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DISSEMINATED INTRAVASCULAR COAGULATION IN BLUNT TRAUMA TO THE CHEST

A Clinical Study +

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Summary: Blunt trauma to the chest is most often the results of an automobile collision or accident but may be produced by a fall or crush injury. The result of blunt chest injury in the acute phase is disrupted tissue perfusion with attendant hypoxia and metabolic acidosis. Trauma causes hemolysis and the red cell stroma may initiate disseminated intravascular coaglutain (DIC). The causes of DIC are hypoventilation, hypoxemia, tissue factor releasing from pulmonary contusion or laceration, hemorrhage and shock. In this study we investigated the tendency to DIC in 25 cases with blunt thoracic trauma. Coagulation test (i.e., platelet count, prothrombin time, thrombin time, partial thromboplastin time, Factor I, fibrin split product, and Factor IV) were studied in two groups. Group I comprised the patients with blunt thoracic trauma, and Group II comprised the control group. The differences of coagulation test between the groups were found statistically significant. DIC was observed clinically in one case. Three tests or more were abnormal in 19 patients, a condition consistend with DIC. Mortality rate was 16 per cent.

Key words: Disseminated intravascular coagulation, chest injury.

Trauma causes more loss of life than any other cause. In the Western World trauma ranks only behind cardiovascular disease and cancer as a casue of death. E.g., in the USA it is the leading cause of death in people under the age of 37years and claims over fifty thousand victims per year. Thoracic traumas are the sole cause of death in 25 per cent of the victims and are a contributory cause in another 50 per cent (25) Traumatic shock and sepsis that cause multiple organ failure are the most common late causes of death following trauma. The most common organ to fail is the lung which develops Adult Respiratory Distress Syndrome (ARDS). ARDS can be caused by DIC with microscopic clots in the lungs (8). An immediate pathologic effect of thoracic injury as presented in Figure 1, is disrupted tissue perfusion with attendant hypoxia and metabolic acidosis (10). Consequently, the developent of DIC is activated. DIC is one of the reason which causes the rise in mortality of the traumatized patients (12). Therefore, while managing the thoracic traumatized patients, DIC must be kept in mind, and the management must be carried out accordingly.

MATERIAL AND METHODS

Our study was carried out on twenty-five pati-

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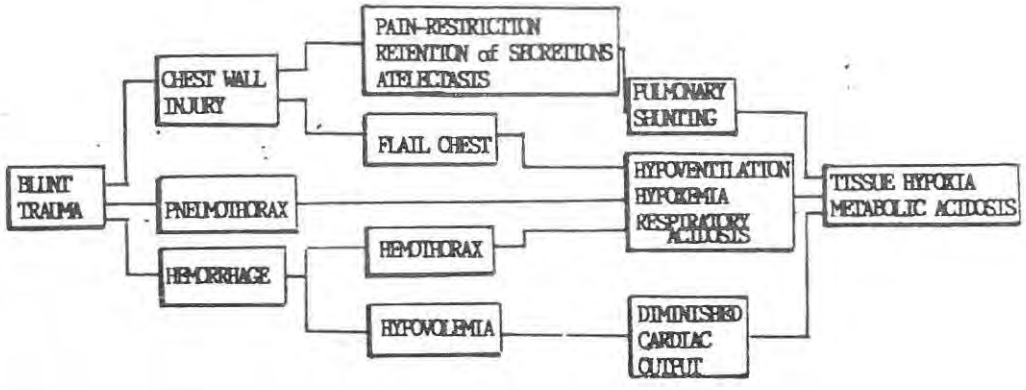

Figure 1. Schematic diagram of blunt thoracic injuries showing how disturbed cardiopulmonary physiologic equilibrium results in tissue acidosis (modified diagram : Hood RM: Trauma to the chest. In Sabiston DC, Jr., Spencer FC (eds), Surgery of the Chest, WB Saunders Co., Philadelphia, 1990, pp. 383-417).

ents with blunt thoracic injury (i.e., Group I). There were two females (8 %) and 23 males (92 %). The average of the patients ages was 47.4 (ranging 7 to 71). The following tests were made for diagnosis of DIC: bleeding time (by Ivy's method), platelet count, prothrombin time (by Quick's method), thrombin time, partial thromboplastin time, Factor I, fibrin split product (FSP), Factor IV (Clark& Collin's method), coagulation time (by Lee-White's method), ethanol gelation test (EGT), hemoglobin, WBC count. These tests were done in the first six hours following admission. The control group (i.e., Group II) was selected from the nontramatized patients who had no illness causing DIC. The chi-square test dues t test was used for statistical analysis.

