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Prognostic Evaluation of Acute Myocardial Infarction Through Blood Chemical Examination

— A Trial to Improve the Efficiency of the Computer Assisted Laboratory Diagnosis (CALD) System of Kawasaki Medical School (KMS) —

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ABSTRACT. To improve the prognostic prediction of acute myocardial infarction by (AMI) CALD (KMS), a computer assisted laboratory diagnostic system used in the Kawasaki Medical School Hospital, new tests for blood cellular elements (WBC, Lym, Plt) were added to those employed to examine 22 types of blood chemical ingredients. The discriminant, the positive or negative values of which refer to prognosis of survival or death, was mathematically derived from the improved CALD. This improved CALD allowed us to predict the prognosis at any time during the clinical course of AMI patients with a considerably good possibility of success. The rate of success 80% level for the surviving cases during the period from the first to the 14th hospitalization day, although it was below the 50% level in the early stage (the first to and second days) of hospitalization.

Key words : computer assisted laboratory diagnosis — clinical chemistry — acute myocardial infarction

Modern medicine has characterized by striking advances in diagnosis and therapy, which have been attained through the extensive use of various elaborate instruments which have been the products of highly advanced engineering. Clinical chemistry is one of the branches of modern medicine that examines and diagnoses patients by means of various instruments for chemical analysis. Clinical chemistry's first aim is to assay the chemical ingredients of body fluids (such as blood, urine, etc.), and then analyzes their numerical values. These are compared and chemical distortions in body fluids due to illness are clinically interpreted. Based on such interpretation, the clinical chemist can appraise the general condition of patients and indicate the character of disturbances of important body organs (the liver, the kidney, the pancreas, etc.). He also provides possible diagnoses (the names of diseases) and predicts prognoses, when possible, without any supportive information regarding a patient's morbid history and the findings of physical examinations.[1,2]

CALD (KMS) [computer assisted laboratory diagnosis of Kawasaki Medical School],[3] which is summarized on examination report sheets which have been given to the wards and the outpatient clinic of Kawasaki Medical School

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Hospital from its central clinical laboratory for the past 10 years, is a representative example of such activity in the field of clinical chemistry. Our experience showed CALD (KMS) to be of satisfactor diagnostic usefulness when it was applied to hepatobiliary diseases, but, to our regret, however it proved to be much less efficient in the diagnosis of acute myocardial infarction.

A correct diagnosis by means of CALD could be made in only 52% of all cases of acute myocardial infarction (AMI). Therefore, the present study was undertaken to improve the diagnostic efficiency of CALD (KMS) for AMI. Seventy-one diagnostically authentic cases of AMI admitted to our hospital were chosen for this purpose. The CALD report sheets, serum isozyme studies and complete blood counts of their medical records were checked for pathologically altered patterns in blood ingredients, both chemical and hematological. After careful study of any distorted patterns in blood ingredients, successful modifications in CALD (KMS) were made. As a result, it has been possible, with subsidiary use of mathematical treatment of the test results with discriminant analysis, to establish diagnoses and to predict prognoses for AMI with far more accuracy than with the original system.

MATERIALS AND METHODS

Materials (Table 1) —— The medical records of 71 acute myocardial infarction (AMI) patients admitted to our hospital during a recent 4 year period (1981–1985) were collected, and CALD report sheets, isozyme tests and hematological tests done during the period of their hospitalization were examined. Their diagnoses had been established according to Oxley's criteria for AMI.[4]

Of these 71 cases, 66 possessed well described medical records with report sheets of various laboratory tests carried out repeatedly in the acute phase.

