

UREA AND SERUM PROTEINS IN CIRCUMSCRIBED PROFOUND BURN ON COMBINED TREATMENT

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The changes of protein metabolism in burn and their importance in the pathogenesis of the disease "burn" are really undoubted for everyone. However, the percentage and degree of burn inducing protein metabolism disorders, and especially the methods and means to correct these disorders, are still an object of discussion (1, 2, 4, 8, 9).

The present communication is a link of a series of research works in which we try to answer some of these questions. In this paper we make an attempt to establish if a small-size (5—10 per cent) profound burn (IIIrd—IVth degree) causes alterations of total protein, protein fractions and serum urea and how protein hydrolysate and hemodex both influences upon these changes.

Material and methods

A standard thermic trauma (IIIrd—IVth degree, 5—10 per cent) was caused by adopted and already described method (5). The experimental animals (rabbits, breed "Velican", 2,8—3,3 kg b. w.) were divided into two groups: Ist burned untreated, and IInd — burned and treated with protein hydrolysate "Hydroprot" and hemodex both. The preparations were injected i. v. in the following terms and doses: immediately after burning — 10 ml/kg hydrolysate and 5 ml/kg hemodex, the same amounts on the 24th hour but on the 3rd day 10 ml/kg hydrolysate only. Serum total protein, its fractions and urea were determined in the following terms: initial, on the 1st, 6th and 24th hour, on the 3rd, 7th, 14th and 28th day. Total protein was estimated after biurete method, its fractions by using paper electrophoresis, and urea after diacetylmonoximone method with "Lahema" tests (CSSR). The results were statistically processed.

Results and discussion

One can see on table 1 that total serum protein decreases in any assessed terms after burn in untreated animals. This reduction is more significant and reliable on the 1st and 6th hour. These results are in unison with literature data available (1, 3, 4, 7). However, it is noteworthy that a relatively small-size burn (5—10 per cent) causes hypoproteinemia that does not restore till the end of the examination although it is not reliable when all the terms are concerned. We can, therefore, suggest that profound circumscribed burns induce protein metabolism changes which require a correction, too. This concept is confirmed by the data from the control group. The combined hydrolysate and hemodex treatment increases the total protein after burn if only for a relatively short period — first 3 terms. After the IIIrd day total protein levels in both groups are almost equal. It seems

logic if one has in mind that medication is rather short and in the acute stage of the illness the vascular permeability is increased and the preparations retain a shorter time in the circulation. It could be concluded that with a view to influence on protein metabolism the protein hydrolysate administration has to be longer than now used. The more so as means used in hypoproteinemia treatment are scanty and rather inefficient (6).

Concerning albumins analogous changes were observed (table 2). In any terms assessed they reduced, most sensitively and reliably on the 14th day after burn. Our data confirm literature ones as well as the fact that hypoproteinemia is mainly determined by hypoalbuminemia. The reasons for reduced blood total protein and albumins both are completely enough explained in the literature as well as in our previous investigations. In general, they can be summarized as follows: protein loss from burned surface by plasmorrhage (both internal and external); protein outflow from circulatory bed into the tissues because of the increased permeability (not only at the place of burn); tissue protein destruction in burn area, first and mainly due to high temperature; protein metabolism disorders at remote places because of an increased proteolytic activity in these tissues; suppressed liver protein synthesis function, etc. (1, 3, 4, 5).

The results are similar to that ones when hydrolysate and hemodex treatment of burned animals is concerned. Albumins do not only reduce but also increase slightly in the first 3 terms. This confirms the statement that total protein changes are directed by albumin ones. In next terms the levels are almost equal in favour of that after therapeutic influence. Therefore, we are allowed to recommend a longer treatment in these cases.

Globulin fractions do not change significantly in spite of the tendency towards an increase in most terms. That is why their data are not presented in the paper. This fact is due mainly to alpha- and beta-globulin levels because gamma-globulins, in general, are more reduced. It is, therefore, especially valuable that both protein hydrolysate and hemodex causes a gamma-globulin increase in about 60 per cent of the terms followed. The importance of this fact is more evident when one has in mind that these preparations reduce increased alpha- and beta-globulins because the latter are considered an expression of tissue damage and infectious-toxic wound influence (2). Urea changes reflect also protein metabolism ones and protein hydrolysate uptake by the organism (6, 9). In any terms assessed serum urea levels increase after burn (table 3). They are the highest and most significant at the 6th and 24th hour. This is completely logic when we bear in mind that early after thermic trauma protein catabolism is most intensive which results in an urea increase. This circumstance effects undoubtedly on whole amino acid assimilation as introduced by means of protein hydrolysate. However, despite urea increase in protein hydrolysate and hemodex treated animals, too, it has to be noted that urea levels are lower that of control burned-untreated animals. It is evident that protein hydrolysate introduction in burns with a view to decrease the catabolism and to stimulate the anabolism of proteins is rather advisable. It is necessary to specify the dosage, terms of introductions and duration of treatment.

