www.jmscr.igmpublication.org
Impact Factor (SJIF): 6.379
Index Copernicus Value: 71.58
ISSN (e)-2347-176x ISSN (p) 2455-0450
crossrefDOI: https://dx.doi.org/10.18535/jmscr/v6i7.05

IGM Publication
Journal Of Medical Science And Clinical Research
An Official Publication Of IGM Publication

Cognitive decline in newly diagnosed Type 2 Diabetes and the effect of good glycemic control on cognitive function

Authors
Sunil Prasobh P[1*], Monisha H[2]
[1]Associate Professor, Department of Internal Medicine, Government Medical College Thiruvananthapuram, Kerala, India
[2]Senior Resident, Department of Internal Medicine, Government Medical College Thiruvananthapuram, Kerala, India
*Corresponding Author
Dr Sunil Prasobh P
Department of Internal Medicine, Government Medical College Thiruvananthapuram-695011, Kerala, India
Email: *sunilprasobh@gmail.com*

Abstract
Background: *Diabetes is a complex metabolic disease with devastating effects on multiple organ systems, including the brain. The latter complication is often referred to as "diabetic encephalopathy" and is characterized by mild to moderate impairments in cognitive function , associated with an increased risk of dementia .The aim of our study was to characterize the clinical profile of patients newly diagnosed with Type2 Diabetes (within six months of onset) and to assess the dementia score of these patients by MMSE(Mini Mental state examination) at presentation and to find out the prevalence of cognitive impairment in this population. Also an attempt was made to study the relation of glycemic control with the dementia scores. These patients were also followed up at one year to assess their MMSE scores.*
Materials and Methods: *140 patients, newly detected with Type2 Diabetes (within six months of onset) were enrolled in the study. Clinical details were obtained with a structured proforma. MMSE scores of the patients were measured at baseline and after one year. The baseline MMSE score was compared with the clinical profile of these patients.*
Results: *The mean age of the cases were 56.5 years with slightly higher male predilection (M:F=1.41:1). On detailed evaluation hypertension was seen in 55%, Coronary artery disease in 35.7%, dyslipidemia in 36.4% , alcoholism in 44.1%, and smoking in 45% of cases. There were also microvascular complications like Diabetic Nephropathy in 17.1%, Diabetic Retinopathy in 10.7% of cases. Most of the cases had poor metabolic control with 75.7% having FBS of >130, 68.6% having PPBS >180 and 78.6% having HbA1c >7. The mean MMSE score at baseline were 24.1 and at one year were 24.6. There was statistically significant difference in dementia scores as assessed by MMSE at baseline and one year in our cases ($p<0.001$).The prevalence of cognitive dysfunction as assessed by MMSE was 30%. There was a statistically significant fall in MMSE with presence of Diabetic Nephropathy (MMSE $p=0.002$) and Diabetic Retinopathy (MMSE $p=0.004$). Poor glycemic control was linked statistically to a low MMSE score. For lower MMSE score there was a statistically significant increase in the PPBS values (MMSE,$p=0.013$) and HbA1c levels (MMSE score,$p=0.002$).*

Conclusion: *A significant proportion (30%) of newly diagnosed Type2 Diabetes (within 6 months) patients had impairment of cognitive function. Cognitive dysfunction in patients with Type2 Diabetes was directly related to poor glycemic status. Among the glycemic targets, Post prandial blood glucose values and HbA1C had better correlation with cognitive decline than Fasting plasma glucose levels. Presence of microvascular complications of Type2 Diabetes such as Nephropathy and Retinopathy predicted cognitive decline. No definite association was seen between cognitive dysfunction and macrovascular disease especially Coronary artery disease in newly diagnosed Type 2 Diabetes. Cognitive dysfunction was independent of other vascular risk factors such as Hypertension, Dyslipidemia and Smoking. Adequate glycemic control in early stages of Type 2 Diabetes may result in improvement in cognitive function.*
Keywords: *Type2 Diabetics, MMSE, Cognitive decline, Glycemic control.*