TABLE 1. Characterization of surviving and dead cases among AMI patients in this investigation

cases / laboratory items	surviving cases	dead cases	total number
number of cases	51	20	71
sex (male : female)	40 : 11	13 : 7	53 : 18
average age	62.1 (36~87)	74.9 (54~93)	65.7 (36~93)
average days of hospitalization	55.5 (23~131)	39.7 (1~128)	51.1 (1~131)
average number of illness	2.2 (1~6)	3.3 (1~5)	2.5 (1~6)
average numbers of examinations /period (days)	9.4 / 24.6	9.0 / 13.0	9.3 / 21.3

(1) Laboratory tests for the diagnosis of AMI——CK•MB and LDH1>LDH2

Serum creatine phosphokinase (CK) specifically increases when heart muscle, skeletal muscle or brain tissue is injured. There are 3 types of CK isozymes (MM: skeletal muscle, BB: brain tissue and MB: heart muscle), and it has been reported in the literature that CK•MB isozyme appears in the blood serum of AMI patients. Therefore, records of CK•MB estimation were scrupulously checked.

TABLE 2. Clinical reference values of the laboratory test items

	laboratory items	normal values
blood chemical laboratory items	*serum protein (SP)	6.5～8.5 g/dl
	*blood sugar (BS)	70～110 mg/dl
	*A/G	1.0～1.8
	*icterus index (II)	4～6
	*total bilirubin (T. Bil)	0.2～1.0 mg/dl
	*direct bilirubin (D. Bil)	40～60 %
	*alkaline phosphatase (AlP)	25～80 IU/ℓ
	*cholesterol (Cho)	131～220 mg/dl
	*γ-glutamyl transpeptidase (γ-GTP)	0～30 IU/ℓ
	*lactic dehydrogenase (LDH)	49～92 IU/ℓ
	*albumin (Alb)	3.5～5.0 g/dl
	*globulin (Glb)	2.5～4.0 g/dl
	*cholinesterase (ChE)	240～490 IU/dl
	*glutamic pyruvic transaminase (GPT)	0～25 IU/ℓ
	*glutamic oxaloacetic transaminase (GOT)	0～20 IU/ℓ
	*creatinine (Crn)	0.8～1.5 mg/dl
	*blood urea nitrogen (BUN)	9～20 mg/dl
	*uric acid (UrA)	2.5～8.0 mg/dl
	*amylase (Amy)	100～400 IU/ℓ
complete blood cell	red blood cells (RBC)	male 410～550, female 360～480 ×10⁴/μℓ
	*hemoglobin (Hb)	male 13.0～17.5, female 11.5～14.5 g/dl
	hematocrit (Ht)	male 39～52, female 34～44 %
	reticulocyte (retic)	0.5～1.5 %
	platelet (Plt)	15～35×10⁴ /μℓ

	laboratory items	normal values
complete blood cell	white blood cells (WBC)	3500～9000 /μℓ
	N. Band	2～10 %
	N. Segment	50～70 %
	neutrophils (neutro)	52～80 %
	eosinophils (eosino)	1～5 %
	basophils (baso)	0～1 %
	monocytes (mono)	1～6 %
	lymphocytes (lympho)	20～40 %
isoenzyme	creatine kinase (CK)	10～70 IU/ℓ
	CK·MM	(+)
	CK·MB	(−)
	CK·BB	(−)
	LDH1	12～28 IU/ℓ
	LDH2	17～36 IU/ℓ
	LDH3	10～23 IU/ℓ
	LDH4	2～7 IU/ℓ
	LDH5	1～6 IU/ℓ
bleeding tendency	protrombin time (PTT)	10.2～11.4 sec
	activated partial thromboplastin time (PATT)	21.3～31.3 sec
	fibrinogen (fibri)	200～400 mg/dl
	fibrin degradation product (FDP)	under 10 μg/mℓ
others	CRP	under 0.6 mg/dl
	ESR	male under 10, female under 15 mm
	creatinine clearance (Ccr)	60～145 mℓ/min

* : laboratory items which are used in CALD

Serum LDH rises in AMI. It has been noted in electrophoretic studies of LDH isozymes that the possibility of a correct AMI diagnosis AMI is greater when isozyme LDH1 is higher in activity than isozyme LDH2. Thus, the results of the LDH isozyme assay were checked carefully with those of CK•MB studies in the medical records.

