ORIGINAL RESEARCH

Assessment of correlation of D-dimer and progressive haemorrhagic injury

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ABSTRACT
Background: Traumatic brain injury is a significant issue for public health. The present study was conducted to assess correlation of D-dimer and progressive haemorrhagic injury.
Materials & Methods: 58 patients of traumatic brain injury (TBI) of both genders were studied. Parameters such as injury time, mechanism of injury, systolic blood pressure, and GCS score were recorded. 5 ml venous blood was taken and the plasma D-dimer level was evaluated.
Results: Out of 58 patients, males were 38 and females were 20.PHI positive and PHI negative patients, mean time from injury to the first CT scan was 1.6 hours and 2.3 hours, time between the first and second CT scan was 8.2 hours and 9.3 hours. D-dimer level was 6.5 mg/L and 2.1 mg/L, APTT was 26.4 seconds and 25.2 seconds, PTINR was 1.05 and 1.01, PLT was 156.3X109/L and 191.2X109/L, PT was 13.2 seconds and 11.7 seconds respectively. The stepwise logistic regression showed that when D-dimer values were dichotomized at 5 mg/L, time from injury to the first CT scan was no longer a risk factor statistically while the OR value of D-dimer to the occurrence of PHI elevated to 11.9.
Conclusion: Several clinical and laboratory data can be used to predict the development of PHI within hours after the injury. A high D-dimer level implies fibrinolysis, and the occurrence of PHI is particularly likely to be predicted by a compensatory response to hypercoagulation at the time of admission.
Key words: Progressive hemorrhagic injury, Traumatic brain injury, D-dimer.

Introduction

Traumatic brain injury (TBI) is a significant issue for public health. It contributes significantly to morbidity and mortality, and survivors frequently experience cognitive or behavioral issues. Numerous studies have shown that anomalies in blood coagulation frequently developed, leading to a predisposition to hemorrhage in head trauma patients. It is obvious that coagulation abnormalities (whatever defined) occurring simultaneously with brain damage significantly contribute to this morbidity and mortality.[1,2] Progressive hemorrhagic injury (PHI) was defined as the appearance of new lesion or an increase in the volume of hemorrhagic lesion, that is, a ≥25% increase compared with the first post-injury CT scan.[3]The treatment of TBI is complex, and the goal is to promote neuroprotection and cerebral perfusion. Rehabilitation is also essential for functional recovery. Short-term and long-term prognostic markers are still needed, even several prognostic factors have been tested.[4] The Glasgow Coma Score (GCS) is the most commonly used scoring instrument for grading TBI severity. Repeated computed tomography (CT) scans play an important role in the management of TBI.[5]D-dimer is a byproduct of fibrinogen breakdown. An abnormal D-dimer level may indicate an imbalance in the coagulation and fibrinolytic systems, which could change how TBI patients respond to treatment. D-dimer's predictive value in TBI patients has been the subject of extensive investigation, although the findings were inconclusive.[6]The present study was conducted to assess correlation of D-dimer and progressive haemorrhagic injury.

Materials & Methods

The present study consisted of 58 patients of traumatic brain injury (TBI) of both genders. All gave their written consent to participate in the study. Data such as name, age, gender etc. was recorded. Parameters such as injury time, mechanism of injury, systolic blood pressure, and GCS score were recorded. AIS scores were determined according to the 1990 revision of the abbreviated injury scale.PHI was defined as the appearance of new lesion or a

conspicuous increase in the size of hemorrhagic lesion i.e., a 25% increase or more versus the first postinjury CT scan. 5 ml venous blood was taken and the plasma D-dimer level was evaluated. Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

Results

Table I: Distribution of patients

Total- 58		
Gender	**Male**	**Female**
Number	38	20

Table I shows that out of 58 patients, males were 38 and females were 20.

Table II: Assessment of parameters

Parameters	PHI positive(42)	PHI negative(16)	P value
Time from injury to the first CT scan (hours)	1.6	2.3	0.05
Time between the first and second CT scan(hours)	8.2	9.3	0.84
D-dimer (mg/L)	6.5	2.1	0.01
APTT (seconds)	26.4	25.2	0.04
PTINR	1.05	1.01	0.04
PLT (10^9/L)	156.3	191.2	0.01
PT (seconds)	13.2	11.7	0.01

Table II, graph I shows that PHI positive and PHI negative patients, mean time from injury to the first CT scan was 1.6hours and 2.3hours, time between the first and second CT scan was 8.2hours and 9.3hours. D-dimer level was 6.5mg/L and 2.1mg/L, APTT was 26.4seconds and 25.2seconds, PTINR was 1.05 and 1.01, PLT was 156.3X109/Land 191.2X109/L, PT was 13.2seconds and 11.7seconds respectively. The difference was significant (P< 0.05).

Graph I: Assessment of parameters

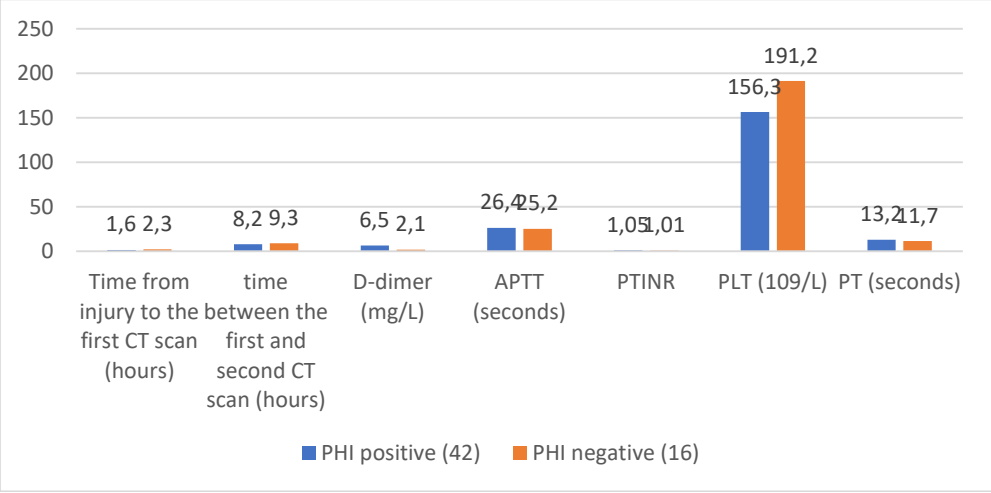

Table III Multivariate logistic regression to identify independent riskfactors for PHI—D-dimer value dichotomized at 5 mg/L

