Plasma Lactic Dehydrogenase and Phosphohexose Isomerase in Leukemia

By M. C. Blanchaer, P. T. Green, J. P. Maclean and M. J. Hollenberg

WIDE ACCEPTANCE of serum glutamic oxaloacetic transaminase estimations1 for the detection of myocardial infarction has stimulated interest in the possible diagnostic uses of other plasma enzymes, including lactic acid dehydrogenase (LD) and phosphohexose isomerase (PHI). Both of these enzymes play a role in the intracellular process of glycolysis.2 Abnormally elevated plasma levels have been found in hepatocellular disease,3,4 myocardial infarction3 and neoplastic disease,3,6 including certain types of leukemia. Plasma LD was found to be raised in acute stem cell leukemia3 and chronic myelocytic leukemia3,5 but was normal in two of the three cases of chronic lymphocytic leukemia5 reported. Israels and Delory7 found that the plasma PHI was increased in chronic myelocytic leukemia, but not in the chronic lymphocytic type nor in the leukocytosis of infections.

In the present work the plasma LD has been examined in various types of leukemia in relation to the plasma PHI levels, the response to treatment and the presence of hemolytic disease.

METHODS

Lactic dehydrogenase and phosphohexose isomerase5 were measured in recentrifuged heparinized plasma. During the collection of blood and in the subsequent manipulations precautions were taken to minimize destruction of the enzyme-rich cells. Venous blood was withdrawn slowly through a large gauge needle and centrifuged at 4 C. The plasma was recentrifuged at 4 C. to remove traces of red and white cells. Such specimens were free of visible hemolysis and no leimkocytes or cellular debris were seen in smears. A further centrifugation yielded no microscopically detectable sediment.

The principle of the LD method is similar to that of the procedures used by previous workers,5,6 but the details differ sufficiently to warrant description. The assay depends on the direct proportionality between the rate of oxidation of reduced diphosphopyridine nucleotide (DPNH2) by pyruvate and the amount of lactic dehydrogenase activity present. The rate of DPNH2 oxidation is followed by the decrease in optical density measured at 340 nm in a Beckman DU spectrophotometer. In the test cuvette are placed 1.0 ml. of 0.1 M phosphate buffer pH 7.4, 0.38 micromoles DPNH2, 0.2 ml. plasma and water to bring the volume to 2.8 ml. The corresponding blank cuvette contains the same constituents except that DPNH2 is omitted. After bringing the reactants to 37 C. and recording the initial optical density reading, the reaction is started by adding 0.2 ml. of 3 mM sodium pyruvate. Readings are taken at half-minute intervals as the reaction proceeds.

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at 37 C. The unit of lactic dehydrogenase activity is based on the density change in the first minute and is expressed as micromoles DPNH₂ oxidized per milliliter of plasma per hour, assuming that a concentration of 1 micromole DPNH₂ per milliliter in a lightpath of 1 cm. has an optical density* of 6.3.

RESULTS

Normals. The normal range of plasma LD and PHI was established by analysis of specimens from 32 apparently healthy medical students and 34 older subjects judged to be healthy from a clinical history and physical examination. The values were independent of age and sex. The plasma LD levels had a mean level of 9.2 units with a standard deviation of ± 1.2. The mean of the plasma PHI values was 17.6 with a standard deviation of ± 4.3. Since the values had an approximately normal frequency distribution, it was assumed that 99 per cent of normal subjects’ enzyme levels would fall in the limits defined by three standard deviations above and below the mean. Thus, the upper limit of normal for the plasma LD was defined as 13 units and that of PHI, 31 units. In both the LD and PHI methods, replicate determinations agreed within 8 per cent.

An analysis of washed red cell suspensions confirmed Bodansky’s finding* that erythrocyte PHI activity is on the average 160 times greater than that of plasma. From this it was calculated that hemolysis sufficient to raise the plasma hemoglobin to 20 mg. per cent would increase the plasma PHI by only two units. In none of the specimens did the plasma hemoglobin exceed 15 mg. per cent, a level of oxyhemoglobin readily detected by eye, but usually confirmed by measurement. The range of LD activity in 41 red cell suspensions was 6 to 20 units per mg. hemoglobin. Hemolysis releasing 20 mg. per cent hemoglobin into the plasma would therefore raise its LD by only 1 to 4 units. It was concluded that hemolysis during and after collection of the specimens did not contribute significantly to the plasma LD and PHI levels. No estimate is available of the amount of these enzymes added to the plasma by leukocyte destruction during blood collection and plasma separation. However, it was felt to be small because of the precautions taken during the manipulations.

