Immediate and Delayed Traumatic Intracranial Hemorrhage in Patients With Head Trauma and Preinjury Warfarin or Clopidogrel Use

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Study objective: Patients receiving warfarin or clopidogrel are considered at increased risk for traumatic intracranial hemorrhage after blunt head trauma. The prevalence of immediate traumatic intracranial hemorrhage and the cumulative incidence of delayed traumatic intracranial hemorrhage in these patients, however, are unknown. The objective of this study is to address these gaps in knowledge.

Methods: A prospective, observational study at 2 trauma centers and 4 community hospitals enrolled emergency department (ED) patients with blunt head trauma and preinjury warfarin or clopidogrel use from April 2009 through January 2011. Patients were followed for 2 weeks. The prevalence of immediate traumatic intracranial hemorrhage and the cumulative incidence of delayed traumatic intracranial hemorrhage were calculated from patients who received initial cranial computed tomography (CT) scan in the ED. Delayed traumatic intracranial hemorrhage was defined as traumatic intracranial hemorrhage within 2 weeks after an initially normal CT scan result and in the absence of repeated head trauma.

Results: A total of 1,064 patients were enrolled (768 warfarin patients [72.2%] and 296 clopidogrel patients [27.8%]). There were 364 patients (34.2%) from Level I or II trauma centers and 700 patients (65.8%) from community hospitals. One thousand patients received a cranial CT scan in the ED. Both warfarin and clopidogrel groups had similar demographic and clinical characteristics, although concomitant aspirin use was more prevalent among patients receiving clopidogrel. The prevalence of immediate traumatic intracranial hemorrhage was higher in patients receiving clopidogrel (33/276, 12.0%; 95% confidence interval [CI] 8.4% to 16.4%) than patients receiving warfarin (37/724, 5.1%; 95% CI 3.6% to 7.0%), relative risk 2.31 (95% CI 1.48 to 3.63). Delayed traumatic intracranial hemorrhage was identified in 4 of 687 (0.6%; 95% CI 0.2% to 1.5%) patients receiving warfarin and 0 of 243 (0%; 95% CI 0% to 1.5%) patients receiving clopidogrel.

Conclusion: Although there may be unmeasured confounders that limit intergroup comparison, patients receiving clopidogrel have a significantly higher prevalence of immediate traumatic intracranial hemorrhage compared with patients receiving warfarin. Delayed traumatic intracranial hemorrhage is rare and occurred only in patients receiving warfarin. Discharging patients receiving anticoagulant or antiplatelet medications from the ED after a normal cranial CT scan result is reasonable, but appropriate instructions are required because delayed traumatic intracranial hemorrhage may occur. [Ann Emerg Med. 2012;59:460-468.]

Please see page 461 for the Editor’s Capsule Summary of this article.
Immediate and Delayed Intracranial Hemorrhage

Editor’s Capsule Summary

What is already known on this topic
Anticoagulant and antiplatelet drugs increase the risk for traumatic intracranial hemorrhage after blunt head trauma.

What question this study addressed
What is the incidence and prevalence of immediate and delayed traumatic intracranial hemorrhage in patients with blunt head trauma who are receiving clopidogrel and warfarin?

What this study adds to our knowledge
In this prospective observational multicenter study of 1,064 patients, the prevalence of immediate traumatic intracranial hemorrhage was 12% for patients receiving clopidogrel and 5.1% for those receiving warfarin. Delayed traumatic intracranial hemorrhage was 0% and 0.6%, respectively.

How this is relevant to clinical practice
In blunt head trauma, patients receiving clopidogrel may be at greater risk of immediate traumatic intracranial hemorrhage than those receiving warfarin. Delayed traumatic intracranial hemorrhage is rare and it may be reasonable to discharge a patient after a normal head CT scan result.

SEE EDITORIAL, P. 469.

INTRODUCTION

Background
The use of anticoagulant and antiplatelet medications, specifically warfarin and clopidogrel, is steadily increasing.1-3 Previous studies suggest that patients receiving either of these medications are at increased risk for traumatic intracranial hemorrhage after blunt head trauma, but the risk in a large, generalizable cohort is unknown.4,6

Importance
The majority of patients with traumatic intracranial hemorrhage are identified on initial cranial computed tomographic (CT) scan. Limited data, however, suggest that patients receiving warfarin are at increased risk for delayed traumatic intracranial hemorrhage (traumatic intracranial hemorrhage diagnosed within 2 weeks of injury after an initially normal cranial CT scan result).7-9 The concern for delayed traumatic intracranial hemorrhage is highlighted by the not uncommon practice of reversing warfarin anticoagulation in patients with head trauma and a normal cranial CT scan result.10 The potential risk for both immediate and delayed traumatic intracranial hemorrhage has generated guidelines recommending routine cranial CT imaging and hospital admission for neurologic observation in head-injured patients receiving warfarin.11-13 These recommendations, however, are not informed by rigorous, prospective, multicenter studies identifying the prevalence and incidence of immediate traumatic intracranial hemorrhage and delayed traumatic intracranial hemorrhage in patients receiving warfarin.

The evidence supporting an increased risk of traumatic intracranial hemorrhage in patients receiving clopidogrel is more limited,11 despite this drug being one of the most commonly prescribed worldwide.15 Although small retrospective studies suggest an increased risk of traumatic intracranial hemorrhage and mortality in head trauma patients receiving clopidogrel,6,16,17 current guidelines do not explicitly recommend routine CT imaging for these patients after blunt head trauma.11-13 In addition, the risk of delayed traumatic intracranial hemorrhage in patients receiving clopidogrel is entirely unknown.

Goals of This Investigation
Knowledge of the true prevalence and incidence of immediate and delayed traumatic intracranial hemorrhage in patients receiving warfarin or clopidogrel would allow clinicians to make evidence-based decisions about their initial patient evaluation and disposition. Therefore, we assessed the prevalence and incidence of immediate and delayed traumatic intracranial hemorrhage in patients with blunt head trauma who were receiving either warfarin or clopidogrel. Warfarin and clopidogrel cohorts were compared. We hypothesized that the prevalence for immediate traumatic intracranial hemorrhage was similar between patients receiving clopidogrel and those receiving warfarin and that the cumulative incidence of delayed traumatic intracranial hemorrhage in both groups was less than 1%.

