Original Article

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Impact of Coagulation Profile on Outcome of Head Injury

Surgery Section

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ABSTRACT

Introduction: Worldwide, head injury is recognized as a major public health problem. Head injury patients often develop consumptive coagulopathy in the absence of other trauma or haemorrhages. The release of tissue factor from the damaged brain is postulated as the cause of coagulopathy.

Aim: To know the impact of coagulation profile derangements and their effect on the outcome of head injury patients.

Materials and Methods: Fifty patients in the age group of 20-70 years admitted with isolated head injury were taken. Samples of complete haemogram (CBC), prothrombin time (PTI), partial thromboplastin time (PTK), D-Dimers and fibrinogen were taken within 24 hour of admission. Coagulopathy was defined as platelet counts < 100,000 cells/mm^2 and PTI >15 seconds or a DIC score more than 4. The outcome in each group was measured according to Glasgow outcome score. Coagulation abnormalities were analysed.

Results: In case of severe head injury, p-values in patients who died with regard to DIC score, Prothrombin time and APTT were found to be significant (p< 0.05). For D-Dimers, fibrinogen and platelets counts the p-value was not significant. In case of moderate head injury, p-values in patients who died with regard to DIC score, platelet count, Prothrombin time, D-Dimer and APTT were found to be significant (0.05). For fibrinogen level it was insignificant. The mean DIC score and mean GOS in the severe head injury patients was significant (p<0.001).

Conclusion: Patients with isolated head injury are at risk of development of coagulation abnormalities, which is associated with poor outcome. Based on our results we also emphasize the importance of early diagnosis of coagulation abnormalities in isolated head injury patients.

Keywords: Coagulopathy, Disseminated intravascular score, Glasgow outcome score

INTRODUCTION

Head injury, is recognized as a major public health problem, worldwide [1]. Head injury patients often develop consumptive coagulopathy in the absence of other trauma or haemorrhages. The release of tissue factor from the damaged brain is postulated as the cause of coagulopathy. The release of tissue factor from the damaged brain is postulated as the cause of coagulopathy. Tissue factor activates the extrinsic coagulation cascade and hence can lead to coagulopathy. The development of coagulopathy in head injury contributes to secondary brain injury [2]. The coagulopathy has been defined as the prolongation of prothrombin time. Prolongation of APTT and decrease in platelet counts. This coagulopathy may present as thrombocytopenia secondary to local platelet consumption after cerebral contusion, anaemia or in its most severe form as disseminated vascular coagulation [3]. Once the patients develop disseminated intravascular coagulation the prognosis becomes very poor.

AIM

To know the impact of coagulation profile derangements and their effect on the outcome of head injury patients.

MATERIALS AND METHODS

This prospective study was carried out on 50 patients of isolated head injury admitted in surgery department of Pt. B.D. Sharma Post Graduate Institute of Medical Sciences, Rohtak, India from January 2009- 2010. Ethical committee approval was taken and consent was taken from the patient attendant.

Patients in the age group of 20-70 years were included in the study. Patients having other associated injuries, having hypertension or any other chronic disease, or having pre-existing coagulopathy, taking anticoagulants or drugs which interfere with the laboratory testing were excluded.

All the selected patients were divided based upon their GCS into two groups based on their GCS. There were two subgroups of patient moderate head injury (GCS 9-13) which included 26 patients and severe head injury (GCS < 9) which included 24 patients.

Initial resuscitation and subsequent management was done as per Advanced Trauma Life Support (ATLS). Samples for complete haemogram (CBC), prothrombin time (PTI), partial thromboplastin time (PTK), D-Dimers and fibrinogen were drawn. The blood was collected by venepuncture in EDTA vacutainers as well as PT tubes containing anticoagulant sodium citrate and processed immediately. Based on results of these blood investigations, DIC score was calculated and severity of the DIC was graded [4]. DIC score was calculated as per [Table/Fig-1]. After calculating the DIC score severity of the DIC was graded as shown in [Table/Fig-2].

	Platelet count (in lac)	PT Time (in secs)	APTT (in secs)	D-Dimer (µg/dl)	Fibrinogen (g/l)	Score awarded
Normal	>1.5	13.5	26-34	<1000	>1	0
Mild derangement	1-1.5	13.5-15.0	>34	1000-2000	<1	1
Moderate derangement	0.60 -1.0	15-18	>39	2000-4000	<1	2
Severe derangement	<0.60	>18	>54	> 4000	<1	3

[Table/Fig-1]: DIC score

DIC Score	Inference
0-3	Normal
3-6	Mild derangement
7-10	Moderate derangement
>10	Severe derangement

[Table/Fig-2]: Severity of the DIC

Outcome was defined by the GOS and the comparison of presenting GCS was done with the GOS and DIC score was done.