Introduction

Type 2 diabetes is a common metabolic disease with a rising global prevalence. According to IDF atlas 2017 around 425 million patients are worldwide with diabetes. In India, diabetes is rapidly gaining the potential status of an epidemic, with India being one of the topmost countries with maximum affection. Kerala is considered to be the diabetes capital, with prevalence nearing 20%.[(1)] Type 2 diabetes mellitus (T2DM) is characterized by abnormalities in carbohydrate and fat metabolism, chronic hyperglycemia, insulin resistance, and a relative insulin secretion defect. Type 2 diabetes mellitus (T2DM) is a heterogeneous metabolic disorder associated with an increased risk for central nervous system disorders.[(2)] Diabetes leads to electrophysio-logical, structural, neurochemical, and degenerative neuronal changes aptly referred to as 'Diabetes Encephalopathy' that lead to cognitive functioning limitations.[(3)]

Diabetes induced cognitive decline is seen to have multifactorial etiology with multiple mechanisms being implicated for the same, including blood glucose and direct effect of chronic hyperglycemia causing brain damage, blood lipid, blood pressure, insulin resistance, hypoglycemia, chronic micro and macro-vascular complications termed the diabetic vasculopathy, limbic-hypothalamic-pituitary-adrenal axis (LHPA) dysregulation as a part of stress response, formation of advanced glycation end products, production of inflammatory cytokines, the effect of oxidative stress on the brain and finally the presence of diabetes-related depression in some individuals.[(4,5,6)] The effects of diabetes on the peripheral nervous system have been well established and well studied but the effects of diabetes on the central nervous system have been less clear.

There are numerous case-control studies done so far showing that cognitive performance of adults with T2DM are on an average 0.6±1.0 SD poorer than that of age- and sex matched non-diabetic adults.[(7)] And also specifically it has been seen that not all domains of cognitive function are impaired by T2DM to an equal extent. Memory function in elderly patients are preferentially more affected and it has been suggested that T2DM promotes accelerated ageing of the brain.[(8)]

Cognitive decline in diabetes is usually seen as an intermediate stage between that of normal ageing and dementia. Since dementia is largely preventable in this group of people if identified in the early stages, it becomes imperative as a matter of public health importance for early identification of cognitive impairment in diabetics. Keeping this in mind, this study was undertaken to characterize any risk factors that might lead to accelerated cognitive decline, and assess the dementia scores of these patients by MMSE (Mini Mental Scale Examination). The main aim of the study was (1) To find out the prevalence of cognitive dysfunction in newly diagnosed Type 2 Diabetes. (2) To assess the association between the level of glycemic control and cognition dysfunction in newly diagnosed T2 DM and also (3) To find out the association between macrovascular and microvasular complications of diabetes with cognitive function in newly diagnosed Type2 diabetes.

Material and Methods

This was a descriptive observational study on the prevalence of cognitive dysfunction in patients with newly diagnosed with Type2 Diabetes and to characterize the clinical profile of those patients. This study was conducted as a hospital based study in the general medical wards and Outpatient Department of the Internal Medicine, Government Medical College Trivandrum, a Tertiary care centre in Kerala for a period of one year. All patients meeting the eligibility criteria, and who reported at the study setting were enrolled in the study (census type) and the effective sample size came to be 140. Patients were diagnosed to have diabetes as per ADA criteria.

The exclusion criteria for the study were

1) Cortical dementia and subcortical dementia due to other causes
2) Neuropsychiatric disorder
3) Encephalopathies like metabolic, hepatic, uremic, viral etc
4) Endocrine dysfunction
5) Severe neurodegenerative disorders
6) Electrolyte imbalance
7) Head injuries and altered consciousness
8) Other diseases interfering with cognitive function

