

## DISSEMINATED INTRAVASCULAR COAGULATION AND CRANIAL TRAUMA.

Coşkun Yolaş M.D. (x)  
Arif Önder M.D. (xx)  
İsmail Hakkı Aydın M.D. (xxx)

### INTRODUCTION :

DIC proceeding cranial trauma seems to have attracted a lot of interest evidenced by a vast number of articles appearing in the literature (1,3,4,11-13,15, 19,21,31,32,35,36). From these reports it can be deduced that DIC in patients with cranial trauma is unexpectedly frequent (4,5,12,15,16,18,21,29,32,33). It is also found that DIC is the underlying mechanism in 4-8 % of the patients suffering from intracerebral hemorrhage associated with cranial trauma (23,31).

The brain tissue, especially the choroid plexus and the meninges are rich in tissue thromboplastin (6,7,22,26,30). When they are injured a vast amount of tissue thromboplastin is intraduced into the circulation which, in turn, activates the extrinsic coagulation system. The resultant disseminated intravascular thrombin give rise to ischemic lesions in various organs (2,7,11,19,34,36). Due to DIC coagulation factors such as platelets and serum fibrinogen decrease while prothrombin time time increas (7,9,10,13,17,20,25,27).

### MATERIALS AND METHODS:

This study is performed on 30 patients with cranial trauma admitted to the neurosurgery department of Atatürk University Medical Faculty. Patients are divided into two groups retrospectively Group-I: survivors (15 patients). Group-II: deceased eventually (15 patients).

Platelets counts, serum fibrinogen and prothrombin time determinations were performed daily for 7 days following the cranial trauma. Additionally EGT was performed once for each patient within the first 12 hours after the trauma(14).

---

(x) Numune Hastanesi Nöroşirurji uzmanı, Erzurum.

(xx) Atatürk Üniversitesi Tıp Fakültesi Nöroşirurji Kliniği, -Yrd. Doç. Dr.

(xxx) Atatürk Üniversitesi Tıp Fakültesi Nöroşirurji Kliniği, Doç

The test was recorded as either positive or negative. The normal fibrinogen, platelet count and prothrombin time were accepted as 200-400 mg per 100 ml, 150.000-300.000 cells per cu mm and 9.5-13.5 sec respectively.

The neurological findings detected in the initial examination were evaluated according to Glasgow coma score for each patient. The laboratory was not informed about the clinical status of the patients. Meanwhile all the necessary means of treatment were kept going on.

### RESULTS :

The age range was 6 to 70 years with a mean of 30.27 of the cases were male and 3 female. At the first examination the mean value of the Glasgow coma score was 15 for group I and 8 for group II. The initial clinical findings and means of treatment are shown in table-1

Table I : Level of consciousness associated lesions with head injury and types of management

		Group I	Group II
Degree of Consciousness	Coma-Precoma	4	12
	Semnlent	8	3
	Bright	3	0
ASSOCIATED LESIONS	Laceratio Capitis	5	7
	Basis Cranii Fracture	9	8
	Depression Fracture	6	6
	Epidural Hematoma	1	4
	Subdural Hematoma	1	3
	Subarachnoid hemorrhage	1	1
	Intracerebral hematoma	2	1
	Contusio Cerebri	7	4
	Laceratio Cerebri	3	5
	Other	6	4
Type of Treatment	Operation	7	11
	Conservative	8	4

In group I the platelet count on the first day was 174.300 cu mm. It decreased to 164 700 cu mm by the third day, after which began to increase gradually reaching 189500 cu mm on the seventh day (Fig. 1 and Table -2)

