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Pseudocholinestrase Activity in Acute Myocardial Infarction: Revisited Old Study

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Abstract

Introduction: *Ischaemic Heart Diseases are becoming progressively commoner in the younger persons of third and fourth decade. A massive rise in the levels of SGOT, LDH, CPK, during myocardial infarction suggest large infarct with poor prognosis. Earlier and rapid fall in the pseudocholinesterase level may have a poor prognosis.*

Aims and Objectives: *To Study incidence, levels and patterns of Pseudocholinesterase Activity In Acute Myocardial Infarction.*

Material and Methods: *75 subjects were included in the study. 25 cases were controls and 50 cases were patients of Acute Myocardial Infarction admitted within 48 hours of onset of Acute Chest Pain in Intensive Coronary Care Unit of Department of Medicine. Out of 50, 44 were confirmed (34 complicated and 10 uncomplicated) and 6 were provisional cases. Subjects were studied for incidence, levels and pattern of pseudochlinesterase activity and data collected were analysed statistically.*

Results and Observations: *1).The normal value of pseudocholinesterase activity ranged from 138.74 to 302.70 µmol/ml (mean value of 192.78 ± 46.64). 2). All 44 (100%) confirmed cases and 4 (66.66%) out of 6 provisional cases of acute myocardial infarction had significant fall in the values of pseudocholinesterase activity at one or other time during the study period. 3). Levels of pseudocholinesterase started falling after 12 hours of chest pain in all 44 confirmed cases and 4 provisional cases. Lowest values were seen in 22 (59.46%) out of 37 cases on 1st day, 17 (39. 53%) out of 43 cases on 2nd day and 5 (11.9%) cases on 3rd day. Levels started rising thereafter and minimum normal values were attained on7th day. Mean lowest value was 80.50±34.59.4). Mean lowest value in complicated group was73.94 ±34.07 while in uncomplicated group it was 96.99 ± 16.53 (P<.01). Lowest values in complicated and uncomplicated groups were seen on 1st and 2nd day respectively. 5). The mean value in complicated group (73.94 ± 34.07, 77.97 ± 20.00 and 92.48 ± 20.88 on 1st, 2nd and 3rd day respectively) was much lower than uncomplicated group (104.28 ± 26.18, 96.99 ± 16.53 and 120.21 ± 22.97 on, 1st, 2nd and 3rd day respectively) following myocardial infarction (P < .00l). 6). 3 (75%) out of 4 cases who expired had lowest value of pseudocholinesterase activity on the day of death.*

Conclusion: *In myocardial infarction, earlier and rapid fall in the pseudocholinesterase level may have a poor prognosis, a persistent low level in patients having complication may be a grave prognosticsign.*

Keywords: *Ischaemic Heart Diseases, pseudocholinesterase activity.*

Introduction

In the affluent countries every third death in sixth decade of life is caused by Ischaemic Heart Disease (WHO 1974). It has become major killer

of mankind in the most fruitful years of life. Ischaemic Heart Diseases are becoming progressively commoner in the younger persons of third and fourth decade. In India too, the incidence of Ischaemic Heart Disease is increasing rapidly in younger person.

The diagnosis of Myocardial Infarction in life was first made by Dr. Adam Hammer in 1876 and since then the diagnosis of Ischaemic Heart Disease has received a constant attention by medical specialists.

The diagnosis of myocardial infarction is made by clinical history taking, twelve lead electrocardiogram and serum enzymes levels. Physical examination does not provide adequate information. The electrocardiogram though has been sheet anchor in diagnosis but has its own limitations. It may take more than 48 hours to develop changes, or may have nonspecific changes. A wide range of biochemical indicators detecting myocardial necrosis are available in the form of various enzymes like Serum Glutamic Oxaloacetic Transaminase, creatinine phosphokinase, hydroxybutyrate dehydrogenase, Aldolases and Lactic dehydrogenase.

Review of Literature

The existence of an enzyme capable of hydrolysing acetylcholine into choline and acetic acid was first suggested by *Dale* (1914) in horse serum and its presence is serum was established by Loewi and *Navratil* (1926). Serum cholinesterase was first discovered in human serum by Stedman and Easson (1932). *Stedman* and Stedman found ant that the enzyme present in the blood (serum) is the specific catalyst for hydrolysis of choline esters, ischolinesterase.

