Predictive Variables for Mortality After Acute Ischemic Stroke

Angela M. Carter, PhD; Andrew J. Catto, PhD; Michael W. Mansfield, DM; John M. Bamford, MD; Peter J. Grant, MD

Background and Purpose—Stroke is a major healthcare issue worldwide with an incidence comparable to coronary events, highlighting the importance of understanding risk factors for stroke and subsequent mortality.

Methods—In the present study, we determined long-term (all-cause) mortality in 545 patients with ischemic stroke compared with a cohort of 330 age-matched healthy control subjects followed up for a median of 7.4 years. We assessed the effect of selected demographic, clinical, biochemical, hematologic, and hemostatic factors on mortality in patients with ischemic stroke. Stroke subtype was classified according to the Oxfordshire Community Stroke Project criteria. Patients who died 30 days or less after the acute event (n=32) were excluded from analyses because this outcome is considered to be directly attributable to the acute event.

Results—Patients with ischemic stroke were at more than 3-fold increased risk of death compared with the age-matched control cohort. In multivariate analyses, age, stroke subtype, atrial fibrillation, and previous stroke/transient ischemic attack were predictive of mortality in patients with ischemic stroke. Albumin and creatinine and the hemostatic factors von Willebrand factor and β-thromboglobulin were also predictive of mortality in patients with ischemic stroke after accounting for demographic and clinical variables.

Conclusions—The results indicate that subjects with acute ischemic stroke are at increased risk of all-cause mortality. Advancing age, large-vessel stroke, atrial fibrillation, and previous stroke/transient ischemic attack predict mortality; and analysis of albumin, creatinine, von Willebrand factor, and β-thromboglobulin will aid in the identification of patients at increased risk of death after stroke. (Stroke. 2007;38:1873-1880.)

Key Words: β-thromboglobulin ■ cerebral infarction ■ mortality ■ stroke ■ von Willebrand factor

Stroke is a major healthcare issue worldwide representing the third most common cause of death in the United Kingdom.1 The Oxford Vascular Study reported the incidence of cerebrovascular events was 1.2-fold higher than coronary events.2 Approximately 50% of cerebrovascular events were in those aged under 75 years despite previous indications that stroke was more a disease of the elderly population.2 Although stroke incidence and subsequent mortality have declined in recent years attributable to improved risk factor management,3 stroke can be considered to represent a greater healthcare burden than acute coronary disease resulting from residual disability. A greater understanding of the underlying causes of stroke and subsequent mortality will be required to establish appropriate prevention and treatment strategies.

Prospective studies have identified risk factors for stroke and stroke mortality, including advancing age, hypertension, diabetes, smoking, and atrial fibrillation (AF).2,4–7 Acute thrombosis is central to the pathogenesis of ischemic stroke and elevated levels of hemostatic factors, including fibrinogen and tissue plasminogen activator (tPA), predict acute stroke.8,9 Case–control studies, including the present cohort, also identified elevated levels of hemostatic factors, including fibrinogen, von Willebrand Factor (vWF), and β-thromboglobulin (βTG), in subjects with acute stroke.10–12 The relationship between biochemical, hematologic, and hemostatic factors and poststroke mortality, however, has not been extensively studied.

The aims of this study were to (1) determine all-cause mortality in a hospital-based cohort with ischemic stroke compared with an age-matched healthy cohort, (2) identify demographic and clinical variables predictive for mortality after ischemic stroke, (3) identify routine biochemical and hematologic factors that add to the predictive value of demographic and clinical variables, and (4) identify hemostatic factors predictive for mortality.

Materials and Methods

Subjects

Consecutive white European patients (n=609) with a clinical diagnosis of acute stroke, defined by World Health Organization clinical
TABLE 1. Demographic and Clinical Characteristics of Patients Surviving During a Median of 7.4 Years Compared With Patients Who Died 30 Days or < and >30 Days After the Acute Event

