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EXPERIMENTAL DIC DEVELOPED IN DOGS WITH PULMONARY AND RENAL INVOLVEMENT INDUCED BY OLEIC ACID AND ANTI-LUNG ANTIBODY.
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In order to have pathogenetic comprehension on the causal relationship between shock state and development of DIC, the following experiments were carried out and compared with the clinical and laboratory findings in human cases with pulmonary and/or renal diseases. An ordinary shock state was invited in dogs by means of exsanguination, depressing mean blood pressure down to 70mmHg and sustaining it for one hour, but almost no changes were realized in coagulation and fibrinolysis parameters including platelet count and functions of blood as well as the heart rate and cardiac index of animals.

The additional injection of a rather small amount of thromboplastin, however, caused typical hematological changes of DIC and demonstrable disturbance of pulmonary-cardiac (p-c) functions which were restored spontaneously.

Repeated injections of oleic acid introduced significant histological changes in the lung and kidney of dogs, but only slight shifts were proved in hematological findings and p-c functions and not so much modification of them was noticed even under the exsanguination shock state. The additional injection of thromboplastin, however, showed exaggerated histological and functional changes.

The long term injections of anti-lung antibody to dogs introduced a certain kind of histological changes, similar to those of so-called "Goodpasture's syndrome" in the lung and kidney as well as blood findings like DIC and p-c disturbance, and the simultaneous manipulation of exsanguination and thromboplastin injection promoted those changes more gravely.

The same kind of exaggerated reactions in blood findings and p-c functions was encountered in clinical cases with lung and/or kidney disorders and suggested that these damages might play much role in the development of DIC.

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COAGULANT AND PROCOAGULANT FACTORS IN ASCITIC FLUID.- ABOUT ETIOLOGY OF DIC INDUCED BY ASCITIC FLUID INFUSION -
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We have experienced two cases of DIC following infusion of ascitic fluid from the peritoneal cavity to the vascular system. We have studied the etiology of this DIC. So, FDP, endotoxin, coagulant factors and procoagulant activity were investigated in ascitic fluid of 11 hepatic cirrhosis cases and 15 cancer cases.

Method and Result; FDP in ascites were more included than in plasma. Endotoxin were positive in about 60% of ascitic fluid. The coagulant factors were recognized a littile except VIII-factor. Only ascitic fluid ded not clott the fibrinogen and did not affect the platlate aggregation. The procoagulant activities were measured by clotting times which the normal plasma (o.1cc) was added with the ascitic fluid or buffer (o.1cc), after 3 minutes incubation, and then added with $^{1}/_{40}$ M $CaCl_2$ (o.1cc).

The clotting time was shortened in the ascitic fluid than buffer (buffer 120.7 ± 7.9 sec, Cancer 82 ± 23.8 sec., cirrhosis 91.4 ± 16.5 sec), and both VII and VIII deficient plasma was shortened too, but X dificient plasma was not coagulated. Also FDP and endotoxin did not shorten the clotting time of normal plasma. Experimentally, the ascitic fluid in dog by binding vena cava in ferior and the ascitic fluid in rat by transplantation of tumor cells shortened the clotting time. Conclusion; Coagulant, fibrinolytic and procoagulant factors were existed in ascitic fluid. We think that DIC induced by ascitic fluid are due to this procoagulant factor and this procoagulant factor may be not tissue - thromboplastin only.

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SPONTANEOUS LIVER RUPTURE IN PREGNANCY ASSOCIATED WITH SEVERE DIC: MANAGEMENT OF THE COAGULATION DISORDER PRIOR TO SURGERY.
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Spontaneous rupture of the liver is a severe complication of pregnancy (maternal mortality: 59%, fetal mortality: 77%). The etiology is unknown, but it is commonly agreed that pre-ecclamptic toxaemia, and vascular damage of the liver play a definite role. Severe coagulation abnormalities which have been previously observed in these patients have as yet not been studied extensively. Recently we investigated two patients with spontaneous liver rupture during pregnancy. Case 1: 34 year old multipara was presented with abdominal pain in the 37th wk of gestation. Suddenly a state of shock was observed. Surgery revealed hemoperitoneum , subcapsular haematoma of the liver and ruptured capsule. The patient survived. Case 2: 30 year old primipara 37 wk of gestation was admitted with severe abdominal pain, jaundice and shock. Autopsy revealed a large subcapsular haematoma with ruptured capsule. Coagulation studies at admission revealed extensive DIC.

Case	plts	AT III	FV	FII	EGT	FDP
1	61	0.54	0.29	0.38	+	2
2	9	0.28	0.25	0.34	+	6
norm:	150-350x10^9/l>	0.80u	0.80u	0.70u-		<1 mg%

Bleeding and DIC were succesfully controlled by the following transfusion regimen: 1) purified human AT III concentrates 2) prothrombin complex 3) cryoprecipitate 4) low dose heparin 5) platelets 6) Fresh Frozen Plasma 7) packed cells. Conclusively, maternal mortality might be reduced by early recognition of symptoms followed by prompt surgical intervention after correction of shock and coagulation defects.

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DISSEMINATED INTRAVASCULAR COAGULATION IN PATIENTS WITH FATTY LIVER OF PREGNANCY. ONSET OF DIC PRIOR TO LABOR.
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Fatty liver of pregnancy (FLP) is a rare syndrome with a high maternal mortality. Bleeding complications are frequent and are associated with disseminated intravascular coagulation (DIC). We have previously demonstrated that DIC in this syndrome can be persistent, lasting 2 to 6 days, and is associated with severe depressions of functional and antigenic antithrombin III (AT III). The severe depression of AT III in this syndrome is believed to secondary to decreased hepatic synthesis and increased consumption from DIC. We have hypothesized that DIC is initiated by active labor, persists because of the low levels of AT III and may be controlled by transfusion of fresh frozen plasma or AT III concentrate. Recently we saw a patient with FLP proven by biopsy who presented with jaundice, nausea, and emesis. The patient was not in active labor. In preparation for surgical delivery, coagulation studies were done which revealed DIC (abnormal screening tests, fibrinogen of 70mg/dl, fibrin split products of 320-640 ng/dl, depressed factors V & VIII and a positive protamine sulfate test). The patient was given 1500 cc of fresh frozen plasma and viable twins were surgically delivered on the second hospital day. Following delivery DIC subsided, but infusions of plasma were continued until the fourth hospital day. Subsequently we measured AT III using a fluorometric synthetic substrate (Protopath-see table above). From our observations on this patient we conclude (1) active labor is not necessary for the initiation of DIC in FLP (2) DIC in FLP can be associated with non-detectable functional AT III (3) transfusions of fresh frozen plasma can significantly increase functional AT III and may control DIC.

Hospital day	1	2	3	4	5	6
Protopath	0	30	42	35	21	26%
Protamine	+	-	-	-	-	-

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