Original Research Paper

Internal Medicine

STUDY OF CARDIOVASCULAR MANIFESTATIONS OF AUTONOMIC NEUROPATHY IN DIABETES MELLITUS

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INTRODUCTION :-
Cardiac autonomic neuropathy(CAN) is defined as the "impairment of cardiovascular autonomic control in patients with established DM following exclusion of other causes. Hyperglycemia and hyperinsulinemia induce metabolic alteration leading to oxidative stress and demyelination. Cardiac autonomic neuropathy independent risk factor for cardiovascular events.

AIMS AND OBJECTIVES:
1. To study the prevalence of cardiac autonomic neuropathy in type 2 diabetes vs type 1 diabetes mellitus.
2. To study the different manifestations of cardiac autonomic neuropathy
3. To study and grade the different signs and symptoms .
4. To study the correlation of clinical profile of the patients with the severity of diabetes cardiac autonomic dysfunction.
5. To study the progression of cardiac autonomic dysfunction with duration if diabetes.

METHOD:
Study design-Observational clinical study

Inclusion Criteria:
50 patients of type 2 diabetes and 10 patients of type 1 with clinical features suggestive features of cardiovascular autonomic neuropathy in age group 15-70 years.

Exclusion Criteria:
• History of alcohol, cigarette addiction
• Severe LV dysfunction / congestive cardiac failure.
• Neurological diseases
• Pregnancy
• HIV/AIDS
• Diabetic ketoacidosis

For evaluation of CAN panel of 5 clinical tests-heart rate response to expiration-inspiration, standing(30:15 ratio), valsava maneuver and blood pressure response to standing ,isometric exercise. ECG, EMG NCV, USG KUB and 2d echo were also carried out. For statistical analysis Z test for quantitative data and chi square for qualitative data.
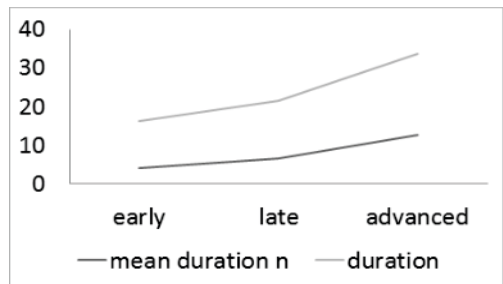
RESULTS:
Age And Sex Wise Distribution Of T1dm Vs T2dm
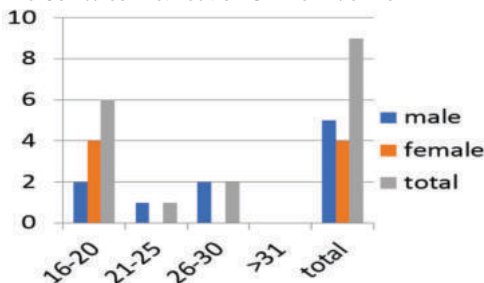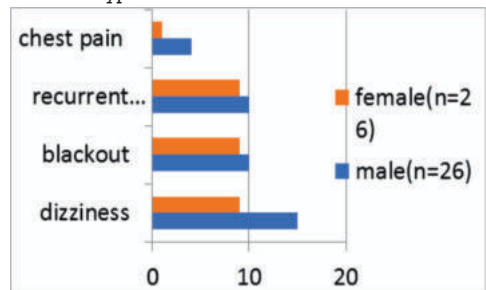

Type 1 Dm
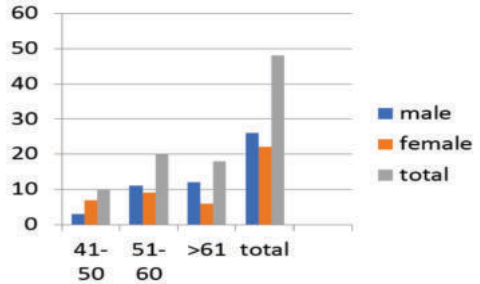
Type 2 Dm

Correlation Of Autonomic Dysfunction In Type 2 Dm With Mean Duration Of Diabetes.
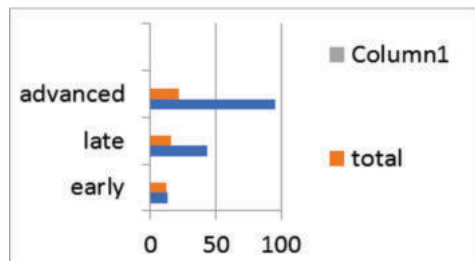