Leukemia. Approximately 350 LD and 310 PHI estimations were made on 30 patients with leukemia. The diagnosis was established in each case by appropriate blood and marrow studies. In the absence of bleeding, a rapidly dropping hemoglobin concentration resistant to transfusion was considered evidence of excessive hemolysis. In such cases the plasma hemochromogen (hemoglobin plus methemalbumin) level* usually fell in the normal range, although elevated values were found during the severe hemolytic episodes described below in patients 12, 19 and 20. An increased rate of erythrocyte destruction was confirmed in a number of patients by Cr⁵¹ red cell survival measurements. Table 1 summarizes the enzyme values found before treatment was begun.

Acute Myelocytic Leukemia. It may be seen that in eight of the nine patients with acute myelocytic leukemia the plasma LD and PHI were elevated, but not in patient 1 who had the aleukemic variety of the disease. With this
PLASMA LD AND PHI IN LEUKEMIA

Table 1.—Plasma Lactic Dehydrogenase (LD) and Phosphohexose Isomerase (Phi) Activity in Leukemia

<table>
<thead>
<tr>
<th>No.</th>
<th>Age &amp; Sex</th>
<th>W.B.C. x 10⁻³/cu.mm.</th>
<th>LD units</th>
<th>PHI units</th>
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<tr>
<td>Normals 32</td>
<td>9.2 ± 1.2 (S.D.)</td>
<td>17.6 ± 4.3 (S.D.)</td>
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<tr>
<td>1</td>
<td>31 M</td>
<td>6</td>
<td>10</td>
<td>5</td>
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<tr>
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<td>35</td>
</tr>
<tr>
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<td>34</td>
<td>39</td>
<td>35</td>
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<td>102</td>
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<td>37</td>
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<td>74 F</td>
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<td>48 M</td>
<td>436</td>
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<td>20</td>
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<td>Leukemia 30</td>
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<td>113</td>
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exception, the plasma LD levels were raised to at least twice the upper limit of normal. Some of the PHI values were markedly elevated, but in four of the nine subjects in this group (cases 3, 4, 5 and 7) the values were within 15 per cent of the upper limit of normal.

No relationship was detected between the enzyme levels and the leukocyte counts or the length of survival after diagnosis when the acute myelocytic leukemia patients were examined as a group. In contrast to this lack of correlation, serial determinations on six of the nine patients (2 to 7) indicated that changes in the enzyme levels in each individual usually reflected changes in the course of this disease. This is shown in figure 1A illustrating the findings in patient 2. After five months of progressive fatigue, this woman of 43 years presented with moderate hepato- and splenomegaly, petechiae, marked sternal tenderness, facial edema and a hemoglobin concentration of
6.7 Gm. per cent (period I). Two units of blood were administered together with a four-day course of 100 mg. 6-mercaptopurine (6-MP) per day. It may be seen that initially both plasma enzymes were grossly abnormal, but that the LD was elevated much more than the PHI. During and shortly after the 6-MP treatment in period II, both enzymes dropped with the leukocyte count. However, no clinical improvement was apparent and there were signs of increasing mediastinal obstruction, including edema of the face, arms and hands, engorged neck veins, dyspnea and a diffuse mottling of the lung fields on x-ray during period II. As these changes progressed, the leukocyte count and the plasma LD began to rise again, but the PHI remained well within the normal range and failed to confirm the deteriorating clinical condition. Seven days after 6-MP had been stopped, a four-day course of ACTH (40 mg. per day) was begun, and then the 6-MP treatment was resumed (period III). There was an immediate improvement in the clinical state which coincided with a sharp drop in the plasma LD, but the decrease in the leukocyte count, and especially in the PHI, was small. Remission continued for one month, but was followed by the terminal relapse.
The leukocyte count and the plasma enzymes increased before death, in spite of renewed ACTH treatment.

The unusually high, initial LD levels in this patient may be related to our unpublished finding that the plasma LD is elevated in a variety of hemolytic diseases, roughly in proportion to the rate of hemolysis as determined by red cell survival studies. In such hemolytic states the plasma PHI was much less elevated than the LD. The presence of excessive hemolysis during periods I and II (fig. 1A), indicated by the rapid drop in the hemoglobin in the absence of gross bleeding and in spite of repeated transfusions, may explain the occurrence of a higher LD:PHI ratio in this subject than in the other 8 patients with overt acute myelocytic leukemia in whom hemolysis was not as obvious.