Coagulopathy- Coagulopathy was defined as platelet counts < 100,000 cells/mm^2 and PTI >15 seconds or a DIC score more than 4. The outcome in each group was measured as discharged (GOS-5) or vegetative state (GOS-2) or dead (GOS-1).

During follow up of the patients GCS, cranial nerve palsy, haematoma formation (surgically operated), local surgical site infection and fever were noted.

STATISTICAL ANALYSIS

Statistical analysis was done by uysing chi square mehthod and student paired t-test. The p-value of <0.05 was taken as significant.

RESULTS

The age of the patients ranged from 20-70 years and was sub-divided in the age groups of 20-30 years (17 patients), 30-40 years (13 patients), 40-50 years (8 patients), 50-60 (7 patients) and 60-70 years age group (5 patients). Total 47 (94%) patients were male and 3 (6%) were female. Accident was the most common mode of injury in these patients and was responsible for 36 (72%) patients. Assault and fall each were responsible for 6 (12%) cases. In 2 (4%) of the cases the mode of injury was unknown. Twenty patients came within 3 hours of injury. Another 20 patients presented within 3-6 hours after injury. Six patients presented within 6-9 hours after injury. Two patients presented 12 hours after injury and in one patient time lag since injury were not known. Total 26 (52%) patients had GCS between 9-13 and 24 (48%) patients had GCS 8 or less. Number of patients having haemorrhagic contusion, extradural haemorrhage, subdural haemorrhage, subarachnoid haemorrhage, depressed fracture skull, diffuse axonal injury and pneumocephalus were as follows; 21 (42%), 11 (22%), 7 (14%), 4 (8%), 4 (8%), 1 (2%) and 1 (2%) respectively. While in 1 (2%) patient had normal CT finding. Forty two (84%) patients were managed conservatively and 8 (16%) required surgical intervention. Out of total 50 patients two patients developed facial nerve palsy and managed conservatively with physiotherapy. One patient recovered while the other patient still had facial nerve palsy at 3rd month.

Parameters	Expired (Group 1) (n=20)	Discharged (Group 2) (n=4)	p-value	Significance
DIC Score	6.3±1.97	4±2.44	<0.001	HS
Platelet	1.64±0.58	1.55±0.31	>0.05	NS
PT Time	15.25±2.98	12.92±1.48	<0.05	S
PTTK	35.84±6.38	28.8±3.85	<0.05	S
Fibrinogen	0.71±0.84	0.40±0.284	>0.05	NS
D-Dimer	2812±1351	2616±1703.86	>0.05	NS

[Table/Fig-3]: Comparison of total DIC scores as well as the individual laboratory tests of expired and discharged patients in the severe head injury group

DIC Score	GOS	p-value	Significance
5.91±2.18	1.54±1.98	< 0.001	HS

[Table/Fig-4]: Correlation of DIC scores with GOS in case of severe head injury patients

Parameters	Expired (group 1) (n-2)	Discharged (Group 2) (n-24)	p-value	Significance
DIC Score	8±1.4	3.92±2.33	< 0.001	HS
Platelets	0.9±0.42	1.75±0.328	< 0.001	HS
PT Time	17.05±1.76	13.75±2.13	< 0.05	S
PTTK	24.05±4.03	34.93±10.71	< 0.001	HS
Fibrinogen	0.47±0.60	0.68±0.66	> 0.05	NS
D-Dimers	4122±883.17	1829.23±1385.15	< 0.001	HS

[Table/Fig-5]: DIC score as well as of individual lab test of expired (GOS-1) and discharged (GOS 3-5) in the moderate head injury group

DIC Score	GOS	p-value	Significance
4.23±2.51	4.62±1.09	> 0.05	NS

[Table/Fig-6]: DIC score with GOS in moderate head injury patients

Patients in severe head injury were divided into two groups. The 1st group included 20 patients which had GOS-1 and 2nd group included 4 patients having GOS–5. In case of severe head injury group patients on analysing the above data of two groups p-value in Case of DIC score was < 0.001 and was highly significant. The p-value in both Prothrombin time and APTT were < 0.05 and were found to be significant. In case of D-Dimers, fibrinogen and platelets the p-value was > 0.05 was not significant [Table/Fig-3].

The mean DIC score was 5.91±2.18 in the severe head injury patients while the mean GOS was 1.54±1.98. On analysing p-value was found to be highly significant (p<0.001). [Table/Fig-4] showing correlation of DIC score with GOS in case of severe head injury patients (n = 24).

