



OBSERVATION OF CARDIAC STATUS IN DIABETICS ABOVE 40 YEARS IN TERTIARY CENTRE OF LUCKNOW, UTTAR PRADESH

Jyoti Verma

Department Of Medicine Integral Institute Of Medical Sciences And Research, lucknow, up, india

ABSTRACT **Objective:** This study was conducted with the aims to evaluate the cardiac status in diabetic mellitus patients above 40 years age and also the relation of aging in diabetic patients to prevalence of cardiac disorders in them. Materials and methods : 30 patients having signs and symptoms of cardiac disease and 30 asymptomatic diabetic patients underwent through echocardiographic examination, exercise stress test equipped with appropriate ancillary techniques and various other non-invasive investigation Majority of the patients lie in age group 51 – 60 years only a few patients presented with signs and symptoms in age above 70 years , mostly men and have duration of diabetes mellitus more than 6 years. Genetic predisposition seems to be the major underlying factor.

KEYWORDS : Diabetes Mellitus , Genetic Predisposition , Cardiac Status.

INTRODUCTION

Diabetes mellitus comprises a group of common metabolic disorders that share the phenotype of hyperglycemia. Several distinct types of DM exist and are caused by a complex interaction of genetics, environmental factors and lifestyle choice. (Alvin C, Powers, 2001).

The prevalence of the disease in adult is around 4% worldwide and this means that 143 million adults are affected worldwide (Park K, 2002) and prevalence will be 5.4% by the year 2025. (Park, K, 2002).

An estimated 30 million person in South East Asia are affected at present, estimated by 2025 there will be nearly 80 million diabetes in this region. The highest among all WHO regions.

Prevalence studies of diabetes mellitus in India utilizing standard WHO criteria for diabetes mellitus, disease prevalence to be 2-4% in rural and 4.0 to 11.6% in urban dweller and impaired glucose tolerance ranging from 3.6 – 9.1% (Park K., 2002).

The disease is more prevalent in the age group of 45-64 years in developing countries as compared to developed countries where it is more prevalent in the age group of more than 65 years. (Park, 2002).

The chronic complication of DM affect many systems. These complication are either vascular or non-vascular.

Vascular complications are further subdivided into macro vascular (coronary artery disease, peripheral vascular disease, vascular disease, cerebrovascular disease) and micro vascular (retinopathy, neuropathy, nephropathy)

Nonvascular complication include gastroparesis, sexual dysfunction and skin changes.

Among the macrovascular complications those affecting CVS disease top the list. In UKPDS, CVS disease accounted nearly 60% of total diabetic deaths. Cardiovascular d/s in diabetic may manifest as coronary artery disease, congestive cardiac failure, cardiomyopathy, disorders of cardiac conduction and rhythm, impaired left ventricular function, other clinical heart disease and autonomic neuropathies affecting the heart.

Laboratory tests in asymptomatic diabetes show a high prevalence of cardiac abnormalities in long standing disease (Bennet, 1975)

Rubler et al. (1972) and Kannel (1974) found microangiopathy in diabetic hearts with unexplained cardiomegaly and heart failure. Stephan et al. (1980) opined about microaneurysms as an important factor in the pathogenesis of heart diseases in diabetes.

Non-coronary involvement of the heart like, cardiomyopathy, preclinical heart disease and autonomic neuropathy are frequently encountered.

Conduction abnormalities namely atrioventricular blocks, Bundle Branch Block, fascicular blocks occurs in diabetes without coronary

artery disease and by hypertension increased incidence of bradycardia, other arrhythmias and third degree AV block is seen commonly in diabetes and they are at particular risk of developing dangerous arrhythmias and sudden death (Nihalani KD, 1986). The exact cause and pathogenesis of development of these conduction defects without features of ischaemia infarction is still debatable. But based on different studies it was concluded that enhanced degenerative process involving conduction tissue because of metabolic disorder associated with diabetes or diabetic micro-angiopathy could be the possible cause of high incidence of cardiac conduction defects seen in diabetes.

The patients of conduction defects are prone to develop dangerous arrhythmias even in well controlled state of the disease.

12- lead electrocardiography remains the most convenient, reliable, cheap and easily accessible method for detection of these defects.

This will help in detecting conduction defects in diabetes and giving them proper advice in appropriate time.

The majority of death certificates (about 75%) listing diabetes also list accompanying cardiovascular condition. Framingham heart study and American heart association has established cardiovascular disease as cause of most of the deaths in diabetes patients.

Ischemic heart disease's are involved in approximately 50-60% of deaths in NIDDM and put upto 15-25% of deaths in IDDM.