The response of the plasma LD and PHI to 6-MP treatment in subject 3 was similar to that seen above in patient 2. Measurement of both enzymes on 12 occasions showed a good correlation between the drop in the white cell count and the fall in the PHI and LD. During the seventh month of treatment a decrease in the 6-MP dosage from 1 to 0.5 mg. per day was followed by an asymptomatic rise in the enzymes and white cell count. Both returned to the normal range on 2 mg. 6-MP per day.

The failure of patients 5 and 6 to respond to treatment was associated with a progressive rise in both enzymes until death, which occurred within 10 days of diagnosis. In patient 5 the LD rose from 50 to 148 and the PHI from 37 to over 300 units in the 3 days immediately before death. In patient 6 the LD increased from 26 to 65 and the PHI from 72 to 122. Patient 7 also failed to respond to 6-MP and survived for only two weeks after diagnosis. However, in contrast to cases 2, 5 and 6, in patient 7, both plasma enzymes in the 12 samples analyzed remained relatively constant until death. In aleukemic subject 1, the LD and PHI also remained within the normal range in the eight specimens analyzed during the month that preceded death. It is of interest that the subcutaneous bleeding apparent in the increasing numbers of petechiae and ecchymoses that appeared as the platelet count dropped terminally was not associated with a rise in the plasma enzymes.

Except for the findings in subjects 1 and 7, it may be concluded that in acute myelocytic leukemia both enzymes, but particularly the LD, usually reflect the course of the disease--falling during remissions and rising during relapses and just before death.

Chronic Myelocytic Leukemia. The findings in five patients with the chronic variety of myelocytic leukemia are shown in table 1. Before treatment, the plasma PHI was strikingly elevated, as has been reported by Israels and Delory. The LD levels were also raised, but not as much as the PHI values, except in patient 11. The high LD:PHI ratio in this subject is of interest, since he also had a severe hemolytic anemia which is discussed below. Four of the five patients in this group were followed with serial LD and PHI estimations through at least one therapeutically induced remission (cases 10, 11, 13 and 14). In each the initially high enzyme levels fell to normal. In patient 10, this fall was succeeded by a rise during a relapse, as shown in figure 1B. This man was first seen three years after the diagnosis of chronic
myelocytic leukemia had been made. In the interval he had become resistant to Myleran (dimethanesulfonyloxybutane). Hepato- and splenomegaly were gross. Dyspnea and ankle edema due to congestive heart failure were also present (period I, fig. 1A). Adenopathy was minimal and his hemoglobin ranged from 9.5 to 12.5 Gm. per cent. Routine treatment for cardiac failure produced some improvement, but the abdominal discomfort and dyspnea continued. X-ray therapy to the spleen produced a dramatic remission with a rapid shrinking of that organ, relief of all symptoms, a sharp fall in the leukocyte count and a decrease in the plasma PHI and LD levels to normal by the end of the radiotherapy course (period II). The subjective improvement lasted six months, but the leukocyte count and the plasma enzyme levels became abnormal again on the third month after radiotherapy and continued to rise (period III). When the progressive splenomegaly and dyspnea had again become incapacitating, a second course of radiotherapy was given. Again the subjective and objective improvement was marked and was associated with a sharp drop in the enzymes (period IV).

Throughout his course this subject showed a good correlation between his clinical status and the plasma levels. The PHI followed the changes in the clinical course closely, but fluctuated markedly during the courses of x-radiation and showed an unexplained decrease just before the second course of radiotherapy was begun. The changes in the LD, while less marked than those in the PHI, followed the clinical course more consistently: falling during remissions, rising to abnormal levels with the first increase in the leukocyte count during period III. Unlike the PHI, the LD remained elevated until the second course of radiotherapy was begun.

Both enzymes were measured on 14 occasions during a nine-month period in patient 13, in whom chronic myelocytic leukemia was diagnosed incidental to an investigation of diabetes of recent onset. As in patient 10 described above, the initial plasma PHI was raised more than the LD, although the latter was also abnormal. On 6 mg. of Myleran per day and an appropriate diabetic diet, the leukocyte count remained stable at about 350,000 for two weeks and then gradually fell to 33,000 during the next two months. In contrast, the response of the plasma enzymes was immediate. Both the PHI and LD began to drop during the first week, although the LD first rose from 52 to 65 in three days before beginning to fall.

