CHANGES OF THE SERUM FIBRIN DEGRADATION PRODUCTS (FDP) IN CHRONIC PYELONEPHRITIS PATIENTS AT STATE OF CHRONIC RENAL FAILURE (CRF)

R. Alahverdyan

Key-words: fibrin degradation products — fibrin monomeric complexes — chronic renal failure — fibrinolysis

Fibrin degradation products (FDP) increase in serum and urine of nephropathy patients is already established by series of investigators (3, 8—10). The study of these products possesses a definite importance for the evaluation of the severity of the disease as well as for the necessity of anticoagulation therapy (11, 12). There are a few of observations in this relation when chronic renal failure (CRF) is concerned (15). The contingents included commonly patients with different primary diseases causing the appearance of renal insufficiency. Thus proceeding from the literature data we set us the task of examining FDP level in various CRF degrees resulted from chronic pyelonephritis only.

Material and methods

The study covered 44 CRF patients divided according to the degree of renal insufficiency into the following groups: 1st — initial — 18 patients; 2nd — expressed — 14, and 3rd — advanced — 12. Selection was made after Razboynikov-Mushmov's classification (6). CRF was the result of chronic pyelonephritis diagnosticated on the basis of the main clinical and paraclinical criteria of the disease (7). The control group consisted in 35 clinically healthy individuals in an almost equal age group as compared with the CRF patients

Serum FDP were determined by using of Merskey's et al. (1966) immunological test, soluble fibrin monomeric complexes (FMC) in plasma after Lipinski's et al. (1968) method and blood fibrinolytic activity (FA) by means of euglobulinolysis (4). Routine coagulation parameters (fibrinogen, blood platelets, prothrombin activity, bleeding and coagulation time) were also examined.

Results and discussion

Our results showed an increase of both FDP and FMC. Their changes were more significant with advancing renal insufficiency. Mean rates of aforementioned indices differed statistically reliably from these of the controls (table 1).

The correlation analysis concerning these diseases demonstrated a strongly positive correlation between FDP and FMC with coefficient of 0.7432. There was a moderately positive relationship of FDP and urea ($r=0.4562$) and FDP and creatinine ($r=0.4374$). The frequency of increased FDP with 1st degree CRF was 38.8 per cent, with 2nd one = 57.7 per cent, and with 3rd one — even 66.6 per cent of the cases. In some patients tending to nitrogen fraction reduction in the course of treatment there was also a decrease of FDP and FMC both, however, still in the range of levels differing statistically significantly from these of the controls.

Table 1

Examined parameters in CRF patients

Parameters/ Groups	FDP mg/l $x \pm \Delta$	FDP (OU) $x \pm \Delta$	FA (min) $x \pm \Delta$
healthy	3.468±0.429	0.363±0.041	223.626±11.850
CRF 1st degree	11.310±4.803 p<0.001	0.445±0.099 p<0,02	232.722±15.710 p<0.05
CRF 2nd degree	15.860±7.426 p<0.001	0.552±0.107 p<0.001	239.285±19.211 p<0.02
CRF 3 rd degree	22.891±13.264 p<0.01	0.641±0.124. p<0.001	261.266±31.631 p<0.01

The rates of these parameters were as followed: 1st degree CRF: FDP 6.062± 1.616 mg/l ($p<0.01$) and FMC 0.445±0.062 U ($p<0.01$); 2nd degree CRF: FDP 10.531±4.103 mg/l ($p<0.01$) and FMC 0.505±0.082 U ($p<0.01$); 3rd degree CRF: FDP 14.027±7.36 mg/l ($p<0.01$) and FMC 0.531±0.120 U ($p<0.01$).

FA showed a statistically reliable time elongation as compared with that of the healthy individuals (table 1). The correlation between FDP and FA was moderately positive with a coefficient of 0.4015.

Fibrinogen concentration increased significantly in all CRF degrees as followed: 1st degree — 4.487±0.56 g/l ($p<0.001$); 2nd degree — 4.763±0.821 g/l ($p<0.001$), and 3rd degree — 4.677±0.678 g/l ($p<0,001$). This index was 2.977± 0.154 g/l in the control group.

