Does Usage of Platelet or DDAVP Prevent Progression of Traumatic Intracranial Hemorrhage in Patients on Anti-Platelet Medication?

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Introduction

Anti-platelet agents(APA) are some of the most commonly used medications in the general population. When these patients suffer traumatic brain injury resulting in an intracranial hemorrhage(ICH), there is a concern for hemorrhage expansion due to their depressed platelet function.(1-5) In post-traumatic ICH(PTICH), the degree of volume expansion with pre-injury APA use is not well delineated, as studies have shown variable results. Current management may involve platelet and/or desmopressin administration; however, clear clinical evidence for their use is lacking.(3) In this study, we have explored the utility of prophylactic administration of platelet and desmopressin administration in preventing the expansion of PTICH.

Objective

To determine whether the adminsitration of platelets or desmopressi is beneficial for patients with traumatic ICH and pre-injury exposure to antiplatelet agents.

Specific Aim

To determine whether platelets or desmopressin prevents the expansion of traumatic ICH when patients are on pre-injury anti-platelet agents.

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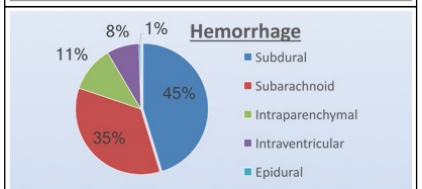
Methods

Medical and radiological records of patients who received treatment at North Shore University Hospital between January 2015-June 2016 for acute traumatic ICH were reviewed. Patients were included if they were on pre-injury APA (aspirin and/or Plavix), but not on anticoagulation therapy. Subjects must have received a non-contrast CT scan between 6-24 hours following the initial scan, without undergoing a surgical intervention between the two scans. The use of desmopressin and platelets was collected. Independent sample t-tests, chi-square and regression analysis were performed to calculate mean and group differences, respectively.

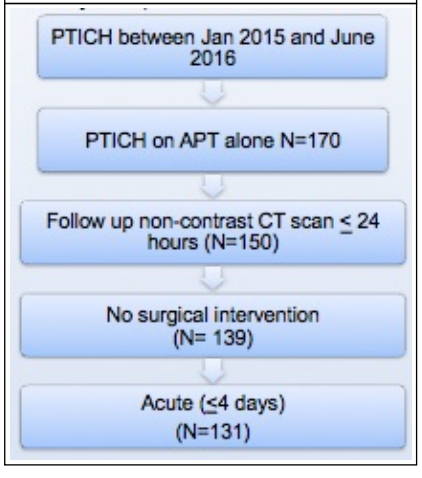
Demographic characteristics depending on overall hemorrhage progression

N=131	Increased	Stable or Decreased	
	Mean (SD)		P-value
Age	83.0 (9.66)	78.7 (11.2)	0.11
Length of stay	14.2 (17.4)	6.7 (5.7)	<0.001
GCS (N=88)	13.4 (2.8)	14.5 (1.2)	<0.001
Platelet (mL)	200 x 10^3 (73.0)	242 x 10^3 (96.4)	>0.05
SBP (mmHg)	145.9 (25.6)	140.7 (24.2)	>0.05
INR	1.06 (.16)	1.16 (1.3)	>0.05
	N (%)	N (%)	P-value
Gender (Male)	15 (68%)	66 (60.5)	0.50
Hypertension	14 (64%)	100 (84.0)	0.003
Prior CVD	3 (0.13)	17 (13.0)	0.9
Smoking History (Yes)	9 (40%)	34(32.0)	0.5



Univariate regression analysis: Effect of therapeutic measure on stabilizing or decreasing the PTICH

N=131	Stable or Decreased (ref)	Increased	OR (95% CI)
Greater than 1 Hemorrhage type	15 (24.6)	46 (75.4)	0.32 (0.12-0.83)
Antiplatelet Agent			
Aspirin	66 (76.7)	20 (23.3)	.17 (0.04-.76)
Plavix	9 (100)	---	--
Aspirin and Plavix	39 (95.1)	2 (4.9)	ref
Therapy			
Desmopressin or Platelet	1 (2.4)	20 (95.2)	0.54 (0.03-9.11)
Desmopressin and Platelet	20 (27.0)	54 (73.0)	0.07 (0.01-0.55)
None	1 (2.4)	40 (97.6)	ref



Multivariable Regression analysis: Effect of therapeutic measure on stabilizing or decreasing the PTICH

N=131	Desmopressin or Platelet	Desmopressin and Platelet	Neither
Model 1 OR (95% CI)	0.54 (0.03-9.11)	0.07 (0.01-0.54)	ref
Model 2 OR (95% CI)	0.56 (0.04-9.37)	0.07 (0.01-0.56)	ref
Model 3 OR (95% CI)	0.70 (0.04-12.63)	0.07 (0.01-0.59)	ref
Model 4 OR (95% CI)	1.01 (0.05-18.95)	0.08 (0.01-0.72)	ref

Outcome: Reference Stable or Decreased
Model 1: Adjusted for type of Antiplatelet (APA) use
Model 2: Adjusted for type of Antiplatelet (APA) use, History of HTN
Model 3: Model 2 + prior CVD , age, admission SBP, male gender
Model 4: Model3 3 + > one regional hemorrhage

Results

Of 131 subjects, 47% had at least two distinct regional hemorrhages and 28% were on dual-antiplatelet therapy. Fifty-five% received both desmopressin and platelet transfsion prior to their follow up CT scan. On follow up, 17% had an expansion of their hemorrhage. While adjusting for the several types of ASA, hisory of hypertension and several other potential confounders, there was a signficant decrease in the likelihood of a stable or decreased hemorrhage when comparing subjects who received platelets and desmopressin to those who did not receive therapy. ($p<0.05$)

Conclusion:

The prophylactic use of platelets and desmopressin does not seem to prevent the progression of acute PTICH in patients exposed to pre-injury APA. In our analysis, the combination of desmopressin and platelets may however worsen the progression. Therefore, the judicious use of these agents is warranted in the acute management of PTICH. Some limitation of our findings include the retrospective nature of the study and the intrinsic biases of the design, a lack of a comparison group of patients not taking antiplatelet agents, and a lack of an equal distribution of sample size across the various therapeutic measures.

References

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