
Soluble fibrin complexes may occur in vivo in a variety of coagulation disorders. The aim of this investigation was to elucidate the in vivo behavior of fibrinogen complexes prepared in vitro in a ratio of 1 part fibrinogen to 30 parts fibrinogen. 12 rabbits (group A) were injected with soluble fibrin complexes containing homologous I-131-fibrin and I-125-fibrinogen. Another group of 12 rabbits served as control (group B) and received I-131-fibrin solubilized in urea and I-125-fibrinogen separately from each other. Studies were performed over a period of 6 days.

The mean distribution volume of fibrin as well as of fibrinogen did not significantly differ between both groups. The elimination characteristics of I-131-fibrin of the soluble fibrin complexes (group A) as well as of the solubilized fibrin (group B) were similar. The fibrinogen elimination did not differ significantly between the groups; a mean T 1/2 of 47.8 h in group A and a T 1/2 of 46.7 h in group B was calculated.

The experiments demonstrate that non-crosslinked soluble fibrin complexes distribute homogeneously in the circulation and dissociate into its subunits. Fibrin is eliminated from the circulating blood without influencing the normal catabolism of fibrinogen.

A STUDY OF THE ASYPTOTIC ERFROBOM RELATED TO HYPERCOAGULABILITY BY DETERMINATION OF SOLUBLE FIBRIN MONOMER COMPLEX (SMC) AND PLASMIN INHIBITOR. T. Suda (Department of Obstetrics & Gynecology, University of Hokkaido, Sapporo, JAPAN), H. Graeff, R. Hafer (Ist Frauenklinik d Universitats München, West-Germany).

Gerbil blood samples from 10 healthy newborns and 19 asphyctic newborns were examined. SPNG and fibrinogen were precipitated from plasma with β-alanine. Agarose gel filtration of redissolved precipitate resulted in separation of SPNG and fibrinogen. Other parameters such as TEG, Prothrombin time (PT), Partial thromboplastin time (PTT), and Plasmin-inhibitors (β2-agreginolbin, A γ-antitrypsin, antithrombin III) were also determined.

(Results)

1. The percent amount of SPNG of total fibrinogen content in asphyctic newborn increased 4.7% ± 1.5%, while the remaining normal infants showed only 3.17 ± 0.55% (P < 0.05).

2. In neither PT nor PTT can a significant difference be seen, although asphyctic newborn showed the tendency of hypercoagulability in TEG.

3. A γ-antitrypsin (9.8 ± 2.3) and antithrombin III (16.8 ± 1.7) levels were much lower in the group with asphyxia.

These results indicate that a low level of Plasma-inhibitors act synergistically with a high activator value.

The low antithrombin III level in particular could be one of the reason for the development Hypercoagulability and DIC in asphyctic newborn infants.


Blood samples from patients with coagulation disorders in obstetrics and with advanced carcinoma of the kidney were examined for the presence of crosslinked fibrinoligomers. Quantitative gel filtration of β-alanine precipitated plasma samples and chain characterization of isolated fibrin derivatives by SDS-PAGE gel electrophoresis after reduction with mercaptoethanol were performed. Electrophoresis of immunoabsorbed material was additionally applied. In all cases of intravascular coagulation crosslinked fibrinoligomers in amounts of β-25 per cent of the total fibrinogen content were observed. Severe carcinoma has a molecular weight pattern of derivatives ranging from 5 million and more down to 45 000. X, Y, D, E and D-dimer were found in the β-alanine supernatant. In 5 patients with advanced renal carcinoma a crosslinked dimeric derivative was observed predominantly.

In vitro produced crosslinked 125-β-fibrinoligomers were injected into rabbits. Radioactivity was measured in gel filtered fractions from β-alanine precipitated samples, and a half-life time of approximately 7 hours of the high molecular weight fraction averaging 5 million daltons was found.

It is concluded that circulating crosslinked fibrinoligomers which differ in regard to complex formation and half-life time to soluble fibrin monomer complexes may indicate intravascular coagulation.