Frequency and Predictors of Stroke After Acute Myocardial Infarction
Specific Aspects of In-Hospital and Postdischarge Events

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Background and Purpose—Stroke is a serious complication after acute myocardial infarction (AMI) and is closely associated with decreased survival. This study aimed to investigate the frequency, characteristics, and factors associated with in-hospital and postdischarge stroke in patients with AMI.

Methods—Eight thousand four hundred eighty-five consecutive patients admitted to a cardiology intensive care unit for AMI, between January 2001 and July 2010. Stroke/transient ischemic attack were collected during 1-year follow-up.

Results—One hundred twenty-three in-hospital strokes were recorded: 65 (52.8%) occurred on the first day after admission for AMI, and 108 (87%) within the first 5 days. One hundred six patients (86.2%-incidence rate 1.25%) experienced in-hospital ischemic stroke, and 14 patients (11.4%-incidence rate 0.16%) were diagnosed with an in-hospital hemorrhagic stroke. In-hospital ischemic stroke subtypes according to the Trial of Org 10172 in Acute Stroke Treatment (TOAST) classification showed that only 2 types of stroke were identified more frequently. As expected, the leading subtype of in-hospital ischemic stroke was cardioembolic stroke (n=64, 60%), the second was stroke of undetermined pathogenesis (n=38, 36%). After multivariable backward regression analysis, female sex, previous transient ischemic attack (TIA)/stroke, new-onset atrial fibrillation, left ventricular ejection fraction (odds ratio per point of left ventricular ejection fraction), and C-reactive protein were independently associated with in-hospital ischemic stroke. When antiplatelet and anticoagulation therapy within the first 48 hours was introduced into the multivariable model, we found that implementing these treatments (≥1) was an independent protective factor of in-hospital stroke. In-hospital hemorrhagic stroke was dramatically increased (5-fold) when thrombolysis was prescribed as the reperfusion treatment. However, the different parenteral anticoagulants were not predictors of risk in univariable analysis. Finally, only 45 postdischarge strokes were recorded. Postdischarge stroke subtypes showed a more heterogeneous distribution of mechanisms. The annual rate of stroke post-AMI remained stable throughout the 10-year study period.

Conclusions—The present study describes specific predictors of in-hospital and postdischarge stroke in patients with AMI. It showed a marked increase in the risk of death, both during hospitalization and in the year after AMI. After hospital discharge, stroke remains a rare event and is mostly associated with high cardiovascular risk. (Stroke. 2014;45:3514-3520.)

Key Words: myocardial infarction • risk factors • stroke

Clinical and epidemiological studies have shown a greater incidence of stroke in patients experiencing acute myocardial infarction (AMI) than in the overall population.1,2 The incidence of stroke after AMI during the hospital stay ranges from 0.7% to 2.2%.3-5 Despite the improvement in reperfusion strategies and the management of cardiovascular risk factors, ischemic stroke (IS) after AMI remains associated with worse short-term and 1-year mortality (30.1% and 36.5%, respectively) when compared with patients without stroke.6,7 Stroke occurred more frequently in the first days after AMI, but incidence progressively decreased over time.5-7 Predictors of an increased risk of IS after AMI have already been described.7,6,8 Hemorrhagic stroke (HS) after AMI is less frequent and such events decreased by 50% between 1998 and 2008, with a 30-day incidence between 0.06% and 0.22%.9 Despite these studies, which mixed ST-elevation and non–ST-elevation MI, one can expect that the falling prevalence of HS is partly explained by the shift in reperfusion strategies from thrombolysis to percutaneous coronary intervention.9 Giugliano et al10 reported 30-day mortality of 65.4% after intracranial hemorrhage, rising to 71.8% at 1 year. Independent predictors of HS have already been described.11 However, many studies included small populations, frequently reported IS and HS simultaneously without discrimination, and were performed...
over several years. Moreover, it has not been established
whether stroke during the hospital stay for AMI and stroke
after discharge are related to the same mechanisms. Thus,
IS and HS should be considered separately, and the impact
of risk factors and acute or chronic AMI therapies on stroke
occurrence should be investigated. Our study aimed to deter-
mine the incidence, the predictive factors, and the prognosis
of in-hospital and postdischarge (1-year follow-up) IS and HS
in a large, unselected population of patients with AMI over a

