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COMPLEMENT PROFILES IN DISSEMINATED INTRAVASCULAR COAGULATION. Y. Furukawa, T. Yoshikawa, M. Murakami, S. Takemura, and M. Kondo. The 1st Department of Medicine, Kyoto Prefectural University of Medicine, Kyoto, Japan.

Complement levels were examined in cases of disseminated intravascular coagulation (DIC), as well as in experimental animal models of DIC, and it was found that complement played a part either in the development or in the early diagnosis of DIC.

Thirteen patients with DIC; 4 associated with bacterial infection, 1 chronic myelogenous leukemia and 8 liver diseases, were serially investigated for their serum complement levels. By the onset of DIC, marked decrease of CH50, C4 and C3 levels were observed in all cases. Those who responded well to anticoagulant therapy revealed a gradual recovery of complement levels, while who failed kept their low serum levels, suggesting that the estimation of complement levels in patients with DIC might be a valuable parameter for their prognosis.

Experimental model of DIC was produced in rats by the continuous intravenous infusion of bacterial endotoxin for 4 hours. Histological examination and several coagulation parameters such as fibrinogen, platelet counts, prothrombin time, PTT and FDP confirmed the presence of DIC. The complement levels, CH50 and C3, were markedly decreased at the onset of DIC and then gradually increased after stopping the infusion of endotoxin. By the pretreatment of rats with heparin, this decrease of serum complement levels by endotoxin was completely prevented.

It is concluded that, although many factors have been known to be involved in the production of DIC, complement might also be an important factor, one by contributing to the coagulation process and the other by being affected by secondary fibrinolysis.

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DISSEMINATED INTRAVASCULAR COAGULATION COMPLICATING THE PERINATAL "DROPPED-DEAD" SYNDROME: LACK OF INFLUENCE OF CARDIO-PULMONARY RESUSCITATION. A. Peliowski, C. Rand, R. Barr and A. Zipursky, Department of Pediatrics, McMaster University, Hamilton, Ontario.

A diagnosis of perinatal "dropped-dead" syndrome (PDDS)-defined as an Apgar score of 0 or 1 at one minute after birth - was made on 301/4800 (6%) babies admitted to a neonatal intensive care unit during the period 1973-1980. This disorder may be complicated by clinical features of disseminated intravascular coagulation (DIC). Laboratory investigation of hemostasis was undertaken on 174/301. DIC was defined as a plasma fibrinogen concentration of $\leq$150mg per dl. or a 2 unit thrombin time of $\geq$ 30 seconds. A retrospective analysis was undertaken in order to determine (a) whether the occurrence of DIC was influenced by the outcome of cardiopulmonary resuscitation (CPR), as reflected in the Apgar score at 5 minutes after birth, and (b) if the features of DIC were transient or persistent. A diagnosis of DIC was made in 69/174 (41%) and it was unrelated to the interval between birth (B) and the time of investigation (I): $p = 0.2687$. Furthermore, DIC was unrelated to change in the Apgar score (between estimates at one and 5 minutes): $p = 0.3260$. Moreover, the change in Apgar score was unrelated to either gestational age (GA): $p = 0.0788$, or birth weight (BW): $p = 0.4776$, although the BI interval was related directly to both GA: $p = 0.0137$ and BW: $p = 0.0052$. Thus, the outcome of CPR is unrelated to degree of prematurity; but the more premature the baby the sooner he/she is studied after birth. DIC is a common complication of PDDS. It is evident even many hours after birth and is not influenced by the outcome of CPR. The efficacy of alternative therapeutic strategies remains to be evaluated.

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DISSEMINATED INTRAVASCULAR COAGULATION: A CLINICAL /LABORATORY CORRELATION IN 48 PATIENTS. R.L. Bick. San Joaquin Hematology Oncology Medical Group, California Coagulation Laboratories. Bakersfield, California, and UCLA Center for the Health Sciences, Los Angeles, California.

Disseminated intravascular coagulation (DIC) is a frequent clinical entity spanning from a moderately severe bleeding disorder to a catastrophic, fulminant, and often fatal form usually associated with hemorrhage or, less commonly,as diffuse thromboses. The clinical and laboratory features of DIC remain confusing and controversial. To critically evaluate the usefulness of coagulation tests in aiding in the diagnosis and monitoring of therapy in DIC the clinical and laboratory findings were summarized in 48 patients with DIC. All patients were subjected to a prothrombin time (PT), activated partial thromboplastin time (PTT), reptilase time (RT), thrombin time (TT), fibrin(ogen) degradation products (FDP), platelet count, protamine sulfate test (PSO_4), fibrinogen determination, and biological antithrombin-III (AT-III) level at the time of diagnosis. In addition, these same laboratory modalities were used to monitor patients during and after therapy. In this series of 48 patients, 38 patients had acute DIC and 10 patients had chronic DIC. In those patients with acute DIC, 100% of patients presented with hemorrhage and 53% of patients had thrombosis; 26% of patients died of their DIC type syndrome. In those patients with chronic DIC, 100% presented with hemorrhage, 80% presented with thrombosis, and none died of their intravascular clotting process. The probability of a pre-treatment abnormality in acute DIC was: FDP > AT-III = platelet count > PSO_4 > TT > PT > fibrinogen level > PTT > RT. The probability of pre-treatment abnormalties in chronic DIC was: FDP > PSO_4 = PT > AT-III = RT > platelet count > fibrinogen level = TT. These studies suggest the FDP level, the AT-III level, PSO_4, and fibrinogen level to be reliable for aiding in the diagnosis of acute DIC . In chronic DIC the fibrinogen level, PSO_4, PTT, and AT-III level appear to be the most reliable indicies.

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CLOTTING ABNORMALITIES DURING THE COURSE OF IMMUNE TYPE GLOMERULONEPHRITIS INDUCED BY $HgCl_2$ IN THE BROWN NORWAY (BN) RAT. A. Michaud, C. Sapin, M. Aiach, P. Druet, F. Forestier. Hôpital Broussais et Hôpital Notre-Dame de Bon-Secours, Paris (France).

This work was undertaken in order to study the clotting process during the course of a biphasic immune type glomerulonephritis (GN) induced by $HgCl_2$ in the Brown Norway (BN) rat. This toxic agent induced, in a first stage, the formation of antiglomerular basement membrane (GBM) antibodies and in a second stage an immune complex type GN.

Antithrombin III (AT III), fibrinogen degradation products (FDP) were evaluated by electroimmunodiffusion using specific antisera. The other classical methods were performed using commercial reagents. The albumin platelet content was evaluated by a radioimmunoassay. The soluble fibrin monomer complexes (FM) were searched for by a chromatographic technic.

Transient clotting proteins variations related to nephrotic syndrome were observed (low AT III level, low factor XII activity, increased factor V activity, and increased fibrinogen level). Disseminated intravascular coagulation (DIC) was suggested by low platelet counts, rise in FDP, and presence of FM. Fibrin thrombi in the glomerular capillaries were observed by immunofluorescence in the rats presenting evidence for DIC and in rats which died during the first phase of the disease. The animals presenting DIC syndrome were the most severy ill.

The hypothesis of an activation of the haemostasis system was supported by a low albumin platelet content and by the existence of high molecular weight fibrinogen derivatives. Complement activation, immune complexes, endothelial lesion might trigger DIC : the mechanism responsable for initiation of DIC are under study.

According to this results, it can be concluded that DIC which is responsable for the death of several animals, plays a major pathogenic role in this experimental autoimmune disease.

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