<table>
<thead>
<tr>
<th>Age, years</th>
<th>Survivors (n=202)</th>
<th>Died &gt;30 Days (n=311)</th>
<th>Died &lt;30 Days (n=31)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male gender</td>
<td>106 (0.53)</td>
<td>154 (0.50)</td>
<td>14 (0.44)</td>
</tr>
<tr>
<td>Smokers</td>
<td>Ex 91 (0.45)</td>
<td>106 (0.34)*</td>
<td>9 (0.28)</td>
</tr>
<tr>
<td></td>
<td>Current 42 (0.21)</td>
<td>55 (0.18)</td>
<td>5 (0.16)</td>
</tr>
<tr>
<td>Stroke subtype</td>
<td>LACI 93 (0.46)</td>
<td>89 (0.28)†</td>
<td>2 (0.08)‡</td>
</tr>
<tr>
<td></td>
<td>PACI 63 (0.31)</td>
<td>105 (0.34)</td>
<td>9 (0.28)</td>
</tr>
<tr>
<td></td>
<td>POCs 22 (0.11)</td>
<td>34 (0.11)</td>
<td>1 (0.03)</td>
</tr>
<tr>
<td></td>
<td>TACI 24 (0.12)</td>
<td>83 (0.27)</td>
<td>20 (0.63)‡</td>
</tr>
<tr>
<td>Previous stroke/TIA</td>
<td>39 (0.19)</td>
<td>116 (0.37)†</td>
<td>7 (0.22)</td>
</tr>
<tr>
<td>AF</td>
<td>16 (0.08)</td>
<td>73 (0.24)†</td>
<td>14 (0.44)‡</td>
</tr>
<tr>
<td>IHD</td>
<td>36 (0.18)</td>
<td>105 (0.34)†</td>
<td>14 (0.44)*</td>
</tr>
<tr>
<td>Diabetes</td>
<td>27 (0.13)</td>
<td>48 (0.15)</td>
<td>6 (0.19)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>87 (0.43)</td>
<td>123 (0.40)</td>
<td>11 (0.34)</td>
</tr>
<tr>
<td>PVD</td>
<td>16 (0.08)</td>
<td>47 (0.15)†</td>
<td>8 (0.25)*</td>
</tr>
<tr>
<td>Aspirin use at time of stroke</td>
<td>36 (0.18)</td>
<td>97 (0.31)*</td>
<td>11 (0.34)</td>
</tr>
</tbody>
</table>

Age presented as median (25th and 75th percentiles); other data as number (frequency).

*P<0.05 compared with survivors after Bonferroni adjustment.
†P<0.001 compared with survivors after Bonferroni adjustment.
‡P<0.05 compared with patients dying more than 30 days after the acute event after Bonferroni adjustment.

Laboratory Analyses

A venous blood sample was taken for analysis of biochemical (albumin, creatinine, cholesterol, triglycerides, glucose, bilirubin, uric acid, total protein), hematologic (hemoglobin, platelet count, white cell count), and hemostatic factors (fibrinogen, factor [F]VII, FVIII, FXIII A and B subunit and activity, vWF, tPA, plasminogen activator inhibitor-1 [PAI-1], and βTG). Biochemical and hematologic analyses were carried out using standard hospital laboratory protocols, and hemostatic factors were analyzed by clotting assays (fibrinogen, FVII, FVIII) or enzyme-linked immunosorbent assay (FXIII, vWF, tPA, PAI-1, and βTG) as previously described.10–15,18 The first 115 patients recruited did not have a sample taken for analysis of hemostatic factors and in a further 15 patients, insufficient blood was obtained for these analyses. There were no differences in demographic, biochemical, or hematologic factors or mortality between those subjects with and without hemostasis samples except for age (no sample: 75 [range, 66 to 81] years; with sample: 72 [range, 65 to 79] years, P=0.03) and platelet count (no sample: 237 [range, 227 to 251]×10^9/L; with sample: 256 [range, 247 to 264]×10^9/L, P=0.04). Samples for analysis of βTG were only taken in 275 patients and 278 control subjects in whom clean venipuncture was performed on the first attempt with minimal venous stasis as previously described.12

Statistical Analyses

The outcome measure was all-cause long-term mortality after acute ischemic stroke. Mortality in control subjects was monitored as a reference. Surviving patients and control subjects were censored on January 19, 2002. Univariate associations between demographic, clinical, biochemical, hematologic, and hemostatic factors and mortality were assessed using Kaplan–Meier survival analysis and significance determined using the log rank test with continuous variables analyzed as quartiles. Hazard ratios for mortality were determined by univariate and multivariate Cox proportional hazards regression analyses with data presented as hazard ratio (compared with the lowest quartile) with 95% CIs. Log minus log plots were evaluated to test the validity of the proportionality of hazards assumption over time; all variables met this assumption. Initial analyses identified the demographic and clinical variables that
independently predicted poststroke mortality. Significant demo-
graphic and clinical variables were entered into subsequent multivariate
Cox regression analyses with biochemical, hematologic, and hemostatic
factors included in the models using forward stepwise selection with a
probability value of 0.05 required for entry into the model. A probability
value of 0.05 or less was considered significant. All statistical analyses
were performed using SPSS for Windows v12.