MATERIALS AND METHODS

Study Design
This was a prospective, observational, multicenter study conducted at 2 trauma centers and 4 community hospitals in Northern California. The study was approved by the institutional review boards at all sites.

Setting and Selection of Participants
Adult (aged ≥18 years) emergency department (ED) patients with blunt head trauma and preinjury warfarin or clopidogrel use (within the previous 7 days) were enrolled. We defined blunt head trauma as any blunt head injury regardless of loss of consciousness or amnesia. We excluded patients with known injuries who were transferred from outside facilities because their inclusion would falsely inflate the prevalence of traumatic intracranial hemorrhage. Additionally, patients with concomitant warfarin and clopidogrel use were excluded.

Data Collection and Processing
The treating ED faculty physicians recorded patient history and medication use, injury mechanism, and clinical examination,
Immediate and Delayed Intracranial Hemorrhage

Nishijima et al

including initial Glasgow Coma Scale score (GCS) and evidence of trauma above the clavicles (defined as trauma to the face, neck, or scalp) on a standardized data form (Appendix E1-E4, available online at http://www.annemergmed.com) before cranial CT (if obtained). Imaging studies were obtained at the discretion of the treating physician and not dictated by study protocol. At each site, approximately 10% of patients (nonrandomly selected) had a separate, independent faculty physician assessment that was masked and completed within 60 minutes of the initial assessment to evaluate the reliability of preselected clinical variables. Data on patients eligible but not enrolled (failures of the study screening process) during ED evaluation were abstracted from their medical records to assess for enrollment bias.

Outcome Measures

Immediate traumatic intracranial hemorrhage was defined as the presence of any intracranial hemorrhage or contusion as interpreted by the faculty radiologist on the initial cranial CT scan. Patients without a cranial CT scan during initial ED evaluation were excluded from the immediate traumatic intracranial hemorrhage calculation. Delayed traumatic intracranial hemorrhage was defined as traumatic intracranial hemorrhage on cranial CT scan, occurring within 14 days after an initial normal CT scan result and in the absence of repeated head trauma. Neurosurgical intervention was defined as the use of intracranial pressure monitor or brain tissue oxygen probe, placement of a burr hole, craniotomy/craniectomy, intraventricular catheter or subdural drain, or the use of mannitol or hypertonic saline solution.

Patients were admitted to the hospital at the discretion of the emergency physician. Patients with normal cranial CT scan results and therapeutic international normalized ratio levels are not reversed at the participating centers. Electronic medical records were reviewed in a standardized fashion by research coordinators and site investigators to assess international normalized ratio results, and did not have traumatic intracranial hemorrhage. Finally, we compared the cumulative incidence of delayed traumatic intracranial hemorrhage, assuming all patients lost to follow-up had a delayed traumatic intracranial hemorrhage.

RESULTS

Characteristics of Study Subjects

Between April 2009 and January 2011, 1,101 patients were enrolled (83.3% of all eligible patients) (Figure). Comparison of patients enrolled and those eligible but not enrolled demonstrated similar characteristics (age, sex, medication use, ED cranial CT, and hospital admission) and outcomes (immediate traumatic intracranial hemorrhage, neurosurgical intervention, and inhospital mortality). Reasons for failures of the study screening process were unknown. Thirty-seven patients were excluded (25 transferred patients and 12 patients with concomitant clopidogrel and warfarin use), leaving 1,064 patients for data analysis. Of the 1,064 patients, 768 patients (72.2%) were receiving warfarin and 296 patients (27.8%) were receiving clopidogrel. There were 364 patients (34.2%) from 2 designated Level I or II trauma centers and 700 patients (65.8%) from 4 community hospitals. The most common mechanism of injury was a ground-level fall (n=887; 83.3%) followed by direct blow (n=59; 5.6%) and motor vehicle crash (n=51; 4.8%).

The majority (n=932; 87.6%) of patients had a GCS score of 15, and 752 (70.7%) patients had physical examination findings of head trauma above the clavicles. The primary indication for warfarin and clopidogrel use was atrial fibrillation (543/768; 70.7%) and coronary artery disease (158/296; 53.4%), respectively. Most patients reported receiving their medication less than 24 hours before injury (warfarin group 660/768, 85.9%; clopidogrel group 252/296, 85.1%). In patients receiving warfarin, 603 of 768 (78.5%) had an international normalized ratio measurement on initial evaluation in the ED (median international normalized ratio 2.5; interquartile range 2.0 to 3.3). The majority of these patients (576/603; 95.5%) had an elevated international normalized ratio (≥1.3), and 458 of 603 (76.0%) had an international normalized ratio (≥2.0).

One thousand of the 1,064 (94.0%; 95% CI 92.4% to 95.3%) received a cranial CT during initial ED evaluation. Hospitalization rates were similar for patients receiving warfarin (271/768; 35.3%) and clopidogrel (93/296; 31.4%). Patient clinical characteristics were similar in both groups, except for headache, concomitant aspirin use,
and evidence of trauma to the neck and scalp laceration, which were more common in the clopidogrel group (Table 1).

**Main Results**

Seventy of the 1,000 patients had immediate traumatic intracranial hemorrhage on ED CT scan. The prevalence of immediate traumatic intracranial hemorrhage was higher in patients receiving clopidogrel (33/276; 12.0%; 95% CI 8.4% to 16.4%) than warfarin (37/724, 5.1%; 95% CI 3.6% to 7.0%; relative risk: 2.31, 95% CI 1.48 to 3.63; *P*<.001) (Table 2). Follow-up was obtained for 63 of 64 of patients not undergoing cranial CT during initial ED evaluation, and none subsequently received a diagnosis of traumatic intracranial hemorrhage. Mortality and neurosurgical intervention rates after immediate traumatic intracranial hemorrhage were not statistically different between cohorts (Table 2).

The majority of patients with immediate traumatic intracranial hemorrhage (45/70; 64.3%) had a normal mental status (GCS score = 15), with similar proportions between the warfarin (23/37; 62.2%) and clopidogrel (22/33; 66.7%) cohorts. Furthermore, in patients with immediate traumatic intracranial hemorrhage, 4 of 37 (10.8%) in the warfarin cohort and 6 of 33 (18.2%) in the clopidogrel cohort had no loss of consciousness, a normal mental status, and no evidence of trauma above the clavicles.