(2) CALD laboratory test items (Table 2)

Laboratory test items included in the CALD of the present investigation are listed (together with their normal ranges) in Table 2. Items with asterisks (*) are laboratory tests belonging to the original CALD (KMS).[3] Items without asterisks, therefore, are tests added to the original system as a result of our investigation. All of them are routinely used in the central clinical laboratory of our hospital.

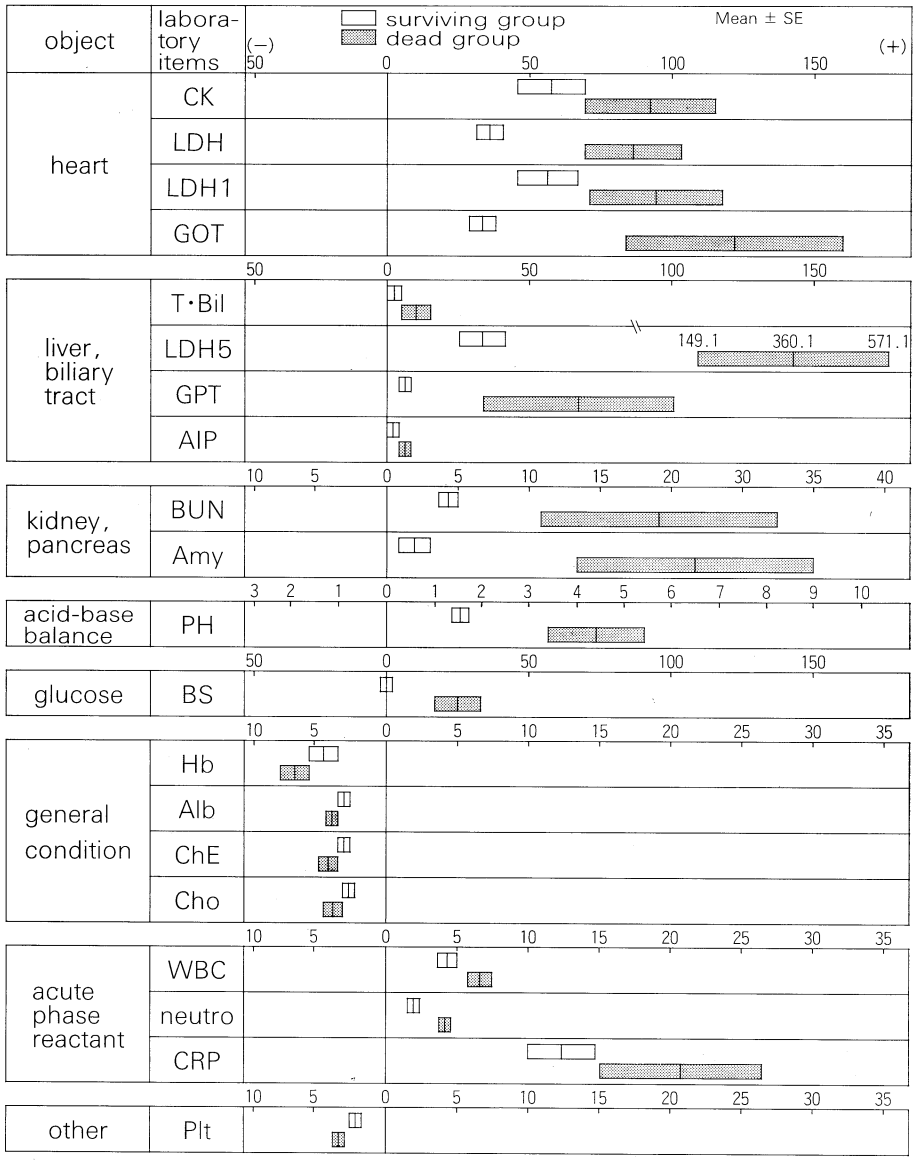
The laboratory test items included in CALD (KMS) have been classified into six groups according to their diagnostic usefulness.[5]