RESULTS

The difference of bleeding time, platelet count, prothrombin time, thrombin time, partial thromboplastin time, fibrinogen, and FSP between the groups were statistically significant. But EGT had not shown any statistical difference between the groups. Table I outlines all these data.

In one case, DIC was observed clinically. Three or more laboratory tests were abnormal in 19 patients (% 76). In six cases (% 24), one to three of tests were abnormal. Three patients of 19 died. In the patient with DIC, prothrombin time, thrombin time and platelet count were the most abnormal coaglutaion tests (Table II).

DISCUSSION

Trauma, that can be called the "neglected disease" of modern society, is the principal cause of death in Americans between ages 1 and 44 and the fourth leading cause of death for all age gropus. Most thracic injuries are from blunt or penetrating trauma. Eighty per cent of blunt traumatic inujuries are caused by automobile accident (24). Tissue injury or necrosis causes the release of a significant amount of thromboplastin into the blood, and starts the coagulation mechanism. There are

Table I. Comparison between patient (I) and control (II) groups.

Tests	Groups	n	X± Sx	SD	t	p
Bleeding time (min.)	I	25	6.36 ± 1.27	1.06	3.12	< 0.05
	II	20	3.05 ± 6.82			
Platelet count (per cubic milimeter)	I	25	94.68 ± 41.77	33.52	4.52	< 0.05
	II	20	246.30 ± 18.15			
Prothrombin time (sec.)	I	25	24.80 ± 1.92	2.00	2.02	< 0.05
	II	20	20.15 ± 5.77			
Thrombin time (sec.)	I	25	31.40 ± 1.18	1.51	13.31	< 0.05
	II	20	11.30 ± 0.33			
Fibrinogen (mg/dL)	I	25	313.90 ± 32.61	19.55	4.07	< 0.05
	II	20	393.55 ± 36.10			
Fibrin split product (> 1:8 dilution, positive)	I	25	21.40 ± 5.12	5.40	3.68	< 0.05
	II	20	1.50 ± 1.71			
Coagulation time (min)	I	25	9.34 ± 9.75	10.21	3.85	< 0.05
	II	20	5.40 ± 5.64			
Factor IV (mg/dL)	I	25	8.90 ± 0.28	0.12	7.09	< 0.05
	II	20	8.50 ± 2.43			
Ethanol gelation test	I	25	positive = 7	negative = 18		> 0.05
EGT, pos./neg.	II	20	positive = 0	negative = 20		> 0.05

four times more thromboplastin in the lung tissue than in the brain tissue (4, 6). The defects of coagulation are frequently found in serious traumatized patients (1). Intrinsic and/or extrinsic coaglutaion factors make the process begin. Abrnormal intravascular coagulation is occured by the simultaneous usage of coaglutaion factors. The process consists of consumption of coaglutaion factors, intravascular lysis of clot, and eventually hemostatic disorder and spontaneous hemorrhage. DIC can be detected by laboratory method, even if there is no clinical finding (2).

DIC is a syndrome of thrombocytopenia, multiple coaglation deficiencies, and secondary fibrinolysis resulting from activation of coagulation systems and platelet aggregation within

Table II. The rates of abnormality of tests on the patients with DIC.

Tests	n	%
Bleeding time	-	-
Platelet count	19	76
Protrombin time	18	72
Thrombin time	25	100
Partial tromboblastin time	-	-
Factor I (fibrinogen)	7	28
Fibrin split product	11	44
Coagulation time	7	28
Factor IV	8	32
Ethanol gelation test (EGT)	7	28

the vascular system. Figure 2 shows pathophysiological mechanism in DIC. Its cause are: (a) hypoxia with endothelial damage, shock, cardiac arrest, septicemia, uremia, heat stroke; (b) multiple trauma, fat emboli, crush injuries, tissue necrosis, burns; (c) intravascular hemolysis, extracorporeal circulation, incompatible blood transfusion, acquired hemolytic anemia, sickle cell anemia, paroxysmal nocturnal hemoglobinemia; (d) metastatic carcinoma; (e) obstetrical problems (abruptio placentae, amniotic fluid embolus, retained death fetus); (f) giant hemangioma; (g) antigen-antibody reaction (11). DIC is a pathological activation of coagulation mechanism and an interval reaction associates the primary diseases (3). Some studies were done in order to show that DIC, the first observations about DIC were made at the beginning of last century on obstetrical cases, was