Myocardial infarction in diabetic is more extensive and post MI recovery and development of collaterals is also poor leading to as many as 20% death in diabetes.

Waller et al. found that diabetics had much greater extent of coronary artery disease than non-diabetic matched as to age, sex and duration of disease.

Recently another interesting aspect of CAD has come to fore and that is silent or asymptomatic CAD. Anginal pain is a poor indicator and underestimates the frequency of significant cardiac ischemia. Many studies have confirmed that silent or atypical presentation of myocardial ischemia is common in diabetes. It was postulated that myocardial ischemia is common in diabetes. It was postulated that myocardial ischemia may also be silent in these patients. Many workers have reported a greater prevalence of silent ischemia in diabetes than non-diabetics.

Approximately 25-30% of diabetic patients suffer from silent ischemia. Absence of pain does not mean absence of underlying heart damage. Heart has some in built reserve capacity to suffer a certain amount of scarring. But any further insult or ischemia leading to cardiomyopathy is the commonest cause of heart failure in USA. So assessing Angina as a sign post of the activity of CAD and detecting silent or asymptomatic CAD may be of particular importance in diabetes given the recognized effects of this disease on the incidence, natural history and therapeutic outcome of CAD in these patients.

It is well known that diabetics also suffer from peripheral and autonomic neuropathy in long standing cases.

Nerve dysfunction may be responsible for inability to perceive pain associated with myocardial ischemia. Diabetic autonomic neuropathy may involve the cardiac afferent sympathetic system, which is a part of the pain perception pathway from myocardial pain receptors to the cerebral cortex. The development of myocardial ischemia in patients with such a neuropathy is associated with more frequent silent myocardial ischemia.

We plan to study symptomatic and asymptomatic diabetic subjects to define the frequency of silent myocardial ischemia. This study is designated to evaluate silent/asymptomatic myocardial ischemia in diabetics by 24 hour ambulatory ECG monitoring and exercise stress test.

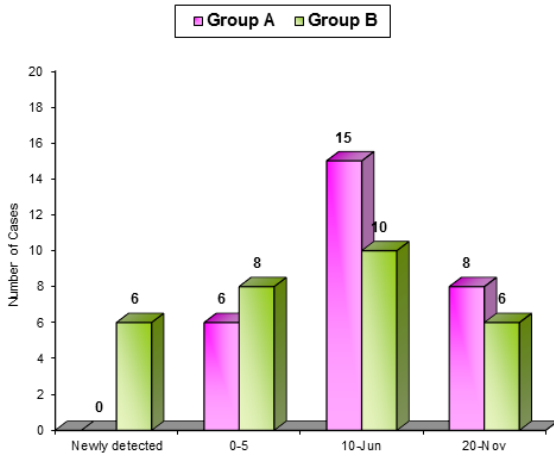
MATERIAL AND METHODS

SUBJECT : Patients were selected from the General Medicine OPDs, Medical wards and staff of the Integral Institute of medical sciences and research, Lucknow, UP.

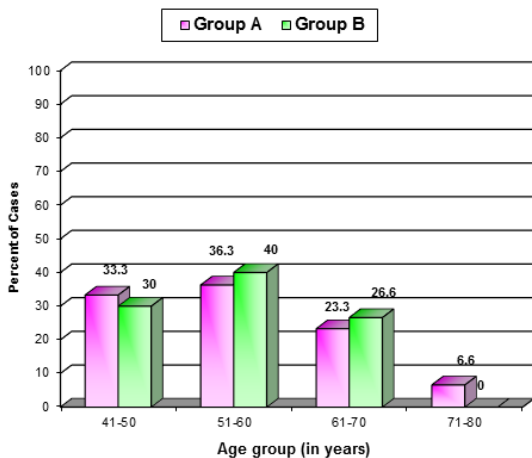
Patients above 40 years were subjected to random blood glucose examination and those who had abnormal random blood glucose levels were subjected to oral glucose tolerance test; 75 g glucose were given to these subjects after an overnight fast and fasting and postprandial venous plasma glucose concentrations were measured. Those declared diabetic then subjected to various tests for the evaluation of cardiac status.