The diagnosis of chronic myelocytic leukemia had been made on patient 12 six years before the present study. Her leukemia had become resistant to Myleran and radiotherapy, herpes zoster was present, the liver and spleen were grossly enlarged and the hemoglobin concentration was 6.6 Gm. per cent. Both plasma enzymes were grossly elevated. In view of the disproportionately raised LD levels associated with the increased red cell destruction noted in patients 2 and 11, it is of interest that patient 12, who had the highest LD levels in the chronic myelocytic leukemia series, also had the most severe hemolytic anemia in this group. Her peak LD levels occurred during a number of post-transfusion hemolytic episodes.

The findings in patients 11 and 14 illustrate that the LD:PHI ratio is more indicative of hemolytic disease than the LD level alone. Eight transfusions
were required in patient 11 (fig. 2) because of impaired erythropoiesis and an increased rate of hemolysis confirmed by a 17-day half-life of Cr\textsuperscript{51} labelled red cells. The return of erythropoietic activity reflected in the reticulocyte response was followed by a gradual rise in hemoglobin concentration that reached 15.5 Gm. per cent in six months. The anemia in patient 14 (fig. 3), although initially as severe as that of patient 11, seemed almost entirely due to decreased red cell production, since Cr\textsuperscript{51} labelled red cells had a nearly normal half-life of 28 days. In spite of these differences, the LD values followed an almost identical course in these subjects. However, the LD:PHI ratio in patient 11, as in other subjects with active hemolysis, was higher than that in patient 14 and others (10 and 13) with no overt hemolytic disease.

A review of all the findings in the chronic myelocytic leukemia group shows that the PHI was usually higher than the LD, relative to their respective upper limits of normal, but that the LD followed the course of the individual

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**Fig. 2.**—Plasma lactic dehydrogenase (LD) and phosphohexose isomerase (PHI) in patient 11. The arrow indicates the labelling of the red cells with Cr\textsuperscript{51}. 
patients more closely than did the PHI. In three of the five subjects (11, 13 and 14) the fall in the LD during therapy preceded a consistent drop in the white cell count. The highest LD values occurred in patient 12, whose condition was the poorest, while the lowest LD was found during a remission in patient 10, who had the best therapeutic response in this group.

**Acute Lymphocytic Leukemia.** Four subjects with acute lymphocytic leukemia were studied (table 1). The initial LD was elevated in all, but the PHI fell within the normal range in three of the four patients. As was also the case in the aleukemic patient (1) in the acute myelocytic leukemia series, the lowest enzyme values in the present group were found in analeukemic patient 15. This was of particular interest since extensive subcutaneous and intracavitary bleeding was present. Postmortem examination within a week of the enzyme measurements revealed leukemic infiltration of the liver, spleen and lymph nodes. In the remaining three cases of acute lymphocytic leu-
kemia the fall in the plasma PHI and LD during treatment and the subsequent rise at the onset of a relapse, seen above in both types of myelocytic leukemia, were also found. Thus, in patient 16, whose initial enzyme levels were the lowest of the three with overt acute lymphocytic leukemia, a remission lasting over six months was induced with prednisone. His improvement was associated with a drop in the LD and leukocyte count to the normal range. In figure 4A are shown the findings on patient 18, a woman of 62, with headache, weakness, phlebitis and petechiae of two weeks' duration. Her leukocyte count and smear had been normal five months before admission, but she had a long history of hypertensive heart disease. During the present study her hypertension was minimal, but left ventricular hypertrophy was present. In the first week after admission to hospital her phlebitis was treated with an anticoagulant until leukemia was diagnosed. During the next week the LD dropped slightly from 50 to 47 before treatment for leukemia was begun, but the PHI showed a much larger unexplained decrease (period I). Both enzymes reached the normal range within a week after the beginning of prednisone therapy. During the remainder of her first hos-

![Figure 4](https://www.bloodjournal.org)

