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SILENT ECG CHANGES IN PATIENTS OF DIABETES MELLITUS

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Contribution
HZ concieved the idea, planned the study and drafted the manuscript. JZ , MIS , AR helped in acquisition of data and did statical analysis. HZ drafted and critically revised manuscript. All authors contributed significantly to the submitted manuscript.

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ABSTRACT

Objectives: To assess the silent ischemic changes in diabetics presented to a tertiary care hospital and to know its association with different variables.

Methodology: A cross sectional study done at Ayub Teaching Hospital (ATH) Abbottabad, from 16th July 2016 to 14th November 2016. Data was collected by filling preformed questionnaire, examining the patients and seeing ECG by interviewers and analyzed using SPSS 23.

Results: Our results shows that out of total 246 patients 45.1% were males with mean age of51.62±11.3years. Of them 11% were type 1 while 89% were type 2 diabetics. About 129(52.4%) of patients had ischemic changes on ECG,19.6% were smokers, 26.8%had family history of DM, 21(8.5%) of hypertension, 99(40.2%) had family history of both DM and hypertension. Among patients 66.3% had raised HbA1C level, 43.1% had dyslipidemia, 7.3% had proteinurea, 22.7% had glycosuria and 18.7% had both proteinuria and glycosuria. Patients having ischemic changes on ECG, 127patients aged more than 30 years and regarding duration of diabetes mellitus (DM) 44 had<5years, 47 had 5-10years, 16 had 10-15years, 17 had 15-20years while 5 had>20years duration of DM.

Conclusion: Ischemic changes were positively associated with age of patients, duration of diabetes, hypertension, raised HbA1C level and hyperlipidemia.

KeyWords: Diabetes mellitus, ECG, silent changes, HbA1C, Lipid profile.

INTRODUCTION

Coronary artery disease (CAD) is highly prevalent and about 8 million people in United States are effected by chronic angina.[1] Diabetic patients have more prevalence of CAD and those having CAD have more occurrence of angina, more severe and extensive disease than non-diabetics with poor outcomes, it even may remain untreated for long time.[2-7] It results in repeated hospitalization, increased economic burden and negatively affect quality of life.[8-10.]

Exercise, controlled blood sugar and blood pressure decreases the risk of CAD, angina, ACS and other atherosclerotic abnormalities. Left ventricular hypertrophy (LVH) is an adaptive response to chronic hypertension i.e. increased peripheral resistance.[17] Patients who develop LVH have great mortality and morbidity related to CAD.[18-21] Cardio-Sis trial showed that lowering of systolic blood pressure to <130 mm Hg in non-diabetic patients having at least one additional risk factor decreases the LVH risk by 39% assessed by electrocardiography.[32]

There are grossly two types of ECG abnormalities minor and major type.[23] Major abnormalities are gross ventricular conduction defects like complete LBBB, RBBB, QRS>120 ms, definite myocardial infarction (MI), possible MI (presence of abnormal minor Q/QS wave plus major ST/T abnormalities), LVH with strain pattern, complete or 2nd degree AV block, pacemaker or AF. While minor abnormalities are Q/QS-wave abnormalities (minor), minor ST/T abnormalities. Tall R waves, right or left ventricular hypertrophy without strain pattern, ST elevation (non-ischemic), incomplete RBBB or LBBB, left or right axis deviation, premature beats (atrial or ventricular), and short PR interval.[24]

Due to neuropathy ischemic heart disease in diabetics is often silent and hence ignored. We conducted our study in order to know the prevalence of silent ischemic changes in diabetics presenting to a tertiary care hospital, and association of ischemic changes with other variables like age, gender, HbA1C, chronicity of Diabetes.

METHODOLOGY

It was a descriptive cross sectional study done at Ayub Teaching Hospital (ATH) Abbottabad, from 16th July 2016 to 14th November 2016 to know silent ischemic changes in ECG of diabetic patients having no complaints regarding cardiovascular system. Patients were selected by using non-probability convenient sampling technique. A pre-tested questionnaire consisting of questions regarding bio data, type of diabetes, history of hypertension, family history of diabetes and hypertension, BMI, ECG changes, HBA1C level, lipid profile, cardiac examination and urine examination was used. All diabetic patients admitted in medicine, cardiology and general surgery ward having nonspecific complaints (complaints other than angina pain) were included in sample population. Patients with known ischemic heart diseases, having angina pain and those having no diabetes were excluded. Data was collected by filling preformed questionnaire through asking the questions given in questionnaire, examining the patients and seeing ECG by interviewers. Data was collected by filling questionnaires after obtaining informed consent and was analyzed using SPSS 23. Descriptive statistics were applied for calculating mean, standard deviation (SD) of continuous variables while frequency and percentages of categorical variables.