The prevalence of immediate traumatic intracranial hemorrhage varied by participating center. The prevalence of traumatic intracranial hemorrhage was highest at the Level I trauma center (12.6%; 95% CI 8.1% to 18.3%) compared with the Level II trauma center (5.0%; 95% CI 2.3% to 9.2%) and the 4 community centers (5.4%; 95% CI 3.9% to 7.4%). All clinical

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**Figure.** Flow of patients in the study.
Table 1. Demographic and clinical characteristics of the study population.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Total (n=1,064)</th>
<th>Warfarin (n=768)</th>
<th>Clopidogrel (n=296)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Demographics</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age, mean (SD), y</td>
<td>75.4 (12.7)</td>
<td>75.3 (13.0)</td>
<td>75.7 (11.9)</td>
</tr>
<tr>
<td>Male sex</td>
<td>502 (47.1)</td>
<td>362 (47.1)</td>
<td>140 (47.3)</td>
</tr>
<tr>
<td><strong>Mechanism of injury</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ground-level fall</td>
<td>887 (83.3)</td>
<td>644 (83.9)</td>
<td>243 (82.1)</td>
</tr>
<tr>
<td>Fall from height</td>
<td>37 (3.5)</td>
<td>23 (3.0)</td>
<td>14 (4.7)</td>
</tr>
<tr>
<td>MVC, &lt;35 miles/h</td>
<td>18 (1.7)</td>
<td>12 (1.6)</td>
<td>6 (2.0)</td>
</tr>
<tr>
<td>MVC, ≥35 miles/h</td>
<td>24 (2.3)</td>
<td>16 (2.1)</td>
<td>8 (2.7)</td>
</tr>
<tr>
<td>Pedestrian struck by automobile</td>
<td>4 (0.4)</td>
<td>4 (0.5)</td>
<td>0</td>
</tr>
<tr>
<td>Bicyclist struck by automobile</td>
<td>4 (0.4)</td>
<td>3 (0.4)</td>
<td>1 (0.3)</td>
</tr>
<tr>
<td>Direct blow</td>
<td>59 (5.6)</td>
<td>45 (5.9)</td>
<td>14 (4.7)</td>
</tr>
<tr>
<td>Concomitant aspirin use</td>
<td>16 (1.5)</td>
<td>13 (1.7)</td>
<td>3 (1.0)</td>
</tr>
<tr>
<td>Unknown mechanism</td>
<td>6 (0.5)</td>
<td>4 (0.5)</td>
<td>2 (0.7)</td>
</tr>
<tr>
<td><strong>Clinical history</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vomiting</td>
<td>45 (4.2)</td>
<td>34 (4.4)</td>
<td>11 (3.7)</td>
</tr>
<tr>
<td>Headache</td>
<td>357 (33.6)</td>
<td>239 (31.1)</td>
<td>118 (39.9)</td>
</tr>
<tr>
<td>Loss of consciousness or amnesia</td>
<td>196 (18.4)</td>
<td>136 (17.7)</td>
<td>60 (20.3)</td>
</tr>
<tr>
<td>Concomitant aspirin use</td>
<td>43 (4.0)</td>
<td>19 (2.5)</td>
<td>24 (8.1)</td>
</tr>
<tr>
<td><strong>Physical examination</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol intoxication</td>
<td>33 (3.1)</td>
<td>26 (3.4)</td>
<td>7 (2.4)</td>
</tr>
<tr>
<td>Any evidence of trauma above the clavicles</td>
<td>752 (70.7)</td>
<td>531 (69.1)</td>
<td>221 (74.7)</td>
</tr>
<tr>
<td>Trauma to face</td>
<td>406 (38.2)</td>
<td>296 (38.5)</td>
<td>110 (37.2)</td>
</tr>
<tr>
<td>Trauma to neck</td>
<td>36 (3.4)</td>
<td>20 (2.6)</td>
<td>16 (5.4)</td>
</tr>
<tr>
<td>Basilar skull fracture</td>
<td>2 (0.2)</td>
<td>1 (0.1)</td>
<td>1 (0.3)</td>
</tr>
<tr>
<td>Scalp abrasion</td>
<td>157 (14.8)</td>
<td>110 (14.3)</td>
<td>47 (15.9)</td>
</tr>
<tr>
<td>Scalp contusion</td>
<td>309 (29.0)</td>
<td>221 (28.8)</td>
<td>88 (29.7)</td>
</tr>
<tr>
<td>Scalp laceration</td>
<td>182 (17.1)</td>
<td>117 (15.2)</td>
<td>65 (22.0)</td>
</tr>
<tr>
<td>Normal mental status (GCS score 15)</td>
<td>932 (87.6)</td>
<td>674 (87.8)</td>
<td>258 (87.2)</td>
</tr>
<tr>
<td>Mild head injury (GCS score 13–15)</td>
<td>1035 (97.3)</td>
<td>747 (97.3)</td>
<td>288 (97.3)</td>
</tr>
<tr>
<td>Moderate head injury (GCS score 9–12)</td>
<td>18 (1.7)</td>
<td>13 (1.7)</td>
<td>5 (1.7)</td>
</tr>
<tr>
<td>Severe head injury (GCS score 3–8)</td>
<td>11 (1.0)</td>
<td>8 (1.0)</td>
<td>3 (1.0)</td>
</tr>
<tr>
<td><strong>ED course</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Initial cranial CT</td>
<td>1000 (94.0)</td>
<td>724 (94.3)</td>
<td>276 (93.3)</td>
</tr>
<tr>
<td>Admitted to hospital</td>
<td>364 (34.2)</td>
<td>271 (35.3)</td>
<td>93 (31.4)</td>
</tr>
</tbody>
</table>

**LIMITATIONS**

Our results should be interpreted in the context of several limitations. This was an observational study; thus, CT scans were not obtained for all patients and ethical considerations prevented CT scanning solely for study purposes. Some patients not undergoing CT scan during initial ED visit potentially had an undiagnosed traumatic intracranial hemorrhage, although none was identified in follow-up. Furthermore, some patients with a negative initial CT scan result may have eventually developed an undiagnosed delayed traumatic intracranial hemorrhage. We did, however, obtain clinical follow-up, which is a reasonable method to evaluate for clinically important outcomes when the definitive test is not ethical or feasible.\(^{22}\)

