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ASSAY OF THE ANTIPROTEASIC ACTIVITY OF SERUM IN DIFFERENT ACUTE AND CHRONIC DISEASES

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Neutral cellular proteases present one of the important selfdefence mechanisms of the organism (1 a, 10, 17). Most of the enzymes implicated in limited proteolytic systems, belong to the great family of serinproteases. Their activity is determined by the presence of serin in their active centre (23). The inhibition of their activity by antiproteases is also related to this structural feature (8, 9, 14). For laboratory investigations serum antiproteases are the most accessible ones (5). Serum contains several proteins with antiproteasic effect. These are alpha-1-antitrypsin, alpha-1-antichymotrypsin, inter-alpha trypsin inhibitor and alpha-2-macroglobulin. The most important one is alpha-1-antitrypsin (7, 16), which constitutes the major inhibitor of plasma protelytic enzymes, representing 90% of serum antiproteasic activity (7).

Normal human serum contains 2—6 g/l alpha-1-antitrypsin, which is synthetized in the liver (1, 6, 5, 4). With knowledge of the important pathogenetic role which proteases have in inducing morphological and functional lesions, the assay of serum antiproteasic activity presents great interest (2). As a first orientation, the performance of standardized proteinogram may be very useful, which shows the presence, decrease or absence of alpha-1-bands. The amount of alpha-1-antitrypsin can be determined by radial immunodiffusion (13), or by immunoassay, respectively (12).

Most useful for for clinical purposes is the determination of serum total antiproteasic activity, which means the capacity of a definite serum volume to neutralize the proteolytic effect of a definite amount of trypsin.

Material and method

We have determined serum total antiproteasic activity in 584 subjects, of which 84 were healthy persons and the rest of 500 were patients suffering from liver diseases (chronic hepatitis, cirrhosis, hepatic fibrosis, hepatoma), infectious states (bronchopneumonia, cholecystitis, rheumatoid polyarthritis, glomerulonephritis, vasculitis, respiratory viroses, herpes simplex), malignant processes (Hodgkin's disease, prostate tumours, malignant lymphomas, multiple myeloma), ischaemic cardiopathy and coeliac disease. The sera investigated were obtained from the material of the Clinical Central Laboratory, Tîrgu-Mureş.

The method we used is based on casein as substrate (11). This is incubated with trypsin in absence, respectively in presence of serum. The activity of trypsin is partially inhibited by serum antiproteases. The difference between the measured activity of trypsin upon casein

in the two tests, indicates the amount of serum antiproteases, expressed in trypsin units. The mean value in healthy individuals is $1,90 \pm 0,25$ U/ml which corresponds to the value found by other authors.

Results and discussions

The results obtained are comprised in Table I.

Table I

Serum antiproteasic activity (SAA) in different diseases.

Nr.	Disease	N.	SAA U/ml	SD	Significance
1.	Chr. hepatitis	72	2,04	$\pm 0,64$	$p<0,001$
2.	Cirrhosis	40	1,96	$\pm 0,84$	$0,7>p>0,6$
3.	Hepatic fibrosis	12	2,05	$\pm 0,65$	$0,2>p>0,1$
4.	Hepatoma	9	2,53	$\pm 0,78$	$p<0,001$
5.	Bronchopneumonia	17	2,14	$\pm 0,34$	$0,1>p>0,01$
6.	Cholecystitis	37	1,92	$\pm 0,63$	$0,9>p>0,8$
7.	Rheumatoid polyarthritis	42	2,20	$\pm 0,61$	$p<0,001$
8.	Glomerulonephritis	21	2,59	$\pm 0,67$	$p<0,001$
9.	Vasculitis	50	1,53	$\pm 1,14$	$0,02>p>0,01$
10.	Respiratory viroses	56	1,98	$\pm 0,57$	$0,4>p>0,3$
11.	Herpes simplex	10	2,22	$\pm 0,49$	$0,1>p>0,01$
12.	Hodgkin's disease	14	2,42	$\pm 0,82$	$p<0,001$
13.	Prostate tumors	6	2,60	$\pm 1,09$	$p<0,001$
14.	Malignant lymphomas	13	2,16	$\pm 0,75$	$0,05>p>0,02$
15.	Multiple myeloma	29	2,07	$\pm 0,56$	$0,1>p>0,05$
16.	Ischaemic cardiopathy	56	1,95	$\pm 0,70$	$0,7>p>0,6$
17.	Coeliac disease	19	2,23	$\pm 0,76$	$0,1>p>0,01$

Considering the changes of serum antiproteasic activity (SAA) in different diseases, the following may be observed:

1. In the group of acute inflammatory diseases, SAA was significantly increased in bronchopneumonia, acute glomerulonephritis, rheumatoid polyarthritis and herpes simplex.

2. In the group of chronic diseases, SAA was significantly increased in chronic hepatitis, hepatic cirrhosis and coeliac disease.

3. Concerning malignant tumours, SAA was significantly increased in hepatoma, Hodgkin's disease, prostate tumours, malignant lymphomas.

4. Significantly decreased values of SAA were detected in the group of 50 subjects suffering from vasculitis.

5. SAA was practically unchanged in subjects with acute respiratory viroses, acute cholecystitis, ischaemic cardiopathy, cirrhosis and multiple myeloma.

6. In some cases increased values of SAA were found in patients with microvasculitis. These may be explained by a sufficient compensation of proteolysis, with satisfying chances for regeneration.

7. Expressed decrease of SAA, paralleled with the presence of grave clinical symptoms, points to the existence of microvascular inflammatory process with reduced chances for regeneration.

8. Presenting well appreciable changes in both directions, the assay of SAA with this simple and rapid method, provides valuable information for diagnosis and prognosis in clinical practice.

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