RESULTS

Our results shows that out of total 246 patients 111 (45.1%) were males 53.6% were from urban. Of them 27 (11%) were type 1 while 219 (89%) were type 2 diabetics. About 12 (4.9%) were between 15-30 years (yrs.) of age, 33 (13.4%) between 31-45 yrs, 135 (54.9) between 46-60 yrs while 66 (26.8%) were more than 60yrs of age (with mean of 51.62±11.3 years). About 91.7% patients had normal BMI. Complaints of patients are given in table1.

Regarding smoking status and ischemia, 48 (19.6%) were smokers, 6 (2.4%) ex-smokers while 3 (1.2%) did not respond to this question, ($p<0.05$). Of total population 66 (26.8%) had family history of DM, 21(8.5%) of hypertension, 99 (40.2%) had family history of both DM and hypertension while 60 (24.3%) had no family history. Physical examination showed that 224(91.1%) were having normal pulse while 9 (3.6%) had bradycardia and 13 (5.3%) had tachycardia confirmed by ECG. Among patients sinus arrhythmia present in 210 (85.4%).

History showed that 106 (43.1%) of patients had duration of DM less than 5 years, 93 (37.8%) had 5-10years,21 (8.5%) had 10-15 years, 20 (8.1%) had 15-20 years while 6 (2.4%) had more than 20 yrs history of DM. About 52.8% were hypertensive, with 72 (29.3%) mild, 44 (17.9%) moderate while 14 (5.6%) had severe hypertension. Examination showed that 73 (29.7%) patients had normal sensory system while 173 (70.3) had abnormal with 115 had only vibration, 17 had abnormal position and 41 were having both abnormal position and vibration sense. Of the population, 206 (83.7%) had normal 1st and 2nd heart sound audible, 12 had 4th, 20 had both 3rd and 4th heart sound, 2 had friction rub while 6 had diastolic murmur audible. About 129 (52.4%) patients had ischemic changes on ECG detail of which are given table 2. Details of other variables are given in table 3.

Table 1: Complaints of study population (n=246)

Complaints + Drugs used	Frequency (n)	Percentage (%)
Epigastric discomfort	53	21.5
Diaphoresis	3	1.2
Shortness of breath(SOB)	54	22
Left arm pain	15	6.1
One sided body weakness	25	10.2
Diabetic foot	15	6.1
Diaphoresis and SOB	20	8.1
Epigastric discomfort and left arm pain	20	8.1
Cough with sputum	19	7.7
Paresthesia	22	8.9
Metformin	84	34.1
Secretagogues	6	1.2
Insulin	81	32.9
Metformin and secretagogues	51	20.7
Diet control only	24	9.8

Table 2: ECG Changes in study population (n=246)

ECG		Frequency n	Percentage (%)
ST segment changes	Normal	186	75.6
	Elevation	32	13
	Depression	28	11.4
T wave changes	Inversion		2.4
	Tall T wave	21	8.5
Significant Q wave		74	30.1
Presence of LVH		17	6.9
Presence of strain pattern		32	13
Blocks	1st degree	14	5.7
	2nd degree	4	1.6
	3rd degree	10	4.1
	LBBB	31	12.6
Arrhythmias	PVCs	9	3.7
	SVT	19	7.7
	AF	2	0.8

Figure: 3: Investigations performed on study population (n=246)

Variable		Frequency (n)	Percentage (%)
HbA1C	Normal	8	33.7
	<10	133	54.1
	10-15	22	8.9
	>15	8	3.3
Lipid profile	Normal	140	56.91
	Raised Cholesterol	57	23.2
	Raised Triglycerides	34	13.8
	Both	15	6.1
Urine	Normal	117	47.5
	Proteinuria	18	7.3
	Glucoseurea	56	22.7
	Ketonurea	9	3.7
	Gl ucosurea and proteinuria	46	18.7