The increased risk of immediate traumatic intracranial hemorrhage in the clopidogrel cohort may be attributed to the higher prevalence of concomitant aspirin use compared with the warfarin cohort (8.1% versus 2.5%). However, we conducted a subgroup analysis excluding patients with concomitant aspirin use, and the clopidogrel cohort maintained a significant increased risk for immediate traumatic intracranial hemorrhage compared with the warfarin cohort. We did not collect data on patients with isolated preinjury aspirin use\(^ {23}\) or patients without preinjury antplatelet or anticoagulation use. Finally, patients receiving warfarin may be more acutely aware of the bleeding risks associated with their medication than those receiving clopidogrel. Therefore, patients receiving warfarin may be more apt to seek emergency care, even with trivial head trauma, and thus have less severe mechanisms of injury compared with patients receiving clopidogrel. We were unable, however, to identify such behavior because the clinical characteristics, mechanism of injury, and CT scan rate were similar overall between the warfarin and clopidogrel groups.
Immediate and Delayed Intracranial Hemorrhage

Table 2. Prevalence of traumatic intracranial hemorrhage, neurosurgical intervention, and mortality.

<table>
<thead>
<tr>
<th>Outcome Measures</th>
<th>Patients, No. (%) [95% CI]</th>
<th>Differences in Proportions, % [95% CI]</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total</strong> (n=1,064)</td>
<td><strong>Warfarin</strong> (n=768)</td>
<td><strong>Clopidogrel</strong> (n=296)</td>
</tr>
<tr>
<td>Immediate tICH*</td>
<td>70/1,000 (7.0) [5.5 to 8.8]</td>
<td>37/724 (5.1) [3.6 to 7.0]</td>
</tr>
<tr>
<td>Inhospital mortality after immediate tICH</td>
<td>15/70 (21.4) [12.5 to 32.9]</td>
<td>8/37 (21.6) [9.8 to 38.2]</td>
</tr>
<tr>
<td>Neurosurgical intervention after immediate tICH</td>
<td>12/70 (17.1) [9.2 to 28.0]</td>
<td>5/37 (13.5) [4.5 to 28.8]</td>
</tr>
<tr>
<td>Delayed tICH†</td>
<td>4/930 (0.4) [0.1 to 1.1]</td>
<td>4/687 (0.6) [0.2 to 1.5]</td>
</tr>
</tbody>
</table>

*Immediate tICH is defined as the presence of tICH on initial cranial CT.
†Delayed tICH is defined as the presence of tICH on cranial CT or autopsy after negative initial cranial CT result without new head trauma.
‡Four patients were lost to follow-up (2 warfarin and 2 clopidogrel) and 1 patient died after discharge from the ED (clopidogrel).

Table 3. Patients with delayed traumatic intracranial hemorrhage (all with preinjury warfarin use).

<table>
<thead>
<tr>
<th>Patient Sex and Age, Years</th>
<th>Mechanism of Injury</th>
<th>Initial GCS Score</th>
<th>Initial INR</th>
<th>Repeated Cranial CT Findings (Days After Initial Cranial CT)</th>
<th>Neurosurgical Intervention/Inhospital Mortality (Days After Initial Cranial CT)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Woman, 63</td>
<td>Ground-level fall, isolated head injury</td>
<td>15</td>
<td>1.15</td>
<td>Massive subdural hematoma with uncal herniation (3)</td>
<td>Mannitol/died (3)</td>
<td>Patient was discharged home from initial ED visit. She was found obtunded at home 3 days later. She was taken immediately to the ED and died in the hospital the same day.</td>
</tr>
<tr>
<td>Man, 63</td>
<td>Ground-level fall, isolated head injury</td>
<td>15</td>
<td>1.50</td>
<td>Small intraparenchymal contusion and subarachnoid hemorrhage (1)</td>
<td>No/no</td>
<td>Patient was admitted to the hospital. Routine repeated cranial CT showed a small tICH. Discharged home HD 4.</td>
</tr>
<tr>
<td>Man, 79</td>
<td>Ground-level fall, isolated head injury</td>
<td>15</td>
<td>4.95</td>
<td>Small intraventricular hemorrhage (7)</td>
<td>No/no</td>
<td>Patient was admitted to the hospital. Repeated cranial CT obtained for a change in mental status on HD 7. Patient improved and was discharged home on HD 8.</td>
</tr>
<tr>
<td>Man, 91</td>
<td>Ground-level fall, isolated head injury</td>
<td>15</td>
<td>1.90</td>
<td>Large intraparenchymal, subarachnoid, and intraventricular hemorrhage with midline shift of 9.3 mm (3)</td>
<td>No/died (7)</td>
<td>Patient was admitted to the hospital. On HD 3, repeated cranial CT obtained for a change in mental status demonstrated a large tICH, and patient was made DNR. Died on HD 7.</td>
</tr>
</tbody>
</table>

INR, International normalized ratio; HD, hospital day; DNR, do not resuscitate.

DISCUSSION

Contrary to our hypothesis, the prevalence of immediate traumatic intracranial hemorrhage in patients with clopidogrel was significantly higher compared with those receiving warfarin despite the cohorts’ having similar characteristics. Additionally, we determined in a large and generalizable cohort of patients receiving warfarin or clopidogrel that the development of a delayed traumatic intracranial hemorrhage after a negative initial cranial CT scan result is rare and does not warrant routine hospitalization for observation or immediate anticoagulation reversal with blood products.