Results

Subjects were followed up for a median of 7.4 years; minimum and maximum follow-up periods for survivors
were 5.3 years and 8.5 years, respectively. During follow up,
343 patients (63%) and 73 control subjects (22%) died with
median times to death of 1.6 and 3.0 years, respectively.

Baseline Patient and Control Subject
Characteristics and Mortality Rates

The characteristics of patients with ischemic stroke who
survived compared with those who died 30 days or less and
those dying more than 30 days are presented in Table 1.
Survivors were younger than those who died. The proportion
of exsmokers was higher in survivors than in those who died;
there was no difference in time since cessation of smoking or

![Figure 1. Kaplan–Meier survival curves comparing patients and control subjects
analyzed by gender. Crosses indicate censored data.](image)

![Figure 2. Kaplan–Meier survival curves indicating the relationship of (A) age,
(B) stroke subtype, (C) albumin, (D) creatinine, (E) β-TG, and (F)
vWF with all-cause mortality after ischemic stroke during a median follow up of 7.4
years. Crosses indicate censored data. LACI indicates lacunar infarction; PACI,
partial anterior circulation infarction; POCS, posterior circulation syndrome; TACI,
total anterior circulation infarction.](image)
pack-years in exsmokers who survived compared with those who died (data not shown). The proportion of subjects with LACI was highest in survivors, whereas the proportion of subjects with TACI was highest in those who died. A greater proportion of subjects with AF, previous stroke, IHD, PVD, and aspirin use at the time of stroke died during follow up. Compared with patients dying after more than 30 days, a greater proportion of patients dying 30 days or less had experienced a TACI and were in AF (Table 1). Subjects dying 30 days or less after the acute event were excluded from subsequent analyses.

The characteristics of the control subjects who died during follow up and survivors are presented in supplemental Table I (available online at http://stroke.ahajournals.org). Those who died were significantly older than survivors. There was no significant difference in gender, smoking, or other clinical variables between those who subsequently died and survivors.

Kaplan–Meier survival curves comparing age-matched patients and control subjects analyzed by gender are presented in Figure 1. Average annual mortality rates in male and female patients were 8.0% and 8.4% compared with 2.6% and 3.2% in male and female controls, respectively. The hazard ratios (95% CI) for mortality in male patients compared with male controls was 3.59 (2.38 to 5.42) and for female patients compared with female controls was 3.14 (2.26 to 4.38).

### Demographic and Clinical Variables Predictive for Long-Term Poststroke Mortality

In univariate analyses, in patients more than 30-day all-cause mortality was significantly associated with age and stroke subtype (Figure 2A, B). Previous stroke/TIA, AF, IHD, PVD, aspirin therapy at the time of stroke, and being a nonsmoker were also significantly associated with mortality (Table 2).
There were no associations between mortality after acute stroke and gender, diabetes, warfarin treatment at the time of acute stroke, hypertension, or admission blood pressure (data not shown). In a multivariate Cox regression analysis, including age, smoking, stroke subtype, previous stroke/TIA, IHD, PVD, aspirin therapy, and AF, independent predictors of poststroke mortality were age, stroke subtype, previous stroke/TIA, and AF (Table 2).

Biochemical and Hematologic Factors Predictive for Poststroke Mortality

In univariate analyses, lower concentrations of albumin and hemoglobin and higher concentrations of creatinine at the time of stroke were associated with higher mortality (Table 3). There were no significant associations between plasma glucose, cholesterol, triglycerides, platelet count, white cell count, bilirubin, plasma uric acid, or total protein and poststroke mortality (data not shown). In multivariate Cox regression analyses, after accounting for age, stroke subtype, previous stroke/TIA, IHD, PVD, aspirin therapy, and AF, independent predictors of poststroke mortality were age, stroke subtype, previous stroke/TIA, and AF (Table 2).

Hemostatic Factors Predictive for Poststroke Mortality

In univariate analyses, elevated levels of fibrinogen, FVIII, βTG, vWF, and tPA and lower levels of FXIII A subunit were each associated with higher poststroke mortality (Table 4). There were no significant associations between PAI-1, FVII, FXIII B subunit or FXIII activity and mortality (data not shown). In multivariate Cox regression analyses, after accounting for age, stroke subtype, previous stroke/TIA, and AF, only βTG and vWF were associated with mortality (Table 4). Kaplan–Meier survival curves for βTG and vWF are presented in Figure 2E and F.