**Fig. 4.**—Plasma lactic dehydrogenase (LD) and phosphohexose isomerase (PHI). Panel A: case 18; panel B: case 17, both acute lymphocytic leukemia. See figure 1 for explanation of symbols.
hospita! admission and for the next month as an outpatient she was moderately well, although some petechiae, lassitude and night sweats persisted (period II). Increasing the prednisone again to 20 mg. per day produced a subjective improvement, but petechiae and ecchymoses continued to increase while the leukocyte count and both enzymes rose (period III). Treatment with 150 mg. 6-MP per day produced a rapid drop in the plasma enzymes, and the white cell count returned to normal; but hemorrhage into the skin increased, her hemoglobin began to drop and she complained of generalized body aches (period IV). Within a week she was moribund with pneumonitis and diffuse bleeding. In spite of the infection, the leukocyte count was only 8,500, and less than 2 per cent of the cells were neutrophils. On the day before her death, the white cell count was 2,200 and consisted mainly of disintegrated cells. By this time the plasma PHI had risen again, but the LD remained low with the leukocyte count. Postmortem examination showed widespread infection, long-standing aortic stenosis, chronic passive congestion of the spleen and liver with a minimum of leukemic infiltration and no enlarged lymph nodes. In reviewing this patient's course it is apparent that the PHI and LD reflected the clinical status and white cell count closely until a week before death, but that neither enzyme warned of the impending 6-MP toxicity.

The findings on a four-year-old boy (patient 17) with acute lymphocytic leukemia, shown in figure 4B, demonstrate that a rise in the plasma LD may occasionally predict a relapse before the PHI or leukocyte count rises. Lassitude had been present for one month before admission, lymphadenopathy was general and both the spleen and liver were enlarged. However, the PHI was normal and only the LD was raised (period I). Transfusions, ACTH and prednisone produced an immediate clinical remission with a fall in the white cell count and both enzymes. In spite of a persistent anemia and the inadvertent withdrawal of prednisone for a month, the patient remained well with a normal leukocyte count and PHI level (period III). However, the LD had begun to rise and continued to do so as the gradual reappearance of adenopathy, splenomegaly and ecchymoses signalled a relapse (period IV). At the time of the final admission (period V) there was an oozing epistaxis, numerous ecchymoses, the liver was 7 cm. below the costal margin and the spleen was felt below the umbilicus. Although the white cell count had increased and the LD was still rising, the PHI remained normal until death. It cannot be decided whether this rise in LD was due to hemolysis, since the pre-terminal fall in the hemoglobin may have been due, at least in part, to external bleeding. Terminally both the leukocyte count and the LD dropped on combined 6-MP and prednisone therapy.

The findings in this patient, if confirmed, illustrate the potential usefulness of the plasma LD in predicting the onset of a relapse (period IV) and the limitations of the PHI measurements in this regard. However, in both this patient and in patient 18 the decreased LD levels found terminally seemed more to reflect the low leukocyte count than the moribund condition of the subject.

**Chronic Lymphocytic Leukemia.** The findings on nine subjects with chronic
lymphocytic leukemia are shown in table 1. With few exceptions, the initial plasma LD and PHI values fell within the normal range. The enzymes were estimated only once in five of the patients; but in subjects 19, 20, 23 and 27 the measurements were made on 25, 20, 8 and 7 occasions, respectively. The values remained remarkably constant for months except in patients 19 and 20 who developed hemolytic anemias requiring numerous transfusions. Although the half-life of their Cr$^{31}$ labelled erythrocytes was shortened to 8 and 12 days respectively during the hemolytic episodes, the LD rose to peaks of only 46 and 23. The plasma PHI levels, however, remained in the low normal range. The resulting high LD:PHI ratios were a better indication of the severity of the hemolytic disease than the LD increase alone. After splenectomy, hemolysis decreased greatly in subject 19 and his plasma LD dropped to 15 units. Other than the relation of LD to hemolysis, no correlation between the levels of the plasma enzymes and clinical course or white cell count was apparent during the course of the individual patients or in the chronic lymphocytic leukemia subjects taken as a group.

**Acute Monocytic Leukemia.** The initial findings in three cases of acute monocytic (Schilling) leukemia are also shown in table 1. In common with the other aleukemic cases (1 and 15 described above), patient 28 had low enzyme levels until death. In the other two patients with monocytic leukemia the plasma LD was increased together with the white cell count. The enzyme levels in case 29 remained stable until death, three months later. The high LD in patient 30, which gradually rose to 173 just before death, may have been related to the severe hemolytic anemia present in this subject.

**DISCUSSION**

The source of the elevated plasma LD and PHI levels in leukemia reported here and by others$^{3-5,7}$ is as yet not firmly established. Evidence has been presented$^{12}$ that the raised plasma PHI in chronic myelocytic leukemia has its origin from the disintegration of enzyme-rich granulocytes. The low PHI values in our aleukemic patients (1 and 28) suggest that the destruction of leukocytes must be intravascular in order to influence the plasma PHI, since other patients who had the same types of leukemia, but with elevated white cell counts, had raised PHI values.