Methods

Patients
The design and methods of the RICO (obseRvatoire des Infarctus de
Côte-d’Or), a French regional survey for acute MI, have been detailed
previously.11 Between January 1, 2001, and July 31, 2010, all the con-
secutive patients hospitalized <24 hours after symptom onset were in-
cluded in the present study. MI was diagnosed according to European
Society of Cardiology (ESC) and American College of Cardiology
(ACC) criteria.12 The present study complied with the Declaration of
Helsinki and was approved by the Ethics Committee of University
Hospital of Dijon. Each patient gave written consent before participa-
tion. The study population is described in Figure 1.

Data Collection
Data including the GRACE risk score14 were collected prospectively.
We carefully recorded treatments prescribed in the first 48 hours after
AMI, or at discharge, especially statins, aspirin (acetylsalicylic acid),
P2Y12 inhibitors, anticoagulants (low–molecular-weight heparin or
unfractionated heparin), and angiotensin-converting enzyme inhibi-
tors. Blood samples were drawn at admission.

Stroke Definition
The WHO definition of stroke was used. Stroke was classified as
hemorrhagic if a focal collection of blood was observed within the
brain parenchyma or ventricular system. Stroke was classified as
ischemic in cases of a focal cerebral, spinal, or retinal infarction or
in the absence of a cerebral collection of blood. The IS subtype was
diagnosed retrospectively by 2 neurologists on clinical and cerebral
imaging data using the original Trial of Org 10172 in Acute Stroke
Treatment (TOAST) criteria.15 Hence, in-hospital and postdischarge
ISs were classified in 5 categories: large artery atherosclerosis, car-
dioembolism originating from atrial fibrillation (AF) or other rhythm
disorders, valve disease, patent foramen ovale or spontaneous intra-
cavitary thrombus, small artery occlusion, stroke of other determined
causes (rare causes of stroke, such as nonatherosclerotic vasculopa-
thy, hypercoagulable states, or hematologic disorders), and stroke of
undetermined cause (≥2 causes identified, negative evaluation, or in-
complete evaluation). If no cerebral imaging was performed because
of hemodynamic instability or death of the patient before imaging,
stroke was classified as undetermined. In-hospital stroke (IHS) was
defined as stroke/TIA occurring between admission to the cardiac in-
tensive care unit for AMI and the patient’s discharge from hospital.
Postdischarge stroke (PDS) was defined as stroke/TIA occurring dur-
ing the first year of follow-up (vital status and major adverse cardiac
events) after the patient’s discharge from hospital.

Short- and Long-Term Follow-Up
Follow-up at 30 days and 1 year was performed to assess survival after
hospital discharge. Follow-up data were obtained either by telephone
interview or by letters sent to the patients, the patient’s relatives, or
the treating physician, or by a review of the patients’ medical records.

Statistical Analysis
Continuous data were expressed as medians (25th–75th percent-
tile) and dichotomous data as numbers (percentages). The normal
distribution of continuous data was tested with the Kolmogorov-
Smirnov test. The categorical variables were analyzed using the
χ2 or Fisher test. Continuous variables were analyzed using the ANOVA
or Kruskal-Wallis test as appropriate. Logistic regression analy-
sis was performed to test for predictors of (1) in-hospital ischemic
stroke (IH-IS), (2) in-hospital hemorrhagic stroke (IH-HS), and (3)
PDS. Improvements in the χ2 and maximum log likelihood stepwise
methods, including Hosmer-Lemeshow goodness of fit χ² estimates,
were used to evaluate the regression model. Variables entered into