Discussion

Patients with ischemic stroke who died more than 30 days after the acute event were at more than 3-fold increased risk of death after the acute event compared with an age-matched healthy cohort, which is in keeping with the results of large population-based studies.5,19 We excluded patients who died 30 days or less after the acute event because death is largely attributable to the acute event itself reflected by the large proportion of patients with TACI in those dying within 30 days in the present study. In contrast, longer-term mortality is generally attributable to vascular disease, in particular cardiovascular disease.5,18

Demographic and Clinical Variables Predictive for Mortality After Ischemic Stroke

In keeping with results of population studies,5,19,20 mortality increased with age and was highest (80%) in subjects aged 75 years or older. Notably, half of those subjects aged less than 75 years also died during follow up. The high mortality rate, irrespective of age, highlights the importance of research into the underlying mechanisms predisposing to stroke and stroke mortality.

### Table 3. Kaplan–Meier and Cox Regression Analysis of Biochemical Variables Associated With Postischemic Stroke Mortality

<table>
<thead>
<tr>
<th>Variable</th>
<th>Univariate Analysis*</th>
<th>Multivariate Analysis†</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Percent Survivors</td>
<td>Hazard Ratio</td>
</tr>
<tr>
<td>Albumin (g/L)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q1 (&lt;38)</td>
<td>26</td>
<td>1</td>
</tr>
<tr>
<td>Q2 (38–40)</td>
<td>32</td>
<td>0.78 (0.56–1.09)</td>
</tr>
<tr>
<td>Q3 (41–43)</td>
<td>41</td>
<td>0.63 (0.44–0.89)</td>
</tr>
<tr>
<td>Q4 (&gt;43)</td>
<td>49</td>
<td>0.45 (0.32–0.65)</td>
</tr>
<tr>
<td>Creatinine (mmol/L)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q1 (&lt;32)</td>
<td>52</td>
<td>1</td>
</tr>
<tr>
<td>Q2 (32–97)</td>
<td>44</td>
<td>1.39 (0.94–2.05)</td>
</tr>
<tr>
<td>Q3 (98–117)</td>
<td>36</td>
<td>1.62 (1.12–2.34)</td>
</tr>
<tr>
<td>Q4 (&gt;117)</td>
<td>20</td>
<td>2.26 (1.58–3.24)</td>
</tr>
<tr>
<td>Hemoglobin (g/dL)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q1 (&lt;12.6)</td>
<td>28</td>
<td>1</td>
</tr>
<tr>
<td>Q2 (12.6–13.6)</td>
<td>32</td>
<td>0.82 (0.59–1.15)</td>
</tr>
<tr>
<td>Q3 (13.7–14.8)</td>
<td>36</td>
<td>0.73 (0.52–1.01)</td>
</tr>
<tr>
<td>Q4 (&gt;14.8)</td>
<td>51</td>
<td>0.49 (0.34–0.70)</td>
</tr>
</tbody>
</table>

*Univariate analysis: percent survival from Kaplan–Meier analysis; hazard ratio and P value from Cox regression analysis.
†Multivariate analysis: hazard ratio and P value from Cox regression analysis, which included age, stroke subtype, previous stroke/TIA, and AF with forward stepwise selection for biochemical/hematologic factors. Hazard ratios only obtained for factors entered into the model.
Q indicates quartile; NE, variable not entered into model.
We used the Oxfordshire Community Stroke Project classification of stroke\textsuperscript{21} to determine the association of stroke subtype with mortality and found an approximately 2-fold overall increased risk of mortality for those with larger infarcts compared with those with LACI (1.6-fold for PACI and 2.6-fold for TACI). These results are in keeping with previous studies, summarized in a recent meta-analysis,\textsuperscript{22} indicating that our hospital-based cohort is representative of larger population-based studies. In keeping with other studies,\textsuperscript{18} we also identified history of stroke/TIA as predicting mortality, independent of stroke subtype, indicating the need for improved management of vascular risk to prevent secondary stroke and subsequent cardiovascular mortality. Univariate analysis indicated a paradoxical association of aspirin use at the time of stroke with subsequent mortality. Aspirin use is likely to reflect a history of atherothrombotic disease supported by the fact that aspirin use was not independently predictive of mortality in multivariate analyses, including history of atherothrombotic disease.

