Prothrombin Time role in Head Injury & Intracranial Hematomas, A prospective Study of 325 cases

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## Abstract

**Objective:** Aimed to asses the role of PT estimation in early diagnosis and predicting the extent and the outcome of head injury with ICerH and/ or Contusion **Method :**PT was measured by Digiclot 818

Group –1: One hundred consecutive head injured patients admitted at Neurosurgical and Al Ramadi teaching hospitals were initially estimated for prothrombin time and subsequently scanned

Group-2: Two hundred twenty five consecutive non scanned head injured patients admitted to Neurosurgical and Al Ramadi teaching hospitals were estimated with prothrombin time at the time of insult and subsequently for the next two weeks

Clinical and neurological evaluation (GCS) score in addition to computerized tomography scan was done for both groups

**Result** <u>:</u>Group -1: Twenty eight (28%) of the initially estimated head injured patients had prolonged prothrombin time in which their subsequent CT scan revealed an ICer H and/or contusion

Seventy six (76%) percent of those with severely head injured patients (GCS $\leq$ 8) were having an initial prothrombin time prolongation...While those with mild

#### Introduction

This work is a laboratorial study with a clinic and radiological correlation between prothrombin time prolongation and the extent, type of intracranial hematomas and prognosis of head injury A close head injury is defined as an injury with an intact inner skull table while an intracranial hematomas can be divided into membranous hematomas which includes SDH,EDH,SAH ...and an intracerebral hematomas and/or contusion <sup>1,2.</sup>

Prothrombin time is a measurement of the extrinsic coagulatory cascades initiated by tissue thromboplastin which is liberated in excessive amount from the assaulted brain through activation of factor 7...in addition to the presence of factor <sup>2,9,10.1,2,3,4,and.5</sup>.

As a result of the movement between the brain anddura and inner table of the skull during the momentum induced by the assault the tissue in addition to the structural brain damage thromboplastin will be liberated in an excessive amount to the circulation.6.

#### Methods

This is a prospective randomized study has been conducted at Neurosurgical and Al Ramadi teaching hospitals from Oct.1999 to Oct.2003 a head injury (GCS $\geq$ 12) only thirty (30%) of them were having the initial prothrombin time prolongation

Group-2 Those patients who survived the initial insult and subsequently thereafter (surgically or conservatively treated) even with a deficit. Daily estimation of prothrombin time showed a decline within the 7-14 days

Those patients who finally died (surgically or conservatively treated) daily estimation of prothrombin time showed a persistent elevation or a fluctuation within the next 7-14 day

<u>Conclusion</u> Prothrmbin time is an important parameter in :

1-Reflecting the severity of head injury (GCS)

3-Early detection of ICerH and /or contusion

2-Prognostic value in cases of of ICerH and /or contusion

Abb : ICerH = Intracerebral hematomas,SDH=subdural hematomas,EDH=epidural hematomas,SAH=subarachnoid

hemorrhages,PT=Prothrombin time,Pts=Patients,CT scan=computarized tomography.

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total of 48 months , during this period three hundred twenty five closed head injured patients were included in this study, mlae to female ratio 3.5/1, with a follow up period of 1-2 weeks.

<u>Group A</u> : includes a 100 consecutive non scanned pts with a close head injury, initial PT was carried out in addition neurological evaluation (GCS score) and a subsequent CT Scan was carried out and the patients were subdivided into three groups.7,8 and 9,.

1-Mild head injury score= (13-15)

- 2-Moderate head injury (9-12)
- 3-Severe head injury ( $\leq 8$ )

Prothrombin time estimation by "Digiclot 818 ". A control level of twelve seconds was standardized a prolongation more than two seconds regarded a abnormal 11...a prolongation more than two seconds was regarded as abnormal. <u>Group B</u> : including 225 pts for those how proved by CT scan to have ICerH , PT was estimated initially and daily for 1-2weeks later, also these patients were subdivided into two groups : Surgically treated and conservatively treated pts

## Results

## Group 1

An Initial Prothrombin time prolongation was observed in 28% of the

Cases in which a follow up CT scan showed an ICerH and/or contusion

| Prothrombin time | patients | Intracranial pathology      | Percent of total |
|------------------|----------|-----------------------------|------------------|
|                  |          | CT Scan                     |                  |
| prolonged        | 16       | ICer H                      | 16%              |
| prolonged        | 12       | contusion                   | 12%              |
| normal           | 46       | Membranous hematoma         | 46%              |
| normal           | 26       | Axonal injury or concussion | 26%              |
| Total            | 100      |                             | 100%             |

