AD NUMBER

AD835850

NEW LIMITATION CHANGE

TO

Approved for public release, distribution unlimited

FROM

Distribution authorized to U.S. Gov't. agencies and their contractors; Critical Technology; 20 NOV 1964. Other requests shall be referred to Army Biological Laboratory, Attn: Technical Releases Branch [TID], Fort Detrick, MD 21701.

AUTHORITY

smufd, d/a ltr 14 Feb 1972

TRANSLATION NO. 1227

DATE: 20 [illegible] 1964

ON THE DISTRIBUTION OF NITROGEN SUBSTANCES IN URINE

[Following is a translation of the final pages of an article by Franz Erben, appearing in the Zeitschrift für Heilkunde (Journal of Therapeutics), Vol 25, 1904, pp 93-95.]

Final Considerations

After the results of each experimental series and the anomalies found for each disease have been summarized above, it remains now to report and to interpret the common [factors] on which all studied cases of infectious diseases are based.

1. In all cases of elevated temperature we find an increase in nitrogen elimination which is chiefly due to an increase in urea; Huppert has pointed out this fact a long time ago.

This increase was higher with higher temperature and it was found to be more pronounced in acutely rising fevers (measles, scarlet fever) than in more moderate ones.

2. The increase in nitrogen is not uniformly distributed between both groups of precipitable and non-precipitable nitrogen, but a sharper increase in precipitable nitrogen and a less pronounced increase in non-precipitable nitrogen is regularly found during the fever and, at times, during the first fever-free days. The rate at which the ratio of precipitable to non-precipitable nitrogen returns to normal after termination of the fever depends perhaps partly on the disease processes causing the fever [and] partly also on the rate of the fever decrease.

3. The increase in ammonia is in all cases parallel to that of the nitrogen and contributes always most [decisively] to the increase in precipitable nitrogen. Its increase may well be caused by another factor than the increase of intermediate albumin degradation products.

4. The uric acid nitrogen, the precipitable and non-precipitable remaining nitrogen can briefly be described as nitrogen of intermediate albumin degradation products.

In fever cases we find a regular increase in this [type of nitrogen]. The degree of this increase differs in the individual diseases.

There is also a difference in the portion which has increased more, the nitrogen precipitable with phosphotungstic acid (xanthine base N, etc.) or the non-precipitable one (amino acid N, etc.). For instance, in the case of measles, both portions are equally increased (time-wise, the precipitable remaining nitrogen reaches its highest peak earlier than the non-precipitable one). Both portions behave similarly in cases of Angina crouposa and Varicella, whereas in the case of scarlet fever, the increase in non-precipitable remaining nitrogen, during and after lysis, which consists mainly of amidoacid nitrogen, is larger by far than that of the precipitable remaining nitrogen; this predominance is not as pronounced in the case of typhoid fever.

An attempt will be made below to explain the cause for this behavior in the cases of scarlet fever and typhoid fever.

5. An infection center in the organism brings about the same changes in the nitrogen distribution in urine as does a fever. It is found also in this case* [that there is] a relative increase in precipitable nitrogen and an increase in ammonia and intermediate albumin degradation products, as well as more pronounced increase, in contrast to fever cases, of the remaining nitrogen which cannot be precipitated with phosphotungstic acid (amidoacid nitrogen).

A third reason for the same circumstances is provided by the resorption of lymphatic tissue.

This is probably a proof for the tremendous increase in non-precipitable remaining nitrogen (amino acid nitrogen, etc.), in the first afebrile days of the studied typhoid case, which is otherwise inexplicable. I believe that this is a reason for the pronounced increase in non-precipitable nitrogen (amino acid N) over that of the precipitable nitrogen, particularly in cases of scarlet fever. After all, the main elimination of the amino acid nitrogen coincides also in this case with the regression of the lymph glands. However, also in this type of tissue resorption (as in the case of suppuration), in contrast to fever cases, a more pronounced increase in non-precipitable remaining nitrogen must be noted.

Whereas tissue suppuration and tissue resorption -- both may be termed as histolysis -- are identical for the chemical processes of the nitrogen secreted in the urine (even if these processes have been determined by a rough method), or whereas their identity or similarity appear to be comprehensible, the similar distribution of nitrogen-containing urea products in [cases of] histolysis and fever is not as easily recognized as being caused by a factor common to both processes. However, if the nitrogen transformation during fever is more closely studied, it is found that, in all investigated cases, much more nitrogen is eliminated by the feverish organism than is the case when no fever prevails and the same food is consumed.

Thus, at any rate, there existed a negative nitrogen balance and the increase in N-elimination is caused by used up albumin of the body. There occurs an autophagy during fever. Von Jaksch (1) found an increase in amido acid N (and precipitable N) in urine, particularly in cases of Diabetes mellitus; Halpern (2) found [such increase] in cases of inanition and carcinom, that is in processes occurring in parallel to exquisite autophagy. Of course, it is possible to prove this increase when the degradation of body albumin has reached somewhat larger proportions. It will not be possible to ascertain that a small increase constitutes an increase.

All this seems to indicate that the following holds: when body albumin decomposes, either by a local process (histolysis) or a non-localized process (autophagy), there occurs an increased elimination of intermediate albumin degradation products in urine.

Literature References

1. von Jaksch, Zeitschrift fur klinische Medizin (Journal for Clinical Medicine), Vol 50, 1903, page 221.

2. Halpern, Zeitschrift fur klinische Medizin (Journal for Clinical Medicine), Vol 50.