Clinical Neurology and Neurosurgery 174 (2018) 163–166

Contents lists available at ScienceDirect

Clinical Neurology and Neurosurgery

journal homepage: www.elsevier.com/locate/clineuro

Efficacy of platelet transfusion in the management of acute subdural hematoma

John Ogunlade[a,b,*], James G. Wiginton IV[a,b], Hammad Ghanchi[a,b], Zein Al-Atrache[c], Margaret Wacker[a,b], Rosalinda Menoni[a,b], Dan Miulli[a,b]

[a] Riverside University Health Systems Medical Center, Moreno Valley, CA, 92555, United States
[b] Arrowhead Regional Medical Center, Colton, CA, 92324, United States
[c] Philadelphia College of Osteopathic Medicine, Philadelphia, PA, 19131, United States

ARTICLE INFO

Keywords:
Subdural hematoma
Acute subdural hematoma
Antiplatelet therapy
Aspirin
Clopidogrel
Reversal therapy

ABSTRACT

Objective: Oral Antithrombotic Therapy has become a well documented predisposing risk factor in the development of traumatic intracranial hemorrhage. Currently, a reversal protocol for antiplatelet therapy remains ill-defined in the management of non-surgical traumatic subdural hematoma and there is no evidence to suggest a clear benefit of platelet transfusion to mitigate the effect of antiplatelet agents. This study aims to establish parameters in which platelet transfusion would be of benefit in patients with non-surgical traumatic subdural hematoma with preinjury antiplatelet therapy.

Patients and methods: This study is a retrospective chart review of patents from 2015 to 2018 at two Level II trauma centers identifying consecutive patients with non-surgical acute traumatic subdural hematomas. Patients with use of aspirin and/or clopidogrel were categorized into subgroups based on transfusion of platelets for antiplatelet reversal therapy, and were compared to a control group. The primary outcome measure was the presence of subdural hematoma expansion.

Results: A total of 72 patients met the criteria for inclusion in this study. The average age of the cohort was 75.4 with a median of 77.5. There were 40 males and 32 females. Chi-square analysis was performed which demonstrated statistical significance for difference between the aspirin and clopidogrel group for percent of hematoma expansion ($p = 0.0284$). Patients on antiplatelet therapy ($n = 36$) were grouped together and compared to patients without antiplatelet therapy ($n = 36$), this demonstrated that the transfusion of platelets for patients on antiplatelet agents ($n = 19/36$) still resulted in a significant hematoma expansion in ($n = 7/19$, 36.8%) compared to patients not on antiplatelet therapy ($n = 3/36$, 8.3%) ($p = 0.0001$).

Conclusion: The results of this study suggest that patients with non-surgical traumatic subdural hematomas on presentation are less likely to expand, however the risk of expansion is greater when the patient is on antiplatelet therapy. There is no clear benefit in the use of platelet transfusion as a reversal agent to mitigate the effects of antiplatelet therapy in the setting of non-surgical traumatic subdural hematomas.

1. Introduction

The incidence of subdural hematoma (SDH) has persistently increased with each passing decade. From 1993 to 2006, Kalanithi reported an increase in admissions for SDH by 154%, with a simultaneous decrease in mortality from 16.4% to 11.6% [1]. Previous studies have identified major risk factors of SDH as male gender and age greater than 50 [2], however oral antithrombotic therapy (OAT) has become a well-documented predisposing risk factor in the development of traumatic intracranial hemorrhage, with an increase in morbidity and mortality when compared to patients without using OAT [3–7]. Antithrombotic therapy can be further divided into anticoagulants and antiplatelet therapy. Reversal agents for anticoagulation have been well researched and documented however a reversal protocol for antiplatelet therapy remains ill-defined [8–10]. In the management of traumatic subdural hematoma (tSDH) which require surgical evacuation, platelet transfusion is often employed as "reversal therapy" for patients who have known or suspected use of antiplatelet medications, as the risk of intraoperative bleeding is high [11]. However, in the management of non-surgical tSDH the benefit of platelet transfusion remains nebulous, as the literature remains unclear of its efficacy in this regard.

A previous prospective study suggests that platelet count can predict

* Corresponding author at: Riverside University Health Systems Medical Center, 26250 Cactus Ave., Moreno Valley, CA, 92555, United States.
E-mail address: j.ogunlade@ruhealth.org (J. Ogunlade).

https://doi.org/10.1016/j.clineuro.2018.09.021
Received 6 August 2018; Received in revised form 9 September 2018; Accepted 12 September 2018
Available online 13 September 2018
0303-8467/ Published by Elsevier B.V.

radiographic and clinical worsening in patients with traumatic intracerebral hemorrhage on antiplatelet therapy [12]. Earlier studies suggest that there is no benefit of platelet transfusion in patients who have mild traumatic brain injuries with intracerebral hemorrhage with pre-hospitalization antiplatelet therapy [13]. Lindbald recently identified increased platelet function with platelet transfusion, but platelet transfusion did not correlate with improved outcome, instead it was a predictor of poor outcome as a reflection of pathological coagulopathy [14]. Research regarding platelet transfusion in non-traumatic ICH continues to surge, however, investigations in the role of platelet transfusion in management of non-surgical tSDH in patients on antiplatelet therapy is lacking [15,16]. This study aims to establish parameters in which platelet transfusion would be of benefit in patients with non-surgical tSDH with preinjury antiplatelet therapy.

The authors hypothesize that the use of platelet transfusions in patients with a nonsurgical, traumatic acute subdural hematoma does not prevent hematoma progression.