1 Diagnostic appraisal of a patient's general condition —— blood hemoglobin concentration (Hb), serum protein concentration (SP), the albumin to globulin ratio (A/G), and blood sugar concentration (BS)
2 Indicators of the degree of jaundice —— the serum icterus index (Ii) and serum total bilirubin (T. Bil)
3 Indicators of hepatic parenchymal injury —— serum enzyme activities, such as lactic dehydrogenase (LDH), globulin (Glb), cholinesterase (ChE), alanine aminotransferase (GPT), and asparate aminotransferase (GOT)
4 Indicators of biliary tract disturbance —— serum enzymes and serum chemical components, such as direct bilirubin (D. Bil), serum alkaline phosphatase (AlP), serum cholesterol (Cho), and glutamyl transpeptidase (γ•GTP)
5 Indicators of urinary tract disturbance —— serum creatinine (Crn), blood urea nitrogen (BUN), and serum uric acid (UrA)
6 Indicator of pancreatic disturbance —— serum amylase activity

All of these laboratory tests were carefully evaluated with regard to their reliability as indicators for prediction of the survival or death of AMI patients. The numerical values obtained from these tests were converted into an SDI (standard deviation index) expression system,[6] and the SDI values of the individual tests were separated into "surviving" and the "dead" classes and plotted on two horizontal straight lines, "surviving" and "dead". The ranges of the distribution of dots in these horizontal lines were compared (Fig. 1). It seems warranted to consider that laboratory tests in which separation of the distribution ranges of dots between the two horizontal lines are apparent and to be superior to those in which overlapping is seen when they are used as indicators for the prediction of prognosis in AMI cases. All the tests listed in Table 2 were examined from this standpoint.

(3) Discriminant for prediction of AMI prognosis (Table 3)

Laboratory tests thought to be helpful in making a prognostic prediction of AMI were chosen and categorized into eight groups as shown in Table 3.



SE : Standard Error

Fig. 1. Distribution of laboratory data expressed in terms of SDI in AMI patients.

The discriminant was derived from the test values of each relevant group.

A careful review of the literature on AMI will lead us to classification of the laboratory tests listed in Table 2 into the following two groups: (1) tests whose values are elevated above the normal range in AMI attacks (BS, T. Bil, D. Bil, AlP, γ•GTP, LDH, Glb, Crn, BUN, UrA, Amy and WBC), and (2) those which are lowered below the normal range (SP, A/G, Cho, Alb, ChE, Hb, Plt and Lym). The discriminant was derived in the following way. Out of 71 AMI cases, 66 were employed as direct resource materials from which to derive the discriminant, because they had received repeated laboratory

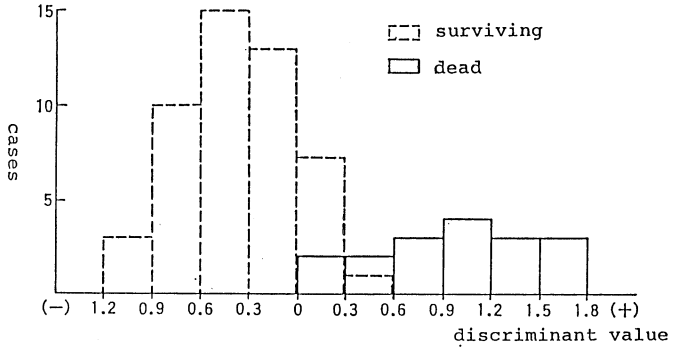
TABLE 3. Correlation rates and rates of success of discriminant values for prediction of AMI prognosis.

