Friday, July 17, 1981

Poster Presentations

Antithrombin III – II

11:00–12:30 h

Grand Ballroom Lobby Boards 209–213

0937

INVESTIGATION OF THE RELATIONSHIP BETWEEN PREOPERATIVE CHROMOGENIC ANTITHROMBIN III ASSAYS AND HEMORRHAGE FOLLOWING HEART SURGERY. J.E. Leveson, J. Winford, J. Iden, A.J. Marengo-Rowe, G.J. Race. Department of Pathology, Baylor University Medical Center, Dallas, Texas 75246.

The above study is part of an ongoing program to determine the mechanisms of hemorrhage in patients who have undergone extracorporeal circulation. Antithrombin III levels were measured in 850 patients (Quantichrom III) prior to undergoing cardiovascular surgery. Following surgery the maximum rate of blood loss was assessed by measuring chest tube drainage at hourly intervals. Patients whose blood loss exceeded 150 ml in any one hour were defined as "bleeders." 257 patients had a maximum rate of blood loss exceeding 150 ml per hour, and this group is called the bleeder group. The bleeder group had a range of antithrombin III values of 16 to 148% and a mode 66% compared to a range of 24 to 170% and a mode 80% for the non-bleeder group. The means of two groups were 66% and 84% ($p < 0.05$) were statistically significantly different. 156 patients had an antithrombin III level of less than 60% of which 81 fell into bleeder group. Possible mechanisms of this relationship are: (1) During massive surgical procedures disseminated proteolysis occurs and those patients with a lower level of proteolytic enzyme inhibitors such as antithrombin III have an impaired ability to control this process, which ultimately leads to hemorrhage. (2) During the massive surgery, an imbalance in protein metabolism occurs. Patients who are poor "protein producers" for pathological, genetic or nutritional reasons are less able to respond to this depletion and they respond less readily to the depletion of proteins such as the coagulation proteins with resultant bleeding. Antithrombin III may be an "indicator protein" for such patients. (3) Reduced levels of antithrombin III reduce the potency of heparin, and thus level of anticoagulation during surgery.

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ESTROGEN THERAPY INDUCED ANTITHROMBIN III DECREASE AND DEFICIENCY IN PATIENTS WITH PROSTATIC CANCER. H.R. Büller, T.A. Boon, N.F. Dabhoiwala, Ch.P. Henny and J.W. ten Cate. Department of Haematology, Division of Hemostasis and Department of Urology. University Hospital "Wilhelmina Gasthuis", Amsterdam, The Netherlands.

Plasma AT III activity was studied in 12 patients with cancer of the prostate receiving estrogen therapy in different dosages. AT III was determined with an automated chromogenic method (normal value 0.80-1.40 u/ml). In six patients with maintenance diethylstilbestrol (DES) therapy (2.5 mg/48 hrs) mean plasma AT III activity was normal ($\bar{X}$ 0.88 u/ml, range 0.74-1.05 u/ml). The dose of DES at 15 mg. daily resulted in a marked AT III activity decrease (mean 0.24 u/ml) in all six patients. In patients with low normal initial plasma AT III activity acquired AT III deficiency, with enhanced risk of thromboembolic complications, may develop. The observations suggest that plasma AT III activity should be monitored before and during estrogen therapy in prostatic cancer patients. In patients with acquired AT III deficiency (<0.80 u/ml) prophylactic administration of low dose heparin subcutaneously should be considered.