2. Patients and methods

Retrospective chart review of patents from 2015 to 2018 from two Level II trauma centers identified consecutive patients with non-surgical acute tSDH. Patients included in the study were adults, at least 40 years of age, who presented as a closed head injury trauma patient following a ground level fall, motor vehicle accident, or assault, and were diagnosed with an acute subdural hematoma on initial computerized tomography of the head (CTH) by an attending radiologist. All acute tSDH in this had a greatest thickness less than 1 cm and midline shift less than 5 mm, and were deemed non-surgical per neurosurgical guidelines [17].

Patients with non-acute and non-traumatic subdural hematomas, history of previous subdural hematoma or intracranial hemorrhage, history of craniotomy or craniectomy, and depressed skull fracture on presentation were excluded, thus eliminating patients with previous intracranial hemorrhage who are predisposed to subsequent hemorrhage and further hematoma expansion as suggested in previous studies [18,19]. Patients with severe poly-trauma requiring emergent surgical intervention by other services on presentation, known history of coagulopathy, renal or liver impairment (on hemodialysis or history of liver cirrhosis), platelet count less than 100 K, abnormal INR, PTT, or PT on admission were excluded from the study, as these characteristics have been well-documented in the literature as risk factors for hematoma formation and expansion [20–23]. Patients with reported use of other anticoagulants, such as warfarin, fondaparinux, rivaoxaban, apixaban, edoxaban, enoxaparin, or dabigatran were also excluded. No patients were on dipyridamole in combination with aspirin or NSAIDs [23].

The following patient demographics were collected for statistical analysis: age, gender, antiplatelet medication used (aspirin, clopidogrel, both, or none), subdural location, hours until stable repeat CTH, number of platelet units transfused, injury mechanism, and platelet count on arrival. The primary outcome measure was no change in the SDH size on the repeat CTH (stable) or interval expansion of the SDH (unstable). The secondary outcome measure was progression of an expanded SDH to surgical criteria.

Data was stratified into two variables: the use of antiplatelet agent versus no antiplatelet agent. A contingency table was constructed with this data to assess whether the hematoma was stable at the initial repeat CT scan versus unstable. Statistical test for independence was derived from this dataset and the patients with antiplatelet use were further stratified into groups of the specific antiplatelet agent being used (aspirin, clopidogrel, or both). Further subgroup analyses were conducted comparing the specific agents to assess for a correlation between the patients being transfused platelets and stability of the SDH.

Study has been approved by an institutional review board for ethical research.

Table 1
Demonstration of patient population. First row demonstrates the age distribution of the patient population. Second row demonstrates the male versus female distribution with *z*-test demonstrating no significant difference in sex among the cohort. Next four sets of data are the subgroups of male and female within the respective antiplatelet agent; "both" indicates that patients were taking both aspirin and clopidogrel. Their respective P value compares male versus female in their respective groups calculated using proportional *Z*-test.

Patient Demographics					
Age	Mean	Median	Minimum	Maximum	
	75.38	77.5	40	100	
Sex	Male		Female		P Value
	40 (56%)		32 (44%)		0.309
Aspirin	14 (54%)		12 (46%)		0.683
Clopidogrel	4 (67%)		2 (33%)		0.405
Both	1 (25%)		3 (75%)		0.317
None	21 (58%)		15 (42%)		0.337

3. Definitions

All patients received standard neurointensive care for acute tSDH including hourly neurological checks for at least 24 h, systolic blood pressure control 100–140 mmHg, repeat CTH 6–8 h after initial scan, seizure prophylaxis, HOB > 30 degrees, and appropriate pain control. The radiologist report final impression was used to classify the first repeat CTH as stable or unstable. These radiologist interpretations were verified by the authors by comparing the two CTH and performing volumetric calculations using the (A + B + C)/2 method. Hematoma volumes greater than 10% of the initial scan were defined as unstable.

4. Results

A total of 253 patient charts were reviewed amongst the two institutions and 72 patients met the criteria for inclusion in this study. The ages of the patients ranged from 40 to 100, with average age of 75.4 and a median of 77.5. There were 40 males and 32 females who were stratified into the four experimental groups (a) aspirin, (b) clopidogrel, (c) both, and (d) none, which showed no difference in distribution of gender in each group (Table 1). The four experimental groups were further stratified into platelet transfusion and no transfusion groups (Table 2).

Chi square test of independence using a contingency table was used to assess for overall statistical significant and was noted to be significant ($p = 0.0459$). Next, chi-square test was used to evaluate for difference between aspirin ($n = 3/26$) and clopidogrel ($n = 3/6$) groups for rate of unstable hematomas which demonstrated a statistically significant difference ($p = 0.0284$) (Table 3), however, no difference was seen in patients on clopidogrel whether they were transfused with platelets or not ($p = 0.414$) (Table 2). No difference was observed in the rate of hematoma progression in single antiplatelet therapy use ($n = 6/32$) compared to no antiplatelet therapy ($n = 3/36$) ($p = 0.1924$). Finally, all patients with antiplatelet therapy, single and dual ($n = 7/36$) were compared to patients with no antiplatelet therapy ($n = 3/36$) and the rate of hematoma expansion was not significant ($p = 0.175$).

Next, further subgroup analysis with *z*-test was performed to assess for differences in the rate of stable tSDH between the patients on aspirin that had platelet transfusion versus no transfusion; this result demonstrated no significant difference ($p = 0.175$). The same was done for patients on clopidogrel and the result demonstrated similar results of no difference ($p = 0.2104$). Of patients on antiplatelet therapy who received platelet transfusion, hematoma expansion was seen in 36.8% ($n = 6/19$) compared to patients not on antiplatelet therapy 8.3% ($n = 3/36$), which was significant ($p = 0.0001$). Hematoma expansion in patients on antiplatelet therapy who were not transfused ($n = 1/17$, 5.9%) were compared to the group of patients not on antiplatelet