To our knowledge, this is the first large, prospective study of head-injured patients with preinjury warfarin or clopidogrel use. We identified 10 warfarin and 3 clopidogrel studies that reported a prevalence of immediate traumatic intracranial hemorrhage.4,6,24-32 The prevalence for immediate traumatic intracranial hemorrhage in patients with preinjury warfarin use ranged from 0% to 65%.4,24-32 The 3 studies evaluating immediate traumatic intracranial hemorrhage in patients with preinjury clopidogrel use demonstrated a prevalence of traumatic intracranial hemorrhage ranging from 36% to 71%.6,25,26 The overall quality of these studies, however, was limited because the majority were small (<100 patients), retrospective registry studies. These studies suffered from significant inclusion bias because the sampled population originated from a trauma registry (patients admitted to a trauma center) and excluded not only patients evaluated and discharged from the trauma center ED but also all patients evaluated at community hospitals. In addition, the prevalence of traumatic intracranial hemorrhage was likely falsely elevated because of the
inclusion of patients transferred to a trauma center. Our study is unique in that a majority of patients were evaluated at community hospitals. Furthermore, we included all patients with any degree of head trauma. Thus, the current study identifies the prevalence of traumatic intracranial hemorrhage in a more generalizable population than those sampled from trauma registries. Numerous case reports and case series described delayed traumatic intracranial hemorrhage, though to our knowledge no previous study evaluated the cumulative incidence of delayed traumatic intracranial hemorrhage.7-9

Current guidelines recommend that patients with head trauma and preinjury warfarin undergo routine cranial CT imaging.11-14 These recommendations are based on theoretical risk and retrospective data because large, prospective studies excluded anticoagulated patients or did not specifically study patients receiving warfarin.33-36 Despite the lower prevalence of traumatic intracranial hemorrhage in this study, the results confirm the substantial risk of traumatic intracranial hemorrhage in patients with blunt head trauma who are receiving warfarin and the benefit of routine cranial CT imaging, even in community hospitals. Previous guidelines, however, do not consider preinjury clopidogrel an indication for cranial imaging.11-13 Despite retrospective data suggesting an increased risk for traumatic intracranial hemorrhage.6,16,17 The current results indicate that the approach to the head-injured patient with preinjury clopidogrel should be similar to that for the head-injured patient with preinjury warfarin use: liberal cranial imaging. Because delayed diagnosis of traumatic intracranial hemorrhage increases morbidity and mortality, early diagnosis of traumatic intracranial hemorrhage is important to initiate treatment, including coagulopathy reversal or neurosurgical intervention.35-39

The prevalence of immediate traumatic intracranial hemorrhage in well-appearing patients is also very concerning. More than 60% of patients with immediate traumatic intracranial hemorrhage in both warfarin and clopidogrel cohorts had a normal mental status (GCS score=15). Additionally, a significant proportion of patients (11% in the warfarin cohort and 18% in the clopidogrel cohort) had no loss of consciousness, a normal mental status, and no physical evidence of trauma above the clavicles. Current National Institute for Health and Clinical Excellence head injury guidelines (updated 2007) recommend urgent (<1 hour) CT imaging in patients with head injury and preinjury warfarin use, provided they sustain loss of consciousness or amnesia.12 In our study, 49 of 70 (70%) patients with immediate traumatic intracranial hemorrhage did not sustain loss of consciousness or amnesia. We recommend routine urgent CT imaging in head-injured patients with previous warfarin or clopidogrel use, even in well-appearing patients without a history of loss of consciousness or amnesia.

The concern for delayed traumatic intracranial hemorrhage in patients with warfarin use stems from case reports and case series,7-9 leading guidelines to recommend routine admission for all head-injured patients receiving warfarin despite a normal cranial CT scan result.14 Moreover, a survey of clinical practices among North American trauma surgeons indicated that 74% of respondents reverse patients receiving warfarin who have blunt head trauma despite a normal cranial CT scan result.10 Furthermore, 66% of respondents reverse these patients with fresh frozen plasma.10 Our results indicate that delayed traumatic intracranial hemorrhage occurs infrequently (<1%) in both populations. Thus, patients receiving warfarin or clopidogrel who have a normal cranial CT scan result and no other indications for admission may be discharged home, albeit

### Table 4. Stratified and sensitivity analyses for immediate traumatic intracranial hemorrhage.

<table>
<thead>
<tr>
<th>Analyses *</th>
<th>Patients, No. (%) [95% CI]</th>
<th>Differences in Proportions, % (95% CI)</th>
<th>Relative Risk (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary analysis</td>
<td>37/724 (5.1) [3.6 to 7.0]</td>
<td>33/276 (12.0) [8.4 to 16.3]</td>
<td>6.8 (2.7 to 11.0)</td>
</tr>
<tr>
<td>Patients 65 y or older</td>
<td>33/594 (5.6) [3.9 to 7.7]</td>
<td>24/217 (11.1) [7.2 to 16.0]</td>
<td>5.5 (3.7 to 7.4)</td>
</tr>
<tr>
<td>Patients with GCS score 13–15</td>
<td>30/703 (4.3) [2.9 to 6.0]</td>
<td>29/268 (10.8) [7.4 to 15.2]</td>
<td>6.6 (2.5 to 10.6)</td>
</tr>
<tr>
<td>Patients with GCS score 15</td>
<td>23/631 (3.6) [2.3 to 5.4]</td>
<td>22/239 (9.2) [5.9 to 13.6]</td>
<td>5.6 (2.2 to 9.5)</td>
</tr>
<tr>
<td>Patients with ground-level fall</td>
<td>30/608 (4.9) [3.4 to 7.0]</td>
<td>27/225 (12.0) [8.1 to 17.0]</td>
<td>7.1 (2.5 to 11.6)</td>
</tr>
<tr>
<td>Patients with evidence of trauma above the clavicles</td>
<td>29/502 (5.7) [3.9 to 8.2]</td>
<td>21/205 (10.2) [6.4 to 15.2]</td>
<td>4.5 (−0.2 to 9.1)</td>
</tr>
<tr>
<td>Patients without concomitant aspirin use</td>
<td>36/705 (5.1) [3.6 to 7.0]</td>
<td>29/252 (11.5) [7.8 to 16.1]</td>
<td>6.4 (2.1 to 10.7)</td>
</tr>
<tr>
<td>Patients evaluated at community hospitals</td>
<td>21/485 (4.3) [2.7 to 6.5]</td>
<td>17/161 (10.6) [6.3 to 16.4]</td>
<td>6.2 (1.1 to 11.3)</td>
</tr>
<tr>
<td>Warfarin patients with INR ≥1.3</td>
<td>35/556 (6.3) [4.4 to 8.6]</td>
<td>33/276 (12.0) [8.4 to 16.3]</td>
<td>5.7 (1.3 to 10.0)</td>
</tr>
<tr>
<td>Warfarin patients with INR ≥2.0</td>
<td>31/441 (7.0) [4.8 to 9.8]</td>
<td>33/276 (12.0) [8.4 to 16.3]</td>
<td>4.9 (0.4 to 9.4)</td>
</tr>
<tr>
<td>Assume patients without cranial CT imaging did not have immediate tICH</td>
<td>37/768 (4.8) [3.4 to 6.6]</td>
<td>33/296 (11.1) [7.8 to 15.3]</td>
<td>6.3 (2.4 to 10.2)</td>
</tr>
<tr>
<td>Assume patients without cranial CT imaging had immediate tICH</td>
<td>81/768 (10.5) [8.5 to 12.9]</td>
<td>53/296 (17.9) [13.7 to 22.8]</td>
<td>7.4 (2.5 to 12.2)</td>
</tr>
</tbody>
</table>

