Observation and identification of lactate dehydrogenase anomaly in a postburn patient

Z-J Liu, Y Zhang, X-B Zhang, X Yang

Objective: Lactate dehydrogenase (LDH) anomaly is one of the macroenzymes. Macroenzymes are enzymes in serum that have formed high molecular mass complexes, either by self polymerisation or by association with other serum components. The aim of this study was to identify the properties of LDH anomaly and observe the changes from admission to discharge in a postburn patient with LDH anomaly in his serum.

Methods: LDH isoenzymes of the serum were electrophoretically fractionated with tereylene cellulose acetate supporting media; LDH anomaly was identified by counter immunoelectrophoresis.

Results: An abnormal LDH-4 band and an extra band on the cathode of LDH-5 were observed in the serum of this patient and were found to be part of an LDH-IgG complex. As his symptoms improved, the patient’s LDH anomaly gradually disappeared. The appearance and disappearance of the anomaly seemed to be related to the progression of the patient’s burns.

Conclusion: In clinical practice, it is important to keep in mind the possibility of an LDH anomaly in patients when the LDH level is abnormally high or does not seem to be related to the clinical state. Early discovery of an LDH anomaly in a patient’s serum may be useful for diagnosis and treatment.

Methods

Instruments and reagents

The Beckman Synchro CX-7 automatic biochemistry analyser (Beckman, USA); Paragon electrophoretic system and appraise densitometer system (Beckman Instruments, Fullerton, CA); and anti-IgG antiserum, anti-IgA antiserum, anti-IgM antiserum were from Beckman. The tereylene cellulose acetate membrane was from the Sijing Biochemical Material Factory, Zhejiang, PRC; and the LDH kit reagent was from Shanghai Long March Company, PRC.

Results

Figure 1 shows the densitometric patterns of LDH isoenzymes in the serum of the postburn patient. Figure 2 shows the result of the counter immunoelectrophoresis. Table 1 shows

Abbreviations: AST, aspartate aminotransferase; CK, creatine kinase; LDH, lactate dehydrogenase; HBDH, α-hydroxybutyrate dehydrogenase; CK-MB, creatine kinase MB isoenzyme; LDH-IgG, lactate dehydrogenase immunoglobulin complex.
the changes in serum enzyme activities of the postburn patient; table 2 shows the LDH activity and isoenzymes of the patient from the time of admission to discharge.

**DISCUSSION**

The LDH-immunoglobulin complex has been reported in various diseases. We describe circulating LDH-IgG complex in a postburn patient; the complex appeared to be related to the progression of burn in this case.

The patient was admitted to our hospital because of 20% burns and his laboratory findings on admission showed high levels of LDH activity (309 U/l, normal 110–240 U/l). After seven days the LDH activity increased to 407 U/l and LDH isoenzyme analysis showed abnormal bands (LDH-1, 11.8%; LDH-2, 15.8%; LDH-3, 17.6%; LDH-4, 42.5%; LDH-5, 5.4%; and extra band, 6.8%).

A broad band around the region of the LDH-4 isoenzyme characterised the patient’s LDH isoenzyme pattern, with the apparent appearance of an extra band on the negative side of LDH-5 (fig 1). In fig 2 it can be seen how we identified the extra band. The precipitate was observed only in the lane for anti-IgG antiserum and the patient’s serum; in the other lanes no precipitate was seen. In a non-substrate test, we used a substrate without lactate but with NAD, methyl thiazolyl tetrazolium, and phenazine methosulphate. After the staining procedure, no colour band was observed on the membrane. Therefore there was no alcohol dehydrogenase present in the sample. When antibodies to albumin were used no precipitate was observed. The patient’s erythrocyte LDH isoenzyme patterns were also normal, therefore it was not a genetic mutation that caused this anomaly because a genetic mutation changes erythrocyte and serum LDH isoenzyme patterns. These results suggest that the anomaly was an LDH-IgG complex. With his symptoms improved, the patient’s serum LDH activity and isoenzymes gradually returned to normal. When he was discharged from hospital, his serum LDH level had normalised to 165 U/l, and LDH isoenzymes were almost within the normal limit (LDH-1, 20.8%; LDH-2, 28.0%; LDH-3, 27.8%; LDH-4, 19.1%; LDH-5, 4.2% and no extra band). The LDH-immunoglobulin complex might be associated with the pathophysiology of this case.

From table 1 we can see that the activities of myocardial enzymes, AST, LDH, HBDH, CK, CK-MB, and MB/CK were all increased at the time of admission, indicating cardiac muscle was injured. Two weeks after admission, the activities of LDH, HBDH, and AST decreased to nearly normal ranges. Although CK was still high, the MB/CK ratio (5%) was less than the standard for myocardial injury (6%). Therefore, the increased CK activity was caused mainly by muscle injury after the burn. If we consider the changes in LDH and isoenzyme in the first week after admission: LDH activity was at its highest (407 U/l),
LDH-4 was 42.5%, and LDH-5’ appeared (6.8%). In the second week, LDH activity returned to normal, LDH-4 was 38.4%, and LDH-5’ was 4%. In the third week, LDH activity was 165 U/l, LDH-4 was 19.1%, and LDH-5’ disappeared. These results indicate that it takes some time for the abnormal band of LDH to return to normal unlike LDH activity. The appearance and disappearance of abnormal LDH bands may be relative to the progression of the patient.

There have been many reports on abnormal isoenzyme patterns of plasma LDH in various diseases. However, there are few reports on LDH-immunoglobulin complexes in postburn patients. Further studies are required to determine the precise significance of the LDH anomaly in the serum of these patients. In clinical practice, it is important to keep in mind the possibility of an LDH anomaly in patients when the LDH level is abnormally high or does not seem to be related to the clinical state.

Authors’ affiliations
Z-J Liu, Y Zhang, X-B Zhang, X Yang, Department of Laboratory Medicine, Southwest Hospital, Third Military Medical University, Chongqing 400038 Peoples’ Republic of China

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