Figure: 4: Association between variable and ECG in study population (n=246)

Variable		Gender	Ischemic patients (n)	Non Ischemic (n)	Total (n)	P-value
Duration of Diabetes Mellitus	<5yrs.	Male	24	33	57	0.04
		Female	20	29	49	
	5-10yrs.	Male	17	13	30	
		Female	30	33	63	
	10-15yrs.	Male	10	2	12	
		Female	6	3	9	
	15-20yrs.	Male	9	0	9	
		Female	8	3	11	
	>20yrs.	Male	2	1	3	
		Female	3	0	3	
Age (In Years)	15-30	Male	0	6	6	0.04
		Female	2	4	6	
	31-45	Male	11	7	18	
		Female	3	12	15	
	46-60	Male	36	27	63	
		Female	42	30	72	
	>60	Male	15	9	24	
		Female	20	22	42	

Results show a strong association between duration of DM and silent ischemic changes on ECG especially when duration is above 5years ($p = 0.004$). Age of patient has also correlation with ischemic changes on ECG ($p = 0.04$) details of which are given in table 4.

DISCUSSION

Diabetes mellitus is an important risk factor for development of IHD, which causes an increased mortality and morbidity and burden on health delivery system.[8-10] Diabetics especially type 2 usually have developed complications at the time of diagnosis.

Autonomic neuropathy is the main cause of silent ischemic changes and MI in the diabetics. Moreover DM also causes secondary hyperlipidemia which is also cause of atherosclerosis also aggravate the ischemic process.

In our study we have screened patients who came for other problems and here diagnosed as cases of silent cardiac ischemia, ECG was used as a tool for silent ischemic changes which reflects varying risk of CVD. Several reports have shown ECG abnormalities as important for diagnosing CVD.[25,8]

Our study shows that a majority of patients (52.4%) had ischemic changes in their ECG which is more than non-diabetic counterparts specially when age increases. This is supported by studies that diabetics have less angina than non-diabetics due to autonomic neurapathy. [22-24,29-31] Studies showed that prevalence of abnormal ECG findings in general population range from 16% to 32% , which also indirectly supports our study because our population was diabetic patients not general population.[32-36]

We also found an important association between increased HbA1C level and cardiac ischemic changes among diabetics, supported by fact that hyperglycemia worsen the myocardial perfusion through modifications in platelets and oxidative stress and can be reduced by glycemic control, even hyperglycemia below diabetic range also increases risk for CVD.[37,38]

In nutshell we found an important association between silent ischemic changes and DM. Also we found that smoking, sedentary lifestyle, hypertension, raised HbA1C level, hyperlipidemia and advance age are associated with silent ischemic changes on ECG of diabetic patients. And it is supported by study which showed that advance age, increase blood pressure, smoking and increase HbA1C are responsible for major ECG changes of diabetic.[24]

CONCLUSION

Ischemic changes were associated with advance age of patients, increased duration of diabetes, hypertension, raised HbA1C level and hyperlipidemias. Moreover DM was more prevalent in females. Proper screening by doing ECG of diabetics at appropriate interval can pick ischemic changes early and timely intervention can prevent catastrophic events and mortality in such risky patients.