Figure 1. Population study. TOAST indicates Trial of Org 10172 in Acute Stroke Treatment.
the multivariable model were chosen according to their univariate relationship with an inclusion cutoff at 5% and exclusion cutoff at 1%. The variables included in the multivariable model for IH-IS were age, female sex, BMI, hypertension, heart rate at admission, previous stroke/TIA, acute antplatelet therapy, left ventricular ejection fraction, percutaneous coronary intervention, new-onset AF, admission glycemia, and C-reactive protein. The variables included in the multivariable model for IH-HS were age, previous renal failure, left ventricular ejection fraction, ST-elevation myocardial infarction, and thrombolysis. The variables included in the multivariable model for PDS were age, female sex, hypertension, previous stroke/TIA, and new-onset AF during AMI. Before the construction of the multivariable models, collinearity between variables was excluded. All multivariable models were tested for multicollinearity (tolerance and variation inflation factor) and were found to be stable. Kaplan-Meier survival statistics and the log-rank test were used to compare survival in patients with IHS with those without stroke. All the tests were 2-sided, and a *P* value <0.05 was considered significant. All analyses were performed using SPSS 20.0 (SPSS Inc, Chicago, Ill).

**Results**

From a total of 8485 patients, 168 (1.98%) had a stroke during the first year after the AMI. Two thirds (1.4% [n=123]) were IHSs, mainly occurring in the first 5 days after admission (87%), with a 30-day mortality of 34.1%. One third (0.64% [n=45] of the 7808 patients who survived the AMI) experienced a PDS during the first year of follow-up (Figure 1). Although the risk of stroke progressively decreases in the months after AMI, the annual rate of post-AMI stroke was stable over the period 2001 to 2010. During the 10-year study period, the incidence of stroke (in-hospital and PDS) remained stable, with no significant year-by-year variation (*P*=0.98) and with no temporal correlation (*R*²=0.01; Figure 2).

**In-Hospital Stroke**

One hundred twenty-three IHSs were recorded; 65 (52.8%) occurred on the first day following admission for AMI and 108 (87%) within the first 5 days. One hundred six patients (11.4%-incidence rate 0.16%) were diagnosed with PDS during the first year after the AMI. Two thirds (1.4% [n=123]) were IHSs, mainly occurring in the first 5 days after admission (87%), with a 30-day mortality of 34.1%. One third (0.64%) of the 7808 patients who survived the AMI experienced a PDS during the first year of follow-up (Figure 1). Although the risk of stroke progressively decreases in the months after AMI, the annual rate of post-AMI stroke was stable over the period 2001 to 2010. During the 10-year study period, the incidence of stroke (in-hospital and PDS) remained stable, with no significant year-by-year variation (*P*=0.98) and with no temporal correlation (*R*²=0.01; Figure 2).

![Figure 2](https://example.com/figure2.png) **Figure 2.** Annual incidence of post-acute myocardial infarction (AMI) strokes (in-hospital, postdischarge, and cumulative events).

![Table 1](https://example.com/table1.png) **Table 1. Patients’ Characteristics in n (%) or Median (Interquartile Range) for In-Hospital (IH) Stroke**

<table>
<thead>
<tr>
<th>Risk factors</th>
<th>IH-Stroke (+) n=123</th>
<th>IH-Stroke (-) n=8362</th>
<th><em>P</em> Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y (65–82)</td>
<td>77 (55–78)</td>
<td>69</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Female</td>
<td>54 (44)</td>
<td>2473 (30)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BMI, kg/m² (22–28)</td>
<td>26 (24–29)</td>
<td>26</td>
<td>0.01</td>
</tr>
<tr>
<td>Hypertension</td>
<td>78 (63)</td>
<td>4450 (53)</td>
<td>0.03</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>28 (23)</td>
<td>1906 (23)</td>
<td>0.92</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>54 (44)</td>
<td>3744 (45)</td>
<td>0.92</td>
</tr>
<tr>
<td>Current smoking</td>
<td>25 (20)</td>
<td>2331 (28)</td>
<td>0.08</td>
</tr>
<tr>
<td>Previous AMI</td>
<td>12 (10)</td>
<td>1100 (13)</td>
<td>0.33</td>
</tr>
<tr>
<td>Previous TIA/stroke</td>
<td>17 (14)</td>
<td>477 (6)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