# Table-1 Initial prothrombin time prolongation in non scanned head injured patients can predict an ICerH and / contusion

In addition an initial prothrombin time estimation for 100 consecutive head injured patients with GCS evaluation showed that the more severe the injury the highest percentage of prothrombin time prolongation

|             | 3                |                      |                  |
|-------------|------------------|----------------------|------------------|
| GCS         | Prothrombin time | Patients             | % prolonged from |
|             |                  | Prolonged from total | total            |
| 12-15       | Prolonged        | 6 /20                | 30               |
| 20 patients | normal           | 14/20                |                  |
| 9-12        | Prolonged        | 23/34                | 68               |
| 34 patients | normal           | 11/34                |                  |
| $\leq 8$    | Prolonged        | 35/46                | 76               |
| 46patients  | normal           | 11/46                |                  |
| Total 100   |                  | prolonged 64         | 64               |
|             |                  | total 100            |                  |
|             |                  |                      |                  |

## Table-2

#### **Initial prothrombin time prolongation in head injured patients in relation to the GCS Initial prothrombin time prolongation was found in 64 out of 100 patients (64%) With severe head injury prothrombin time prolongation is more pronounced** Group 2

Those cases with ICeH were Followed with subsequent daily estimation of prothrombin time.

For those who survived the initial insult with or without a deficit whether treated conservatively or surgically showed that their prothrombin time would return to normal within 7-14 days.

## Fig-1, Fig -2.

While subsequent daily estimation of prothrombin time for those who finally died whether treated conservatively or surgically showed that their prothrombin time would remain high or fluctuated. Fig-1, Fig-2

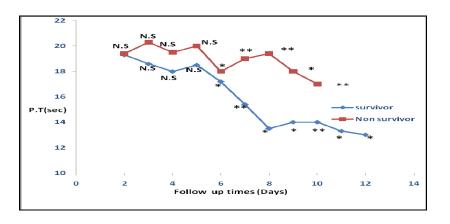


Fig-1(Prothrombin time estimation in <u>surgically</u> treated patients with ICer H and survived more than 8 days from the initial prolongation)

Those who remained alive their Prothrombin time return toward norm control level after the first week, while those who finally died their Prothrombin time remained elevated \* = p < 0.05

\*\* = p < 0.001

NS=not significant

Each dote represent at least a mean Prothrombin time estimation of at least 15 out of 23 patients for those who survived and 14 out of 22 patients for those who finally died

Surgery within 48 hours of insult, total 45 patients

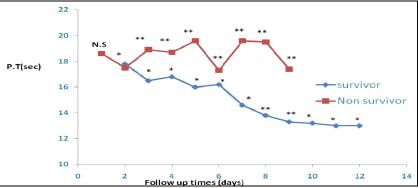


Fig-2(Prothrombin time estimation in <u>conservatively</u> treated patients with ICerH and or contusion and survived more than 8 days from the initial prolongation)

Those who survived even with a deficit their prothrombin time return toward norm control level after the first week, while those who finally died their Prothrombin time remained elevated Total 102 patients

\* = p < 0.05. Statistically significant

\*\* = p <0.001... Statistically significant

NS=not significant

Each dote represent at least a mean prothrombin time estimation of at least 60 out of 118 patients for those who survived and 30 out of 62 patients for those who finally died

# Discussion

In this study we try to throw some light on the relation of the prothrombin time with the pattern and sequally of head injury

Initial prothrombin time prolongation in non scanned closed head injured patients was found in 28 out of 100 (28%) in which this can predict an ICerH or contusion table- 1 ,Vander Sand JJ et al concluded that coagulation study was more sensitive than CT scan and clinical changes in demonstrating brain contusion 6.12.

The initial prothrombin time prolongation after ICerH and/or contusion is due to the liberation of tissue thromboplastin which is present in large amount in brain tissue, consequently this process will trigger the extrinsic cascades of coagulation which depends mainly on factor VII the final result will end with a decrease factor VII so reflected by prolonged

prothrombin time (prothrombin time control for healthy persons were 12+,-2 seconds..Control healthy persons).