A detailed history taken including past history of any comorbidites sought for. Patients were clinically evaluated and examined focusing on presence of other complications of T2DM. The clinical assessment of cognitive function will be graded according to the Mini Mental State Examination (MMSE) which is a brief 30-point questionnaire test that is used to screen for cognitive impairment. It is also used to estimate the severity of cognitive impairment and to follow the course of cognitive changes in an individual over time. It consists of 19 questions in 5 categories: Orientation, Memory, Calculation, Language and 3 step command. The severity distribution of MMSE scores on admission was divided into 3 categories as Severe <18, Mild 18-23, and No impairment 24-30 and repeat assessment done after a follow up period of one year. The validity of the MMSE as a screening tool for detecting dementia has been extensively studied.[9] Laboratory tests included complete Hemogram, Liver function test, Renal Function Test, Fasting Lipid profile, Electrolytes, FBS, PPBS, HbA1C, Urine Protein: Creatinine and Fundus examination. Glycemic targets were set at FBS <130, PPBS <180 and HbA1C <7.0%

Statistical Analysis

The data was collected and analyzed using statistical tools. Mean, median, percentage sampling were used for quantitative analysis and Statistics Package for Social Sciences, SPSS (version 16) was used for qualitative analysis to derive the values of probability (p value). Chi square test was used appropriately for analysis of variables.

Results

Of the 140 patients studied, the age varied from 32 years to 77 years. The Mean age was 56.5 ± 9.53.The maximum patients was seen in the age groups between 51 to 60 (57%) and 61 to 70(42%). (Table 1). There were 82 males (58.6%) and 58 females(41.4%) enrolled in the study. (Table 2).About 3.65% patients were illiterate. The number of patients who had education in the form of primary ,high school, higher secondary, degree and above were 47.1%, 31.4%, 12.9%, 5% respectively.(Table 3).About 45% of patients had hypertension and 36.4% had dyslipidemia. About 45% gave history of smoking. The patients who had a FBS≤ 130mg% were 24.3 % and PPBS ≤ 180mg% were 52.9%.Only 21.4% of patients had an HbA1c ≤ 7%.(Table 4,5,6). 35.7% had coronary artery disease at the initial stage. Te patients who had Diabetic nephropathy and retinopathy were 17.1% and 10.7% respectively. (Figure 1,2). 62.9% of patients were treated with oral drugs alone while 37.1% were treated with drugs and insulin.(Figure 3).

About 30% had MMSE scores <24, thus demented at baseline. None had scores less than 10, ence no severe dementia. The mean MMSE

score was 24.1 at baseline and 24.6 at one year follow up. (Table 7) (Figure 4). There is statistically significant difference ($p<0.001$) in MMSE scores at baseline and one year follow up.

No statistical correlation was seen with MMSE score and presence of CAD. (Table 8). There is significant statistical correlation between diabetic nephropathy and retinopathy and cognitive decline in T2DM as evidenced by MMSE scores.29.When patients with moderate cognitive decline were evaluated, 29.2% had diabetic nephropathy while 33.3% had diabetic retinopathy. (Table 9,10). There is no statistical correlation between baseline FBS and MMSE score. (Table 11) There is statistical correlation between adequately controlled diabetes as evidenced by PPBS <180 and HbA1c <7% and baseline MMSE scores. (Table 12,13).

Table 1: Percentage distribution of the sample according to age

Age	Frequency	Percent
<40	9	6.4
41-50	24	17.1
51-60	57	40.7
61-70	42	30.0
>70	8	5.7
Total	140	100.0

Mean ± SD 56.5 ± 9.53

Table 2: Percentage distribution of the sample according to sex

Sex	Frequency	Percent
Male	82	58.6
Female	58	41.4
	140	100.0

Table 3: Percentage distribution according to level of education

Education	Frequency	Percent
Illiterate	5	3.6
Primary	66	47.1
High school	44	31.4
Higher secondary	18	12.9
Degree and above	7	5.0
Total	140	100.0

Table 4: Percentage distribution according to Fasting blood glucose values

FBS	At presentation		Follow up at one year	
	N	%	N	%
≤130	34	24.3	71	50.7
>130	106	75.7	69	49.3
Total	140	100.0	140	100.0

Table 5: Percentage distribution according to Post prandial blood glucose

PPBS	At presentation		Follow up at one year	
	N	%	N	%
≤180	44	31.4	74	52.9
>180	96	68.6	66	47.1
Total	140	100.0	140	100.0

