EXCHANGES OF NUCLEOPROTEINS AND NUCLEIC ACIDS IN THE COURSE OF BOTKIN'S DISEASE

A. V. Andryushechkina and L. P. Nikolaeva

From the Department of Biological Chemistry (Chairman: Prof. N. N. Ivanovsky) Saratov State Medical Institute

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In a previous communication [1], we noted the marked lowering of nucleoproteins (NP) and of nucleic acids (NA) in the liver of humans dying as a result of acute hepatic dystrophy. In the present investigation we determined the NP and NA content of the blood in normal healthy humans (donors) and in those ill with Botkins disease.

The determination of NP and NA in the blood was done according to the method of O. P. Chepinoga, E. B. Skvirskaya and L. P. Rukina [4]; the quantity of NP and NA was judged by the phosphorus determined by the colorimetric method (Table 1).

Simultaneously we investigated certain end products of the metabolism of NP and NA in the urine of humans ill with Botkin's disease; the uric acids by the titrating method, and the purine urinary products by precipitating them in the form of copper salts. The number of purine bonds was judged according to the nitrogen determined by the Kjeldahl method.*

TABLE 1

Phosphorus Content of Nucleoproteins and Nucleic Acids in Blood of Healthy and Ill (Botkin's Disease) Humans

Material studied	Nucleo proteins	Ribonucleic acid	Desoxyribonucleic acid
	Phosphorus (in mg %)		
Healthy blood (20 samples)	3—9	2.5—5.5	0.8—1.3
Blood from patients with Botkin's disease (6 cases)	3—5	1.8—2.4	Traces

The data obtained, as shown in Table 1, demonstrate that in Botkin's disease the NP and NA levels are 1.5 - 2 times lower than normal: the blood of patients with Botkin's disease averages 4 mg %, while in the healthy, 6 mg % of NP phosphorus.

We analyzed the urine of patients with Botkin's disease and the results, shown in Table 2, demonstrate that the daily output of purine compounds is increased 1.7 times: on the average the urine of patients contains 0.487 g of purine nitrogen against 0.28 g in the healthy control humans (per day).

The fall of the daily purine output in the urine down to 0.188 g was seen only in one patient who had extreme hepatodystrophy, and later died.

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TABLE 2

Purine Nitrogen Content in the Urine of Healthy Humans and Those Ill with Botkin's Disease

Material studied	Nitrogen of all purine compounds (in g)	% purines, oxidized into uric acids
Urine of healthy:		
a) mixed food	0.235—0.330	78—85
b) low purine diet	0.070—0.210	95—96
Urine of Botkin's disease patients (22 cases)	0.331—0.527	10.8—20.5

The data in Table 2 convince us that the process of oxidizing the purine compounds into uric acids is retarded 4-10 fold in cases of Botkin's disease as compared with the healthy organism.

Thus, the quantity of uric acids excreted decreases most sharply at the height of the disease. Recovery is accompanied by a rise in the uric acid output. In some cases the uric acid output reaches almost normal (Table 3).

TABLE 3

The Content of Uric Acids in Human Urine of Patients With Botkin's Disease as Related to the Intensity of the Disease Process

Patient	Diagnosis and General Condition	Urinary acids, daily output (in g)
D	Suspicion of sub-acute dystrophy	0.15
	improvement	0.34
	clinical recovery	0.59
G	Severe hepatodystrophy with coma	0.18
	improvement	0.37
K	Botkin's disease, severe	0.17
	improvement of condition	0.48
B	Botkin's disease	0.26
	improvement of condition	0.54
N	Botkin's disease, severe	0.11
	improvement	0.35
G	Botkin's disease	0.34
	improvement	0.50

The results obtained testify to the diminution of the oxidation of purine to uric acids as Botkin's disease appears, this agreeing with results obtained by A. Ya. Myasnikov [3], L. S. Shvarts [6] and others on the diminution of oxidative-restorative processes in the course of Botkin's disease.

The disturbances in the oxidative-restorative processes seen in the course of Botkin's disease have also been seen by a number of other investigators who believe that the process of fatty acid oxidation is inhibited, [2], the evacuation of urinary oxygen is increased [6] and there occurs a fall in the intensity of oxidative phosphorylation [8].

The sharp drop in the NP and NA content of the liver in patients dying of acute hepatic dystrophy, the diminished content of NP and NA in the blood of patients ill with Botkin's disease, and the increase in the amount of urinary nitrogen resulting from the presence of purines, all testify to the disturbed synthesis of NP and NA during the course of this disease.

It might be supposed that the upset in the metabolism of NP and NA is one of the reasons underlying the diminution of the oxidative-restorative processes in the tissues of patients suffering from Botkin's disease.

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* In Russian.