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Assessment for the value of serum lactic dehydrogenase in protein-energy malnutrition

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With 1 figure and 1 table

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Measurement of enzyme activities in biological fluids offered an efficient and significant tool for detecting the biochemical processes carrying in the body. The microanalytical methods for enzyme assays became increasingly useful in diagnostic and prognostic evaluations. During the last decade, much enzyme assays have been used as a reliable index for cell degeneration in various affected organs (4).

Serum lactic dehydrogenase (LDH) enzyme received much importance as a diagnostic test for cardiac and hepatic damages (4, 10). Recently, *Markert* and *Moeller* (11) demonstrated that LDH exists in distinct molecular forms which could be separated by electrophoretic techniques. Each molecular form or isoenzyme is present in greater amounts in certain tissue; while the electrophoretic form designated as LDH_1 is predominating in extracts of cardiac muscle, LDH_5 predominates in extracts of liver and skeletal muscles.

Serum LDH activities have been reported by several authors (2, 3, 17) to be significantly increased in cases suffering from protein-energy malnutrition (PEM).

The aim of the present work is basically to assess the value of serum LDH isoenzyme fractionation in relation to various other serum enzymes, namely, glutamic-pyruvic transaminase (GPT), glutamic-oxalacetic transaminase (GOT), creatine phosphokinase (CPK) and hydroxybutyrate dehydrogenase (HBDH) in PEM. These enzymes were selected since they are known to be altered in sera due to muscle and liver cells damages in several pathological states. Such studies may lead to a better understanding for the nature of hepatic and cardiac affections in PEM.

Material and methods

Sixteen cases suffering from Kwashiorkor of the mild-moderate severity according to *Jelliffe* classification (8), within an age range between 12 and 36 months, were included. A control group of twelve normal children of the same age range and socioeconomic status were also included for comparison. Fasting blood samples were collected and sera were separated. Enzyme assays were carried out on the same day of sampling.

Serum GPT and GOT were assayed according to the colorimetric method described by *Frankel* and *Reitman* (6); CPK essentially according to the method of *Nuttal* and *Wedin* (14); HBDH by *Rosalki* method (16) and serum total LDH was determined according to the colorimetric method described by *King* and *Wootton* (9). Agar gel electrophoresis was applied for separation of serum LDH isoenzymes according to *Weine* (20) technique. LDH isoenzyme fractions were evaluated semiquantitatively by visual mean according to its intensities.

The results obtained were statistically analyzed according to the *Student* t test.

Results and discussion

The results obtained showed that total serum LDH activities are higher in all PEM cases studied if compared with the control group. This finding agreed well with those reported by several authors (2, 3, 17) and this suggested that the pathological lesion present in these cases is either cardiac, hepatic, or skeletal muscle in nature.

Several techniques were tried to differentiate the organ specificity of LDH isoenzymes, amongst these criteria is the heat stability index for heart LDH devised by *Wroblewski* and *Gregory* (22). The first approach for the evaluation of serum LDH fractionation was given by *Hill* (7) and stimulated a progress in this respect. Serum-LDH-isoenzyme pattern in cases suffering from PEM showed that LDH_2 and LDH_3 were relatively very intense; LDH_1 intense; LDH_4, moderate and complete absence of LDH_5 in all PEM cases studied. This picture denotes that heart LDH_1 isoenzyme did not vary significantly from normal patterns. In this connection, *Abdin* (1) has reported that electrocardiographic studies failed to reveal any cardiac changes in PEM cases although serum total LDH activities were high and the heat-stability index was either normal or below

Table 1. Blood serum enzyme activities in normals and PEM cases (units mU/ml).

Group	GPT	GOT	CPK	HBDH	Total	LDH 1	2	3	4	5
Normals (12)*										
Range	4–15	8–20	10–36	65–125	65–155					
Mean	8	11	23	91	105	+	++	++	+	+
S.D. ±	2.88	3.30	6.88	16.80	25.60					
S.E. ±	0.84	0.95	2.00	4.86	7.42					
PEM (16)*										
Range	8–33	12–52	32–50	79–288	121–248					
Mean	19	33	42	189	191	+	+++	+++	+	−
S.D. ±	8.78	14.49	5.48	59.60	37.80					
S.E. ±	2.18	3.74	1.38	14.90	9.40					
P <	0.01	0.01	0.01	0.01	0.01					

* Number of cases studied.
+++ = Very intense, ++ = intense, + = moderate, − = absent.