Parameters	OR value	95% CI	P value
D-dimer (>5 mg/L)	11.9	5.4-23.1	0.01
Thrombocytopenia	4.3	1.09-36.4	0.02
Fg abnormal	3.0	1.4-7.4	0.03

The stepwise logistic regression showed that when D-dimer values were dichotomized at 5 mg/L, time from injury to the first CT scan was no longer a risk factor statistically while the OR value of D-dimer to the occurrence of PHI elevated to 11.9.

Discussion

In the United States, there are between 180 and 250 traumatic brain injuries (TBI) per 100,000 people each year.[7,8] It is a significant burden on global public health and a major source of illness and mortality in young adults. An estimated 1.5 million TBI sufferers pass away every year. Vehicle collisions and falls were the main causes of TBI.[9]TBI has been linked to acquired coagulation problems because of an imbalance between anti- and procoagulant factors, platelets, endothelial function, and fibrinolysis. The main causes of aberrant coagulation are tissue injury, hypoperfusion, hypothermia, and acidaemia.[10] Researchers have looked at the correlation between coagulation and fibrinolytic markers and the outcome of TBI in recent years.[11]The present study was conducted to assess correlation of D-dimer and progressive haemorrhagic injury. We found that out of 58 patients, males were 38 and females were 20. Zhang et al[12]evaluated the prognostic role of D-dimer level upon admission in patients with traumatic brain injury (TBI). Eleven studies with 2761 patients were included. Eight studies examined the predictive role of higher D-dimer level for the risk of PHI, and the pooled OR was 1.72. Three studies examined the predictive role of higher D-dimer level for the risk of 3M GOS <3, and the pooled OR was 2.00. Significant between-study heterogeneities were observed, and sensitivity analyses and subgroup analyses were performed. No significant publication bias was found. We found PHI positive and PHI negative patients, mean time from injury to the first CT scan was 1.6 hours and 2.3 hours, time between the first and second CT scan was 8.2 hours and 9.3 hours. D-dimer level was 6.5 mg/L and 2.1 mg/L, APTT was 26.4 seconds and 25.2 seconds, PTINR was 1.05 and 1.01, PLT was $156.3X10^9$/L and $191.2X10^9$/L, PT was 13.2 seconds and 11.7 seconds respectively. Tian et al[13]described and evaluated relationship between D-dimer values and progressive hemorrhagic injury (PHI) after traumatic brain injury (TBI). A cohortof 194 patients with TBI was evaluated in this clinical study. Eighty-one (41.8%) patients suffered PHI as determined by a second CT scan. The plasma D-dimer level was higher inpatients who demonstrated PHI compared with those who did not. Using a receiver–operator characteristic curve to predict the possibility by measuring the D-dimer level, avalue of 5.00 mg/L was considered the cut-off point, with a sensitivity of 72.8% and a specificity of 78.8%. Eight-four patients had D-dimer levels higher than the cut point value(5.0 mg/L); PHI was seen in 71.4% of these patients and in19.1% of the other patients (P<0.01). Factors with P<0.2 on bivariate analysis were included in a stepwise logistic egression analysis to identify independent risk factors for TBI coagulopathy. Logistic regression analysis showed thatthe D-dimer value was a predictor of PHI, and the odds ratio(OR) was 1.341 with per milligram per litre. The step wise logistic regression also identified that time from injury to the first CT shorter than 2 hours, PLT counts lesser than 100×109/L and Fg lower than 2.0 g/L were risk factors for the development of PHI. When D-dimer values were dichotomized at 5 mg/L, time from injury to the first CT scan was no longer a risk factor statistically while the OR value of D-dimer to the occurrence of PHI elevated to 11.850. We found the stepwise logistic regression showed that when D-dimer values were dichotomized at 5 mg/L, time from injury to the first CT scan was no longer a risk factor statistically while the OR value of D-dimer to the occurrence of PHI elevated to 11.9. Xu et al[14]included192 TBI patients. Plasma D-dimer and fibrinogen were measured, and subsequently D/F ratio was calculated. A total of 43 patients (22.4%) experienced PHI. Both Glasgow coma scale (GCS) score (odds ratio [OR], 0.565; 95% CI, 0.464–0.689) and D/F ratio (OR, 4.026; 95% CI, 2.219–7.305) were the two independent predictor for PHI. Area under ROC curve (AUC) of D/F ratio was similar to that of GCS score (AUC, 0.816; 95% CI, 0.754–0.868 vs. AUC, 0.834; 95% CI, 0.773–0.883; $P = 0.699$). Moreover, D/F ratio significantly improved AUC of GCS score to 0.928 (95% CI, 0.881–0.960; $P < 0.001$). The limitation the study is small sample size.

Conclusion

Authors found that several clinical and laboratory data can be used to predict the development of PHI within hours after the injury. A high D-dimer level implies fibrinolysis, and the occurrence of PHI is particularly likely to be predicted by a compensatory response to hypercoagulation at the time of admission.

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