Rudolph V La Matta et al (1968) studied and separated 5 isoenzymes of pseudocholinesterase by Starch Gel Electrophoresis method. *La Mote* et al (1970) further studied the different form of pseudocholinesterase and their molecular weight. *Kirk* (1969) studied the pseudocholinesterase activity in vascular system. *La Du and Dewald* (1971) studied the genetic regulation of human cholinesterase in man. *Wilfred and Copenhower* (1981) had tried to visualize the cholinesterase reactions in cardiac conduction pathways.

Pseudocholinesterase Activity in Acute Myocardial Infarction

Martti Oka (1954) in his study of 7 patients had concluded that measurement of plasma cholinesterase activity seems to be a careful test when following the course of myocardial infarction and also making its prognosis. He suggested that a constant decrease in the plasma cholinesterase activity is undoubtedly a bad prognostic sign. On the other hand increasing values indicate that recovery is proceeding. No definite mechanism of fall of pseudocholinesterase activity has yet been explained in acute myocardial infarction. *Basu* et al (1972) presumed that synthesis of enzyme by the liver may be deranged due to hypoxic condition of liver in infarction or there may be more utilization of pseudocholinesterase enzyme.

In their study *Charles B. Moore* et al (1957) found that the earliest fall in the levels of pseudocholinesterase was seen on the 1st day of infarction. Minimum values were found to be on 4^{th} day and normal values were attained on 12^{th} day. *Basu* et al (1972) ascertained that earliest fall in the levels of pseudocholinesterase was seen on 1^{st} day. Minimum values were also seen on the same day. *Vachharajani* et al (1976) found earliest fall in the levels of pseudocholinesterase on 1st day. The minimum values were attained on the same day. Normal values were attained on 14^{th}day. *G.S. Sainani* et al (1979) and *Chawhan* et al (1982) found lowest value of pseudocholinesterase on 2^{nd} day.

Pseudocholinesterase in Relation to Complication and Mortality

It is well known that a massive rise in the levels of SGOT, LDH, CPK, during infarction suggest large infarct with poor prognosis. Charles, B. Moore (1957) reported that earlier and rapid fall in the pseudocholinesterase level may have a poor prognosis, a persistent low level in patients having complication may be a grave prognostic sign. In

his study all the fatal cases had marked fall in pseudocholinesterase levels before death. Mutalik et al (1976) found that in complicated cases the levels of pseudocholinesterase were much lower than uncomplicated. G.S.Sainani et al (1979) studied the patients of infarction having cardiac arrhythmias and found that patients having tachyarrhythmias had little higher value in comparison to patients with bradyarrhythmias though the difference was not significant statistically.

Material and Methods

The study comprised of patients of Acute Myocardial Infarction admitted within 48 hours of onset of Acute Chest Pain in Intensive Coronary Care Unit of Department of Medicine, L.L.R.M. Medical College and associated hospital, Meerut from June 1983 to May 1984. Besides patients of Acute Myocardial Infarction 25 controls were also studied for pseudocholinesterase activity estimation.

Selection of Cases

All cases (n=75) studied were grouped as follows.

Group I Controls (n=25)

Group II: Cases of Acute Myocardial Infarction (n=50)

A. Confirmed Cases (n=44) i.e. patients with typical ECGchanges:

(i) Complicated(n=34)

(ii) Uncomplicated(n=10)

B. Provisional Cases (n=6) patients with normal or nonspecific. changes in EKG, but with classical clinical features and course.

Criteria for Exclusion and Inclusion

Patients admitted after 48 hours of onset of acute chest pain were excluded from the study. A thorough clinical check up was done in every case and the cases with liver diseases (Hepatitis, Cirrhosis, Malignancy) malignancies, chronic renal failure, Dermatomyositis, Nephrotic Syndrome and toxic goitre were excluded from the study. Patients receiving sympathomimetic drugs, phenothiazine derivative and atropine or its derivatives were excluded from study. However, patients receiving infrequent administration of these drugs were included in the study.

The study was designed in the following fashion

1. Clinico-Electrocardiographic study.
2. Haematological study.
3. Biochemical study.

Pseudocholinesterase Activity Estimation

In Controls: Pseudocholinesterase estimation was done once in age and sex matched normal persons.