**Clinical data**

- **STEMI**
  - 82 (67)
  - 4878 (58)
  - 0.08
- **Anterior wall location**
  - 69 (56)
  - 3234 (39)
  - <0.001
- **Cardiogenic shock**
  - 29 (23.6)
  - 692 (83.3)
  - <0.001
- **Killip class>2**
  - 23 (18)
  - 632 (7)
  - <0.001
- **PCI**
  - 54 (44)
  - 5329 (63)
  - <0.001
- **Thrombolysis**
  - 21 (17)
  - 1577 (19)
  - 0.67
- **Heart rate, bpm**
  - 87 (76–100)
  - 77 (65–90)
  - <0.001
- **LVEF, %**
  - 46 (35–55)
  - 55 (45–62)
  - <0.001
- **GRACE risk score**
  - 173 (150–200)
  - 144 (119–171)
  - <0.001
- **New-onset AF**
  - 26 (21)
  - 742 (9)
  - <0.001

**Biological data on admission**

- **Glucose, mmol/L**
  - 7.8 (6.7–11.7)
  - 7 (5.9–9.1)
  - <0.001
- **CRP, mg/L**
  - 18.7 (5.7–79.7)
  - 6.1 (2.9–19)
  - <0.001
- **Creatinine clearance, mL/min**
  - 52 (31–73)
  - 72 (49–98)
  - <0.001
- **NT-proBNP, pg/mL**
  - 4895 (2277–11508)
  - 903 (247–3137)
  - <0.001

**Chronic treatments**

- **VKA**
  - 6 (5)
  - 515 (6)
  - 0.69
- **ASA**
  - 30 (24)
  - 1579 (19)
  - 0.15
- **Clopidogrel**
  - 19 (15)
  - 784 (9)
  - 0.03

**Acute treatments**

- **Beta blocker**
  - 60 (61)
  - 4571 (78)
  - <0.001
- **ACE inhibitor**
  - 41 (41)
  - 3559 (61)
  - <0.001
- **Statin**
  - 60 (61)
  - 4395 (75)
  - 0.002
- **VKA**
  - 3 (2.4)
  - 166 (2)
  - 0.97
- **ASA**
  - 104 (85)
  - 7801 (93)
  - <0.001
- **Clopidogrel**
  - 80 (65)
  - 6700 (80)
  - <0.001
- **LMWH**
  - 35 (28)
  - 4683 (56)
  - <0.001
- **UFH**
  - 87 (70)
  - 4628 (55)
  - <0.001

ACE indicates angiotensin-converting enzyme; AF, atrial fibrillation; AMI, acute myocardial infarction; ASA, acetylsalicylic acid; BMI, body mass index; bpm, beats per minute; CRP, C-reactive protein; GRACE, Global Registry Acute Coronary Events; LMWH, low–molecular-weight heparin; LVEF, left ventricular ejection fraction; NT-proBNP, N-terminal pro-BNP; PCI, percutaneous coronary intervention; STEMI, ST-elevation myocardial infarction; TIA, transient ischemic attack; UFH, unfractionated heparin; and VKA, vitamin K antagonist.

These patients had a lower left ventricular ejection fraction, a higher GRACE risk score, and were more likely to have new-onset AF in the intensive care unit than patients without
IHS. Repерfusion therapies such as percutaneous coronary intervention were less common in patients experiencing an IHS than in patients without stroke. Furthermore, during the acute phase, acetylsalicylic acid and P2Y12 inhibitors were less frequently prescribed in the IHS group.

Biological data showed a higher level of admission glycemia, C-reactive protein, and N-terminal-proBNP levels in IHS patients. After multivariable backward regression analysis, female sex, previous TIA/stroke, new-onset AF, left ventricular ejection fraction, and C-reactive protein were independently associated with IHS. When antiplatelet and anticoagulation therapies within the first 48 hours were introduced into the multivariable model, we found that the implementation of these acute treatments (≥1) was an independent protective factor of IHS. Moreover, female sex was no longer an independent predictive factor of IHS. The addition of antiplatelet therapy did not impair the stability of the model (HL, 9.53; P=0.30). On the contrary, acute anticoagulation was not independently associated with IHS.