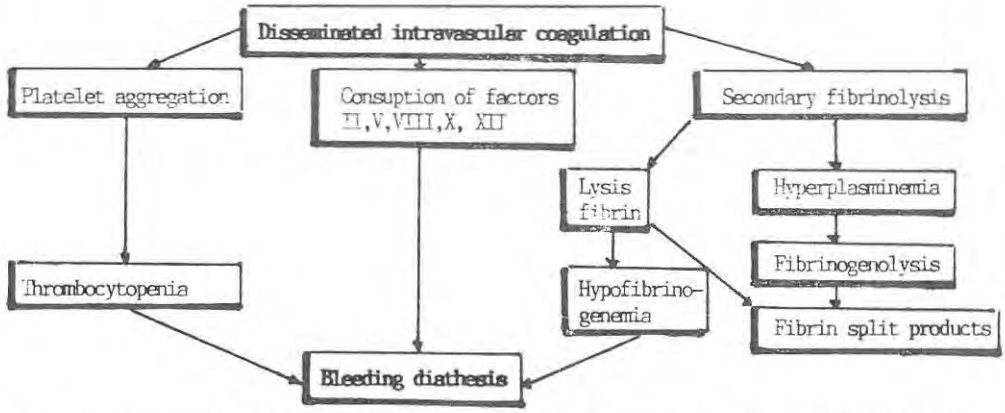

Figure 2. Pathophysiological mechanism in DIC (from: Hussey C and Wilson SD: Acute coagulation disorders. In: Condon RE, Nyhus IM (eds). Manual of Surgical therapeutics. Little, Brown and Company, Boston, pp. 341-351).

associated with other diseases. In a patient, bleeding around catheter after the uretral dilation was observed but foci of bleeding during cystostomic scopy was not seen, with benign prostatic hyperplasia, some coagluation abnormalities such as lenghtening of prothrombin and thrombin times, thrombocytopenia and decreasing of factors II, V, VIII, XII supporting DIC were found (21).

In the other study about children with bacterial menengitis, DIC was found in the rate of five per cent. In all these cases, prothrombin time and partial thromboplastin time have prolonged, factor II, V, VIII decreased and FSP's increased (23).

Arterial aneurysm may also be associated with DIC (16, 20). In these patients with aneurysm, prothrombin time and partial thromboplastin time had prologed, and fibrinogen and platelet count had decreased. Immediately after the surgical management of aneurysm together with preoperative heparine treatment, all abnormal coagulation tests became normalized. Here the provoking factor of DIC was thrombi within aneurysm. The thrombin time was one of the most valuable tests of DIC (14).

Clark and his colleagues (4) observed DIC in cranial and spinal traumatized patients. They found the following outcomes: decreased platelet count, prolonged prothrombin time, and brain tissue within lateral and sagittal sinus in necropsies. They concluded that the reason for prolonged prothrombin time was the release of thromboplastin to the circulation from traumatized brain and probably from traumatized lung tissue, and the activation of extrinsic coagulation system. In the study of 87 children with cranial trauma, one or more coagulation test were abnormal in % 71 of the cases in the first two hours following trauma. Mortality rate was four times more in the cases with DIC compared to patients without DIC (15).

Hardaway (7, 8, 9) claimed that shock associated with DIC in critical patients frequently, and shock itself, had started DIC and also continued with it. Death from traumatic shock has been associated with loss of blood internally or externally. However, even though blood restoration is adequate, many patients die after trauma. Death is often due to ARDS. Death and ARDS have been associated with DIC and microclots in the lungs.

Colman et al (5) described that dyspnea, hemoptysis, rales, and diffuse infiltration in chest film resulted in interrupted pulmonary circulation as a preterminal complication of DIC. Disseminated intravascular coagulopathy is the reasons for microcirculatory occlusion.

In a retrospective study (22), the frequency of DIC in all hospitalized patients was 1/1000. Approximately one fifth of DIC cases was silent clinically but these cases were found with laboratory findings.

Hypoxia, hypovolemia, hemorrhage, shock resulting from thoracic trauma and tissue factor releasing from contusion or laceration of lung tissue are among the reansons for DIC. The clinical feature of DIC varies according to primary disease and the degree of coaglutaion disorder. However, anemia, hypotension, oliguria and hemorrhage are seen in a majority of cases with DIC was clinically evident in one of our cases.

There was a significant difference of FSP between the gropus at it is seen on Table I. This was quite similar to the other authors' criteria (11, 13, 15, 17, 19). Again in the same table, there was some differences between the groups in the point of bleeding time, prothrombin time, platelet count, thrombin ti-

me, and these difference were very close to the criteria of DIC in the other authors' studies (4. 6, 11, 15, 17, 19). EGT did not show any difference between the groups (Table I). This was like the other studies (6).

DIC rates varies between 56 to 70 per cent in literature (18). This rate is % 76 in our series. Our mortality was 16 per cent. This rate is 25-67 % in literature (6).