REFERENCES

1. Go AS, Mozaffarian D, Roger VL, Benjamin EJ, Berry JD, Borden WB, et al. Heart disease and strokestatistics—2013 update: a report from the American Heart Association. Circulat 2013;127(1):56-245.
2. Peterson PN, Spertus JA, Magid DJ, Masoudi FA, Reid K, Hamman RF, *et al*. The impact of diabetes on one-year health status outcomes following acute coronary syndromes. BMC Cardiovasc Disord 2006;6:41.
3. Morrow DA, Scirica BM, Chaitman BR, McGuire DK, Murphy SA, Karwatowska-Prokopczuk E, *et al*. Evaluation of theglycometabolic effects of ranolazine in patients with and without diabetes mellitus in the MERLIN-TIMI 36 randomized controlled trial. Circulation 2009;119(15):2032-9.
4. Beltrame JF, Weekes AJ, Morgan C, Tavella R, Spertus JA. The prevalence of weekly angina among patients with chronic stable angina in primary care practices: the Coronary Artery Disease in General Practice (CADENCE) study. Arch Intern Med 2009;169(16):1491-9.
5. Maddox TM, Reid KJ, Spertus JA, Mittleman M, Krumholz HM, Parashar S, *et al*. Angina at 1 year after myocardial infarction: prevalence and associated findings. Arch Intern Med 2008;168(12):1310-6.
6. Duarte R, Castela S, Reis RP, Correia MJ, Ramos A, Pereira AP, *et al*. Acute coronary syndrome in a diabetic population--risk factors and clinical and angiographic characteristics. Rev Port Cardiol 2003;22(9):1077-88.
7. Herlitz J, Wognsen GB, Emanuelsson H, Haglid M, Karlson BW, Karlsson T, *et al*. Mortality and morbidity in diabetic and nondiabetic patients during a 2-year period after coronary artery bypass grafting. Diabetes Care 1996;19(7):698-703.
8. Brown N, Melville M, Gray D, Young T, Munro J, Skene AM, *et al*. Quality of life four years after acute myocardial infarction: short form 36 scores compared with a normal population. Heart 1999;81(4):352-8.
9. Brorsson B, Bernstein SJ, Brook RH, Werko L. Quality of life of patients with chronic stable angina before and four years after coronary revascularisation compared with a normal population. Heart 2002;87(2):140-5.
10. Arnold SV, Morrow DA, Lei Y, Cohen DJ, Mahoney EM, Braunwald E, *et al*. Economic impact of angina after an

acute coronary syndrome: insights from the MERLIN-TIMI 36 trial. Circ Cardiovasc Qual Outcomes 2009;2(4):344-53.

11. Antzelevitch C, Belardinelli L, Zygmunt AC, Burashnikov A, Di Diego JM, Fish JM, *et al*. Electrophysiological effects of ranolazine, a novel antianginal agent with antiarrhythmic properties. Circulation 2004;110(8):904-10.
12. Chaitman BR, Skettino SL, Parker JO, Hanley P, Meluzin J, Kuch J, *et al*. Anti-ischemic effects andlong-term survival during ranolazine monotherapy in patients with chronic severe angina. J Am Coll Cardiol 2004;43(8):1375-82.
13. Rousseau MF, Pouleur H, Cocco G, Wolff AA. Comparative efficacy of ranolazine versus atenolol for chronic angina pectoris. Am J Cardiol 2005;95(3):311-6.
14. Chaitman BR, Pepine CJ, Parker JO, Skopal J, Chumakova G, Kuch J, *et al*. Effects of ranolazine with atenolol, amlodipine, or diltiazem on exercise tolerance and angina frequency in patients with severe chronic angina: a randomized controlled trial. JAMA 2004;291(3):309-16.
15. Chisholm JW, Goldfine AB, Dhalla AK, Braunwald E, Morrow DA, Karwatowska-Prokopczuk E, *et al*. Effect of ranolazine onA1C and glucose levels in hyperglycemic patients with non-ST elevation acute coronary syndrome. Diabetes Care 2010;33(6):1163-8.
16. Timmis AD, Chaitman BR, Crager M. Effects of ranolazine on exercise tolerance and HbA1c in patients with chronic angina and diabetes. Eur Heart J 2006;27(1):42-8.
17. Prisant LM. Hypertensive heart disease. J Clin Hypertens (Greenwich) 2005;7(4):231-8.
18. Koren MJ, Devereux RB, Casale PN, Savage DD, Laragh JH. Relation of left ventricular mass and geometry to morbidity and mortality in uncomplicated essential hypertension. Ann Intern Med 1991;114(5):345-52.
19. Levy D, Garrison RJ, Savage DD, Kannel WB, Castelli WP. Prognostic implications of echocardiographically determined left ventricular mass in the Framingham Heart Study. N Engl J Med 1990;322(22):1561-6.
20. Bikkina M, Levy D, Evans JC, Larson MG, Benjamin EJ, Wolf PA, *et al*. Left ventricular mass and risk of stroke in an elderly cohort. The Framingham Heart Study. JAMA. 1994;272(1):33-6.
21. Sokolow M, Perloff D. The prognosis of essential hypertension treated conservatively. Circulation 1981;23(5):697-713.
22. Verdecchia P, Staessen JA, Angeli F, de Simone G, Achilli A, Ganau A, *et al*. Usual versus tight control of systolic blood pressure in nondiabetic patients with hypertension (Cardio-Sis): an open-label randomised trial. Lancet 2009;374(9689):525-33.
23. Prineas R, Blackburn H. The Minnesota Code Manual of Electrocardiographic Findings: Standards and Procedures for Measurement and Classification. Boston, MA: John Wright-PSG; 1982.
24. Soliman EZ, Backlund JY, Bebu I, LiY, Zhang ZM, Cleary PA, et al. Progression of Electrocardiographic Abnormalities in Type 1. Diabetes During 16 Years of Follow-up: The Epidemiology of Diabetes Interventions and Complications (EDIC) Study. J Am Heart Assoc 2016; e002882.
25. Denes P, Larson JC, Lloyd-Jones DM, Prineas RJ, Greenland P. Major and minor ECG abnormalities in asymptomatic women and risk of cardiovascular events and mortality. JAMA 2007;297(9):978-85.
26. Auer R, Bauer DC, Marques-Vidal P, Butler J, Min LJ, Cornuz J, *et al*. Association of major and minor ECG abnormalities with coronary heart disease events. JAMA 2012;307(14):1497-505.
27. Denes P, Garside DB, Lloyd-Jones D, Gouskova N, Soliman EZ, Ostfeld R, *et al*. Major and minor electrocardiographic abnormalities and their association with underlying cardiovascular disease and risk factors in Hispanics/Latinos (From the Hispanic Community Health Study/Study of Latinos. Am J Cardiol 2013;112(10):1667-75.
28. Soliman EZ, Prineas RJ, Roediger MP, Duprez DA, Boccara F, Boesecke C, *et al*. Prevalence and prognostic significance of ECG abnormalities in HIV-infected patients: results from the Strategies for Management of Antiretroviral Therapy study. J Electrocardiol 2011;44(6):779-85.
29. Marchant B, Umachandran V, Stevenson R, Kopelman PG, TimmisAD. Silent myocardial ischemia: role of subclinical neuropathy in patients with and without diabetes. J Am Coll Cardiol 1993;22(5):1433-7.
30. Murray DP, O'Brien T, Mulrooney R, O'Sullivan DJ. Autonomic dysfunction and silent myocardial ischaemia on exercise testing in diabetes mellitus. Diabet Med 1990;7(7):580-4.
31. Chiariello M, Indolfi C, Cotecchia MR, Sifola C, Romano M, Condorelli M. Asymptomatic transient ST changes during ambulatory ECG monitoring in diabetic patients. Am Heart J 1985;110(3):529-34.
32. Sutherland SE, Gazes PC, Keil JE, Gilbert GE, Knapp RG. Electrocardiographic abnormalities and 30-year