In moderate head injury group patients on analysing the above data of two groups p-value in case of DIC score was <0.001 and was found to be highly significant. The p-value in case of platelet count was <0.001 and was highly significant. The p-value in case of prothrombin time was, 0.05 and was significant. The p-value in case of APTT was, 0.001 and was highly significant. The p-value in Fibrinogen was > 0.05 and was not significant. The p-value in D-Dimer was <0.001and was highly significant. Comparison of Total DIC score as well as of individual lab test of expired (GOS-1) and discharged (GOS 3-5) in the moderate head injury group [Table/Fig-5].

The mean DIC score was 4.23±2.51 while the mean GOS was 4.62±1.09 in the moderate head injury group. On analysing p-value was found to be non significant (p> 0.05). [Table/Fig-6] showing correlation of DIC score with GOS in moderate head injury patients (n=26). The overall mean DIC score of all the patients were 5.04+2.49 while the overall mean GOS in these were 3.1±1.9. On analysing both, p-values were found to be highly significant (p< 0.001).

DISCUSSION

Head injury incidence is increasing day by day, now a days, most commonly due to increase in the number of road traffic accidents which in turn is due to the increasing number of vehicles on the road [5]. Out of 50 patients (9.12, mean GCS), 48 % (n=24) had severe head injury while 52% (n=26) had moderate head injury which is at par with BK Gan et al., (8.3, mean GCS) but not with Carolina et al., in which 62.8% patients had severe head injury [6,7].

In the present study majority of the patients had road side accident 72% (n=36) which is at par with Carolina et al., [7]. Mortality in present study was 42% which was at par with Carolina et al., and Greuters et al., [7,8]. Coagulopathy developed in 76% (n=38) patients in our study which was in concordance with Carolina et al., but more than those of Greuters et al., and Harhangi et al., in which it was 54% and 33% respectively [7-9]. This variation in the prevalence of coagulopathy may be due to the variations in the definition of coagulopathy, diversity in the level of injury severity among various studies.

The mean platelet value in the severe head injury group patients was 1.63±0.54 lacs while it was 1.69±0.40 lacs in patients of moderate head injury patients in our study. This is further supported by the study of Martin et al., they observed thrombocytopenia to be an independent risk factor for progressive head injury. They observed a significant decrease in platelets counts in patients having progressive head injury [10].

The mean values of PTI/INR in the severe head injury group were 1.06+0.12 and 1.26+0.24 in the discharged and the expired

patients respectively while it in the moderate head injury group it was 1.11+0.19 and 1.35+0.14 in the discharged and expired patients respectively. Our findings were in concordance with the findings of Vineet et al., [11]. The patients of both moderate and severe head injury group who died had higher INR values as compared to the discharged ones.

The mean D-dimers value was 2377.20+1468.14 µg/dl in the present study. The mean value in severe and moderate head injury group was found to be 2779.73+1375 µg/dl and 2005.63+ 1478.08 µg/dl. Observations in present study with respect to D-dimer were similar with the findings of Scherer R et al., who also reported increase in D-dimer concentration in 20 head injured patients in comparison to other trauma patients [12]. Similar results were seen in the study of Kuo et al., [13]. They observed that D-Dimer values > 1496 µg/dl was associated with poor prognosis in head injury patients.

Seventy six percent of the patients in the present study had moderate to severe DIC scores. Selladurai et al., observed that 38 percent of their patients had moderate to severe DIC scores [14] while Vineet et al., observed 63% of their patients had moderate to severe DIC scores [11]. The variation in the percentage of patients having such wide range of scores can be explained on the basis of differences in the severity of injuries of patients in the different studies and due to reason that different studies they use different criterion for calculating the DIC score. In the moderate head injury group the mortality was 8% while in the severe head injury group the mortality was 79%. In those patients who developed coagulopathy the mortality was 55%. The overall mortality in the present study was 42% while it was 34% and 60% in Carolina et al., and Selladurai et al., [7,14].

On correlating DIC score with the outcome of the patients we observed the DIC score to be significant (p-value was < 0.001). Carolina et al., and Greuters et al., also observed that presence of coagulopathy was related to a fatal outcome and on correlating coagulopathy with outcome observed the p-value was < 0.01 in the former <0.04 in the latter study respectively [7,8].

CONCLUSION

Incidences of head injury are rising day by day and the mortality and morbidity is high in cases of head injury. From the present study we concluded that patients with isolated head injury are at a risk of development of coagulation abnormalities, which is associated with poor outcome. Based on our results we also emphasize the importance of early diagnosis of coagulation abnormalities in isolated head injury patients. However, further studies are warranted to strengthen our study.

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