\begin{table}
\centering
\caption{Kaplan–Meier and Cox Regression Analysis of Hemostatic Factors Associated With Postischemic Stroke Mortality}
\begin{tabular}{lllll}
\hline
 & \textbf{Univariate Analysis}\textsuperscript{*} & & \textbf{Multivariate Analysis}\textsuperscript{†} & \\
 & \textbf{Percent Survivors} & \textbf{Hazard Ratio} & \textbf{P Value} & \textbf{Hazard Ratio} & \textbf{P Value} \\
\hline
\textbf{Fibrinogen (g/L)} & & & & \\
Q1 (<3.53) & 53 & 1 & NE & 0.657 \\
Q2 (3.53–4.27) & 46 & 1.18 (0.79–1.76) & 0.411 \\
Q3 (4.28–5.37) & 35 & 1.55 (1.05–2.27) & 0.026 \\
Q4 (>5.37) & 29 & 1.92 (1.32–2.81) & 0.001 \\
\textbf{FVIII (U/mL)} & & & & \\
Q1 (<1.68) & 51 & 1 & NE & 0.764 \\
Q2 (1.68–2.12) & 49 & 1.10 (0.73–1.67) & 0.643 \\
Q3 (2.13–2.69) & 36 & 1.54 (1.03–2.29) & 0.035 \\
Q4 (>2.69) & 25 & 2.06 (1.40–3.02) & <0.001 \\
\textbf{FXIIIa (%)} & & & & \\
Q1 (<94) & 26 & 1 & NE & 0.311 \\
Q2 (94–113) & 38 & 0.70 (0.49–0.98) & 0.039 \\
Q3 (114–138) & 55 & 0.44 (0.30–0.63) & <0.001 \\
Q4 (>138) & 48 & 0.51 (0.35–0.72) & <0.001 \\
\textbf{\(\beta\) TG (ng/mL)} & & & & \\
Q1 (<31.3) & 72 & 1 & 1 & \\
Q2 (31.3–43.9) & 41 & 2.80 (1.60–4.90) & <0.001 & 2.58 (1.37–4.88) & 0.004 \\
Q3 (44.0–64.6) & 38 & 2.74 (1.57–4.78) & <0.001 & 1.32 (0.67–2.62) & 0.419 \\
Q4 (>64.6) & 18 & 4.86 (2.94–8.33) & <0.001 & 3.33 (1.78–6.22) & <0.001 \\
\textbf{vWF (IU/mL)} & & & & \\
Q1 (<1.35) & 62 & 1 & 1 & \\
Q2 (1.35–1.85) & 40 & 2.02 (1.25–3.29) & 0.004 & 1.48 (0.83–2.63) & 0.184 \\
Q3 (1.86–2.43) & 34 & 2.25 (1.39–3.64) & 0.001 & 1.53 (0.84–2.78) & 0.166 \\
Q4 (>2.43) & 15 & 3.92 (2.46–6.23) & <0.001 & 2.15 (1.18–3.91) & 0.012 \\
\textbf{tPA (ng/mL)} & & & & \\
Q1 (<9.0) & 50 & 1 & NE & 0.349 \\
Q2 (9.0–12.2) & 55 & 0.92 (0.58–1.45) & 0.72 \\
Q3 (12.3–15.9) & 40 & 1.32 (0.87–2.02) & 0.20 \\
Q4 (>15.9) & 31 & 1.70 (1.12–2.56) & 0.012 \\
\hline
\multicolumn{5}{l}{\textsuperscript{*}Univariate analysis: percent survival from Kaplan–Meier analysis; hazard ratio and P value from Cox regression analysis.}
\multicolumn{5}{l}{\textsuperscript{†}Multivariate analysis: hazard ratio and P value from Cox regression analysis, which included age, stroke subtype, previous stroke/TIA, and AF with forward stepwise selection for hemostatic factors. Hazard ratios only obtained for factors entered into the model.}
\text{Q indicates quartile; NE, variable not entered into model.}
\end{tabular}
\end{table}
tension were undiagnosed at the time of recruitment, because presence of these risk factors was determined with reference to case notes and current use of antihypertensive and antihyperglycemic agents. Nor can we exclude misreporting of smoking status, which may be a source of error in our analyses. It must also be recognized that the present hospital-based cohort is relatively small in comparison with population-based studies, which may contribute to the observed discrepancies.