In our study, all patients had prothrombin time estimation soon after the insult, prothrombin time prolongation was seen in 64 out of 100 (64%),this elevation was detected within the first 48 hours after the insult table-2,

Tissue thromboplastin liberation is directly proportional to the amount of tissue damages again reflected by prothrombin time prolongation as the severity of tissue damaged increases, 30% prothrombin time prolongation in mild head injury while 76% in severe head injury table-2

Peck S D *et al* advocated that coagulative system abnormality could have a profound effect on patient's outcome as mentioned previously, more tissue damage affecting the extrinsic coagulatory cascades 10.13.

Prothrombin time estimation subsequently and daily in those patients who survived even with a deficit whether conservatively or surgically treated as far as 7-14 days after the initial rise showed a return of prothrombin time to its control basic level around the end of the first week.Fig-1, Fig2

In survivors prothrombin time returned to normal because of the limited process of damages with no secondary factors (ischemia,raised ICP, hypoxia) so prothrombin time will soon resettled toward normal

While in those who finally died again conservatively or surgically treated prothrombin time remained high or fluctuated Fig-1..Fig-2

In those who finally died the effect of primary severe damage of the secondary accumulative factor (edema, hypoperfusion,) will maintain a constant liberation of thromboplastin from the damaged brain cells leading to a consumption coagulopathy.

Therefore the curve of the dead group did not follow the pattern of the survivor group, these changes were statistically significant P<0.001.

## Conclusions

prothrombin time is simple highly valuable parameter in:

1-Predicting an ICerH and /or contusion in non scanned head injured patients

2-Estimating the extent and severity of brain damage

3-Has a significant prognostic value

## **Recommendation**

The introduction of prothrombin time estimation to all causality departments in Iraqi hospital can: 1- Select which head injured patient need CT scan specially when there is a limited access to the CT scan 2-Be a valuable tool in predicting the prognosis in head injured patients.

#### References

1-Astrup T Assay and content of tissue thromboplastin in different organs. Thromb Haemost 1989; 14:401-416 2-Pondaag W, Disseminated intravascular coagulation related to the outcome in head injury, Acta Neuro chir (wein) [supp]1996;28:98-102

3-Bick R L , Baker Wf. Diagnosis efficiency of the Ddimmer assay in disseminated intravascular coagulation (DIC).Thromb Res 1997; 65:785-790.

4-Tovi D ,Fibrinolytic activity of human brain a histochemical study Acte Neuro /Sand 2003 ;49:152-162

5-Richard Leblance,M,D.Augustin M.O.Gorman M.B.,B,Ch.FRCP© Neonatal intracranial haemorrhage J Neurosurgery8-Richard Leblance,M,D.Augustin M.O.Gorman M.B.,B,Ch .FRCP© Neonatal intracranial hemorrhage J Neurosurgery:642-651,Nov 1990.

6-Vander Sand JJ,Velkamp JJ Bockhout-Mussert RJ ,Vielvoye GJ Hemostasis and computerized tomography of the head injury, their relationship to clinical feature's Neurosurg 1981;55:718-724

7-Levali A,MariaL,Fariva,(eds)Prognosis of severe head injury J Neurosurgery 57:779-783.1982

8-Taesale,G Jennett,B:Assessment of coma and prognosiser head injury.Acute Neurochin (wein),34,45-55.1996

9-Taesale, G Murry,G,Parker,L et,al,:Adding up the GSC Score Acute Neurochin(wein),Suppl29)13-16,1996

10-Peck S D .Disseminated intravascular coagulation .How often have you missed it? Rocky Mt Med J 1997; 67:25-31

11-Biomerio company standardization

12-Young CW: CTand clinical criteria fir conservative treatment of suprasellar traumatic intracerebral hematomas Acte-Neurochir-(WEIN) 1995; 135(3-4):131-5.

13-Copper,P,R,:Post-Traumatic intracranial mass lesion .In Copper,P,R,ed:Head injury :3<sup>rd</sup>.Baltimore,Williamsand Wilkins,1993,pp.355-371

14-Miller, J, D,, Evaluation and treatment of head injury in adults, Neurosurgery, Q.12:28-43, 1992

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