Table 6: Percentage distribution of the sample according to HbA1c

HbA1C	At presentation		Follow up at one year	
	N	%	N	%
≤7	30	21.4	55	39.3
>7	110	78.6	85	60.7
Total	140	100.0	140	100.0

Table 7: Dementia assessment at baseline and after one year using MMSE(Mini Mental state examination)

MMSE	At baseline		Follow up at one year	
	N	%	N	%
Moderate 10-18	14	10.0	12	8.6
Mild 19-23	28	20.0	24	17.1
Normal >/= 24	98	70.0	104	74.3
Total	140	100.0	140	100.0

McNemar test p=0.064

Table 8 Distribution of CAD and baseline MMSE

CAD	MMSE						**Total**	
	Moderate		Mild		Nil			
	N	**%**	**N**	**%**	**N**	**%**	**N**	**%**
Yes	6	12.0	6	12.0	38	76.0	50	100.0
No	8	8.9	22	24.4	60	66.7	90	100.0
Total	14	10.0	28	20.0	98	70.0	140	100.0

χ^2 =3.200 df =2p=0.202

Table 9 Distribution of Diabetic nephropathy and baseline MMSE

Nephropathy	MMSE						**Total**	
	Moderate		Mild		Nil			
	N	**%**	**N**	**%**	**N**	**%**	**N**	**%**
Yes	7	29.2	5	20.8	12	50.0	24	100.0
No	7	6.0	23	19.8	86	74.1	116	100.0
Total	14	10.0	28	20.0	98	70.0	140	100.0

χ^2 =12.306 df =2 p=0.002

Table 10 Distribution of Diabetic retinopathy and baseline MMSE

Retinopathy	MMSE						**Total**	
	Moderate		Mild		Nil			
	N	**%**	**N**	**%**	**N**	**%**	**N**	**%**
Yes	5	33.3	1	6.7	9	60.0	15	100.0
No	9	7.2	27	21.6	89	71.2	125	100.0
Total	14	10.0	28	20.0	98	70.0	140	100.0

χ^2 =10.880df =2 p=0.004

Table 11 Distribution of FBS and baseline MMSE

FBS	MMSE						**Total**	
	Moderate		Mild		Nl			
	N	**%**	**N**	**%**	**N**	**%**	**N**	**%**
≤130	3	8.8	11	32.3	20	58.8	34	100.0
>130	10	9.4	19	17.9	77	72.6	106	100.0
Total	13	10.0	30	20.0	97	70.0	140	100.0

χ^2 =3.22 df =2 p=0.19

Table 12 Distribution of PPBS and baseline MMSE

PPBS	MMSE						**Total**	
	Moderate		Mild		Nil			
	N	**%**	**N**	**%**	**N**	**%**	**N**	**%**
≤180	1	2.3	5	11.4	38	86.4	44	100.0
>180	13	13.5	23	24.0	60	62.5	96	100.0
Total	14	10.0	28	20.0	98	70.0	140	100.0

χ^2 =8.679 df =2 p=0.013

Table 13 Distribution of HbA1c and baseline MMSE

HBA1C	MMSE						**Total**	
	Moderate		Mild		Nil			
	N	**%**	**N**	**%**	**N**	**%**	**N**	**%**
≤7	0	0.0	1	3.3	29	96.7	30	100.0
>7	14	12.7	27	24.5	69	62.7	110	100.0
Total	14	10.0	28	20.0	98	70.0	140	100.0