Prevalence Of Symptoms Associated With Cardiac Autonomic Dysfunction In Type 2 Dm.
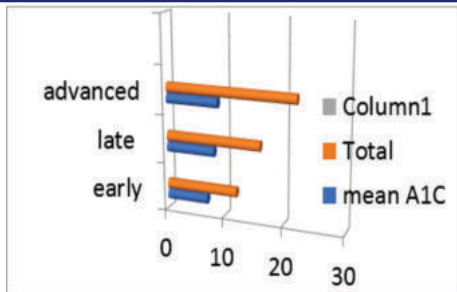

Correlation Of Ophthalmological Manifestations:
Normal fundus examination was predominant in early CAN while NPDR and PDR were predominant in advanced CAN. Severe retinopathy is an independent predictor of CAN.

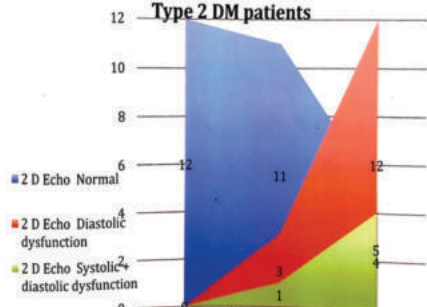Correlation Of Mean Urine Microalbuminuria Values With Can .

Correlation Of Mean A1c With Autonomic Dysfunction

1.   WHO diabetes programme.
2.   Federation of IDF Diabetes atlas 2012 update.
3.   Kannel WB, mc gee dl diabetes and cardiovascular diseases.
4.   Ewing dj, Campbell, clarke-natural history of diabetic autonomic neuropathy.
5.   Harrisons principle of internal medicines

Correlation of 2D Echo findings with CAN in Type 2 DM patients

RESULTS AND DISCUSSION

1.   In both type 1 and 2, there was increase in severity of CAN with increase in duration of diabetes, waist hip ratio ,age and HbA1c
2.   In type, there is positive correlation between urine micro-albuminuria and CAN.
3.   In type 2 DM,
•   Elevated triglycerides,
•   Presence of LV dysfunction
•   Orthostatic hypotension
•   Prolonged QTc(>440ms)
•   Resting pulse rate >100
•   Are independent predictors of advanced CAN.

CONCLUSION:

The vagus nerve tends to be involved early In CAN resulting in reduction of parasympathetic activity and sympathetic predominance which continues till the latest stage of CAN when sympathetic denervation ensues which relates clinically with postural hypotension.

The clinical manifestations range from-
•   Resting tachycardia (90-130bpm)
•   Exercise intolerance.
•   Orthostatic hypotension.
•   Silent ischemia.
•   Cardiomyopathy and LV dysfunction.
•   Cardiac arrhythmias.

•   CAN is a very common and often undiagnosed complication of DM. It is associated with significant increase in morbidity and mortality and plays an important role in pathogenesis of diabetic cardiomyopathy and micro vascular complication.
•   Before CAN is symptomatic and evident clinically ,pts might have subclinical CAN for years
•   Recent guidelines have recommended screening of CAN in DM patients. Use of HRV and spectral analysis has simplified testing but it is still time consuming.
•   Improving glycemic control, lifestyle changes and CVD risk factors management are the mainstay of treatment which slow the progression of CAN rather than reverse it
•   However, better understanding of CAN pathogenesis is required in order to develop disease modifying treatements.

REFERENCES