AF is associated with an approximately 5-fold increased risk for the development of stroke.\textsuperscript{24,25} Stroke in subjects in AF is generally associated with embolization of thrombus formed within the left atrium.\textsuperscript{26} Previous studies showed that AF predicted poor outcome at 1 month and 1 year in comparison with subjects in sinus rhythm,\textsuperscript{24,27} with mortality rates of 50\% at 1 year. In keeping with previous studies,\textsuperscript{28,29} AF was a significant predictor of mortality in the present study, independent of stroke subtype and other measured demographic and clinical factors. Only 16\% of patients in AF survived during follow up, and only 34 subjects in AF (33\%) were taking antithrombotic treatments at the time of acute stroke.

Biochemical and Hematologic Variables Predictive for Mortality After Ischemic Stroke

Of the routine factors analyzed, only albumin and creatinine were associated with poststroke mortality after accounting for demographic and clinical variables. Low albumin concentrations predicted mortality, which is consistent with the findings of previous studies.\textsuperscript{28,29} It has been reported that low albumin concentrations only predict mortality in smokers,\textsuperscript{28} and it has been suggested that smokers with decreased albumin are particularly sensitive to smoking-induced inflammation and presumably, therefore, also its consequences.\textsuperscript{28,30} In contrast to these studies, we found that low albumin (<38 g/L) was predictive for mortality irrespective of smoking history. Decreased albumin levels may be associated with poor nutritional status\textsuperscript{29}; however, total protein was not associated with subsequent mortality in the present study. Albumin levels could be decreased attributable to an acute inflammatory response to stroke or ongoing chronic inflammation; however, the association between decreased albumin and mortality was independent of stroke subtype and white blood cell count was not associated with stroke mortality, suggesting that these are unlikely to explain the observed association. Elevated creatinine at the time of acute stroke also predicted long-term mortality. These data suggest that patients with increased creatinine and/or decreased albumin may have impaired renal function, possibly attributable to advancing age and hypertension. Such patients should be investigated further to determine underlying causes of these biochemical disturbances to facilitate appropriate management.

Hemostatic Factors Predictive for Mortality After Ischemic Stroke

Fibrin formation and platelet activation occur in the acute phase of stroke.\textsuperscript{31} We previously reported elevated levels of fibrinogen, FVIII, vWF, PAI-1, and βTG in subjects with acute stroke.\textsuperscript{10–13} Of the hemostatic factors analyzed, only vWF and βTG were independently associated with poststroke mortality during follow up. vWF is stored in endothelial cell Weibel-Pallade bodies and platelet α-granules and is released in response to endothelial cell and platelet activation. vWF plays an essential role in platelet adhesion to subendothelial matrix, exposed at sites of endothelial disruption, promoting thrombus formation. βTG is released on platelet activation and is considered a specific marker of ongoing platelet activation. Results from the present study indicate that vWF and βTG are predictive for long-term mortality after the acute event independent of stroke subtype and other predictors of mortality and are likely to reflect both endothelial disruption and platelet activation.

Limitations of the Study

We analyzed only data collected at the time of presentation with stroke in this study to determine the predictive value of variables that could readily be obtained after stroke. The information on treatment at discharge was not obtained, and consequently, we may have underestimated the influence of variables on stroke mortality. We only analyzed total mortality in this study because of the recognized inconsistencies in cause of death reporting by death certificate.

Summary

The results of this study indicate that, in addition to well-recognized risk factors for poststroke mortality, including advancing age, more severe strokes, AF, and previous stroke/TIA, low albumin and elevated creatinine predict mortality after stroke accounting for other factors, which may indicate impaired renal function in these patients. Furthermore, we have shown that the hemostatic factors vWF and βTG predict poststroke mortality after accounting for other factors, likely indicating ongoing vascular damage and platelet activation. We acknowledge that βTG requires particularly stringent sample handling and is therefore unlikely to be adopted as a routine hospital measurement. However, vWF antigen measurement is straightforward and could be considered as a routine assessment when evaluating patients with acute stroke to identify patients in whom ongoing vascular damage and platelet activation is likely to be occurring. These subjects may reflect a subgroup of patients at particular risk for subsequent thrombotic events in whom combined antiplatelet treatment may be most beneficial. The results of the present study indicate that in subjects with acute ischemic stroke, analysis of albumin, creatinine, vWF, and βTG will aid in the identification of patients who are at increased risk of death after stroke.

Sources of Funding

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Disclosures

None.

References


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