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A TWO YEARS PROSPECTIVE STUDY ON HEMORRHAGIC PROGRESSION OF CEREBRAL CONTUSION IN RELATION OF COAGULOPATHY

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ABSTRACT Cerebral contusions are common radiological findings in traumatic head injury. Patients with associated coagulopathy may have risks of progression of contusions in post trauma days. In this prospective study on 54 patients with contusional head injury, we tried to correlate radiological progression of contusions in relation with some common laboratory investigations on coagulopathy. We concluded that repeat CT scan is necessary in patients with contusions in every case of clinical deterioration to look for hemorrhagic progression irrespective of having normal laboratory study reports on coagulation.

KEYWORDS :

INTRODUCTION:

Cerebral contusions are one of the most common form of traumatic brain injury. This most commonly occur in ares where sudden deceleration of the head causes the brain to impact on bony prominences like frontal, temporal and occipital poles in coup or contrecoup fashion. It need surgical treatment if there is progressive neurological deterioration, contusion volume is more than 50 cm^3 or Glasgow Coma Score is 6-8 with frontal or temporal contusion volume is more than 20cm^3 with midline shift is more than 5mm. Hemorrhagic progression of contusions is due to continued bleeding of microvessels fractured at the time of primary injury.[2] When head trauma results in a cerebral contusion, the hemorrhagic lesion often progresses during the first several hours after impact, either expanding or developing new, non-contiguous hemorrhagic lesions, a phenomenon termed hemorrhagic progression of a contusion (HPC).[15]This concept has given rise to the notion that continued bleeding might be due to an overt or latent coagulopathy.[1] In the Traumatic Brain Injury, coagulopathy is often broadly defined as any perturbation in a patient's coagulation parameters, and may include a prolongation of the prothrombin time (PT), an elevation of the International Normalized Ratio (INR), an elevation of the activated partial thromboplastin time (aPTT), or a decrease in the platelet (PLT) count.[3] Presence of coagulopathy is a causative risk of progression of hemorrhage. Although coagulopathy after TBI has long been recognized to be a non-specific indicator of poor prognosis,[4] review of the literature does not support a simple causative relationship between coagulopathy and progressive delayed contusional hemorrhage.

AIMS AND OBJECTIVES:

The aim of our study is to find any relationship between coagulation disorder in the form of post traumatic abnormalities in Platelet count, Prothrombin time (PT), International Normalised Ratio(INR), actvated Partial Thromboplastin time(aPTT) and hemorrhagic progression of contusions.

MATERIALS AND METHODS:

We performed this study in our Institute, a semi referral centre for head injury patients from January 2012 to December 2013. Among the head injured patients, we selected the patients admitted with CT scan done within 12 hours of injury shows contusions. All patients with genetic coagulation disorder and patients on any type of anticoagulative medications were excluded from this study. Platelet count, aPTT, PT and INR were studied on second hospital day and repeated on seventh day. Repeat CT scan was done on third post trauma day. We tried our best to perform the coagulation tests from a same laboratory in a very few cases we had to avail reports from another laboratory. We restricted the normal values for our study like Platelet count:1,50,000-4,50,000, aPTT: 26.3-39.4S and INR above 1.5 was determined as elevated.

RESULT:

Among the brain injured patients we selececed 54 patients of mean age 26years(15-67 years) for our study. These 54 patients were with cerebral contusions at their initial CT scan performed within six hours of injury and were admitted in our Institute within 12 hours of injury. Male patients outnumbered females as male-female ratio was 8:1(48 male and 6 female patients).The mode of injury was mainly road traffic injury followed by fall, fall of heavy object over head and assault.

Mode of injury	Male	Female
RTA	30(62.5%	4(66.6%)
Fall from height	12(25.0%	2(33.3%)
Fall of object	4(8.3%)	--
Assault	2(4.17%)	--

There were associated injuries along with brain contusions like othe obvious brain injuries detected in CT, long bone injury, facial injury, ophthalmic injury etc. as tabulated below.

Associated injury	Male	Female
Ac Subdural hge	2	--
SAH	2	1
Skull #(Linear/depressed)	4	--
Long bone #	3	1
Chest injury	1	--
Facial injury	1	--
Ophthalmic injury	1	--

Areas of brain involved are as follows:

Area of brain	Male (n=48)	Female (n=6)	HPC (n=13)
Frontal	15(31.25%)	3(50.0%)	7(M=6, F=1)
Temporal	9(18.75%)	2(33.3%)	1(Male)
Fronto temporal	13(27.08%)	1(16.7%)	5(M=3,F=2)
Parietal	7(14.58%)		-
Occipital	4(8.33%)		-

Coagulation parameters on 2nd and 7th Admission day:

Coagulation Parameter	2nd Adm day n(%)	7th Adm day n(%)
Normal Platelet count	46(85.18%)	39(72.22%)
Low platelet count	8(14.81%)	15(27.78%)
Normal aPTT	44(81.48%)	38(70.37%)
Elevated aPTT	10(18.52%)	16(29.63%)
Normal PT	40(74.07%)	35(64.81%)
Elevated PT	14(25.92%)	19(35.18%)
Normal INR	42(77.78%)	34(62.96%)
Elevated INR	12(22.22%)	20(37.04%)

Glasgow Coma Score on admission and hemorrhagic progression of contusion:

