

Supplementary Abstracts

Factor XIII

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INCOMPLETE FIBRIN FORMATION AND HIGHLY ELEVATED FACTOR XIII LEVELS IN MULTIPLE MYELOMA

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In some patients suffering from multiple myeloma a defect in fibrin formation has been suggested as one cause for bleeding tendency. However nothing is known about the commonness of this disorder or about the correlation to factor XIII activity.

As pointed out in this investigation the defect in fibrin formation, as proven in SDS-PAGE electrophoresis, is due to a lack of α -chain polymerization in 5/11 patients with IgG-myeloma and 2/5 with IgM paraproteinemia. No disturbed fibrin formation could be observed in IgA myeloma (n=6). Factor XIII concentration of subunit A and to a smaller extent of subunit S (Laurell technique) were highly elevated in all cases with regular fibrin formation. Comparable values were obtained measuring the transamidase activity as incorporation of C 14 labelled putrescine into casein. Levels up to 600 % of normals could be evaluated. In contrast all patients with a lack of α -chain polymerization had a factor XIII activity within the normal range. Addition of factor XIII concentrate to plasma with defective fibrin formation led in 5 out of 7 cases to a partial crosslinking of α -monomers. A complete α -chain polymerization could not be achieved, even when high concentrations of factor XIII were used. We conclude that in some cases paraproteins can inhibit the factor XIII activity and prevent its action on fibrin. Patients, who are able to produce factor XIII, especially the active part subunit A in excess, can achieve a regular fibrin formation. Treatment with factor XIII in case of bleeding should be taken into consideration.

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CONCENTRATIONS OF SEVERAL HORMONES IN A FACTOR XIII PREPARATION OF PLACENTAL ORIGIN. W. Krämer, G.-H. v. Mittelstaedt, R. Schmidt, C.L. Heene. I. Department of Internal Medicine, Department of Clinical Chemistry, University of Heidelberg, Faculty for Clinical Medicine Mannheim, D. 6800, FRG.

A 13 year old male patient suffering from an inborn factor XIII deficiency was treated by a factor XIII concentrate to cure a pseudotumor at the leg. Under treatment the patient showed clinical symptoms which were regarded as not yet observed hormonal side effects. The used preparation was made of human placenta. It seemed to be instructive to analyse in this concentrate the amount of several placental hormones.

Three randomised charges of this factor XIII preparation were investigated for the hormonal activity of LH, prolactin, FSH, cortisol, HPL, free oestriol, total oestriol and testosterone. All determinations were carried out by RIA. Elevated concentrations for testosterone and up to the reach of the second period of pregnancy for LH and total oestriol were found.

It is necessary to pay attention to hormonal side effects in treating patients with factor XIII preparations made of placental origin.

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THE HORMONE - DEPENDENT CONCENTRATION CHANGES OF FACTOR XIII (FIBRINOLIGASE) DURING PREGNANCY.

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The effects of hormones on blood coagulation and fibrinolysis have been studied extensively since oestrogen / progesterone preparations were used for contraceptive purposes. There is a general agreement that such preparations cause a rise in plasma levels of fibrinogen, prothrombin and factors VII, IX, and X. In comparison, most of the coagulation factors synthesized by the liver show a gradual increase during normal pregnancy. Only factor XIII and calcium reveal a tendency to decrease. In 90 patients out of a normal population the plasmatic factor XIII concentration declined from 0.39 units in the first trimester to 0.29 and 0.23 units in the second and third trimester. Since the fibrinogen concentration is higher than during normal menstrual cycles the reduction of factor XIII may be caused by a decreased synthesis or an increased clearance rate from circulation. The abnormal haemostatic conditions during gestation could be interpreted as a physiological response to the increase of oestrogen and progesterone leading to a hypercoagulable state. Reduced levels of factor XIII may counterbalance the increased rate of fibrin formation and serve as protection against thromboembolic complications.