The normal plasma PHI in chronic lymphocytic leukemia is considered$^{12}$ to reflect both the low level of this enzyme in leukemic lymphocytes and their relatively long life span compared with that of leukemic granulocytes.$^{13,14}$ The possibility that leukemic infiltration of PHI-rich tissues* such as liver, spleen, bone and brain might raise the plasma PHI seems unlikely in view of the relatively low plasma values found in patients with clinical and post-mortem evidence of such tissue invasion (cases 1, 3, 15 and 17).

In all except the aleukemic subjects and those with chronic lymphocytic leukemia, the plasma LD activity usually rose when the disease was active and fell during remissions. The low LD levels in chronic lymphocytic leukemia, like the low PHI, may be explained by the long life span of the lymphocytes in this condition and their low LD content$^{15}$ relative to that of leukemic granulocytes. The normal LD in our aleukemic patients and the tendency
for the LD and leukocyte count to change together in leukemias other than the lymphocytic type indicates that the elevated plasma LD levels may arise, at least in part, from the intravascular disintegration of leukocytes. Nevertheless, there were a sufficient number of discrepancies between the leukocyte count and the plasma LD in our series to suggest that some of the plasma enzyme may arise from other LD-rich tissues, including erythrocytes.

The red cell as a source of the elevated plasma LD in leukemia was suggested by our unpublished finding of high levels in erythroblastosis fetalis, paroxysmal nocturnal hemoglobinuria and other types of hemolytic disease. In the present series, the plasma LD was not raised significantly by hemolysis of extravasated blood following tissue bleeding, but was always increased in patients in whom red cell survival studies showed an abnormal rate of erythrocyte destruction. In a more detailed examination of this relationship, to be presented elsewhere, it was observed that the rate of hemolysis is better correlated with the increase in the plasma LD:PHI ratio than with the LD activity alone (subjects 11, 14, 19, 20). The cause of this high ratio peculiar to hemolytic states is being studied.

**Summary**

Two enzymes, lactic dehydrogenase (LD) and phosphohexose isomerase (PHI), were measured in the plasma of 30 patients with leukemia and compared with the findings in 66 control subjects. Abnormally elevated PHI levels were found in both acute and chronic myelocytic leukemia, but not in lymphocytic leukemia. The plasma LD was increased above normal in acute and chronic myelocytic leukemia, in acute lymphocytic, but not in chronic lymphocytic leukemia. Both enzymes were normal or only slightly raised in three patients with the aleukemic type of the disease. Hemolytic anemia in seven leukemic patients was associated with high plasma LD values in the presence of relatively low PHI levels.

Results of serial enzyme studies from the time of diagnosis until death indicated that both plasma enzymes, but especially the LD, usually reflected changes in the course of the disease—failing during remissions and rising during relapses. In most cases this enzyme paralleled the leukocyte level but occasionally indicated the onset of a relapse or remission before the white cell count had begun to change.

**Summario in Interlingua**

Le duo enzymas, dishydrogenase de acido lactic (DL) e isomerase de phosphohexosa (IPH), esseva mesurate in le plasma de 30 patientes con leucemia e comparate con le constatationes in 66 subjectos de controlo. Anormalmente alte nivellos de IPH esseva trovate tanto in acute como etiam in chronic leucemia myelocytic sed non in leucemia lymphocytic. Le DL del plasma esseva augmentate a supra le norma in acute e chronic leucemia myelocytic e in acute sed non in chronic leucemia lymphocytic. Ambe enzymas esseva normal o levemente elevate in tres patientes con le typo aleuemic del morbo. Anemia hemolytic in septe patientes leucemic esseva associate con alte valores pro le DI. del plasma, durante que le nivellos de IPH esseva relativamente basse.
PLASMA LD AND PHI IN LEUKEMIA

Studies del enzymas executate in series ab le tempore del diagnose usque al morte indicava que DL e IPH, sed specialmente DL, reflecte usualmente le alterationes que occurre in le curso del morbo. Le concentration del enzymas descendе durante remissiones e ascende durante recidivas. In le majoritate del casos, le concentration de DL in le plasma esseva correlationate con le nivello del leucocytos, sed in certe casos le enzyma indicava le declaration de un recidiva o de un remission ante que le numeration del leucocytos manifestava ulle alteration.

REFERENCES

Plasma Lactic Dehydrogenase and Phosphohexose Isomerase in Leukemia

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