*Based on patients who received a cranial CT scan on initial evaluation after head injury.
Immediate and Delayed Intracranial Hemorrhage

In summary, ED patients with blunt head trauma and preinjury clopidogrel use have a significantly higher prevalence of immediate traumatic intracranial hemorrhage compared with those with preinjury warfarin use. Routine cranial CT imaging is generally indicated in patients with blunt head trauma who are receiving clopidogrel or warfarin, regardless of the clinical findings. The cumulative incidence of delayed traumatic intracranial hemorrhage is very low for both groups, suggesting that in patients with a normal cranial CT scan result, anticoagulation reversal is unnecessary and discharging them home from the ED may be reasonable. Because delayed traumatic intracranial hemorrhage may rarely occur, routine follow-up and appropriate discharge instructions are necessary.

Supervising editor: Robert A. De Lorenzo, MD, MSM

Author contributions: DKN had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. DKN, ASR, and JFH conducted the analysis and interpretation of data. All authors contributed to the study conception and design, acquisition of data, drafting and critical revision of the article, obtaining funding, and approval of the final article. DKN takes responsibility for the paper as a whole.

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The views expressed in this article are solely the responsibility of the authors and do not necessarily represent the official view of NCRR, NIH, or Kaiser Permanente. Information on the NCRR is available at http://www.ncrr.nih.gov/. Information on Re-engineering the Clinical Research Enterprise can be obtained from http://nihroadmap.nih.gov/clinicalresearch/overview-translational.asp.

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REFERENCES

IMAGES IN EMERGENCY MEDICINE

DIAGNOSIS:

Traumatic mydriasis with hyphema. In blunt ocular trauma, the anterior chamber is compressed, forcefully dilating the pupil. This can injure the iris sphincter, dilator muscles, or nerves of the ciliary plexus to form a traumatic mydriasis, which manifests as a moderately dilated pupil with diminished accommodation and reactivity. Visual acuity can be normal or impaired, and intraocular pressure may range from low to high. Treatment with ophthalmic cycloplegics is typically initially used to relax the iris and ciliary body in this often permanent defect.1,2

Like traumatic mydriasis, hyphemas may herald significant intraocular injuries. This patient was found to have an associated cyclodialysis—a tear between the uveal tissue and sclera commonly caused by blunt ocular trauma, which allows additional flow of aqueous humor from the anterior segment into the suprachoroidal space.3

REFERENCES

**APPENDIX E1.** Data collection form for the emergency department.

### Coumadin/Plavix Head Trauma Study v.7.30.09

**Trauma Code:**
- □ 911
- □ 922
- □ 933
- □ no code

**Date of ED arrival:** □/MM/DD  □ AM □ PM

**Date of injury:** □/MM/DD  □ AM □ PM □ Unknown date and time of injury

**Patient contact phone numbers (please check in EMR and patient to confirm contact info):**
- □ Verified
- □ Not verified
- □ If no number in EMR → phone no. ( ) __________ □ ________

**Mode of arrival:**
- □ Private car
- □ Ambulance
- □ Transfer from outside hospital/ED?
  - □ Yes
  - □ No if yes → date/time arrived at outside ED __/__/___ □ AM □ PM

**Reported Med(s):**
- □ warfarin/coumadin
- □ aspirin
- □ clopidogrel/plavix

**Why is patient taking coumadin/warfarin?**
- □ atrial fibrillation (heart arrhythmia)
- □ DVT or PE
- □ heart valve replacement
- □ in-dwelling catheter
- □ other _________
- □ unknown

**Why is patient taking clopidogrel?**
- □ coronary artery disease (CAD)
- □ stroke (CVA)
- □ peripheral artery disease (PAD)
- □ other _________
- □ unknown

**Mechanism of injury:**
- □ fall from standing height or less
- □ fall from greater than standing height (ex. ladder or stairs)
- □ direct blow to head (ex. assault, hit head on table, etc)
- □ unknown mechanism of injury
- □ MVA > 35 MPH
- □ MVA ≤ 35 MPH
- □ pedestrian vs. auto
- □ bicyclist vs. auto
- □ other mechanism___________

**Evidence of head trauma (trauma above the clavicles)?**
- □ Yes (complete below) □ No (skip to GCS question)
  1. Is there trauma to face? □ Yes □ No
  2. Is there trauma to neck? □ Yes □ No
  3. Is there trauma to scalp (from above eyebrows to the occiput)?
     - □ depressed skull fracture
     - □ contusion/hematoma
     - □ signs of basilar skull fracture
     - □ abrasion
     - □ none

**Initial GCS in ED (please fill out):** □ GCS 15 (If Less than 15, complete below)

<table>
<thead>
<tr>
<th>Eye</th>
<th>Verbal</th>
<th>Motor</th>
</tr>
</thead>
<tbody>
<tr>
<td>4 Spontaneous</td>
<td>5 Oriented</td>
<td>6 Follow commands</td>
</tr>
<tr>
<td>3 Confused</td>
<td>4 Inappropriate words</td>
<td>5 Localizes pain</td>
</tr>
<tr>
<td>2 Pain</td>
<td>3 Withdraws to pain</td>
<td>4 Abnormal flexure posturing</td>
</tr>
<tr>
<td>1 None</td>
<td>2 Incomprehensible sounds</td>
<td>3 Abnormal extension posturing</td>
</tr>
<tr>
<td>1 None</td>
<td>2 None</td>
<td>1 None</td>
</tr>
</tbody>
</table>

1. Has the patient vomited? □ Yes □ No □ Unknown
2. Does the patient have a headache? □ Yes □ No □ Unknown
3. Did (or does) the patient have amnesia or loss of consciousness? □ Yes □ No □ Unknown
4. If GCS is < 15 and the patient has dementia, do you think the dementia is the sole cause of the abnormal GCS?
   - □ Yes
   - □ No
   - □ Unknown
5. Is patient clinically intoxicated? □ Yes □ No □ Unknown