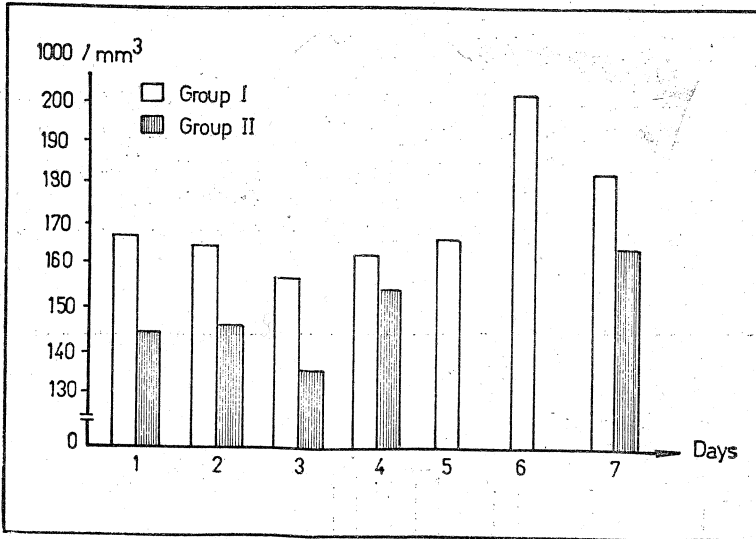


Fig. 1: Platelet counts per cu mm x 1000 from first to seventh day after head injury in 30 patients.

Table 2: The average platelet counts in the both groups.

Days	GROUP I			GROUP II		
	n	Mean (1000)	SD	n	Mean (1000)	SD
1	15	174.3	32.6	14	149.4	54.6
2	15	170.3	29.8	11	150.9	75.7
3	10	164.7	31.2	5	140.3	47.9
4	9	169.7	22.5	2	161.5	44.2
5	9	174.6	34.6			
6	6	210.0	60.3			
7	10	189.5	36.2	2	170.0	73.5

SD : Standart deviation

Similar changes were also observed in the second group but when a day by day comparison was made, the values in group II were found to be lower than the first group. In group, I, the differences in platelet counts between the first and third

days and between the third and seventh days were statistically insignificant ( $t: 0,60$  and  $P>0,05$ ;  $t: 1,52$  and  $P>0,05$  respectively). The same was also true for group II and the differences in the platelet counts between the first and third and between the third and seventh days were statistically insignificant ( $t: 0,46$  and  $P>0,05$ ;  $t: 0,15$  and  $P>0,05$  respectively).

The mean fibrinogen concentration in group I on the first day was 242,5 mg per 100 ml which increased to 289 mg per 100 ml on the third day and reached 295 mg per 100 ml on the fifth day, decreasing there after to 268 mg per 100 ml on the seventh day. In group II fibrinogen concentration was 216, 240, 250 and 260 mg per 100 ml on first, third, fourth and seventh days respectively (Fig. 2 and Table-3).

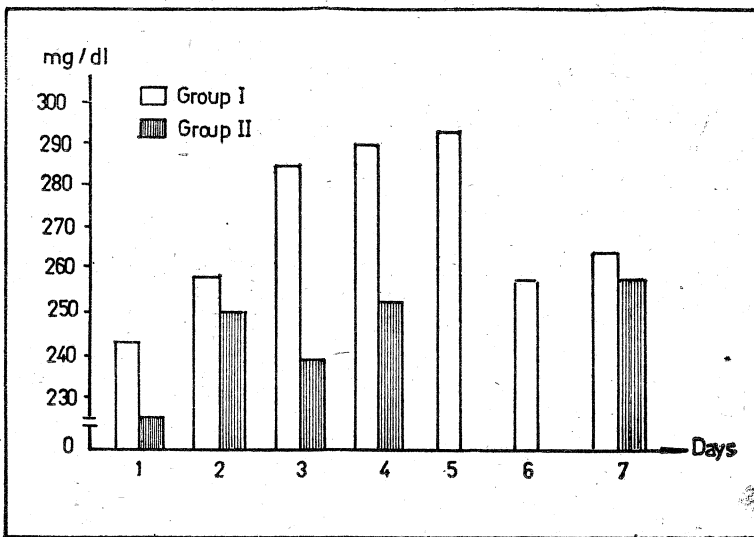


Fig. 2: Fibrinogen values in mg per 100 ml from first to seventh day after head injury in 30 patients.

In group the difference in fibrinogen concentration between days 1 and 4 was statistically significant ( $t: 3,14$  and  $P<0,01$ ). Where as the difference between fourth and seventh days was insignificant ( $t: 1,38$  and  $p>0,05$ ). In group II the differences between day 1 and 4 and between day 4 and 7 were insignificant ( $t: 0,64$  and  $P>0,05$ ;  $t: 0,14$  and  $P>0,05$  respectively).

