 0.773)		
Overall			442	36	295	39	0.55

* Chi square test; *ROC* receiver operating characteristic; *CI* confidence interval

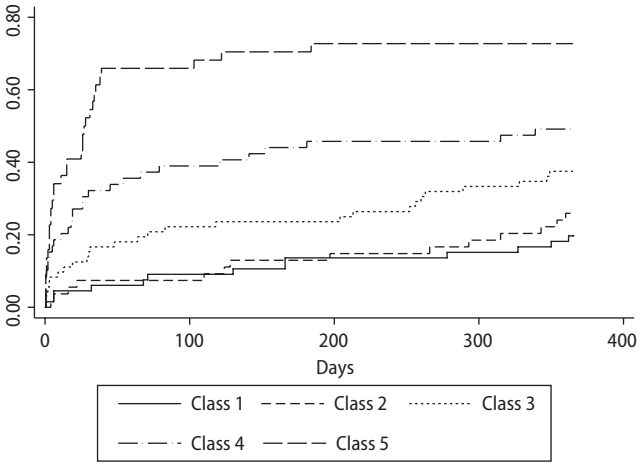

Fig. 1 Mortality during the first year following hospital admission in the validation cohort, stratified according to risk class category in the 2-variable model (risk classes 1 to 5)

comorbidity index and stroke type best predicted 1-year mortality. In all models a rising risk score was associated with increasing one-year mortality.

The models in the present study were derived and validated in a sample of unselected patients in a hospital with a geographical catchment area, being the only hospital providing care for stroke patients in this area. In contrast, some previous models were developed in tertiary care referral hospitals or based on multicenter registry data [8, 24–26].

We included both patients with ischemic and hemorrhagic stroke in our sample, whereas most other studies have developed separate models for ischemic or hemorrhagic stroke [8, 24–27]. In the present study, we used a reasonably large sample size for deriving the model and validated the model. To our knowledge our study is the first to establish a reliable and validated prognostic index by means of a point system for patients with ischemic and hemorrhagic stroke.

The selection of outcome in prediction models after stroke varies considerably. Some studies of mortality have focused on shorter term outcomes such as 3-month or 100-day mortality [8, 25–27], and only a few studies have reported on 1-year mortality as in the present study [22, 24]. Hence, despite the large number of prediction models following stroke, there are actually few previous studies of 1-year mortality for comparison.

For example, 30-day and 1-year mortality risk indices were developed in an Australian tertiary care referral hospital setting. The resulting risk index for 1-year mortality used neurological deficits (impaired consciousness, dysphagia, or urinary incontinence), bilateral affection, comorbidity (ischemic heart disease, peripheral vascular disease or diabetes mellitus), hyperthermia and hyperglycemia without known diabetes as covariates. The results in the present study are similar to the Australian results [24, 25].

Two studies from the German Stroke Study Collaboration have reported that age, stroke severity and fever predicted mortality over 100 days following ischemic stroke [9, 28], while fever was omitted in a third study [26]. Those findings are similar to the 2-variable risk score model in the present study, a single-center study also including hemorrhagic strokes and with 1-year mortality as outcome.

Other studies of stroke outcomes have focused on functional outcome or used the results of imaging studies in conjunction with clinical variables as covariates in the models. Therefore, they are difficult to compare with the present study. Body temperature did not predict mortality in the present study, in contrast to some previous studies. Both elevated [12, 14] and reduced body temperature [2] have been associated with increased mortality.

Previous prognostic models for mortality following stroke have often been logistic regression models that have been difficult for practitioners to use, although recently some simpler predictive models have appeared [24, 25]. In the present study, the models were not based on prospectively collected data in a study, but on retrospectively collected information from routine medical records. We think this supports the utility of the models in clinical practice.

Some methodological aspects should be noted. The present study was retrospective and recorded data on predictors by medical record abstraction, which has inherent limitations in data quality and reliability. However, previous studies in patients with stroke have indicated that retrospectively collected variables remain reliable and valid [19, 29].

Another limitation of the study is the lack of stroke subtype differentiation among patients with ischemic stroke, for example small vessel disease, large vessel disease, or cardioembolic stroke which have been shown to influence the prognosis [17].

The models in the present study were based on patients > 60 years of age from one hospital, also including severely ill patients. Moreover, the time from stroke onset to hospital admission varied, about 90 % of the patients were admitted to hospital within 48 hours after symptom onset. Therefore, one should be careful about generalization beyond this age group and to other settings or countries. The optimal method for translating regression coefficients into an integer-based simple scoring rule is far from certain [23]; however we have used a standard and commonly used method. The use of a split-sample approach enabled us to validate the models in an independent sample. Validation might have been done in a completely different population outside our base cohort, which would have strengthened the external validity of our findings. However, only some sim-

ilar studies have validated their models in independent samples.

Implications and conclusions

In a derivation cohort of stroke patients >60 years, we have developed simple models that reliably predicted 1-year mortality in the validation cohort. The models included easily available variables, such as major comorbidities and vital signs from the routine assessment at hospital admission including the neurological examination. These models could be used in practice, because the variables can reliably be obtained retrospectively from medical charts. In a clinical setting, the models can be used to identify patients with a high mortality risk, in order to tailor and guide treatment and avoid exposing patients with a good prognosis to potentially dangerous treatments. The information from such models can also be useful when informing patients or relatives about prognosis and treatment, and in planning of discharge and rehabilitation. Further, the models can be useful in clinical trials, to guide estimates of sample size, to improve risk stratification and patient selection, and to adjust for case-mix in comparisons of outcome between studies or institutions. However, the final models and corresponding risk scores should be validated externally in other samples and preferably in prospective studies.

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