

ANTITHROMBIN III IN PATIENTS TREATED WITH SUBCUTANEOUS OR INTRAVENOUS HEPARIN. J. Conard, T. Lecompte, M.H. Horellou, B. Cazenave, M. Samama. Laboratoire Central d'Hématologie, Hôtel-Dieu, Paris, France.

Plasma Antithrombin III (AT III) has been measured by Mancini method and serum antithrombin (AT) activity by Von Kaulla's method in 49 patients treated with intravenous (10 cases) or subcutaneous (39 cases) heparin at therapeutical doses. Among the 39 subcutaneous treated patients, 19 were re-examined after cessation of heparin.

A statistically significant decrease in plasma AT III and in serum AT activity has been observed in both groups of heparin-treated patients. In addition, in the 19 subcutaneous treated patients, the plasma AT III and serum AT activity which were decreased under treatment, increased within normal limits after cessation of the treatment: consequently, a congenital AT III deficiency could be ruled out in these patients.

In conclusion, we did find a statistically significant decrease in plasma AT III and in serum AT activity in heparin-treated patients, whatever the route of administration.

CROSSED IMMUNOELECTROFOCUSING OF ANTITHROMBIN III IN HEALTHY AND IN CONGENITAL AND ACQUIRED DEFICIENCY. G. Leone, V.M. Valori, G. Traisci, B. Bizzi. Istituto di Clinica Medica-Università Cattolica del Sacro Cuore, Rome, Italy.

Isoelectrofocusing was carried out in LKB Multiphor apparatus with pH 4-6.5 carrier ampholites using polyacrilamide gels. Specimens of human purified antithrombin, normal plasma pool, plasma with congenital and acquired AT-III deficiency were isoelectric focused. Purified Antithrombin showed two peaks. The isoelectric points of these antithrombin peaks were 4.6 and 4.9. Next the polyacrilamide gel slabs were placed on glass plates coated with agarose containing 4% anti human antithrombin antibody; crossed electrophoresis was then carried out. Normal pool plasma exhibits two peaks, which correspond to the isoelectric focusing position of the purified AT-III. Plasmas of two families with congenital AT-III deficiency exhibit only one peak at pH lower. On the contrary plasmas of patients with acquired AT-III deficiency (hepatic cirrhosis, asparaginase therapy) exhibit two peaks. Considering that microheterogeneity is fundamentally due to a difference in glycosylation (Borsodi and Nerasimhan, Brit. J. Haemat. 1978, 39,121) it is possible to hypothesize a defect in glycosylation in congenital defect.

BIOLOGICAL ACTIVITY OF ANTITHROMBIN III IN BLOOD PLASMA AND EDEMATOUS FLUIDS OF PATIENTS WITH A NEPHROTIC SYNDROME. L.V.Podorolskaya, G.V.Andrenko, L.R.Polyantseva. Laboratory of Enzymatic Fibrinolysis, Moscow State University, Moscow, USSR

The biological activity of antithrombin (AT) III was tested by the Abilgaard method in blood plasma of 149 patients with renal lesions of various etiology, of 27 donors and in 17 samples of edematous fluid of patients with nephrotic syndrome (NS). The majority of patients had a decreased AT III content in blood plasma, which was especially well pronounced in NS patients irrespective of etiology. In edematous fluids the level of AT III was 10% of that in the plasma. The functional activity of AT III in the plasma was well-correlated with NS manifestations, i.e. 24 hr-proteinuria, hypoalbuminemia, hypercholesterinemia) and with hemocoagulation and fibrinolysis parameters (SFMC content, plasma tolerance to heparin, contents of heparin, plasminogen activator and kallikrein and total antitryptic activity). A reactive increase of AT III in NS patients with venous occlusion is of essential prognostic value. Heparin therapy results in an increase of the originally low AT III content and in positive dynamics of NS.

THE EFFECT OF LOW DOSE INTRAVENOUS HEPARIN ON ANTI-FACTOR Xa AND ANTITHROMBIN III. S.A. Jennings, B.P. Heather and R.M. Greenhalgh. Professorial Department of Surgery, Charing Cross Hospital, London W6 8RF, Great Britain.

Preoperative blood samples from 17 patients undergoing major abdominal surgery were examined by the thrombelastograph saline dilution test, which has previously been shown to be a predictor of the risk of early postoperative deep vein thrombosis (DVT)(Heather et al 1980). By this test 8 patients were predicted to be at low risk of developing a DVT and received no special prophylaxis. 9 patients were considered to be at risk and were treated with a subcutaneous dose of 1000 units of heparin with the premedication together with a low dose of intravenous heparin infusion from the induction of anaesthesia until 2 hours after operation. Plasma antithrombin III (ATIII) concentration and anti-factor Xa activity were measured preoperatively, on day 1 and on day 3. No early DVT occurred, as assessed by I¹²⁵ fibrinogen scanning, in either the untreated low risk patients or in the high risk patients receiving heparin infusion. The high risk patients had lower levels of ATIII before operation than the low risk patients (75±9%; 98±39%) and significantly lower levels on day 3 (64±25%; 106±34% p<0.02). However, these lowered levels of ATIII appeared in the high risk group to be augmented by the significant increase in anti-factor Xa activity 127±64% before operation, 217±89%, on day 1 (p<0.02). Furthermore, on day 3 the high risk patients had significantly greater activity than those patients in the low risk group (212±76%; 106±34% p<0.02).

These results show that those patients at risk of developing a postoperative DVT had a significantly enhanced activation of anti-factor Xa, as a result of intravenous low dose heparin with the subsequent abolition of early venous thrombosis.