In cases of Acute Myocardial Infarction: Pseudocholinesterase activity estimation was done at the time of admission and then on 1st, 2nd, 3rd, 5th, 7th and 10th day of Acute Chest Pain.

Observations

In the present study a total of 75 cases were studied, out of which 50 cases were of acute myocardial infarction and 25 were controls.

Table 1: Showing Pseudocholinesterase Activity in Controls

Age group (years)	Total No. of cases	Male			Female		
		No. of cases	PsCHE (µ mol/ml)		No. of cases	PsCHE (µ mol/ml)	
			Mean +	S.D.		Mean +	S.D.
< 40	5	3	189.48	62.63	2	204.75	5.58
40-49	7	6	194.67	56.91	1	199.43	-
50-59	8	7	186.87	54.62	1	167.48	-
60-69	3	3	206.83	64.57	-	-	-
>70	2	1	187.53	-	1	190.60	-

Table 2: Showing incidence of fall of pseudocholinesterase levels in cases of acute myocardial infarction.

Subjects	Total No. of cases	Incidence of fall of PsCHE in No. of Cases	%
I-Acute Myocardial Infarction	50	48	96
Ia-Confirmed cases	44	44	100
(i) Complicatedcases	34	34	100
(ii) Uncomplicatedcases	10	10	100
Ib-Provisional Cases	6	4	66.66

Table 3: Showing pattern of pseudocholinesterase in patients of acute myocardial infarction

Time Interval (In days)	PsCHE in controls (µ mol/ml)	No. of Patients	PsCHE (µ mol/ml)		
			Mean	± S.D.	P
On Adm. (within 12 hrs)	192.78 ± 46.64	29	134.49	25.91	<.001
1		37*	80.50	34.59	<.001
2		43*	82.39	20.72	<.001
3		42*	99.08	24.26	<.001
5		41*	121.60	25.72	<.001
7		40	144.20	34.30	<.001
10		40	162.22	37.48	>.05

*4 Cases expired during the study one each on 1st, 2nd, 3rd and 5th day respectively

Table 4: Showing pattern of pseudocholinesterase in uncomplicated and complicated cases of acute myocardial infarction

Time Interval (Days)	Total No. of Cases	PsCHE (µ mol/ml)						P Value
		Uncomplicated cases			Complicated cases			
		No.	No. of cases with lowest activity	Mean + SD	No.	No. of cases with lowest activity	Mean + SD	
On Adm. (within 12 hrs)	29	6	1	151.20±29.23	23	-	130.14±23.76	>.05
1	37*	8	3	107.28+26.18	29*	19	73.94+34.07	<.001
2	43*	10	5	96.99+16.53	33*	12	77.97+20.00	<.001
3	42*	10	2	120.21+22.97	32*	3	92.48+20.88	<.001
5	41*	10	-	132.51+23.86	31*	-	118.08+25.67	>.05
7	40	10	-	162.98+27.54	30	-	137.94+34.42	>.05
10	40	10	-	174.98+29.15	30	-	156.77+38.75	>.05

*4 Cases expired during the study one each on 1st, 2nd, 3rd and 5th day respectively

Discussions

We studied 75 cases for pseudocholinesterase activity estimation. 25 age and sex matched patients served as controls. Pseudocholinesterase enzyme activity was estimated only once in controls. 50 were cases of acute myocardial infarction. Pseudocholinesterase activity was estimated on admission and then on 1st, 2nd, 3rd, 5th, 7th, 10th day following acute chest pain.

Normal value in our set up ranged from 138.74 to 302.70 µmol/ml with a mean activity of 192.78 + 46.64. These values were in agreement to the values found by other authors (J.de la Huerga, 1952; Mutalik et a1, 1976; Vachharajani,1976; Sainani et a1,1979). There was no significant difference in the pseudocholinesterase activity in different age groups in this study.