As expected, IHS-HS were dramatically increased (5-fold) when thrombolysis was prescribed as the reperfusion treatment. However, the different parenteral anticoagulants were not predictors of risk in univariable analysis. Only age, thrombolysis, and previous renal failure were independently associated with IHS-HS. Again, the fitting of the model was good (HL, 14.98; P=0.08).

In patients who had an IHS, in-hospital and 30-day mortality were increased, and at 1 year, this increased risk was maintained as shown by the Kaplan-Meier survival analysis (Figure 3). Finally, 1 year after AMI, 57 patients from the IHS group had died versus 1056 patients from the group without stroke (46.4% versus 12.6%; P<0.001). After adjustment for other prognostic factors after AMI, IHS were still associated with a significant increase in mortality (odds ratio, 1.82; 95% confidence interval, 1.05–3.15; P=0.031).

### Postdischarge Stroke

Forty-five PDSs were recorded: 43 patients (95.6%-incidence rate 0.64%) experienced PDS, and 2 patients (4.4%-incidence rate 0.03%) were diagnosed with an HS. PDS subtypes showed a more heterogeneous distribution of mechanisms (Figure 1). Patients with PDS were older and more frequently women than patients without stroke. They were more likely to have a history of treated hypertension, previous myocardial infarction, diabetes mellitus, and prior stroke.

#### Table 2. Patients’ Characteristics in n (%) or Median (Interquartile Range) for Postdischarge (PD) Stroke

<table>
<thead>
<tr>
<th></th>
<th>PD-Stroke (+) n=45</th>
<th>PD-Stroke (−) n=7023</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>78 (69–83)</td>
<td>67 (55–77)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Female</td>
<td>24 (53)</td>
<td>1989 (28)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>26 (23–30)</td>
<td>26 (24–29)</td>
<td>0.74</td>
</tr>
<tr>
<td>Hypertension</td>
<td>37 (82)</td>
<td>3645 (52)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>13 (29)</td>
<td>1526 (22)</td>
<td>0.33</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>26 (58)</td>
<td>3180 (45)</td>
<td>0.13</td>
</tr>
<tr>
<td>Current smoking</td>
<td>9 (20)</td>
<td>2057 (29)</td>
<td>0.23</td>
</tr>
<tr>
<td>Previous AMI</td>
<td>12 (27)</td>
<td>900 (13)</td>
<td>0.01</td>
</tr>
<tr>
<td>Previous TIA/stroke</td>
<td>11 (24)</td>
<td>381 (5)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>New-onset AF</td>
<td>18 (22)</td>
<td>555 (8)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LVEF, %</td>
<td>54 (45–65)</td>
<td>55 (45–63)</td>
<td>0.68</td>
</tr>
<tr>
<td>GRACE risk score</td>
<td>156 (135–173)</td>
<td>142 (117–167)</td>
<td>0.02</td>
</tr>
<tr>
<td>CRP, mg/L</td>
<td>9.9 (3.8–41.8)</td>
<td>6 (2.9–17)</td>
<td>0.03</td>
</tr>
<tr>
<td>Creatinine clearance, mL/min</td>
<td>55 (37.4–72.9)</td>
<td>73.7 (51.7–100)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HbA1C, %</td>
<td>6.1 (5.7–6.8)</td>
<td>5.8 (5.5–6.4)</td>
<td>0.04</td>
</tr>
</tbody>
</table>

AF indicates atrial fibrillation; AMI, acute myocardial infarction; BMI, body mass index; CRP, C-reactive protein; GRACE, Global Registry Acute Coronary Events; LVEF, left ventricular ejection fraction; and TIA, transient ischemic attack.
infarction, and previous TIA/stroke. During the acute phase of myocardial infarction, new-onset AF in the intensive care unit was more frequently diagnosed in the PDS group than in the other group. Percutaneous coronary intervention was less frequently performed in patients who had a stroke after than in patients without stroke (Table 2).

