ORIGINAL

Interpretation of Lesser E.C.G. Changes In Diabetes Mellitus

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Introduction

The manifestations of cardiovascular complications in diabetes are manifold. It may be angina pectoris, myocardial infarction, ventricular failure, syncope and sudden death. Although coronary artery disease affects diabetics excessively it fails to account for all the cardiopathy. Disease of small intramural arteries and arterioles, neurological dysfunction and disturbed metabolic milieu of heart muscle and conducting tissues may all contribute to the disordered cardiac function. Simple assumption that all heart disease in the diabetics is secondary to coronary insufficiency may be misplaced.

As observed according to the Minnesota code the major E.C.G. changes are Q wave abnormalities, precordial R wave abnormalities, and left bundle branch block.

Apart from this there are certain lesser E.C.G. changes limited to ST segment and T wave abnormalities following the same Minnesota code.

Study of electrocardiograms in a multinational study in vascular disease revealed following findings regarding Indian diabetics. Employing Minnesota code, coronary probable was 6.6% in males and 3% in females and coronary possible was 16.6% males and 21.4% females. (W.H.O. 1980).

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The coronary possible is the one that is associated with lesser E.C.G. change due to abnormalities of repolarisation and limited to ST and T changes as mentioned earlier. These ST and T changes in E.C.G. are not always due to ischaemic heart disease. The other different causes implicated may be regional variations, infection and anaemia and may be also due to cardiomyopathy. It has also been contended that transient and reversible metabolic factors associated with diabetes may have contributory role in the E.C.G. abnormalities.

Material and Methods

For this study, 100 diabetics, age group 35-54 years were screened for—

1) Nutritional status by total body mass index.

2) Biochemical examination

This consists of estimation of haemoglobin, total leucocyte count, ESR, blood sugars (Fasting and post- prandial), blood urea, serum sodium and potassium and serum cholesterol using standard method.

3) Cardiac status

This was evaluated by E.C.G. and plain skiagram of the chest for evaluation of cardio-thoracic ratio.

Those showing lesser E.C.G. changes (coronary possible) were given standard exercise test as well as systolic time interval (employing 4 channel minograph recorder) was determined

The lesser E.C.G. changes are interpreted according to the following criteria.

For ST segment

1. ST elevation more than 1mm in L_1 , L_2 , avl, avf, V_5 , V_6 .
2. ST elvation more than 2mm in V_1 , V_2 , V_3 , V_4 .
3. Juctional ST depression + upward sloping in L_1 , L_2 , avl, avf, V_1 - V_6 .
4. ST depression less than —0.5mm horizontal or downwards in L_1 , L_2 , avl, avf, and V_1 - V_6 .
5. ST depression more than 0.5mm and less than 1mm horizontal or downwards in L_1 , L_2 , avl, avf and V_1 - V_6 .
6. ST depression more than 1mm horizonatal or downwards in L_1 , L_2 , avl, avf, and V_1 - V_6 .

For T wave

1. Negative T 5mm in L_I , L_2 , avl, and $V_2 - V_6$.
2. Negative T 3 or 4mm in L_I , L_2 , avl and $V_2 - V_6$.
3. Negative T 1 or 2mm in L_I , L_2 , avl, $V_2 - V_6$.

Such patients were also administered atropine 0.025 mg/kg body weight (Wheeler 1973) and later Isoprenaline 0.06 ug/kg body weight (infusion for 3 minutes) to evaluate the autonomic function. 5 non-diabetic subjects, age-sex matched with normal E.C.G., were employed to provide data for controls, again 5 diabetics, age-sex matched with normal E.C.G. were similarly tested for comparison.

Results

Table:- 1 : Total diabetes cases screened——100

Age group	Duration of diabetes		
	1 -6 years	7 - 13 years	<14 years
35 - 41 M	8	4	3
F	10	6	4
42 - 48 M	6	5	2
F	8	5	4
49 - 54 M	4	6	5
F	6	6	8

Table:- 2: E.C.G. changes and duration of diabetes

X — coronary probable

Y — coronary possible

Age		1 — 6 years		7 — 13 years		> 14 years	
		X	Y	X	Y	X	Y
35 - 41	M	0	1	0	0	0	0
	F	0	1	0	1	0	0
42 - 48	M	0	3	2	1	0	0
	F	0	1	0	1	0	1
49 - 54	M	0	1	0	1	1	2
	F	1	1	1	3	0	2

Table:- 3: Details of abnormalities in 20 diabetes cases with coronary possible findings.