---

**GESTALT BOX (PLEASE HAVE ANY OF FOLLOWING FILL OUT):**
- □ EM Attending
- □ EM R3
- □ EM R2

1. Clinical suspicion for the presence of intracranial hemorrhage on CT (regardless of whether a CT was obtained):
   - □ <1% □ 1-5% □ 6-10% □ 11-50% □ >50%
2. Clinical suspicion for intracranial hemorrhage requiring neurosurgery (regardless of whether a CT was obtained):
   - □ <1% □ 1-5% □ 6-10% □ 11-50% □ >50%
3. Were these two questions completed prior to knowledge of CT results?
   - □ Yes □ No

---

**PLEASE PLACE COMPLETED FORM IN MARKED FOLDER IN EMR ROOM!!!**
APPENDIX E2. Data collection form for follow-up.

**Study ID:** ____________

**MRN:** __________________

**Name of Patient:** ____________________________

**Disposition from ED:** ☐ home/care facility ☐ ICU ☐ floor/telemetry ☐ AMA ☐ Transfer

☐ immitch ☐ delayedich ☐ died2wksdcED ☐ died2wksdcall ☐ all mort

***ND= not documented***

**Patient information:**

Age (at time of arrival): ________ ☐ ND ☐ Male ☐ Female

DC date from hospital: __ / ___ / ____ ☐ ND ☐ DC home ☐ LOS: ______ Days

**Initial CT HEAD:** Date: ___ / ____ / _________ - may be from outside hospital

☐ initial CT from outside hospital ☐ initial CT not from outside hospital

☐ normal head CT ☐ abnormal head CT

- Skull fracture: ☐ unknown or none ☐ yes
- SDH: ☐ unknown or none ☐ yes
- EDH: ☐ unknown or none ☐ yes
- IPH: ☐ unknown or none ☐ yes
- IVH: ☐ unknown or none ☐ yes
- SAH: ☐ unknown or none ☐ yes
- Shift: ☐ unknown or none ☐ yes
- Herniation: ☐ unknown or none ☐ yes

1 = normal CT, 2 = no mass > 25 cc, normal cisterns, no shift, 3 = no mass > 25 cc, absent or compressed cisterns, no shift > 5 mm; 4 = same as 3 but with shift > 5 mm, 5 = mass > 25 cc with surgical evacuation; 6 = mass > 25 cc without surgical evacuation

Marshall score (1-6): ____________

**Repeat CT head:** ☐ unknown or none ☐ yes Date: ___ / ____ / _________

Repeat CT: ☐ same ☐ abnormal

Repeat CT head: ☐ normal ☐ abnormal

- Skull fracture: ☐ unknown or none ☐ yes
- SDH: ☐ unknown or none ☐ yes
- EDH: ☐ unknown or none ☐ yes
- IPH: ☐ unknown or none ☐ yes
- IVH: ☐ unknown or none ☐ yes
- SAH: ☐ unknown or none ☐ yes
- Shift: ☐ unknown or none ☐ yes
- Herniation: ☐ unknown or none ☐ yes

Marshall score (1-6) ____________

**LABS:**

Platelet count (initial, may be from outside hospital): __________ per microliter ☐ ND

INR level (initial, may be from outside hospital): __________ per microliter ☐ ND

Direc/415tions:
For patients discharged from ED or admitted to hospital: Fill out page 1-2
For patients with intracranial hemorrhage: Fill out all pages
Follow up phone call
- at least 2 weeks after seen in ED; if has been in-hospital for 2 weeks no need for phone call (fill out “Results”)

☐ dc home from ED ☐ dc home from hospital < 14 days ☐ dc home from hospital ≥ 14 days

Consent:
Patient contacted ☐ yes ☐ no Family contacted ☐ yes ☐ no EMR contact ☐ yes ☐ no
Number of attempts: _____ Date of phone call: _____ / _____ / _____
Patient/family consent ☐ yes ☐ no If consent declined, reason: __________ Unable to contact ☐ yes ☐ no

Symptoms:
post injury problems ☐ yes ☐ no headache ☐ yes ☐ no
nausea/vomiting ☐ yes ☐ no dizziness ☐ yes ☐ no
weakness ☐ yes ☐ no passing out ☐ yes ☐ no
other ____________________________

still taking medication (warfarin or plavix)? ☐ yes ☐ no ☐ unknown

Follow up:
seen by PMD ☐ yes ☐ no date of PMD _____ / _____ / _______
seen in ED ☐ yes ☐ no date of ED _____ / _____ / _______
name of ED _______________________
reason for visit to ED _______________________
admitted to hospital ☐ yes ☐ no date hosp _____ / _____ / _______
reason for admission _______________________
name of hospital _______________________
number of days admitted _____________

Results:
CT head repeated ☐ yes ☐ no
Repeat CT head: ☐ normal ☐ abnormal

Skull fracture: ☐ unknown or none ☐ yes
SDH: ☐ unknown or none ☐ yes
EDH: ☐ unknown or none ☐ yes
IPH: ☐ unknown or none ☐ yes
IVH: ☐ unknown or none ☐ yes
SAH: ☐ unknown or none ☐ yes
Shift: ☐ unknown or none ☐ yes
Herniation: ☐ unknown or none ☐ yes

Marshall score: _____________
death of patient ☐ yes ☐ no
neurosurgery done ☐ yes ☐ no
type of NS _______________________ Date of NS _____ / _____ / _______
**Patients with traumatic ICH: Fill out below**

**Isolated head:**  □ yes  □ no  
*AIS score:* 1 = minor, 2 = moderate, 3 = serious (non-life threatening), 4 = severe (life threatening, survival probable), 5 = critical (survival uncertain), 6 = unsurvivable

AIS head and neck (0-6): __________________

AIS face (0-6): _________________

AIS chest (0-6): _________________

AIS abdomen (0-6): _________________

AIS extremities (0-6): _________________

AIS external (0-6): _________________

ISS (0-75): __________________

**LABS:**

INR level (1st) even if out of hospital: _______ D/T INR level: _____/___/___ _____:_____ □ ND

INR level (2nd) : _______ D/T INR level: _____/___/___ _____:_____ □ ND _____ min from initial