χ^2 =13.00 df =2 p=0.002

Figure1: Percentage distribution according to Diabetic Nephropathy

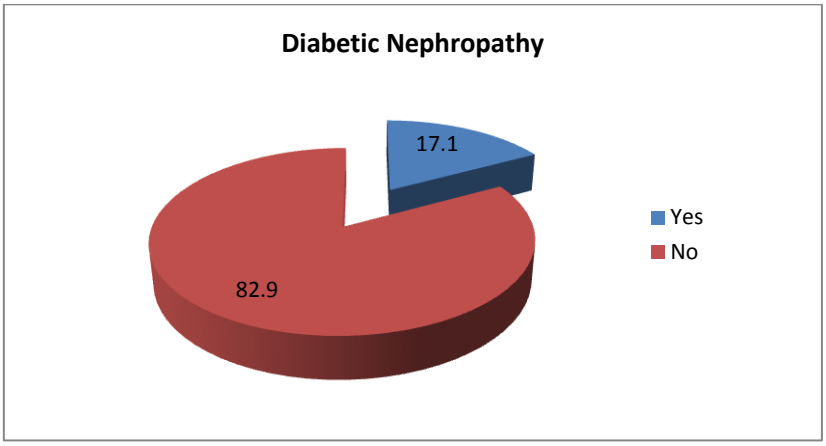


Figure 2 Percentage distributions according to diabetic retinopathy

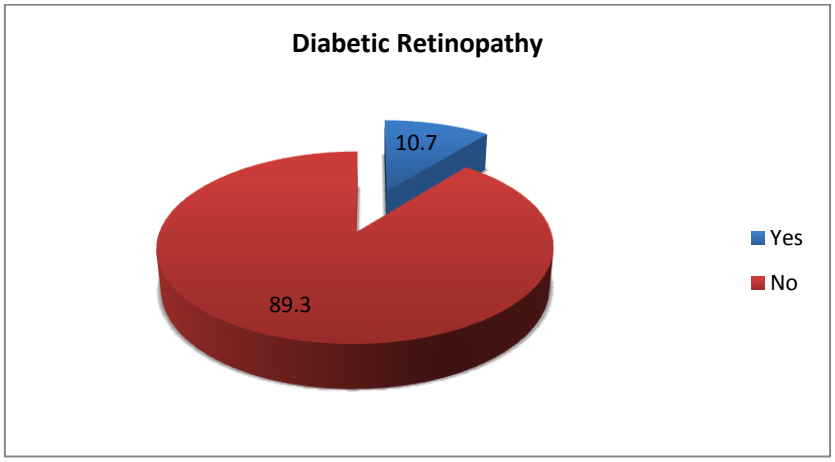


Figure 3 Percentage distributions according to diabetes treatment

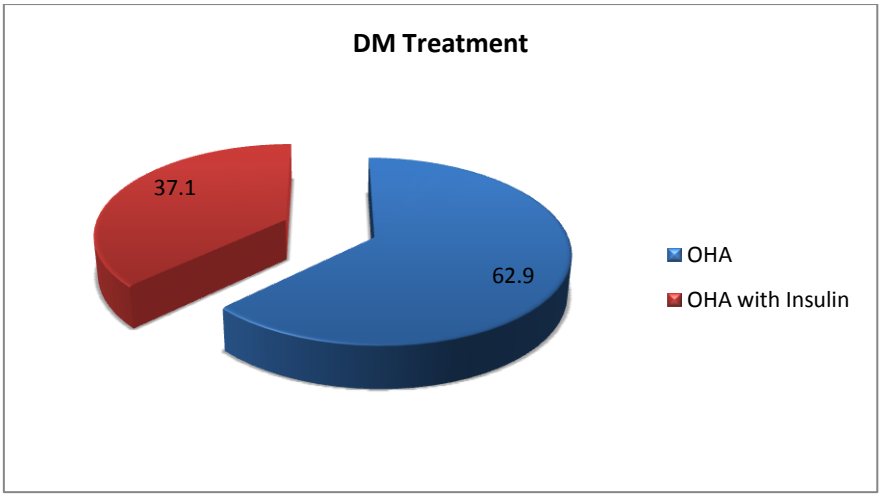
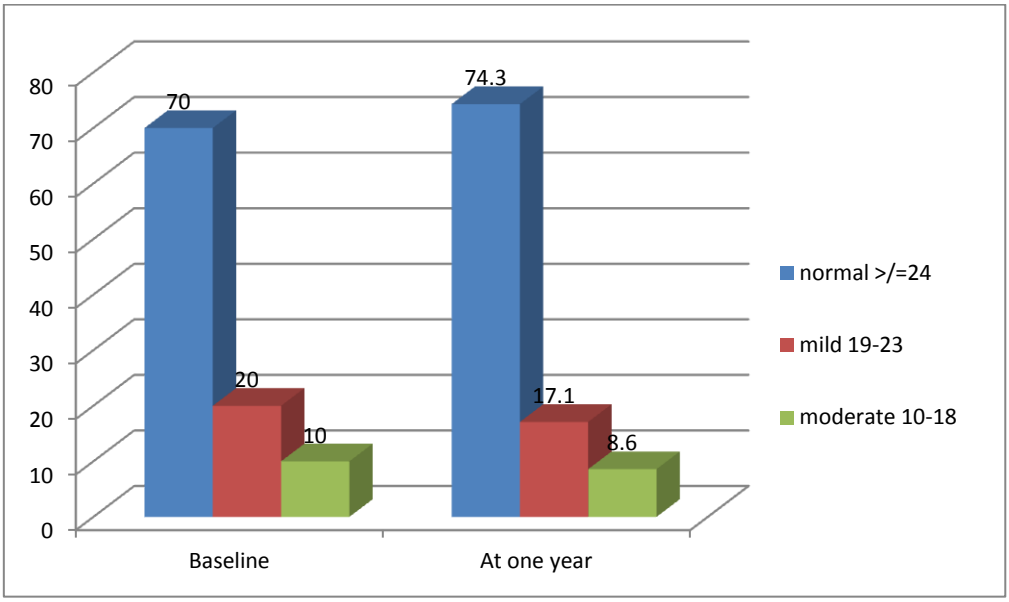