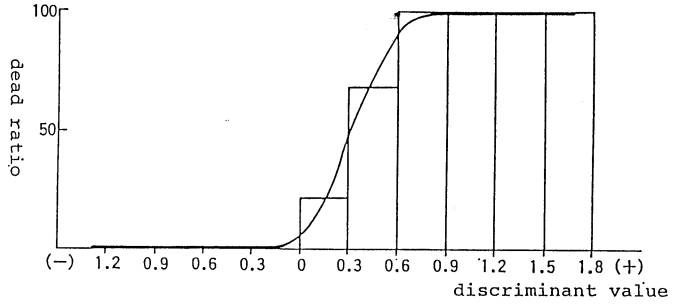
No.	number of items	number of cases	laboratory items		correlation ratios	success rates of discriminant values	
1	3	57	maximum value	T. Bil, LDH, Lym	0.4539	surviving cases	81.4%
			minimum value			dead cases	92.9%
2	7	66	maximum value	BS, T. Bil, LDH, GOT, BUN, Amy, Lym	0.5978	surviving cases	79.6%
			minimum value			dead cases	88.2%
3	7	66	maximum value	BS, T. Bil, LDH, GOT, BUN, Amy	0.6043	surviving cases	83.7%
			minimum value	Lym		dead cases	88.2%
4	7	57	maximum value	BS, T. Bil, LDH, GOT, BUN, Amy, Lym	0.5681	surviving cases	79.1%
			minimum value			dead cases	92.9%
5	9	57	maximum value	BS, T. Bil, LDH, ChE, GOT, BUN, Amy, Lym, LDH5	0.5852	surviving cases	81.4%
			minimum value			dead cases	92.9%
6	18	69	maximum value	BS, T. Bil, D. Bil, AlP, γ·GTP, LDH, Glb, GPT, GOT, Crn, BUN, UrA, Amy	0.6284	surviving cases	86.3%
			minimum value	SP, A/G, Cho, Alb, ChE		dead cases	88.9%
7	18	66	maximum value	BS, T. Bil, D. Bil, AlP, γ·GTP, LDH, Glb, GOT, BUN, UrA, Amy	0.7144	surviving cases	81.6%
			minimum value	SP, A/G, Cho, Alb, ChE, Plt, Lym		dead cases	100 %
8	22	66	maximum value	BS, T. Bil, D. Bil, AlP, γ·GTP, LDH, Glb, GPT, GOT, Crn, BUN, UrA, Amy, WBC	0.7157	surviving cases	83.7%
			minimum value	SP, A/G, Cho, Alb, ChE, Hb, Plt, Lym		dead cases	100 %

examinations during their period of stay in the hospital. The laboratory tests of the CALD were numbered 1, 2, 3, —— so that they might be dealt with systematically in a computer. The values or results of the needed tests were inserted into the computer to be memorized, one case after another, in the order of the dates of examinations. Extraction of maximum and minimum values of individual blood components that had been assayed in chronological succession was made by the computer. Maximum values were obtained for the group of blood components showing elevation over the normal range in AMI, and minimum values for those exhibiting decent below the normal range.

Then, the results of prognostic observation of AMI cases made on each relevant day were classified into "surviving" 1 and "dead" 2 and put into the computer.

$$\text{discriminant value} = 0.269\times(\text{SP})+0.273\times(\text{BS})-0.076\times(\text{A/G})+0.185\times(\text{T. Bil})+0.062\times(\text{D. Bil})+0.328\times(\text{AlP})+0.147\times(\text{Cho})-0.216\times(\gamma\cdot\text{GTP})-0.359\times(\text{LDH})-0.125\times(\text{Alb})-0.206\times(\text{Glb})+0.107\times(\text{ChE})-0.010\times(\text{GPT})+0.491\times(\text{GOT})-0.030\times(\text{Crn})+0.261\times(\text{BUN})-0.079\times(\text{UrA})+0.142\times(\text{Amy})-0.019\times(\text{Hb})-0.243\times(\text{Plt})-0.002\times(\text{WBC})-0.198\times(\text{Lym})$$





reference value (%) at cut off (0.000)

discriminant success rate	surviving	83.7	predictive value	surviving	100.0
	dead	100.0		dead	98.0
			efficiency		87.9

Fig. 2. Distribution of discriminant values of surviving and dead cases among patients with AMI

Finally, a discriminant analysis package[7] was introduced into the computer to obtain discriminants useful for determining "surviving" and "dead" statuses. In this way, the computer printed out the values of the coefficients for the terms of individual blood components (e.g., 1 —— SP, 2 —— BS, 3 —— A/G, ——) automatically. Then, it calculated and printed out the coefficients for the types of blood components. The case of group #8, for example, the coefficients were 0.269 for 1 —— SP, 0.273 for 2 —— BS, and 0.076 for 3 —— A/G. Employing these coefficients as material, the discriminant for group #8 was derived as shown in Fig. 2. Namely, the equation (1) refers to the discriminant for CALD group #8.