Age and Sex Distribution

While observing the sex distribution, it was found that males outnumbered the females. The male: female ratio was 5.2: 1.0. The male preponderance in ischaemic heart disease is well recognised (Semantary and Somani, 1975). Male female ratio was found to vary from 3.4:1 to 8:1 (Vakil et a1,

1963; Mathur et a1, 1960). Disparity in the incidence of myocardial infarction in males and females is greatest before the age of forty and diminishes after the age of seventy years (Annastessiades, 1961). Lesser susceptibility of women to myocardial infarction in early age has been attributed to the effects of estrogens or other sex hormone (James et al, 1955). In the present study there was only one female less than the age of 40 years. Average age (Mean) of females was little higher than males in this study (Male 54.1 years, females 55.8 years). Similar results were observed by Annastassiades et al (1961). Maximum number of patients was in the 6th decade of life followed by 5th and 7th decade (32%, and 26% each respectively). Master et al (1939), Mintz and Kartz (1947) in their study also observed similar results.

There was no significant difference seen in the incidence of *complications* in either sex (Male: female ratio is 1:1 approximately)

Incidence and Pattern of Pseudocholinesterase Activity in Acuie Myocardial Infarction

In the present study fall in the levels of pseudo-cholinesterase was observed in all 44(100%) confirmed cases of acute myocardial infarction. This finding was consistent with the findings of other workers (Basu et a1,1972; Mutalik et a1, 1976; Vachharajani et al, 1976; Sharma et a1, 1978; Sainani et al, 1979, Chawhan et a1,1982).

In this study we have observed a significant fall in the levels of pseudocholinesterase on 1st day following infarction and low values were seen on very same day in most of the cases. Low values following infarction have been observed by various authors on 1st day (Basu et a1,1972, Vachharajani, 1976; Mutalik et a1, 1976; Sharma et a1, 1978; Sainani et al, 1979; Chawhan et al, 1982)

It is very difficult for us to comment whether pseudo- cholinesterase levels start falling in early hours of infarction as none of our patients in the study group came to hospital before 8 to 10 hours of chest pain. In this study out of 29 *complicated* cases arrived in 24 hours of chest pain, 19 (65.51%) had lowest value while remaining 10 and 2 out of five cases, who arrived on 2nd day had lowest activity on 2nd day, remaining three patients had lowest value on 3rd day. In *uncomplicated* group 3 (37.5%) out of 8 patients who arrived on 1st day, had lowest activity on 1st day, remaining 5 (62.5%) had lowest activity on 2nd day and remaining two who came on 2nd day of infarction had lowest activity on 3rdday.

Duration of Low Activity of Pseudocholinesterase

In present study low levels of pseudocholinesterase activity persisted for 7 days in both complicated and uncomplicated groups. However, in complicated group 15 (50%) out of 30 patients had low activity on 7th day and minimum normal value in rest was attained on 10th day. In 6(15%) out of 40 survived cases, values of pseudocholinesterase activity remained lower than the minimal normal value even on the 10th day. Our observations are consistent with the findings of Mutalik et al (1976), Sainani et al (1979) and Chawhan et al (1982).

Pseudocholinesterase Activity in Complicated and Uncomplicated Cases

The present study the levels of pseudocholinesterase started falling after 12 hours of chest pain and the minimum values were seen on 1st day in complicated cases (73.94 + 34.07) while on 2nd day in uncomplicated cases (96.99+16.53). Minimum normal values in complicated and uncomplicated groups were attained on 10th and 7th day respectively.

In complicated and uncomplicated group the pseudocholinesterase activity was 73.94 ± 34.07 and 104.28+26.13 on1st day, 77.97 + 20.00 and 96.99+16.53 on 2nd day, 92.48+20.8 8and120.21+22.97on 3rd day respectively. These values were highly significant statistically (P <.001) (Table 5). These findings are similar to the observations of Basu et al (1972), Vachharajani at a1. (1976) Mutalik et al(1976).

It was observed in this study that minimum normal values in patients with arrhythmias and

pump failure were attained on 7th and 10th day respectively. While Chawhan et al (1982) had inferred that patients with left ventricular failure and arrhythmias had minimum normal values on 10th and after 10 days respectively. Out of 6 cases who had lowest activity of pseudocholinesterase on 10th day, 2 cases expired in further follow up, and 4 patients improved and were discharged in satisfactory condition.

Summary & Conclusions

In myocardial infarction, earlier and rapid fall in the pseudocholinestrase level may have a poor prognosis, a persistent low level in patients having complication may be a grave prognostic sign, so we suggest that determination of pseudocholinesterase in patients of acute myocardial infarction before discharge of patient may be an additional useful test in such type of cases.

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