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HEAD INJURY;
INCIDENCE OF HAEMOSTATIC ABNORMALITIES

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ABSTRACT... Back Ground: Abnormal coagulation and fibrinolysis is a complication observed in individuals having traumatic brain injury. **Objectives:** To evaluate the incidence of this complication. **Design:** Retrospective and comparative study. **Setting:** Lahore general Hospital Lahore. **Period:** From 1995 to 2000. **Materials & Methods:** One hundred adults, both male and female, having traumatic brain injury, admitted in Lahore general Hospital Lahore were subjected to estimation of prothrombin time, activated partial thromboplastin time and fibrin degradation products 48 hours after injury. **Results:** Fifty control and thirty three patients with severe head injury were studied. Prothrombin time in 17 patients was prolonged. Which was statistically significant. APTT was prolonged in four cases when compared with normal. FDP were increased in 51% cases. This elevation was again significant statistically. **Conclusion:** Significant changes in prothrombin time and fibrin degradation products were observed after 48 hours of brain injury.

INTRODUCTION

To understand coagulation disorders in patients with head injury, it is essential to understand normal haemostatic mechanism. Haemostatic system is a balanced ongoing activity of promotion of local haemostasis and prevention of generalized thrombosis. Both activities go hand in hand in such a way that blood is retained in blood vessels and keeps on flowing in normal intact vessels and bleeding is arrested from injured vessels[1,2].

Normal haemostasis depends on a delicate, balanced and complex interaction between blood vessels, platelets, plasma coagulation proteins, coagulation inhibitors and fibrinolytic system. When blood vessel is severed due to trauma, haemostatic plug is formed at the site of trauma in few minutes. This process involves vascular responses, platelet adhesion and aggregation and activation of blood coagulation mechanisms.

In vessels having smooth muscles contraction occurs by neurogenic spasm and myogenic vessel rigor, while contraction in micro-circulation appears to be due to local release of vaso-constrictive substances[3].

Endothelial cells of blood vessels possess both thrombotic and antithrombotic factors and because of balanced activity of these factors, a non thrombotic surface is maintained. Antithrombotic factors on endothelial cells are thrombomodulin and heparin like

molecules, which protect against the unchecked action of thrombin[4].

Plasminogen activator converts plasminogen to plasmin which in turn lyses fibrin. Coagulation is promoted by endothelial cells by release of $\alpha 2$ macroglobulin, platelet activating factor, factor 5, tissue factor (thromboplastin) and tissue plasminogen activator inhibitors. The subendothelial connective tissue is also thrombogenic by promoting platelet adherence through Von-Willebrand's factor.

During platelet plug formation, platelets undergo adhesion formation, shape change and platelet aggregation under influence of different factors in circulation and locally released.

Immediately after platelet plug formation, coagulation of procoagulant proteins takes place, end result of this phenomenon is conversion of fibrinogen to fibrin in presence of thrombin which is derived from prothrombin.

Fibrinolytic system removes fibrin fragments and dissolves clots, ensuring a free flowing vascular system. Plasmin (Derivative of plasminogen) is responsible for this fibrinolysis.

These coagulant and anticoagulant activities may be assessed by determining prothrombin time, activated partial thromboplastin time, thromboplastin time and fibrin degradation products. It has been observed that triggering factors for coagulant and anticoagulant cascade are not only released from peripheral circulatory system but also from brain independently[5].

This study was conducted to observe the incidence of coagulopathy in patients who had only heady injury.

Selection criteria

Age: 3.5 - 75 years
Sex Male and female
History of severe heady injury
Glasgow coma scale <9
No history of blood transfusion
Normal control subjects, both male and female of 3.5 to 75 years of age.

Exclusion criteria

Patients with head injury and other parts injury
Blood transfusion within 48 hours
Glasgow coma scale >9

Blood sampling

4.5 ml blood with 0.5 ml trisodium citrate was collected in a test tube for determination of prothrombin time (PT), activated partial thromboplastin time (APTT) and fibrinogen / fibrin degradation products (FDP) estimation.

RESULTS

Thirty three patients were included in this study, having severe head injury. Prothrombin time in control was 13.6±1.01 with a range of 12-15 seconds while prothrombin time in patients was 16.82±5.28 with a range of 13-38 seconds.

Table-I. Prothrombin time in control

Time	No
13.6±1.01 (12-15)	50

Table-II. Prothrombin time in patients

Time (Second)	No of pts	%age
<12	-	-
12-15	20	60.61%
15.1-16.4	5	15.15%
16.5 - 19.4	2	6.06%
>19.5	6	18.18%

Table-III. Comparison of prothrombin time (The values are expressed as mean±SD, the range values are given in parentheses)	
Subjects	**Range Values**
Control(n=50)	13.6±1.01 (12-15)
Patients (n=33)	16.82 ± 5.28 (13 - 38)
Statistical analysis patients vs control = p<0.001)	

The rise in patient"s prothrombin time was statistically very highly significant.

The APTT mean in control subjects was 33.32±2.23 with a range of 30 - 39 seconds. The APTT in patient's samples was 34.66±8.63 with a range of 27 - 87 seconds. When compared with normal group, changes were statistically non significant.

Table-IV. Activated partial thromboplastin time in control	
Time	**No**
33.32±2.33 (33.39)	50

Table-V. Activated partial thromboplastin time in patients (n=33)		
APTT	**No of pts**	**%age**
<20 seconds	-	-
26-29	1	3.03%
30-39	28	84.85%
40-43	-	-
44-58	2	6.06%
59-70	2	6.06%
Statistically non significant.		

Table-VI. Comparison of activated partial thromboplstin time	
Subjects	**Values**
Control (n=50)	33.32±2.33 (33 - 39)
Patients (n=33)	34.66±8.63 (27 - 87)
Statistically non significant.	

Table-VII. Fibrin degradation products in control	
No of subjects	50
Serum FDP (ug/ml)	<10 ug/ml

Table-VIII. Distribution of fibrin degradation products in pts		
Serum FDP (Ug/ml)	**No of pts**	**%age**
<10ug/ml	16	48.49%
10-20	6	18.18%
21-40	4	12.12%
41-80	3	9.09%
>80	4	12.12%

Serum fibrinogen / fibrin degradation products (FDP) level in control group was <10µg/ml. None case having risen level of FDP was recorded. FDP level was very significantly high in patients with severe head injury.

DISCUSSION

Damage to brain tissue leads to the release of coagulation promoting substances which may enter the vascular system and activate the enzyme cascade leading to clot formation. In presence of widespread brain damage extensive coagulation is more likely. This study was conducted to evaluate the incidence of coagulopathy in patients with brain injury responsible for unconsciousness of GCS <9.

Mean values of prothrombin time remained prolonged in 70% cases after 48 hours of brain injury. This observation is in accordance[7]. This persistent prolonged PT may be due to persistent release of higher concentration of tissue thromboplastin from severely traumatized brain as suggested[8].

Mean values of APTT after 48 hours of injury remained prolonged in 12% of cases, this prolongation percentage is highly significant and in accordance[9].

Very strong relationship between severity of brain injury and rise in FDP was observed. These findings are in accordance with Preston et al.

CONCLUSIONS

In presence of significant changes in PT, APTT & FDP in patients with severe traumatic brain injury, it is recommended that these patients must be critically evaluated for these abnormalities before and after surgery to avoid life threatening complications.

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