OBSERVATION AND CONCLUSION :

Showing distribution to duration of diabetes mellitus

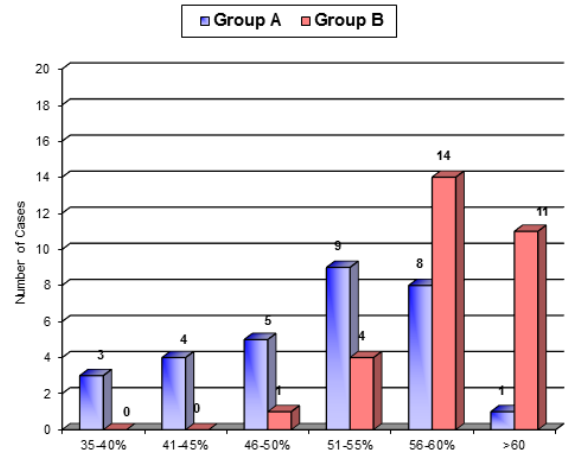


Showing distribution of patients according to age groups

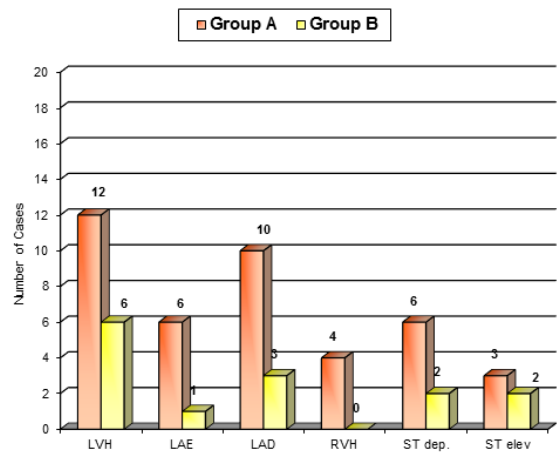


Showing distribution of patients according to autonomic dysfunction

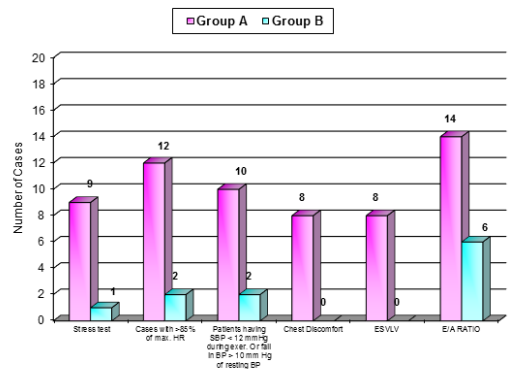
Showing observation of ejection fraction at rest by echocardiography in study group



Showing distribution of patients according to ECG abnormalities



Observations of treadmill testing in study group



Inclusion and exclusion criteria were followed as per decided. Selection criteria is as follows :

Group A : Diabetic patients above 40 years with sign and symptom of cardiac disease.

Group B : Diabetic patients above 40 years without sign and symptom of cardiac disease.

AGE IN GROUP A AND B :

Inoguchi T, Yamashita T, Umeda F, Mihara H (2000 January) has found that elderly NIDDM patients (aged over 60 years) had an extremely high prevalence (estimated 26.3%) of silent myocardial ischemia.

In present study, maximum number of patients (in group A) lie in the age group 51 – 60 years while in group B it is also in the same age group. So, this shows that investigations should be started in early age as soon as patients are declared diabetic.

SEX DISTRIBUTION :

Framingham Heart Study, 1974, in this prospective study of 5000 individuals over an 18 years follow up period, the frequency of congestive heart failure was more than twice as high in diabetic men than in the nondiabetic cohort, while it was increased fivefold in diabetic women.

In present study, dominance of male patients in number may be a reflection of their openness to visit hospital and may reflect sex distribution in employment.

DURATION OF DISEASE :

Hamby et. al. (1974), noted increased incidence of diabetics with idiopathic cardiomyopathy. On a study on 73 patients, he found duration of diabetes were quite variable ranging from 1 year to 11 years.

In present study, majority of patients has duration of diabetes more than 6 years in both group A (80%) and group B (53.3%).

ECG MANIFESTATIONS :

Group A study sample showed maximum number of patients (40%) having left ventricular hypertrophy (P-value > 0.05, Not significant). 33.3% had left axis deviation (P-value < 0.05; significant) and 20% had ischemic changes in ECG. 20% patients showed left atrial enlargement (P-value < 0.05, significant), thus changes of cardiomyopathy in these patients. ST elevation is present only in 10% patients (P-value < 0.001, Highly significant). Similarly, group B showed 20% patients having left ventricular hypertrophy and 10% patients had left axis deviation. Only 6.6% had ST elevation during their examination.

EJECTION FRACTION :

Various studies showed reduced ejection fraction in diabetics. Seneviretne et. al. in (1977) shown diabetic patients with evidence of microangiopathy had lower fractional shortening as compared to control group. Shapiro et. al. (1980) and Abenmovoli et. al. (1981) reported similar findings.