When the mean fibrinogen concentration of each group were compared on the same day, the values of group II were lower than group I, but the differences were statistically insignificant. For the difference on day 1,  $t: 1,13$  and  $P>0,05$ ; on day 4,  $t: 0,086$  and  $P>0,05$  and on day 7,  $t: 0,13$  and  $P>0,05$ .

Table-3: The average fibrinogen values in the both groups.

Days	GROUP I			GROUP II		
	n	Mean	SD	n	Mean	SD
1	14	242.5	57.3	15	216	69.1
2	15	260.3	56.5	11	249.1	61.4
3	10	289	44.8	5	240	58.7
4	9	292.8	13.9	2	250	70.7
5	9	295	450			
6	6	258.3	49.2			
7	7	268	55.1	2	260	70.7

SD : Standart deviation

Prothrombin time reached to its shortest duration on the fourth day in both groups and tended to a slight prolongation towards the seventh day. When compared day by day prothrombin time was longer in group II than in group I (Fig.3 and Table-4).

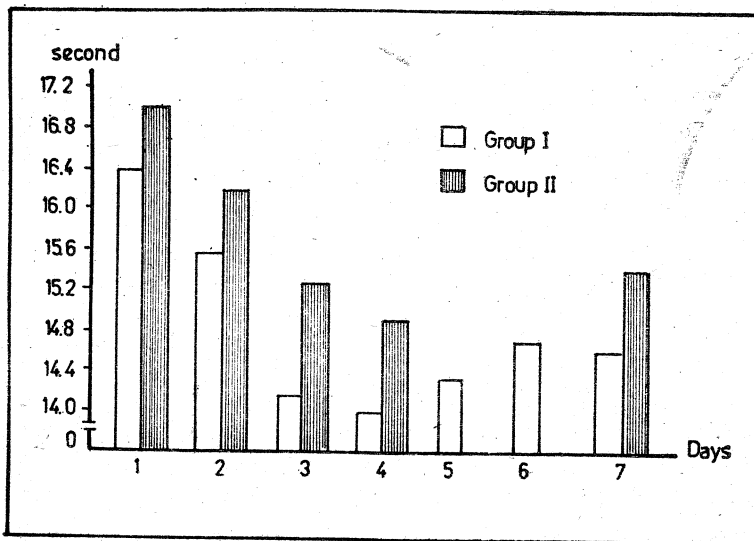


Fig. 3: The daily course of the average values of prothrombin time in both groups.

The shortening of prothrombin time was statistically significant only in group I and only between days 1 and 4 ( $t: 2,05$  and  $P > 0,05$ ). For group II the difference, either between days 1 and 4 or between days 4 and 7, was insignificant ( $t: 0,58$  and  $P > 0,05$  ;  $t: 0,28$  and  $P > 0,05$  respectively).

EGT was negative for all cases in group I, while three patients of group II had positive results. These patients were lost within two days following the head injury.

Table 4: The average values of the prothrombin timei in the both groups.

Days	GROUP I			GROUP II		
	n	Mean	SD	n	Mean	SD
1	15	16.4	5.25	13	17	5.72
2	15	15.7	2.13	11	16.3	3.52
3	10	14.2	0.42	5	15.2	16.40
4	9	14.0	0.00	2	15	1.40
5	9	14.4	0.73			
6	6	14.3	0.93			
7	10	14.7	1.06	2	15.5	2.12

SD : Standart deviation

## DISCUSSION

In 1972 Druskin demonstrated a case developing DIG after head injury (12). In 1973 Keimowitz and Armis reported another similar case. These authors emphasized that in spite of a vast number of articles about DIC accompanying wide tissue destruction, the literature was devoid of reports concerning DIC preceding cranial trauma (19). In 1974 Preston reported 3 cases and claimed that DIC syndrome should be carefully searched in cases with head injury (22). In a study of 13 patients with brain tissue injury, Goodnight et al. demonstrated evidences of defibrination in 4 cases and hypofibrinogenemia and thrombocytopenia in others. They concluded that this defect in hemostasis could be a factor enhancing the bulk of intracranial hematomas(15). In 1975 Vecht et al. studied 34 patients. In 8 patients who died within the first 24 hours prothrombin time was prolonged. EGT was negative in all 34 cases (35). In 1978, in a similar study covering 30 patients, Auer reported conspicuous changes in platelet counts and fibrinogen concentrations. He found that platelets decreased significantly right after the head injury and exhibited a gradual slight increase towards the seventh day. Fibrinogen concentrations were also found to be decreased initially which reached to a normal level around the fifth day. In conclusion, Auer noted a correlation between the brain lesions and the changes in platelet counts and fibrinogen concentrations(3). Van Der Sande, in a group of 150 cases, deduced a relation between the severity of the neurological deficits and laboratory results. 12 of his 150 patients exhibited unequivocal evidences of DIC, 10 cases had hypofibrinogene-

mia and another 12 were found to have a positive EGT. In addition, he emphasized that the laboratory findings were abnormal in all of the patients who eventually died (32).

