

PREVENTION BY PHOSPHOLIPASE C OF PULMONARY INSUFFICIENCY SYNDROME INDUCED BY TISSUE THROMBOPLASTIN IN RATS. K-E GIERCKSKY M.D. INSTITUTE OF MEDICAL BIOLOGY, TROMSØ, NORWAY

Purified phospholipase C (PLC) is a potent inactivator of tissue thromboplastin in vitro (1,2). Rats injected with a lethal dose of purified human tissue thromboplastin (3) survived when given PLC i.v. before the thromboplastin injection (4,5). PLC i.v. also led to a striking reduction in ^{125}I -fibrin and ^{51}Cr -platelets in the lungs when given just before a sublethal infusion of thromboplastin (5). Rat adipose tissue was homogenized and centrifuged to give 3 fractions, of which one had a marked procoagulant, tissue thromboplastin-like activity. Infusion of this fraction led to an accumulation of ^{125}I -fibrin and ^{51}Cr -platelets similar to that following infusion of tissue thromboplastin. LD_{50} for purified PLC in rats have been determined (6). Doses smaller than 10 % of LD_{50} protected rats against otherwise lethal doses of the procoagulant from adipose tissue and reduced the accumulation of fibrin and platelets in the lungs to nearly control levels. PLC does not alter the primary bleeding time, platelet half-life or thrombin-induced platelet aggregation. 1. Otness et al E.J.B. 1972, 2. Bjørklid et al TDH 1973, 3. Bjørklid et al BBRC 1973, 4. Giercksky et al S.J.H. 1976, 5. Giercksky & Bjørklid, S.J.H. 1976, 6. Otness et al S.J.C. lab. 1976.

COMPARATIVE HEMATOLOGY: STUDIES ON CLASS AVES, DOMESTIC TURKEY, MELEAGRIS GALLOPAVO. Jessica H. Lewis, Ute Hasiba and Joel Spero. School of Medicine, University of Pittsburgh and Central Blood Bank of Pittsburgh, Pittsburgh, Pennsylvania, U.S.A.

This study compared turkey and human blood in various coagulation and cellular parameters. Turkey blood clotted slowly (\pm 60 minutes) in either glass or siliconized tubes and the formed clots retracted very slightly or not at all. Prothrombin times, using mammalian brain, were 12-14 seconds for human plasma and over 60 seconds for turkey plasma. On the other hand, turkey brain clotted turkey plasma in 12 to 14 seconds and human plasma in over a minute. Russell Viper Venom clotted both plasma in 17 to 22 seconds. The activated partial thromboplastin time on turkey blood was over two minutes. Bovine thrombin clotted turkey plasma in about 30 seconds compared to 15 for human. Turkey clotting times with human thrombin or reptilase were very much longer than human. Thromboplastin generation tests with all human components gave substrate clotting times of 8.6 to 9.1 seconds; with all turkey components they were greater than 40 seconds. Turkey fibrinogen averaged 450mg/dl. Assayed in systems used for human coagulation factors, turkey factor XIII was high normal, factor VIII low normal (0.48U/ml), factors II and V present in traces and the others, VII, X, IX, and XII, absent or not measurable. Turkey thrombocytes were oval, nucleated cells which aggregated during clotting but not with adenosine diphosphate, mammalian or turkey collagen or ristocetin. TEM showed thrombocytes to contain microtubules, an open canalicular system, and a few mitochondria as well as a large nucleus. Hemostasis in turkeys appears dependent upon an extrinsic pathway in which tissue factor from an injured area initiates thrombin formation, which, in turn, causes thrombocyte aggregation and fibrin formation.

A COMPARISON OF THE ANTICOAGULANT ACTIONS OF COMMERCIALY AVAILABLE CONTRAST MEDIA (CM).

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Previous studies have reported on the anticoagulant effect of commercially available contrast media used in diagnostic radiology. The purpose of this study is to compare the anticoagulant actions of these agents in vitro. Eight commercially available contrast medias were supplemented to citrated human plasma (CNP) in 1:10, 1:20 and 1:50 proportions; isomolar sucrose, glucose, sodium chloride, and saline supplemented CNP were used as controls. Prothrombin time (PT), partial thromboplastin time (PTT), thrombin time (TT), antithrombin-III, plasminogen/plasmin and factor assays were performed at 0 time, 30 minutes and 2 hours after incubation at 37°C. No significant alteration of the coagulation parameters were observed at 1:50 dilution, however at 1:10 and 1:20 dilution, most contrast media produced aberration of clotting parameters. The antithrombin potency of these contrast media at a 1:10 dilution ranged from 0.3-1.3 u/ml heparin. In addition, this antithrombin activity was synergistic with heparin. The antithrombin activity of these agents was not neutralized by protamine sulfate, polybrene or toluidine blue in the amounts which neutralized 1 u/ml heparin. Analysis of various clotting factors revealed that factors II, V, VII and XII were not affected by contrast media. Factors VIII and IX were depressed significantly. These changes were mainly dependent on the concentration of meglumine in the contrast media. Similar studies on the blood obtained from patients infused with contrast media for diagnostic purposes are in progress in our laboratory.