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INTEREST OF THE STUDY OF PREKALLIKREIN AND FIBRINOLYSIS IN DIABETIC PATIENTS. J.C. Adjizian, D. Janody, C. Droulle, G. Jeunehomme, M. Leutenegger, G. Potron. Clinique médicale II et Laboratoire central d'Hématologie, Centre Hospitalier et Universitaire, rue Alexis Carrel, 51100 REIMS, France.

For the comprehension of macro and microangiopathy diabetes physiopathology, it appeared to us interesting to study the contact factors and plasmatic fibrinolysis. The study covered 144 patients with all types of diabetes.

Measurement of prekallikrein, plasminogen and fast α_2 antiplasmin were carried out on chromogen substrates (S 2302 and 2251 - Kabi) using an automatic apparatus (Kem O Mat 2 Coultronics).

At the same time, we studied the glycemia response, A_1C hemoglobin and Willebrand factor.

A dynamic and evolutive study was made on patients with an artificial pancreas. Results showed variations due to age, diabetes equilibration and in particular low rates of prekallikrein with a decrease of the fibrinolytic system in diabetic patients with macroangiopathy.

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EFFECTS OF ASPIRIN (ASA) AND SULFINPYRAZONE (SPZ) ON THE COAGULOPATHY OF DIABETIC NEPHROPATHY (DN). I. Mariscal, J. Fernandez de Castro. Department of Medicine. Hosp Gomez Farias ISSSTE. Guadalajara, Mexico.

In diabetics, changes in the vessel wall and platelet hyperaggregability are well documented. Fg/Fn deposits in the glomeruli among other changes as well as cDIC have been described in DN. Based on these data this study was planned to explore the role of platelets in the coagolopathy of DN.

Eighteen patients with DN characterized by mild to severe urea retention, significant proteinuria and creatinine clearence of 67 ml/min or less participated in a study which was prospective, double blind, random in distribution and subject to the patient's own control. ASA 1.0 gm or SPZ 600 mgs or placebo 3 pills/day was used. Coagulation and fibrinolysis tests were done regularly.

ASA shortened thrombin time (Biggs) from 22.0 to 19.3 sec *, - raised plasminogen (Mancinni-M Partigen Plasminogen) from 12 to 13 mgs P=NS and decreased FDP (Merskey) in serum from 56 to 32 Ug ** and in urine from 2.8 to 1.0 Ug **. SPZ raised plasminogen from 12 to 13 mgs **.Both ASA and SPZ decreased fibrinogen (Ingram) P=NS and produce no change on PT (Quick), PTT (Proctor) and platelet count (Bretcher & Cronkite).

SPZ changes were interesting but barely significant up to now.

The changes induced by ASA are suggestive that cDIC was inhibited. Since ASA has no direct effect on the modified parameters, it can be considered that the action was due to ASA antiplatelet activity.

We can propose that platelets play an important role in maintaining the coagulopathy of DN and possibly in initiating it.

* $P < 0.05$ ** $P < 0.01$

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to improve
the understanding
and treatment of
thromboembolic
diseases

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