In this study, group A patients showed 30% with ejection fraction in range between 51 – 55%. 26.6% patients had ejection fraction 56 – 60%. Thus, group A patients had lowered ejection fraction because of impaired systolic performance of left ventricle due to cardiomyopathy. Cardiomyopathy can be because of ischemia and interstitial fibrosis.

13% cases in Group A had ejection fraction of 41-45% (P-value < 0.05, significant).

3.3% cases in Group A and 36.3% cases in Group B had ejection fraction > 60%. (P-value < 0.005; highly significant).

DISCUSSION ON CONDUCTION BLOCK IN DIABETICS :

First degree AV block, right bundle branch block (RBBB), Right bundle branch block with left anterior hemi block. (LAHB) and left bundle branch block (LBBB), Mobitz type II block were detected – observations are as follows –

	Group A	Group B
1° AV Block	- 10%	6.6%
RBBB	- 13%	6.6%
RBBB + LAHB	- 6.6%	0%
Mobitz type II block	- 6.6%	0%

P-value > 0.05 (Not significant).

Various studies have shown high prevalence of cardiac conduction defects in normotensive non-ischemic diabetics. Coronary artery disease has been accepted in the past as an important cause of heart block in diabetics (Harris et. al. 1969). However, other studies showed that areas of fibrosis involving the conduction system with or without scarring/fibrosis of the myocardium also resulted into heart block (Zoob and Smith, 1963). M.J. Davies (1971) opined about idiopathic bundle branch fibrosis. RBBB was observed in 10% and 6.6% in group A and group B respectively. Dutta et. al. (1975), had reported 6.11% BBB amongst 817 diabetes patients without evidence of myocardial infarction out of this 5.87% were having RBBB.

Dutta et. al. (1975), had opined that high incidence of RBBB could be due to degenerative changes affecting the slender right bundle branch selectively as a part of diabetic process.

Zoneraich et al. (1977), have shown electro-cardiographic abnormalities in diabetes are independent of duration of the disease, treatment and degree of glucose control.

Singh et al. (1978), reported 9.25% of conduction defects in diabetics with no clinical or electrocardiographic evidence of ischemic heart disease compared to those diabetics with definite evidence of CAD (4.6%). They observed RBBB in 5.3% of diabetics.

Ahuja MMS (1983) has quoted that diabetic cardiac disease is related neither to the duration nor to the quality of control of diabetes mellitus. It was quoted that microangiopathy of the heart may be responsible for complications like conduction defects in diabetes. Blandford and Burden (1984) opined about microangiopathic rather than arteriosclerotic changes in the diabetic heart responsible for bundle branch defects. They reported cardiac conduction defects in 15.5% of normotensive non-ischemic diabetes as compared to the patients of severe hypertension (9%).

K.C. Samal et. al. (1986) detected ECG changes in (25.5%) cases of type II diabetes. Mishra et. al. (1984) reported RBBB in 7.33% of diabetics. They had evaluated 41 cases of diabetes without any history or evidence of angina, myocardial infarction, hypertension, smoking or obesity.

Nahalni KID (1986) had opined that bradycardia, arrhythmia and third degree AV block are commonly seen in diabetics and they are prone to develop dangerous arrhythmias and sudden death. Both coronary artery disease are primary degeneration and fibrosis of conduction system are responsible for these changes.

Gupta R.C. et. al. (1987) reported conduction defects in only 3.4% of the 292 normotensive ambulatory diabetic and none in controls. They have shown that ECG abnormalities in diabetes patients were not related to duration of the disease, type of the treatment and blood glucose levels. They also convicted subclinical myocardial/coronary vessel disease for electrocardiographic abnormalities in diabetic hearts without detectable cardiac involvement.

Dutta et. al. (1991) have opined about idiopathic fibrosis extending from bundle of HIS to A-V node along with fibrosis of adjacent myocardium without myocardial infarction as a cause of chronic heart block.

Thus, enhanced degenerative process involving A-V node and bundle of his system because of metabolic disorder associated with diabetes or diabetic microangiopathy could be the possible cause of conduction tissue involvement in diabetes without evidence of coronary artery disease.

In present study, RBBB plus left anterior hemi block is present in 6.6% of group A patients but group B patients had shown none. Also, Mobitz type II block is present in 6.6% of group A study sample but none in group B shows much.

ECHOCARDIOGRAPHIC MEASUREMENTS :

Robert Hamby and Co-workers in 1974 in their study on patients of cardiomyopathy, found a significantly increased association with diabetes. They noted that LV end diastolic volume at rest was abnormally increased in diabetic patients.

Zoneraich et al. 1977 found LV end systolic volume in diabetes without clinical heart disease to be higher than control but this was not statistically significant.

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