mortality among white and black men of the Charleston Heart Study. Circulation 1993;88(6):2685-92.

33. De-Bacquer D, De-Backer G, Kornitzer M. Prevalence of ECG findings in large population based samples of men and women. Heart 2000;84(6):625-33.
34. Strogatz DS, Tyroler HA, Watkins LO, Hames CG. Electrocardiographic abnormalities and mortality among middle-aged black men and white men of Evans County, Georgia. J Chronic Dis 1987;40(2):149-55.
35. De-Bacquer D, Martins-Pereira LS, De-Backer G, De-Henauw S, Kornitzer M. Prevalenceand correlate of ECG abnormalites in the adult Belgian population. J Electrocardiol 1995;28(1):1-11.
36. Vitelli LL, Crow RS, Shahar E, Hutchinson RG, Rautaharju PM, Folsom AR. Electrocardiographic findings in a healthy biracial population. Atherosclerosis Risk in Communities (ARIC) Study Investigators. Am J Cardiol 1998;81(4):453-9.
37. Deedwania P, Kosiborod M, Barrett E, Ceriello A, Isley W, Mazzone T, *et al*. Hyperglycemia and acutecoronary syndrome: a scientific statement from the American Heart Association Diabetes Committee of the Council on Nutrition, Physical Activity, and Metabolism. Circulation 2008;117(12):1610-9.
38. Khaw KT, Wareham N, Bingham S, Luben R, Welch A, Day N. Association of hemoglobin A1c with cardiovascular disease and mortality in adults: the European Prospective Investigation into Cancer in Norfolk. Ann Intern Med 2004;141(6):413-20