No significant difference was observed between PDS and no-PDS patients regarding antiplatelet and anticoagulation treatment prescribed at discharge. Given the small number of HSs in PDS patients (2 events), it was not possible to conduct separate analyses for ischemic and hemorrhagic subtypes in the PDS group. The multivariable backward regression analysis (Table 3) showed that age, previous TIA/stroke, and a history of hypertension were independently associated with an increased risk of PDS after AMI. Again, the fitting of the model was good (HL, 8.82; P=0.36).

Discussion

For IH-IS, only 2 types of stroke were identified more frequently: as expected, the leading subtype of IH-IS was cardioembolic stroke (n=64, 60%), the second was stroke of undetermined pathogenesis (n=38, 36%). These results are important because (1) subtypes of stroke have rarely been reported in previous studies, (2) as expected, the cardioembolism mechanism is the major cause of IH-IS, underlining that not only chronic but also transient cardiac disorders are responsible for emboli migrating from the heart. Moreover, the rapid decrease in the relative risk for stroke in the days after AMI clearly suggests that the acute cardiac abnormalities after the coronary artery occlusion are associated with an excess risk. These abnormalities include AF, ischemic mitral valve regurgitation, hypotension, or cardiogenic shock, all of which lead to intracardiac thrombus formation. Among these, AF is of particular interest because recent studies have demonstrated that silent AF is 3 times more frequent than symptomatic AF. In patients with a high risk of stroke, silent or subclinical AF is a frequent event and is associated with the onset or the recurrence of ischemic accidents. In the Asymptomatic Atrial Fibrillation and Stroke Evaluation in Pacemaker Patients and the Atrial Fibrillation Reduction Atrial

### Table 3. Predictors of In-Hospital Ischemic Stroke (n=8485), In-Hospital Hemorrhagic Stroke (n=8485), and Postdischarge Stroke (n=7068)

<table>
<thead>
<tr>
<th></th>
<th>In-Hospital Ischemic Stroke</th>
<th>In-Hospital Hemorrhagic Stroke</th>
<th>Postdischarge Stroke</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Univariate Analysis</td>
<td>Multivariate Analysis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>OR 95% CI</td>
<td>OR 95% CI</td>
<td>OR 95% CI</td>
</tr>
<tr>
<td>Age</td>
<td>1.03 (1.02–1.05)</td>
<td>1.86 (1.30–2.67)</td>
<td>2.65 (1.58–4.46)</td>
</tr>
<tr>
<td>Female</td>
<td>1.86 (1.30–2.67)</td>
<td></td>
<td>2.52 (1.05–2.21)</td>
</tr>
<tr>
<td>BMI</td>
<td>0.93 (0.89–0.98)</td>
<td></td>
<td>1.02 (1.00–1.02)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1.52 (1.05–2.21)</td>
<td></td>
<td>0.96 (0.95–0.97)</td>
</tr>
<tr>
<td>Heart rate</td>
<td>1.02 (1.00–1.02)</td>
<td></td>
<td>0.96 (0.95–0.97)</td>
</tr>
<tr>
<td>Previous TIA/stroke</td>
<td>2.65 (1.58–4.46)</td>
<td></td>
<td>2.65 (1.58–4.46)</td>
</tr>
<tr>
<td>LVEF</td>
<td>0.96 (0.95–0.97)</td>
<td></td>
<td>0.96 (0.95–0.97)</td>
</tr>
<tr>
<td>Acute antiplatelet therapy (aspirin or P2Y12 inhibitors)</td>
<td>0.26 (0.15–0.46)</td>
<td>0.26 (0.15–0.46)</td>
<td>0.26 (0.15–0.46)</td>
</tr>
<tr>
<td>PCI</td>
<td>0.45 (0.31–0.64)</td>
<td></td>
<td>0.45 (0.31–0.64)</td>
</tr>
<tr>
<td>New-onset AF</td>
<td>2.75 (1.77–4.27)</td>
<td></td>
<td>2.75 (1.77–4.27)</td>
</tr>
<tr>
<td>CRP</td>
<td>1.01 (1.01–1.01)</td>
<td></td>
<td>1.01 (1.01–1.01)</td>
</tr>
<tr>
<td>Glycemia on admission</td>
<td>1.07 (1.04–1.10)</td>
<td></td>
<td>1.07 (1.04–1.10)</td>
</tr>
</tbody>
</table>

