BEHAVIOUR OF SOLUBLE DES-AB FIBRIN AND FIBRINOGEN IN MAN.

I. Mahn and G. Müller-Berghaus, Dept. Clin. Immunol. & Blood Transfusion and Dept. Internal Medicine, Justus-Liebig-Universität, Giessen, West-Germany.

Human des-AB fibrin was generated from I-131-fibrinogen by clotting with thrombin in the presence of EDTA and aprotinin, and solubilized in buffered urea. About 1 to 2 mg of fibrin was injected simultaneously with I-125-fibrinogen dissolved in buffered urea into 14 male healthy volunteers.

The mean distribution volume of the soluble fibrin was 48.5 ml/kg in comparison to 46.1 ml/kg of the injected fibrinogen demonstrating a homogeneous distribution of both the labelled proteins in the circulating blood. The elimination characteristics of the soluble fibrin did not represent a monophasic exponential decay. 24 hours after injection a mean of 24.3 % of the initial value of the fibrin was traced in the blood in comparison to a mean of 53.3 % of the fibrinogen at that time. The terminal slope of the fibrin elimination curve between 2 and 8 days after injection was a monophasic exponential curve with a mean $T_{1/2}$ of 2.3 days. The fibrinogen elimination curve showed a $T_{1/2}$ of 4.2 days during that period of time.

These experiments indicate that solubilized fibrin injected into man can be traced in the circulating blood for a long period of time. The catabolic behaviour of fibrin differs significantly from that of fibrinogen.

SOLUBLE FIBRIN MONOMER COMPLEXES IN PATIENTS WITH RENAL CELL CARCINOMA.:

K. Andrassy, R. Gärtner, G. Riedasch, R. Zimmermann, E. Ritz, R. Hafter, Med. + Urolog. Universitätsklinik Heidelberg and Univ.-Frauenklinik, München, F.R.G.

Patients with renal cell carcinoma (r.c.) are known to exhibit retention of bromsulphalein, increase in alkaline serum phosphatase (a.p.) and diminution of prothrombin time (Stauffer-syndrome). These abnormalities were thought to reflect impaired liver function. However, the increase in a.p. was shown to be due to appearance of Regan's isoenzyme. The present study was designed to test the hypothesis that coagulation abnormalities in the patients result from low grade (intratumoral) intravascular coagulation. 26 patients with r.c. with/without metastases were studied preoperatively: Assay of coagulation tests: soluble fibrin monomer complexes (SFMC) by ethanol gelation and by precipitation with β -alanin and subsequent agarose chromatography according to Hafter and Graeff. - Thrombin coagulase time (t.c.) was abnormal in virtually all patients (23/26); in addition reptilase time (r.t.) (15/26), FDP as measured by TRCHII (8/26) and staph. clump-test (4/26) and normotest (6/26) were abnormal. T.c. and r.t. were particularly abnormal in 12/26 patients in whom increased conc. of SFMC were found. It is concluded that low grade intravascular coagulation, possibly within the vascular bed of the highly vascular tumor, and to a lesser extent fibrinolysis accounted for the coagulation abnormalities in (non)-metastasing renal cell carcinoma.