The SD values obtained by calculation of the discriminants are useful for determining the prognosis of AMI. The prognosis is good (survival), is positive ($D>0$). On the contrary, the prognosis is poor (dead), if D is negative ($D<0$).

The reliability of such prognoses is evaluated by a correlation ratio (assuming the value ranges from 0 to 1) which is printed out by the computer together with D.

RESULTS AND DISCUSSION

(1) Usefulness of CK•MB (+) and LDH1>LDH2 for the CALD of AMI (Table 4)

As shown in Table 4, the blood sera of AMI were positive for CK•MB and, simultaneously, the yielded LDH1 activity was larger than that of LDH2 in 84.6% cases of the AMI cases examined. When the instances in which either only CK•MB(+) or LDH isozyme distortion (LDH1>LDH2) was found were combined with those in which cases were simultaneously positive for CK•MB(+) and LDH1>LDH2, the isozyme abnormality was found to be existent in 94.4% of all cases. About 90% (≒88.8%) of the AMI cases showed a single CK•MB(+) abnormality.

Structurally, the LDH molecule is a tetramer consisting of a single or two types of subunits. The subunits are L (proper to the liver cells) and H (proper to the cardiac muscle cells). Therefore, there are 5 different kinds of LDH, namely LDH1=HHHH (found in cardiac muscle, the kidney and erythrocytes), LDH2=HHHL (contained in the kidney, erythrocytes and cardiac muscle), LDH3=HHLL, LDH4=HLLL and LDH5=LLLL.[8] In AMI, LDH1=HHHH and LDH2=HHHL are exuded by damaged muscular cells of the heart into the blood. As a result, LDH activity is elevated in blood serum, and isozyme studies of serum reveals a LDH1>LDH2 pattern in which LDH1 is more activated than LDH2. This is one of the diagnostically significant findings peculiar to AMI.[9]

In addition, creatine phosphokinase (CK),[10] which is distributed in the heart, the skeletal muscle and brain, is released from injured cardiac muscular cells into the blood, and this results in elevation of CK activity in blood serum.[11] The CK molecule is dimeric, being composed of a single type or two types of subunits, namely subunit B related to the brain and subunit M concerned with skeletal muscle.[12] Therefore, three kinds of isoenzymes are

TABLE 4. Diagnostic evaluation of serum isozyme study (CK·MB and LDH) for detection of AMI cases

section	findings CK·MB	findings LDH1>LDH2	total number of cases surviving (%)		dead (%)		total (%)	
positive group	+	+	41	(80.0)	19	(95.0)	60	(84.6)
	+	−	1	(2.0)	1	(5.0)	2	(2.8)
	−	+	3	(6.0)	0	(0)	3	(4.2)
	+	⊹	1	(2.0)	0	(0)	1	(1.4)
	⊹	+	1	(2.0)	0	(0)	1	(1.4)
	subtotal		47	(92.0)	20	(100.0)	67	(94.4)
negative group	−	−	0	(0)	0	(0)	0	(0)
	−	⊹	1	(2.0)	0	(0)	1	(1.4)
	⊹	−	1	(2.0)	0	(0)	1	(1.4)
	⊹	⊹	2	(4.0)	0	(0)	2	(2.8)
	subtotal		4	(8.0)	0	(0)	4	(5.6)
	total		51		20		71	

(+ : positive, − : negative, ⊹ : no examination)

found, i.e. BB in brain tissue, MB in cardiac muscle cells and MM in skeletal muscle cells. CK·MB is absent in normal serum, but it appears in serum during an AMI attack.[11] Accordingly, adoption of CK·MB into the framework of test items included in the CALD (KMS) will enhanced the reliability and sensitivity of computer assisted diagnosis of AMI, because CK·MB is highly specific for this disease. It is thus probable that a correct diagnosis using the newly improved CALD can be in 90% of AMI cases.