GCS	n(%)	HPC n(%)
Minimal, GCS 15	2(3.70%)	-
Mild, GCS 14	10(18.52%)	3(30%)
Moderate, GCS 9-13	36(66.67%)	9(25%)
Severe, GCS 5-8	4(7.41%)	1(25%)
Critical, GCS 3-4	2(3.70%)	-

Relationship of coagulopathy (as reported on second day and seventh day of trauma) and progression of contusions:

HPC	Platelet count		aPTT		PT		INR	
Total HPC 13	Normal	Low	Normal	High	Normal	High	Normal	High
Second day	10	3	8	5	5	8	9	4
Seventh day	9	4	9	4	4	9	7	6

In our study, we found that total 13 patients were suffered from hemorrhagic progression of contusions(HPC) and out of 13, number of female patients were three. According to the GCS status, the two critically injured patients admitted expired within 12 hours of admission (one post operative), and we found that even upto 30% of mild head injured patients developed HPC followed by equal fear of 25% chance of HPC in moderate head injury and severe head injury patients.

We did not get any conclusive evidence of any relationship between coagulation parameter and HPC. Out of 13 patient with HPC, 10(76.92%) and 9(69.23%) patients were having normal platelet count on second and seventh day of admission respectively. Similarly, aPTT was normal in 8(61.54%) and 9(69.23%) patients on second and seventh day of admission respectively. Retrospectively we found that initial (second day) PT was high in eight patients when we repeated CT scan on third day and among the 13 HPC cases detected on repeat CT on third day, 9 patients(69.23%) were still having elevated PT. But among 13 HPC cases detected on third post admission day, INR was normal in nine(69.23%) and seven(53.85%) patients on second and seventh day of admission.

DISCUSSION:

Depending on the severity of injury, time of testing, sensitivity of clotting tests, and the specific parameter being measured, the incidence of clotting abnormalities in TBI patients is reported to vary from 15–100% [5]. More recent studies show that up to 45% of severe traumatic brain injury patients become coagulopathic.Coagulopathy can develop up to 5 days after injury, and the incidence appears to be linearly correlated with increasing severity of injury.[6]

Mechanisms that account for coagulopathy in TBI have not been fully elucidated, and disagreements persist as to the cause. Tissue factor (tissue thromboplastin) is abundant in the brain, and may be released in large quantities following trauma. Diffuse activation of the extrinsic coagulation pathway may lead to disseminated intravascular coagulation (DIC). Subsequent consumption of clotting factors may cause a bleeding diathesis. Another explanation denotes that coagulopathy is more likely to occur when both tissue injury and hypoperfusion are present, with the protein C pathway perhaps playing an important role.[7]

The reported incidence of an elevated PT or INR in the context of TBI varies widely. A study of moderate and severe TBI reported an incidence of 5%, [8]but another study found an overall incidence of 26%. 1Several studies have reported on PT measurements and the incidence of radiologic progression of hemorrhagic contusions. Tian and co-workers in 2010 observed that only 7.4% of the patients who showed progressive injury on CT had an elevated PT.[9] A widely cited report by Oertel and colleagues , in which serial coagulation tests were obtained, found that 57% of patients who had elevated PT on a first coagulation panel showed progression on CT, and 60% who had elevated PT on a second coagulation panel did not show progression .[10] Engstrom and co-workers found no difference in PT values at any time between groups that would develop or would not develop hemorrhagic progression of contusions .[11] Allard and associates observed that 42% of patients with normal INR values showed evidence of lesion progression in follow up CT scans. So, from statistical analysis we can not blame elevated INR or PT as a sole cause of progression of hemorrhagic contusions in follow up CT scan of head injury patients.

The incidence of abnormal aPTT is 1-30% of all traumatic brain ijury patients. It shows a definite relationship of progression of contusions in follow up CT scans. Variability of results may be due to severity of injury, time of testing and difference of different laboratory values. Allard and coleagus showed 48% patients with elevated aPTT developed incresed contusions.[12]Tian and coleagues fond it only 4.9% [8]whereas Oertel and coleagues found it to be 3.4% [9]and Stein et al showed that of all patient with progression of contusions on Ct, 12% were with abnormal aPTT.[5]

An IMPACT study in 2007 observed that 7% of TBI patints shows thrombocytopenia.[1] Different studies sow variable results of incidence of progression of hemorrhagic contusion in relation with thrombocytopenia. Engstrom and associates showed no relationship between thrombocytopenia and hemorrhagic progression of contusions.[3] Allard et al found progression of contusions in 91% of patints with low platelet count and 44% with normal count [12]Tian et al found it to be 13.6% [9]and Stein et al found it to be 11%.[5]Out of the 38 patients with HPC in the report by Patel and colleagues (Patel et al., 2000), only 2 (5.2%) were coagulopathic.[14]Though some of these studies documented some association between thrombocytopenia and progresssion of hemorrhagic contusions, but it appears that only low platelet count is not sole responsible for progression of contusions rather platele dysfunction may be more determining risk factor than count.[13]

To conclude, we can say from this study that mere presence of measurable coagulopathy is neither a definite risk factor for progression of hemorrhagic contusions, nor its' absence reduces the fair chance of progression of contusions. It is better to recommend to have a repeat CT scan of brain in every case of neurological deterioration in patients with traumatic cerebral hemorrhagic contusions.

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