There was, therefore, a parallelism between FDP concentration and frequency of increase, on the one hand, and the severity of renal insufficiency in different CRF stages, on the other. Diminution of nitrogen fractions induced a reduction of the level of this index. The positive correlation between FDP, on the one hand, and urea creatinine, on the other, argued also for this statement. Our observations were in concordance with Hedner's et al. (1973) data. The increased fibrinogen concentration reported by ourselves confirmed the investigations of other authors, too (1, 2).

FA in the circulation was suppressed in CRF patients (2, 5). This fact could be due to the increased activity of fibrinolysis inhibitors. The time elongation of euglobulinolysis as established in our study did not allow us, however, to affirm emphatically that there was FA depression with these cases because by using of this method an information was provided about fibrinolysis activators only and inhibitor influences were ignored.

FDP enhancement in CRF despite of FA depression in the common circulation could be due most likely to local fibrinolytic processes resulted from renal intravascular coagulation. Our assumption is confirmed by the strongly positive correlation between FDP and FMC found out in this study.

On the basis of our own observations we can consider blood FDP increase in CRF most probably a result from secondary fibrinolysis. The level of these products rises with advancing renal insufficiency and correlates with that one of serum urea and creatinine.

REFERENCES

1. Андреенко, Г. Фибринолиз (биохимия, физиология, патология). М., Московский университет, 1979. — 2. Делийскн, М. Автореферат канд. дис. Варна, 1979. — 3. Ена, Я. *Сов. мед.*, 1975, № 7, 85—88. — 4. Енев, Н. Лабораторен контрол при провеждане на антикоагулантна и фибринолитична терапия. В: Антикоагулантна и фибринолитична терапия. С., 1970, 84—112. — 5. Карастанев, И., К. Атанасов. *Урол. и нефрол.*, 1976, № 3, 19—21. — 6. Ненов, Д. Хронична бъбречна недостатъчност. Във: Вътрешни болести. С., Мед. и физк., 1980, 1, 263—265. — 7. Ненов, Д. Пиелонефрит. Във: Вътрешни болести. С., Мед. и физк., 1980, 2, 422—429. —8. Astedt, B., I. M. Nilsson. *Folia Haematol.*, 1972, No 1/2, 84—94. — 9. Haanen, C., A. Holdrinet, P. Wijdeveld. *Scand. J. Haematol.*, 1971, *Suppl.* 13, 337—343. — 10. Hedner, U., I. M. Nilsson. Les maladies du rein et produits de degradation du fibrinogene. In: Actualites nephrologiques de l'hopital Necker. Paris, Flammarion, 1973, 257—287. — 11. Lagrue, G. *Pathol. Biol.*, 23, 1973, No 6, 486—491. — 12. Lipinski, B., K. Worowski. *Thromb. Diathes. Haem.*, 1968, No 1, 44—49. — 13. Merskey, C., G. Kleiner, A. Johnson. *Blood*, 1966, No 1, 1—18. — 14. Michielsen, P., L. Koele, J. Vanreterghem, et al. *Clin. Nephrol.*, 1976, 105—111. — 15. Nilsson, I. M. *Scand. J. Haematol.*, 1971, Suppl. 13, 357—365.

ИЗМЕНЕНИЯ УРОВНЯ СЫВОРОТКИ ФИБРИНДЕГРАДАЦИОННЫХ ПРОДУКТОВ (ФДП) У БОЛЬНЫХ ХРОНИЧЕСКИМ ПИЕЛОНЕФРИТОМ В СТАДИИ ХРОНИЧЕСКОЙ ПОЧЕЧНОЙ НЕДОСТАТОЧНОСТИ (ХПН)

Р. Алахвердян

РЕЗЮМЕ

Исследованы фибриндеградационные продукты, растворимые фибринмономерные комплексы и эуглобулиновый лизис (как метод для определения фибринолитической активности крови) у больных с начальной, выраженной и далеко зашедшей почечной недостаточностью, наступившей в результате хронического пиелонефрита. Устанавливается, что с прогрессированием почечной недостаточности параллельно возрастает и количество фибриндеградационных продуктов и фибринмономерных комплексов. Стоимости этих показателей показывают статистически значимую разницу по сравнению с показателями исследования клинически здоровых людей. У больных, при которых отмечается тенденция к снижению азотистых фракций в результате лечения основного заболевания, устанавливается также уменьшение количества фибриндеградационных продуктов.