(2) Evaluation of the usefulness of the CALD test items for prognostic prediction in patients with AMI

Fig. 1 illustrates the ranges of distribution in results the principal tests included in CALD for the 71 AMI cases studied. The test results are expressed in terms of the SDI system to make comparison of the "surviving" class with the "dead" class feasible. Scrutiny of this figure reveals that there is a discrete separation of distribution ranges between the two classes with regard to GOT, LDH, GPT, BUN, Amy, and other items.

We therefore believe that these test items will be helpful in the prediction of prognosis for AMI cases.

On the contrary, test items for the appraisal of general condition, such as Hb, Alb, ChE and Cho did not show any clear-cut discrimination of distribution ranges between the "surviving" and "dead" classes.

As for the cellular components of blood WBC (leucocyte count and neutrophylic leucocytes), which are the acute phase reactants, there was a clear-cut distribution. A distinction in similar levels was also observed in the distinction ranges of Plt (thrombocyte count) between the "surviving" and "dead" cases. Therefore, these test items may be considered useful for prognostic prediction of AMI.

(3) Discriminant for prediction of prognosis of patients with AMI (Table 3)

The discriminant for predicting prognosis of survival or dead for the 66 patients with AMI was derived by evaluation of the results of selected combinations of CALD test items. There were 8 groups of selected combination of laboratory test items, and these are listed in Table 3. Several examples of discriminant equations derived for these groups are mentioned below.

Group #3 (7 test items)

$$D = 0.356 \times (SP) + 0.333 \times (T.\ Bil) - 0.506 \times (LDH) + 0.562 \times (GOT) + 0.330 \times (BUN) + 0.168 \times (AMI) + 0.231 \times (Lym) \quad \cdots\cdots (2)$$

Group #6 (18 test items)

$$D = 0.073 \times (SP) + 0.259 \times (BS) + 0.031 \times (A/G) + 0.212 \times (T.\ Bil) + 0.070 \times (D.\ Bil) \times 0.264 \times (AlP) + 0.046 \times (Cho) - 0.114 \times (r \cdot GTP) - 0.417 \times (LDH) - 0.152 \times (Alb) - 0.017 \times (Glb) + 0.010 \times (ChE) - 0.180 \times (GPT) + 0.650 \times (GOT) - 0.002 \times (Crn) + 0.280 \times (BUN) - 0.059 \times (UrA) + 0.240 \times (Amy) \quad \cdots\cdots (3)$$

Group #8 (22 test items)

The discriminant is expressed as (1) which is shown in Fig. 2. Equation (2) was obtained by analysis of 5 test items which proved to be distinctly different in the distribution ranges between the surviving and dead AMI cases (such as BS, LDH, GOT, BUN and Amy), plus 2 test items (T. Bil and Lym) which were less salient but different in their distribution ranges. This discriminant (2) enabled us to predict the prognosis of AMI correctly in 83.7% of surviving cases and 88.2% of dead cases. The reliability was about 0.60 when examined by the correlation ratio. The results are shown in Table 3.

On the other hand, when test item totaling 22 in number formed by 4 tests concerned with the blood cellular elements and 18 tests belonging to the original CALD (KMS) were employed as material, equation (1), as shown in Fig. 2, was derived as the discriminant for the CALD (E: extended). By using this CALD (E) discriminant, future recovery from AMI could be presumed with 83.7% accuracy and forthcoming death due to this disease with a 100% accuracy. The correlation ratio (the reliability of prediction) was about 0.72 (Table 3).