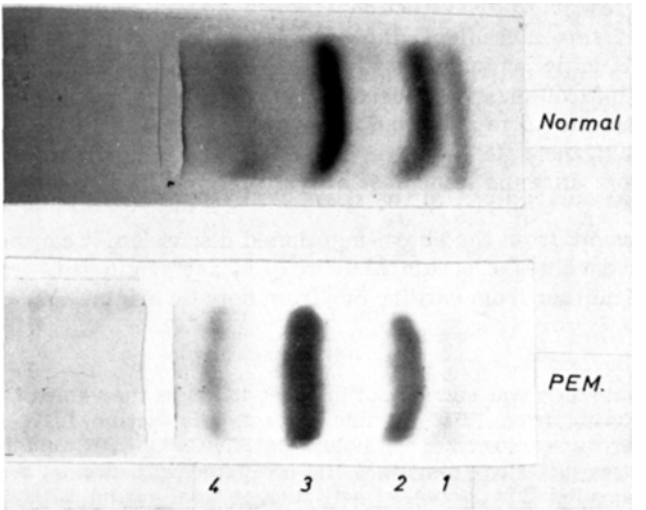

Fig. 1. Serum-LDH-isoenzyme pattern in normals and PEM cases.

the normal ranges. Thus our findings offer further support for the absence of cardiac LDH changes in PEM cases.

Serum LDH isoenzyme pattern met with PEM in the present study is similar to that reported by *Nerenberg* (13) in cases suffering from anaemia, rheumatic and muscle dystrophy. *Sandstead* (18) has reported that various degrees of megaloblastic changes in the bone marrow are seen in PEM. Skeletal muscles which are reported to contain large amounts of LDH (12) may be the source for this elevated serum LDH in PEM, especially that muscle wasting is a characteristic symptom in PEM (19).

Serum GPT showed a mean value slightly higher in PEM when compared with normal and 50 per cent of the PEM cases studied showed values below the upper normal range. This indicates that tendency for increased activities is higher for GOT than GPT. In this connection, *Rosalki* (15) has reported that serum GOT: GPT ratio is below one indicates a state of hepatic cirrhosis. In the present study, this ratio amounted to about 1.80 which may denote that PEM cases did not suffer from liver cirrhosis. This suggestion was supported by the histopathological studies carried out by *El-Nabawi* (5) on the liver of PEM cases.

Serum CPK activities are reported to be significantly increased in patients suffering from cardiac damage and skeletal muscle dystrophy (21). The relatively increased values of serum CPK activities in PEM are suggestive to be of skeletal muscle origin rather than of cardiac since serum-LDH-isoenzyme patterns could not reveal any cardiac changes in these cases.

Serum HBDH has been reported to be more specific than total serum LDH for the diagnosis of myocardial affections (15) and in the present study there is a good agreement between HBDH and LDH.

In the present study, although HBDH showed higher activities than normals, yet this increment did not go parallel with LDH_1 isoenzyme. HBDH: LDH ratio which is used for the differential diagnosis between myocardial infarction and other diseases, especially progressive muscular dystrophy, amounted to about 0.6 in normals and 1.0 in PEM cases studied. *Elliot* and *Wilkinson* (4) have reported that this ratio increased in cases suffering from anaemia and muscle dystrophy while it is normal in liver cirrhosis.

In conclusion, from the above-mentioned discussion, it can be suggested that increased values of serum LDH in PEM are originating from skeletal muscles and neither from cardiac nor from hepatic origin.

Summary

The present study was carried out in order to assess the value of serum LDH in cases suffering from PEM. In this respect, total serum LDH and its isoenzyme pattern in conjunction with serum GPT, GOT, CPK, and HBDH were assayed in cases suffering from PEM and compared with normal control group. The study revealed that increased activities of total serum LDH in PEM are neither of cardiac nor of hepatic origins. The activities and significance of other enzymes studied suggested that skeletal muscles are the main sources for these increased activities of serum LDH in PEM. The study offered a strong evidence for discrimination of liver cirrhosis in PEM.

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