In our present study, platelet counts and fibrinogen concentrations exhibited a decrease and prothrombin time was prolonged in both groups during the first days following cranial trauma. These findings which are very similar to those observed by Auer(3) and Van Der Sande (32) denote an unequivocal derangement in the normal coagulation system. We believe that the sharp decline in these parameters which is proceeded d by a gradual restoration is ar reflection of DIC which is also brought into action abruptly after the injury and resolves sloyly by the well knoun compensatroy mechanisims.

On the other hand, all of the parameters we studied denote a worse coagulation defect in group II. The patients in this group had suffered from more severe degrees of head injury and they eventually died. Thus we presume that the severe the cranial trauma the ominous will be the DIC and its results.

3 of our patients with a positive EGT were lost and it seems that the EGT positivity may be regarded as an unfavorable prognostic sign.

During the study, none of the individually examined patients exhibited any clinical findings which could raise the suspicion of DIC, in spite of the many laboratory evidences in favor of it.

We counclude that head injuries are frequently proceeded by DIC which is usually compensated and, thus, is subclinical and the severity of this coagulation derangement is directly proportional to the severity of the initial trauma.

#### *SUMMARY :*

The correlation between disseminated intravascular coagulation (DIC) proceeding cranial trauma and the severity of the trauma is eveluated, and an attempt is made to draw prognostic implications from the data obtained.

The study is undertaken on 30 patients, 15 of whom survived (group-I) and the remaining 15 died eventually (groupII). In each patient platelet counts, serum fibrinogen and prothorombin time determinations were made daily for 7 days. Additionally ethanol gelation test(EGT) was performed for each patient within the first 12 hours following the trauma.

Daily differences in the mean values of the laboratory data are compared within each group as well as between two groups. The platelet counts and serum fibrinogen decreased while prothrombin time was prolonged in almost all of the patients in the first days. These changes were more conspicuous in the second group.

It is concluded that DIC proceeding cranial trauma is directly proportional to the severity of the cerebral lesion.

## REFERENCES :

1. Allen JG, Glotzer ET: Acute disseminated intravascular coagulation and fibrinolysis. *Arch Surg* 88: 694-698, 1964.
2. Anderson JM, Braun JK: Brain ischemia and disseminated intravascular coagulation, *Lancet* 1: 373-374, 1972.
3. Auer L: Disturbances of the coagulatory system in patients with cerebral trauma. I. *Acta Neurochir* 43: 51-59, 1978.
4. Auer L, Ott E: Disturbances of the coagulatory system in patients with severe cerebral trauma II. Platelet function. *Acta Neurochir* 49: 219-226, 1979.
5. Becker DP, Miller JD Greenberg RP: Prognosis after head injury. In: Youmans JR (Ed). *Neurological Surgery Philadelphia, WB Saunders Co, 1982, pp 2137-2174.*
6. Bell, WN, Alton HG: A brain extract as a substitute for platelet suspensions in the thromboplastin generation test. *Nature* 174: 880-881, 1954.
7. Clark JA, Finelli RE, Netsky MG: Disseminated intravascular coagulation following cranial trauma: Case report. *J. Neurosurg* 52: 266-269, 1980.
8. Clauss A: Gerinnungsphysiologische Schnell methode zur Bestimmung des Fibrinogenes. *Acta Haemat (Basel)* 17: 237-246, 1957.
9. Colman RW, Robboy SJ, Minna JD: Disseminated intravascular coagulation: An approach. *Am J Med* 52: 679-689, 1972.
10. Deykin, D: The clinical challenge of disseminated intravascular coagulation *N Eng J Med* 283: 636-644, 1970.
11. Drayer BP, Poster CM: Disseminated intravascular coagulation and head trauma: Two cases studies. *JAMA* 231: 174-175, 1975.
12. Druskin MS, Drijansky R: Afibrinogenemia with severe head trauma. *JAMA* 219-755-756, 1972.
13. Gerrits WBJ, Prakke EM, Meer JVD, et al: Causes of a negative ethanol gelation test in diffuse intravascular coagulation. *Thrombus Diathes Haemorrh (Stuttg)* 31: 299-307, 1974.
14. Godal HC, Abildgaard U: Gelation of soluble fibrin in plasma by ethanol. *Scand J Haemat* 3: 342-350, 1966.
15. Goodnight SH, Kenoyer G, Rapoport SI, et: Defibrination after brain tissue destruction. *N Eng J Med* 290: 1043-1047, 1974.
16. Gudeman KS, Wheeler CB, Miller JD, et al: Gastric secretory and mucosal injury. Response to severe head trauma. *Neurosurgery* 12: 175-179, 1983.