Conventionally, however, blood cell counts in clinical laboratories are carried out in a hematology section which is operated independent of the clinical chemistry section. Therefore, it is only natural that the staff of the clinical chemistry section want, if possible, to get discriminant through the CALD (KMS) based exclusively on clinical chemistry data. With this in mind, equation (5) was derived by using solely the blood chemical test results belonging to CALD (KMS) as material. The rates for prognostic prediction by this discriminant were 86.3% and 88.9% for the "surviving" and "dead" cases, respectively. The correlation ratio was 0.6284 (Table 3).

With employment of the data described above, the rate of success in prognostic prediction and the correlation ratio should become higher with the increase in the number of CALD laboratory test items (hematological as well as clinicochemical) which are used for derivation of the discriminant. In fact, a 100% rate of success in prognostic prediction for the "dead" AMI cases

has already been attained. It is worth mentioning, however, that this rate of success was achieved only at the final stage of consecutive laboratory observations when all data required for prognostic prediction had already been obtained clinically. This aspect is shown in Fig. 2.

(4) Re-evaluation of the discriminant through its application to a new group of AMI cases

It is natural that the discriminants obtained in the previous section, equation (1) pertaining to group #8 and others, provided us with a high rate of success in prognostic prediction, since it was applied to the original group of 66 AMI cases, which were the real material for the derivation of the equations. Therefore, we felt that the reliability of this discriminant with another group of AMI cases should be examined to see if an equally excellent rate of success could be obtained.

For re-elevation, this discriminant was applied to a new group of AMI cases ("dead" 7 and "surviving" 15) presented in Table 5. The results of this examination were as follows.

new cases	"surviving" group	"dead" group
22 cases	80.0%	100%
original cases pertaining to (1)		
(66 cases	83.7%	100%)
	for the purpose of comparison	

Accordingly, it is clear that this discriminant held equally well in the new group of AMI cases as in the original group.

TABLE 5. Characterization of surviving and dead cases among new AMI patients for the appraisal of reliability of the discriminant equation derived in this investigation

cases / laboratory items	surviving cases	dead cases	total number
number of cases	15	7	22
sex (male: female)	12 : 3	7 : 0	19 : 3
average age	60.3 (46~81)	71.0 (42~95)	64.5 (42~95)
average days of hospitalization	57.2 (26~140)	9.7 (3~18)	42.1 (3~140)
average number of illness	2.1 (1~4)	4.0 (2~6)	2.2 (1~4)
average numbers of examinations/period(days)	9.1 / 28.1	5.1/ 5.8	7.7/21.6

(5) Prognostic prediction of AMI cases by the discriminant through observation of their successive hospitalization days

A clinical chemist who is in change of providing clinicians at the bedside of AMI patients with CALD report, feel regret when a prognostic prediction must be issued from the laboratory in the last stage of a patient's clinical course, at which time a prognosis may have already been clearly made by the

clinicians without laboratory cooperation. He may consider such a final stage prediction to be of no value to either clinical chemists or the clinicians at the bedside. Clinicians on hospital wards continuously desire information from the laboratory regarding the prospects of their AMI patients. Therefore, the discriminant (1) obtained in our study was applied to the first, second, 7th and 14th hospital days of the patient's course in order to check the degree of infallibility of its prediction about the original group of 66 AMI cases and the new group of 20 AMI cases. The results are shown in Tables 6 and 7.

From these two tables, it is apparent that the rate of success prognostic prediction by equation (1) for the AMI cases with a benign course, however was poor (35.3–57.1%) on the first hospitalized day, and did not benign to improve until the 7th hospitalized day (65–100%). Therefore, the discriminant (1) is excellent for determining the benign prognosis of AMI patients, but it is not so successful in predicting early days of the death of patients in the hospitalization. If we take this amply into consideration, we can predict the prognosis of AMI cases successfully through CALD (E: extended) by use of discriminant (1). Prognostic prediction through CALD (KMS) with, which takes into account only tests of blood cellular elements, successful somewhat less of data useful for judgment is less abundant. However, discriminant (3) is satisfactory for diagnostic prediction of AMI cases in routine laboratory work.

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