Sex		Low BMI	Low HB% <10gm%	Raised ESR > 40	Raised Sr. cholesterol > 250mg%	C.T. ratio > 1	creatinine	Low K
M	8	1	1	0	2	1	0	0
F	12	2	9	7	8	1	1	2

Table:- % 4: Response to various cardio function tests

Group	STI P/L ratio	Response to Atropine Heart rate	Response to Isoprenaline Heart rate
I - Normal non diabetic No ECG changes	0.26-0.34	80-125 (56.2%)	98-182 (85.7%)
II - diabetics with normal ECG	×	82-112 (32.5%)	90-135 (50%)
III - Diabetics with lesser ECG changes	0.35-0.50	88-112 (28.4%)	96-124 (29.1%)

Discussion

In the study carried out for over all incidence of lesser E. C. G. changes in diabetics as per Minnesota code, it was seen that 20% had such changes. Samuel in 1977 has also recorded non-specific ST-T changes in 27% of diabetic patients. In the retrospective study the total incidence was 38%. The discrepancy might be due to the difference in the number of cases studied.

The distribution according to sexes was 60% for females and 40% for males. Regarding the prevalence of females over males, Sarvotham in 1968 has recorded similar findings. He further went on to attribute this difference between male and female on the basis of anaemia in child bearing period.

The incidence of lesser E.C.G. changes and duration of diabetes mellitus was inversely proportion in present study. Ahuja in 1965 has pointed out the possibility of existing

cardiovascular complications without overt diabetes. Moreover he has clearly mentioned that these changes need not necessarily go together with long duration of diabetes. Talwarker in 1971 has also pointed out that duration of diabetes is not related to the cardiovascular manifestation.

Nutritional (significant anaemia) was 50% and infective causes (ESR more than 45) contributing to non-specific ST changes could be attributed in 35%. Keen in 1979 has attributed some of the lesser E. C. G. changes to anaemia and infection.

Sr. cholesterol was high in 50% with lesser changes. The exercise test was positive in 7 out of 20 cases (35%). In these 7 cases only 4 (20%) had hypercholesterolemia. Since we have studied cholesterol level in peripheral blood only no comment can be made on the cholesterol levels of ventricular wall. High cholesterol content in the ventricular wall was demonstrated by Regan in 1974 in experimental studies in dogs.

In assesment of STI in those cases of lesser changes, 35% had P/L ratio more than 0.345 and in all these cases exercise test was positive. Thus we can conclude that in those cases showing positive exercise test there is LV dysfunction also.

Lloyd in 1975 has postulated the autonomic disturbances of heart in diabetes. He has mentioned the impairment of vagal function as the most easily recognised abnormality.

The response to atropine and isoprenaline was needed to test parasympathetic and sympathetic functional state. In diabetics of longer duration, a relationship on loss of receptor activity could be summarised as responsiveness (as adjudged by heart rate) was minimal in such instances. Again blood pressure response in normal control after initial fall becomes higher than initial value while same value is not seen in the diabetic group. Again, diastolic pressure fall in the diabetics with no recovery while in the normal recovery to initial value is forthcoming.

Conclusions

Although coronary angiography was not done, by clinical assessment it is highly unlikely that the subjects in the present study had any significant coronary artery disease. The lesser E. C. G. Changes, abnormal stress test response, and left ventricular dysfunction in high percentage among diabetic thus could be due to other factors such as nutrition, anaemia, infections or miscellaneous metabolic factors. The parasympathetic and sympathetic system dysfunction was directly related to duration of diabetes while there was no correlation of E. C. G. changes to duration of diabetes.

Bibilography

1. Ahuja, M. M. S. Verma, V. M. and Shanker, U.: Asymptomatic diabetes mellitus in patients with coronary artery diseases. J. Assn. Phys. India, 13, 631, 1965.
2. Keen, H. Jarret, R. J. (1) WHO multinational study of vascular disease in diabetes. (2) Macrovascular disease prevalence. Diabetes Care, 2, 187, 1979.
3. Lloyd, R. H.: Mostyn and Watkins, P. G.: Defective innervation of heart in diabetic autonomous neuropathy. Brit. Med. J., 3, 15, 1975.
4. Regan, T. J. et al.: Altered myocardial function and Metabolism in chronic diabetes mellitus with ischaemia in dogs. Circulation Research. 35, 222, 1974.
5. Samuel Zoneraich, Olga Zoneraich, Jai, J. Rhee, Chung Lio, Mahmood M. Patel: Electrocardiographic findings in diabetic patients with-out clinical evidence of heart involvement. N. Y. State J, Med. 77, 8, 1254, 1977.
6. Sarvotham, S. G. and Berry, J. N.: Prevalance of coronary artery disease in an urban population in north Indian diabetics. Circulation, 37, 939, 1968.
7. Talwalker, C. V.: Inter-relationship between IHD and diabetes mellitus. J. Assn. Phys. India, 19, 139, 1971.
8. Wheeler, T., and Watkins, P. J.: Cardiac denervation in diabetes. Brit. Med. J, 4, 584, 1973.
9. W. H. O. Expert Committee on Diabetes Mellitus. Technical Report series 646, W. H. O., Geneva, 1980.