

Supplementary Abstracts

Antithrombin III Heparin

ANTITHROMBIN III ACTIVITY LEVELS IN SERUM: RESULTS OF A LARGE SURVEY STUDY USING A NEW METHOD OF SAMPLE PREPARATION
A. Gray, K. Uhlmeier, R. LaBonte and L. Bailey Jr. Abbott Laboratories, North Chicago, Illinois and Bio-Diagnostics Medical Laboratories, Springfield, Illinois.

The determination of Antithrombin III (AT III) levels in serum is of major interest because the serum AT III level will reflect both the level of AT III in the circulation (plasma AT III) and the level of procoagulant factors in the circulation (which consume the AT III during serum formation *in vitro*). Serum samples are also more readily available for testing in reference laboratories who normally receive very few citrated plasma samples. A collaborative study was performed to test AT III levels on all serum samples sent in to the Bio-Diagnostics Medical Laboratory for chemistry panel testing during a one month period.

It has been recognized that the level of prothrombin consumption during serum formation will vary widely depending on the sample handling conditions. Only a small amount of thrombin generation is required for complete defibrination of a blood sample. Some serum samples collected in glass without an anticoagulant still had normal plasma levels of prothrombin and AT III. This may indicate early removal of platelets by centrifugation, low levels of contact factors or rapid anticoagulation kinetics. A new method was devised to standardize the complete activation (and thus AT III consumption) of samples received from approximately 800 offices and clinics collecting and mailing the samples under various conditions. To 100 μ l sample serum was added 10 μ l of commercial thromboplastin/calcium ion solution to activate the sample extrinsically. After at least five minutes, the samples were assayed for their AT III activity using the Qanticheck AT III assay on an Abbott VP. A total of 5800 samples were assayed with a mean value of 83 ± 1 (2 S.D.). There were 132 samples outside of the normal range of 74 ± 20 % AT III (determined on 99 normal adults).

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ANTITHROMBIN III INFUSION DURING FULMINANT HEPATIC FAILURE
S. Braude, J. Arias, R.D. Hughes, J. Canalese, A.E.S. Gimson, R. Williams, M.F. Scully*, V.V. Kakkar* Liver Unit, *Thrombosis Research Unit, King's College Hospital Medical School, London, England.

The antithrombin III (ATIII) levels in 17 patients with fulminant hepatic failure due to viral hepatitis or paracetamol overdose were found to be 25.8 ± 12.80 of normal on admission. The levels did not correlate with eventual survival or death and remained essentially unchanged for up to 7 days.

In an attempt to assess the role of the low levels of AT III during the course of hepatic failure and in relation to treatment by charcoal haemoperfusion we have infused patients with commercially purified ATIII. Preliminary measurements of ATIII (chromogenic substrate method) were made and ATIII infused to achieve a plasma concentration of 50 to 70%. Infusion was by an initial bolus of 1500-2000 units followed by up to 500 units every 6 hours. To date 3 patients in Grade IV hepatic coma have been treated, one died 1 day after admission and the other two survived. In the latter the return of the prothrombin time to normal was similar to that in patients without the addition of ATIII. In one of the survivors the platelet count did not fall, suggesting ATIII may have had a protective effect on platelet consumption. There was also an indication, that there was a more uniform and better response to heparin anticoagulation during haemoperfusion than found previously without ATIII infusion.

Further patients will be treated to evaluate whether AT III substitution can reduce the consumptive coagulopathy and platelet destruction which occurs in the course of fulminant hepatic failure.

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ANTITHROMBIN-III DEFICIENCY CAUSING DEEP VEIN THROMBOSIS AND PULMONARY EMBOLISM IN A YOUNG MALE
K. Genth, J. Schaefer, J. Frank, W. Krämer, B. Weinel, D. Heene. Klinikum Mannheim, University of Heidelberg, West Germany.

A 34 year old male was admitted to the hospital with typical clinical symptoms of acute pulmonary embolism caused by deep vein thrombosis in the upper leg detected by phlebography. Pulmonary embolism was verified by the lung-perfusion-scintigram. The patient developed an infarct pneumonia with hemoptoe. Episodic thromboembolic phenomena occurred due to antithrombin-III deficiency (AT-III). The method, using homogenic substrates exhibited low AT-III activity of 8.6 IU/ml (25°C) due to a familiar AT-III deficit. Fiberoptic pulmonary catheter was placed into the pulmonary artery to measure pulmonary artery pressure (PAP, PCP), right ventricular pressure (RVP) and to determine cardiac output (CO) using the dye dilution technique. Heart rate (HR), central venous pressure (CVP) and aortic pressure (AOP) were recorded continuously. Patient received immediately fibrinolytic therapy, initiated by an initial dose of streptokinase (SK) (250 000 IU/20 min.), followed by a maintenance dose (100 000 IU/h), lasting 3 days. Measured values are given in the schedule:

	HR	CVP	AOP	PAP ₁	PAP ₂	PCP	RVP	CO
before SK	120	18	110/60	107	36	40	103/11	3.6
after SK	88	13	120/80	94	34	20	90/14	4.8

M-mode echocardiography detected before SK a moderate enlarged right ventricle and a small left ventricle, indicating a low output. After SK these values were improved. In conclusion, this case demonstrated a serious thromboembolic disorder, related to AT-III deficit. SK-therapy improved the hemodynamic situation.