17. Hamilton PJ, Stalker AL, Douglas AS: Disseminated intravascular coagulation A review . J Clin Pathol 31: 609-619, 1978.
18. Kaufman HH, Hui KS, Mattson JC, et al: Clinicopathological correlations of disseminated intravascular coagulation in patients with head injury. Neurosurgery, 15: 34-42. 1984.
19. Keimowitz RM, Armis BL: Disseminated intravascular coagulation associated with brain injury. J Neurosurg 39: 178-180, 1973.
20. Kruup MA, Chatton MJ: Current medical diagnosis and treatment. California, Lange Medical Publications 1977, pp 318-319,
21. Mc Gauley JL, Miller CA, Penner JA: Diagnosis and treatment of diffuse intravascular coagulation following cerebral trauma: Case report. J Neurosurg 43: 374-376, 1975.
2. Preston FE, Malia RG, Sworn MJ, et al: Disseminated intravascular coagulation as a consequence of cerebral damage. J Neurol Neurosurg Psychiatry 37: 241-248, 1974.
23. Pretorius ME, Kaufman HH: Rapid onset of delayed traumatic intravascular coagulation and fibrinolysis. Acta Neurochir 65: 103-109, 1982,
24. Quick AJ: Hemorrhagic Diseases and thrombosis, 2nd ed Lea and Febiger, Philadelphia, 1966, p 39
25. Robboy SJ, Mihm MC: The skin in disseminated intravascular coagulation Brit J Dermatol 88: 221-229, 1973,
26. Sawaya R, Donlon JA: Chronic disseminated intravascular coagulation in metastatic brain tumor: A case report and review of literature. Neurosurgery 12: 580-584, 1983.
27. Sharp AA: Diagnosis and management of disseminated intravascular coagulation. British Med Bul 33: 265-272, 1977.
28. Shurin S, Recate H: Disseminated intravascular coagulation as a complication of ventricular catheter placement. J Neurosurg 54: 264-267, 1981.
29. Spallone A, Mariani G, Rosa G, et al: Disseminated intravascular coagulation as a complication of ruptured intracranial aneurysms: Report of two cases. J Neurosurg 59: 142-145, 1983.
30. Takashima S, Koga M, Tanaka K: Fibrinolytic activity of human brain and cerebrospinal fluid. Br J Exp Pathol 50: 533-539, 1969
31. Van Der Sande JJ, Emeis JJ, Lindeman J: Intravascular coagulation a common phenomenon in minor experimental head injury. J Neurosurg 54: 21-25, 1981

32. Van Der Sande JJ, Veltkamp JJ, Boekhout-Musedt RT, et al: Head injury and coagulation disorders. *J Neurosurg* 49: 357-365, 1978
33. Van Der Sande JJ, Veltkamp JJ, Bouwhuis-Hooger Werf ML: Hemostasis and intracraial surgery. *J Neurosurg* 58/93-698, 1983.
34. Vecht CJ, Smit Sibinga CT: Head injury and defibrination. *Lancet* 2: 905,1974
35. Vecht CJ, Smit Sibinga CT, Minderhoud JM: Disseminated intravascular coagulation and head injury. *J Neurol Neurosurg Psychiatry* 38: 567-571,1975.
36. Winston K, Conner S: Successful of subdural hematoma in the precence of severe coagulopathy. *Neurosurgery* 11: 277-279, 1982.