AF indicates atrial fibrillation; BMI, body mass index; bpm, beats per minute; CI, confidence interval; CRP, C-reactive protein; LVEF, left ventricular ejection fraction; MI, myocardial infarction; OR, odds ratio; PCI, percutaneous coronary intervention; and TIA, transient ischemic attack.
Pacing (ASSERT) study, subclinical AF was associated with a worse prognosis, characterized by a 2.5-fold increased risk of stroke or systemic embolism. Several hypotheses have been put forward to explain the increase in silent AF. These include excess platelet activity, radical species, or inflammatory reactions. In this context, the protective influence of antiplatelet and anticoagulation therapy highlighted in our statistical analysis, together with the increased risk associated with higher plasma concentrations of C-reactive protein, assessed at the time of hospital admission, suggest that in situ thrombosis is also a determinant of IH-IS.

The second main pathogenesis was the undetermined pathogenesis subtype. This subtype includes ≥2 identified causes (n=14, 13.2%), where the neurologist is unable to make a final diagnosis; a negative evaluation (n=19, 17.9%), where the cause of the stroke cannot be determined with any degree of confidence, and, for some patients, no likely pathogenesis despite a comprehensive evaluation. Only 5 patients (4.7%) had an incomplete evaluation. Our results together with previous studies fuel the debate on the exact pathophysiology of stroke after AMI, which is still unclear.

Predictors of Stroke

For PDS, our findings were that (1) despite an increased risk of stroke after AMI, the absolute number of cases of stroke in our surviving population was low (0.6%); (2) mechanisms leading to stroke were heterogeneous; and that (3) the ratio between the subtypes resembled an all-cause stroke population. Interestingly, the ratio was similar to that of a recent article published by our study group, although we did not use the TOAST classification to classify types of stroke. PDS was associated with 3 independent factors: age, previous TIA/stroke, and systemic hypertension. The features of the myocardial infarct or prescriptions at discharge were not associated with stroke events after hospital discharge. Thus, PDS was related more to the global cardiovascular risk than to the clinical presentation of the infarct.

Risk of Stroke After AMI

In our study, stroke incidence during the first year after AMI was 2%, with no significant reduction during the decade of inclusion. Previous studies often reported lower post-AMI stroke rates of 1% to 1.5% during the first year. Lichtman et al reported a stroke rate after AMI at 6 months of 2.5% and determined an estimated annual rate at 5% per year. However, our study proved that the temporal distribution of stroke after AMI is not linear. However, most events occurred in the first days after AMI (64% of all strokes recorded occurred within the first 5 days after AMI).

Limitations

This study showed that between 2001 and 2010, stroke incidence (in-hospital and after hospital discharge) remained stable. However, a recent study clearly reported that the risk of stroke during the first year post-AMI decreased between 1998 and 2008, especially in patients with diabetes mellitus (from 7.1% to 4.7% in patients with diabetes mellitus and from 4.2% to 3.7% in patients without diabetes mellitus). Our study did not find this trend probably because of the relatively small number of strokes recorded in our study.

Conclusions

Stroke after AMI severely impairs the prognosis. Acute but also chronic cardiac conditions and inflammation could play a role in short-term stroke events. However, cardioembolism mechanisms remain the leading cause of stroke, despite optimization of acute reperfusion. In contrast, PDS is less common and mostly related to a high cardiovascular risk.

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Disclosures

None.

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