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Character of Laboratory Data in Infection Induced DIC

Yoshinobu TAKEMOTO, Shigeru TANAKA, Yoshihiro NAKAMURA, Jun TANABE, Kunichi KIDO, Chiiho FUJII and Akitsugu KOHAMA

Department of Emergency Medicine, Kawasaki Medical School Kurashiki 701-01, Japan

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ABSTRACT. 1. A comparative study of laboratory data on DIC due to infections and non-infections was performed. It showed normal levels of fibrinogen and elevated FDP levels to be prominent in infection-induced DIC and a better recovery from infection-induced DIC than from non-infection induced DIC.

2. DIC due to sepsis showed the characteristic features of laboratory data with low levels of α_2-PI and AT-III at the time of diagnosis.

Key words : Disseminated intravascular coagulation — Sepsis — Antithrombin-III — α_2-Plasmin inhibitor

Experience in our emergency department and others has led to the conclusion that the development of disseminated intravascular coagulation (DIC) at the early stage of various disorders should be strictly monitored that primary therapy for DIC should be begun as soon as possible. In emergency departments, decompensated DIC at the time of diagnosis has been considered to result in a poor prognosis. In our previous report, it was indicated that infections are the major cause of DIC.[1] DIC, however, may be caused by non-infections. The purpose of the present study was to compare the differences in DIC in two groups of patients : those with DIC due to infections and those with DIC arising from non-infections.

METHODS

Of 1563 cases admitted to our clinic, 50 patients (3.1 percent) ranging in age from nine to eighty-eight years old have developed DIC. Thirty-six of these cases were male, and fourteen were female. Laboratory data examined were platelet count, prothrombin time (PT), activated partial thromboplastin time (aPTT), fibrinogen, serum fibrin degradation product (FDP), antithrombin-III (AT-III) and α_2-plasmin inhibitor (α_2-PI). DIC was diagnosed in the same manner as in our previous study.[1]

RESULTS

1. Causative disorders of DIC (Table 1)

Twenty-five cases developed DIC following infectious disorders. Of these,

武元良整，田中　茂，中村義博，田辺　潤，木戸訓一，藤井千穂，小濱啓次

15 cases were diagnosed to be due to sepsis because of a positive blood culture, while 10 cases were diagnosed as DIC due to infections without sepsis.

TABLE 1. Causative disorders of 50 cases with DIC.

Infections		25
sepsis	15	
pneumonia	4	
hepato-biliary tract infection	2	
panperitonitis	2	
meningitis	1	
pyelonephritis	1	
Hemorrhagic shock		9
Burns		5
Multiple fracture		3
Head trauma		2
Respiratory arrest		1
Cardiac arrest		1
Dissecting aneurysma		1
Unknown		3

2. Character of laboratory data in DIC due to infections

Table 2, consisting of 25 DIC cases caused by infections, summarizes the abnormal data at the time of diagnosis for DIC. The mortality rate and the ratio of recovery from DIC are also shown. A high incidence of thrombocytopenia (below $12 \times 10^4/\mu l$) and elevated FDP levels were observed in infections without sepsis (10 cases), whereas there was a decrease in α_2-PI (<4.32 mg/dl), the platelet count and AT-III (<18 mg/dl) in septic patients. The ratio of improvement from DIC was 70 percent in cases with sepsis and 73 percent in those without sepsis.

TABLE 2. 25 cases of DIC caused by infectious disorders.

Tests	Values	Sepsis (15)	Infections (10)
		percent of cases (%)	
α_2 -PI	<4.32 mg/dl	83	40
Platelet count	$<12 \times 10^4/\mu l$	78	100
AT-Ⅲ	<18 mg/ dl	75	50
FDP	≧10 μg/ml	67	90
Protamine sulfate test positive		67	60
PT	≧15 sec	29	20
fibrinogen	<200 mg/dl	26	22
aPTT	≧50 sec	14	10
mortality (%)		60	40
improved cases of DIC (%)		70	73

3. Comparison of laboratory data for infection and non-infection induced DIC

Differences in laboratory data in DIC caused by infection and non-infection are compared in Table 3. Both forms of DIC showed a high incidence of thrombocytopenia, 87 percent for both. There was a tendency toward a high FDP in infectious diseases, on the other hand, decreased α_2-PI was frequently seen

in non-infections. There were no differences in mortality rate. The rate of recovery was 72 percent for DIC caused by infections and 56 percent for non-infection induced DIC.

TABLE 3. Comparison between infections and non-infections (DIC 50 cases).

Tests	Values	Infection (25)	Non-infections (25)	Total (50)
		percent of cases		
Platelet count	$<12\times10^4/\mu l$	87	87	87
α_2 -PI	<4.32 mg/dl	63	85	76
Protamine sulfate test	positive	63	72	68
AT-III	<18 mg/dl	62	71	67
FDP	≧10 μg/ml	77	45	59
fibrinogen	<200 mg/dl	25	41	33
PT	≧15 sec	20	28	24
aPTT	≧50 sec	13	24	18
mortality (%)		52	60	56
improved cases of DIC (%)		72	56	68

DISCUSSION

1. Laboratory data and underlying disorders of DIC

DIC is frequently associated with patients of sepsis complicated by shock, although, infections without shock are also thought to develop in DIC.[1] The mechanism of DIC has been considered to be the result of damage to platelet and vascular endothelial cells by endotoxin and of a hypercoagulable state caused by a reaction with granulocytes.[2] It has been suggested that a progressive and rapid decrease of AT-III and α_2-PI might be seen in sepsis more than in the other infections. Such a decrease may be observed in Table 2.

Hypofibrinogenemia is a well-known complication in typical DIC. The incidence of hypofibrinogenemia among our 25 cases of infection-induced DIC was 26 percent in sepsis and 22 percent in other infections. These findings suggest that it is necessary to gain a better understanding of the causative diseases of DIC. Thus, it should be emphasized that coagulation factors increase with compensatory production of fibrinogen in inflammatory conditions at the acute stage.

2. Infections and non-infections (Table 3)

There is a tendency towards elevated FDP and normal levels of fibrinogen in infections. This implies that the DIC induced by infections is compensated DIC, since inflammation promotes the hypercoagulable state by increasing fibrinogen and produces the increment of fibrin and fibrinogen degradation product at the inflammatory sites.[3]

Not only hypofibrinogenemia (41%) but also low levels of α_2-PI (85%) are apt to appear with non-infections. These results are indicative of consumption coagulopathy or increased secondary fibrinolysis. Therefore, it seems possible to conclude that the majority of cases of advanced decompensated DIC are cases of DIC due to non-infections.

It was observed that most cases of DIC due to non-infections had a lower ratio of improvement (56%) than those of DIC due to infections (72%). This lower ratio might be responsible for decompensated DIC. By contrast, DIC

due to infections improved satisfactory if diagnosis was made early at the acute stage and therapy could be started as soon as possible.[4)]

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