Consequently, there is a tendency towards DIC in thoracic traumatized patients. Therefore, the test concerning DIC must be done during the treatment of these patients, and as soon as the diagnosis is proved, the treatment must be started with heparine.

References

1. Adams HD: Thoracic injuries. ***Surg Clin North Am*** *41: 619-622, 1961.*

2. Bowie BM, Charles AO Jr., Waiter EJ: Surgical hemostasis. ***J Neurosurg*** *51: 137-196, 1979.*

3. Bozer AY, İliçin G: Şok. ***Hacettepe Üniversitesi Yayınları****, Ankara 1980, ss. 10-36.*

4. Clark JA, Finelli RE, Netsky MG: Disseminated intravascular coagulation and head injury. ***Neurol Neurosurg Psychiatri*** *38: 567-571, 1980.*

5. Colman RW, Robboy SJ, Minna JD: Disseminated intravascular coagulation. ***An Rev Med*** *30: 359-364, 1979.*

6. Hamilton PJ, Stalher AL, Douglas AS: Disseminated intravascular coagulation. ***J. Clin Pathol*** *31: 609-619, 1978.*

7. Hardaway RM: Disseminated intravascular coagulation in experimental and clinical shock. ***Am J Cardiol*** *20: 161-173, 1967.*

8. Hardaway RM, Williams CH: A new treatment for traumatic shock and ARDS. ***Resuscitation*** *19: 61-76, 1990.*

9. Hardaway RM, Williams CH, Marvasti M: Prevention of adult respiratory distress syndrome with plasminogen activator in pigs. ***Crit Care Med*** *18: 1413-1418, 1990.*

10. Hood RM: Trauma to the chest. In Sabiston DC Jr, Spencer FC (eds), Surgery of the Chest, WB Saunders Co., ***Philadelphia****, 1990, pp. 383-417.*

11. Hussey C, Wilson SD: Acute coagluation disorders. ***In Condon RE,Nyhus LM (eds), Manual of Surgical Therapeutics, Little, Brown and Company****, Boston, 1978, pp. 341-351.*

12. Kaspar CK: Thromboembolic complications of Factor IX concentrates. ***Haemorrhagea*** *33: 640-644, 1975.*

13. Mant JM, King GE: Severe acute disseminated intravascular coagulation. ***Am J Med*** *67: 557-562, 1979.*

14. Merskey C, Johnson AJ, Kleinder CJ et al: The defibrination syndrome: Clinical features and laboratory diagnosis. ***Brit J Haemat*** *13: 528-549, 1967.*

15. Miner ME, Kaufmann HH: Disseminated intravascular coagulation and fibrinolytic syndrome following head injury in children: Frequency and prognostic implications. ***J Pediatr*** *41: 687-691, 1982.*

16. Nyui S, Inoue S,Sato T, et al: Repair of aortoiliac aneurysm associated with chronic disseminated intravascular coagulation-a case report. ***Jpn J Surg*** *21: 110-113, 1991.*

17. Pondaag W: Disseminated intravascular coagulation in head injured patients. ***Advances in Neurosurgery 6, Springer-Verlag****, Heidelber, 1978, pp. 159-162.*

18. Saldeen T: The microembolism syndrome. ***Microvascular Research*** *11: 277-259, 1979.*

19. Sande JJ, Welthamp JJ, Welthamp JJ, Boechout-Mussert RJ et al: Head injury and coagulation disorders ***J Neurosurg*** *49: 357-265, 1978.*

20. Satiani RM, Janin J, Ewans EW: Consumption coagulopathy associated with arterial aneurysm ***J Cardiovasc Surg*** *20: 273-279, 1979.*

21. Sherman LA: Therapeutic problems of disseminated intravascular coagulation. ***Arch Intern Med*** *132: 446-453, 1973.*

22. Sigeal T, Seligsch U, Aghai E, et al: Clinical and laboratory aspectes of disseminated intravascular coagulation. ***Throm Haemost*** 39: 122-129, 1978.

23. Tanyeri G, Tanyeri K: Akut promyelosit lösemi ve yaygın damariçi pıhtılaşması. Akut bakteriyel menenjitli çocuklarda yaygın damariçi pıhtılaşması. ***Türk Hematoloji Cemiyet VIII. Kongresi İzmir****, 26-28 Nisan 1973.*

24. Thomas AN: Management of injured patient: Thoracic injuries. ***In WayLV(ed), Current Surgical Diagnosis & Treatment****, Libraire du Liban, Beirut, 1983, pp. 210-232.*

25. Trinkle JK: Management of blunt thoracic injuries. ***In Jackson JW, Copper DKC (eds), Rob & Smith's Operative Surgery - Throacic Surgery,*** *Butterworths, London, 1986, pp. 22-38.*