INR level (3rd) : _______ D/T INR level: _____/___/___ _____:_____ □ ND _____ min from initial

INR level (4th) : _______ D/T INR level: _____/___/___ _____:_____ □ ND _____ min from initial

INR level (5th) : _______ D/T INR level: _____/___/___ _____:_____ □ ND _____ min from initial

**TREATMENT:**

PRBCS:  □ no or unknown  □ received RBC during first 48 hours _____ units of PRBCs in 1st 48 hours □ ND

FFP:   □ no or unknown  □ received FFP during first 48 hours _____ units of FFP in 1st 48 hours □ ND

Vit K:   □ no or unknown  □ received Vit K during first 48 hours _____ mg of Vit K in 1st 48 hours □ ND

FVIIa:  □ no or unknown  □ received FVIIa Weight (kg): _____ □ ND

FVIIa: _____ mcg of FVIIa in 1st 48 hours □ ND

FVIIa: _____ mcg/kg of FVIIa in 1st 48 hours □ ND

Time from arrival at UCDMC to drug dosing: ________ minutes □ ND
Study ID: ______________

Prothrombin complex: ☐ no or unknown ☐ received PTC ☐ Weight (kg): _____ ☐ ND
PTC: ________ mcg of PTC in 1st 48 hours ☐ ND
PTC: ________ mcg/kg of PTC in 1st 48 hours ☐ ND
Time from arrival at UCDMC to drug dosing: ________ minutes ☐ ND

Neurosurgical intervention: ☐ unknown or none ☐ yes
☐ ICP monitor (bolt): ☐ unknown or none ☐ yes
☐ Burr hole placed: ☐ unknown or none ☐ yes
☐ Craniotomy done: ☐ unknown or none ☐ yes
☐ Intraventricular catheter done: ☐ unknown or none ☐ yes
☐ Subdural drain done: ☐ unknown or none ☐ yes
☐ Use of mannitol for ICP: ☐ unknown or none ☐ yes
☐ Use of hypertonic saline for ICP: ☐ unknown or none ☐ yes

Other neurosurgical intervention (describe): ______________________________________

Date and time of neurosurgical intervention: _____/_____/______ _____:_____
If no exact date and time documented, give best estimate in minutes: _______________ minutes
☐ no documentation at all of time of neurosurgical intervention

OUTCOMES:

Mortality: ☐ yes ☐ no
Mortality in ED: ☐ unknown or none ☐ yes or suspected
Mortality in 48 hours: ☐ unknown or none ☐ yes or suspected
Mortality in 30 days: ☐ unknown or none ☐ yes or suspected
Discharge home: ☐ unknown or none ☐ yes or suspected
Discharge to SNF (Skilled Nursing): ☐ unknown or none ☐ yes or suspected
Transfer to outside hospital: ☐ unknown or none ☐ yes or suspected
Length of ICU stay: ________________ days ☐ ND
TE (thromboembolism): ☐ unknown or none ☐ yes or suspected
ETT: ☐ yes ☐ no ☐ Days on mechanical ventilation (ETT): _______________ days ☐ ND
Discharge GCS (3-15): _____________ ☐ ND

1 = death 2 = persistent vegetative, minimal responsiveness, 3 = severe disability, conscious but disabled, dependent for daily support, 4 = moderate disability, disabled but independent, can work in sheltered setting, 5 = good recovery, resumption of normal life despite minor deficits

Glasgow outcome score at discharge, (1-5): _________ ☐ ND
**APPENDIX E3.** Data collection form for inter-rater reliability.

### Data Collection Form

**Location:**
- □ UCDMC
- □ Kaiser Sac
- □ Kaiser SSC
- □ Kaiser Ros
- □ Kaiser SRF
- □ Kaiser SSF
- □ Kaiser RWC

**Initial GCS in ED (please fill out)**
- □ GCS 15 (If less than 15, complete below)

<table>
<thead>
<tr>
<th>Eye</th>
<th>Verbal</th>
<th>Motor</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>Spontaneous</td>
<td>6</td>
</tr>
<tr>
<td>3</td>
<td>Verbal</td>
<td>4</td>
</tr>
<tr>
<td>2</td>
<td>Pain</td>
<td>3</td>
</tr>
<tr>
<td>1</td>
<td>None</td>
<td>2</td>
</tr>
</tbody>
</table>

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>None</td>
<td>2</td>
</tr>
<tr>
<td>1</td>
<td>None</td>
<td>None</td>
</tr>
</tbody>
</table>

**Evidence of head trauma (trauma above the clavicles)?**
- □ Yes (complete below)
- □ No (skip to GCS question)
- □ Unknown

1. Is there trauma to face? □ Yes □ No
2. Is there trauma to neck? □ Yes □ No
3. Is there trauma to scalp (from above eyebrows to the occiput)? (if yes, fill out below)
   - □ Depressed skull fracture
   - □ Signs of basilar skull fracture
   - □ Abrasion □ None
   - □ Contusion/hematoma
   - □ Laceration
   - □ Other ________________________

**Has the patient vomited?** □ Yes □ No □ Unknown
**Does the patient have a headache?** □ Yes □ No □ Unknown
**Did (or does) the patient have amnesia or loss of consciousness?** □ Yes □ No □ Unknown

If GCS is < 15 and the patient has dementia, do you think the dementia is the sole cause of the abnormal GCS?
- □ N/A (GCS 15)
- □ Yes, abnormal GCS is caused by dementia alone
- □ No, abnormal GCS is due to injury
- □ Unknown

**Is patient clinically intoxicated?** □ Yes □ No □ Unknown
APPENDIX E4. Data collection form for missed eligible patients.

Data sheet 4: Missed Eligibles

MRN: _________       Name of Patient:________________   MD: __________________

Location
UC Davis
Kaiser North Sacramento
Kaiser South Sacramento
Kaiser Roseville
Kaiser San Rafael
Kaiser South San Francisco

1. Patient information (Based on initial ED):

Age (at time of arrival):_________          □ ND   Gender: □ Male □ Female
Date of ED arrival:  __ /___/____  Time of arrival: ________ □ AM □ PM
GCS _____ □ ND
History of warfarin use: □ unknown or none □ yes or suspected
History of plavix use: □ unknown or none □ yes or suspected
History of aspirin use: □ unknown or none □ yes or suspected