According to our study we can conclude that unpreventable hypoproteinemia and dysproteinemia after profound burn is favourably influenced by protein hydrolysate and hemodex treatment.

Table 1
Total serum protein after burn and its treatment ($\bar{x} \pm Sx$)

Groups	Number	1 st hour	6 th hour	24 th hour	3 rd day	7 th day	14 th day	28 th day
Controls	24	6.16±0.12	6.16±0.12	6.16±0.12	6.16±0.12	6.16±0.12	6.16±0.12	6.16±0.12
Burned and untreated	9	5.58±0.34+	5.64±0.22+	5.74±0.21	5.86±0.31	5.71±0.26	5.76±0.25	5.87±0.18
Burned treated with prot. hydrox. a. hemodex	8	6.27±0.35	5.77±0.31	5.99±0.38	5.71±0.19	5.76±0.4	5.74±0.44	5.86±0.42

Sign+ means $p < 0.05$ as compared to the controls

Table 2
Albumin changes after burn and its treatment ($\bar{x} \pm Sx$)

Groups	Number	1 st hour	6 th hour	24 th hour	3 rd day	7 th day	14 th day	28 th day
Controls	19	58.48±2.41	58.48±2.41	58.48±2.41	58.48±2.41	58.48±2.41	58.48±2.41	58.48±2.41
Burned and untreated	7	57.44±3.21	61.0±3.30	57.65±3.07	57.92±3.76	51.82±2.89	49.26±2.76+	56.92±1.13
Burned treated with prot. hydrox. a. hemodex	7	59.50±3.0	64.73±3.75	58.81±3.59	54.27±4.48	52.26±3.61	49.79±5.01	57.79±1.74

Sign + means $p < 0.05$ as compared to the controls

Table 3
Serum urea after profound circumscribed burn and its treatment ($\bar{x} \pm Sx$)

Groups	Number	1 st hour	6 th hour	24 th hour	3 rd day	7 th day	14 th day	28 th day
Controls	24	26.48±1.97	26.48±1.97	26.48±1.97	26.48±1.97	26.48±1.97	26.48±1.97	26.48±1.97
Burned and untreated	11	29.19±3.47	44.63±3.50++	50.33±4.37++	30.14±5.72	28.42±3.02	28.84±3.73	32.74±4.13
Burned treated with prot. hydrox. a. hemodex	8	27.99±4.15	52.51±7.16++	48.33±6.96++	29.44±4.11	29.64±4.60	27.48±4.0	27.76±4.24

Sign+ means $p < 0.001$ as compared to the controls

REFERENCES

1. Арьев, Т. Я. Ожоги и отморожения. Л., Медицина, 1971, 285 с. — 2. Герасимова, Л. Г. Дисс. канд. М., 1966. — 3. Заец, Т. Л. В: Ожоговая болезнь. Киев, Здоров'я, 1966, 78—81, 245. — 4. Клячкин, Л. М., В. М. Пинчук. Ожоговая болезнь. Л., Медицина, 1969, 479 с. — 5. Козарев, И., И. Попдимитров. *Хирургия*, 1978, № 3, 181—185. — 6. Кремер, Ю. Н., С. П. Витолина, О. Я. Пуле, Франк. *Вопр. питан.*, 1972, № 1, 47—51. — 7. Похно, М. М., О. Г. Печерская. — В: Ожоговая болезнь, Киев, Здоров'я, 1966, 72—75, 245. — 8. Waiteister, R., et al. *Scand. J. Plast. Reconstr. Surg.*, 13, 1979, No 1, 185—188. — 9. Kagan, R., et al. *Ann. Surg.*, 195, 1982, No 1, 70—74.

МОЧЕВИНА И СЫВОРОТОЧНЫЕ БЕЛКИ ПРИ КОМБИНИРОВАННОМ ВОЗДЕЙСТВИИ ПОСЛЕ ОГРАНИЧЕННОГО ГЛУБОКОГО ОЖОГА

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РЕЗЮМЕ

Произведен ожог III б и IV степени 5—10 % поверхности тела кроликов. Подопытным животным внутривенно вводился белковый гидролизат (10 мл/кг) и гемодекс (5 мл/кг) сразу после ожога, 24 часа после него (такая же доза) и в третий день (10 мл/кг) гидролизата. Простеживались: общий белок, сывороточные белковые фракции и мочевины в первом, шестом и двадцать четвертом часу в третий, седьмой, четырнадцатый и двадцать восьмой день после ожога.

Установлено, что после ожога наступает диспротеинемия — уменьшается количество общего белка и альбуминов в указанные сроки, увеличиваются глобулиновые фракции, главным образом — альфа- и бета-глобулины. О нарушении белкового обмена говорит и увеличенное количество мочевины.

При введении в организм обожженных животных гидролизата и гемодекса общий белок и альбумины увеличиваются, а глобулины уменьшаются в ближайшие сроки. Уменьшается и количество мочевины.

Авторы делают вывод, что белковый гидролизат и гемодекс оказывают благоприятное влияние на нарушения белкового обмена после ожога.