Figure 4 Dementia assessment at baseline and after one year using MMSE



Discussion

The clinical profile of the patients were studied and their cognitive dysfunction were rated using MMSE score. Also their dementia scores were compared with their vascular risk factors and level of glycemic control. The following discussion elucidates various findings described under Observation and analysis. Out of the 140 patients studied, the mean age was 56.5 yrs. Only thirty five percent of the individuals were elderly above the age of 60 years. No statistically significant difference in MMSE scores were found when comparing elderly diabetics (>60yrs) with other age groups (p=0.95). This was in accordance with the study by Priyam et al in 2012 where there was no significant impact of age on cognitive function. This could be because we compared the cognitive score with age- adjusted normative data instead of taking a separate control group for comparison similar to the study mentioned above.[10] Study by Reaven et al published in Diabetes care in 1991 has shown more cognitive impairment in elderly diabetics when compared with controls.[11] But the mean age of the study population here was 69.8 years .Similar results were also seen in Rotterdam study.[12] We did not get similar reports in our study probably because we studied a much younger population and only 5.7% of our study population were above the age of 70 years.

Of the 140 patients studied, 41.4% were females and 58.6% were males. There is a slightly higher male predilection (1.43:1) in our study. There is no statistically significant sex difference in MMSE scores among male and female diabetics (p=0.965). Fifty five percent were hypertensives and forty five percent were not. There is no statistically significant (p=0.398) difference in MMSE found between diabetics with or without hypertension**.** In our study population, 36.4% had dyslipidemia along with a diagnosis of Diabetes**.** No statistically significant correlation (p=0.243) in MMSE scores was found due to presence or absence of dyslipidemia in diabetic subjects**.** History of Coronary artery disease was seen in 35.7% along with a diagnosis of Diabetes**.** No statistically significant correlation (p=0.202) was found in MMSE scores either due to presence or absence of concomitant CAD in diabetic patients. These results were consistent with that of a study conducted by Ruis et al in 2009, one of the few studies on cognitive function in early Diabetes, where neither sex nor blood pressure, cholesterol levels, or BMI was significantly related to cognitive performance. However, in this study a history of macrovascular disease, was associated with reduced information-processing speed.[13]

In our study 45% had history of smoking. No statistical correlation (p=0.531) in MMSE score

difference due to smoking in diabetics was found. In this study, Non Alcoholics, Reformed and Addicted Alcoholics were studied. Of these, 44.1% were alcohol consumers**.** No statistically significant correlation (P=0.473) seen between MMSE scores and consumption of alcohol in diabetics. This is in accordance with the The Doetinchem Cohort Study where people with incident diabetes had statistically significant cognitive impairment irrespective of their smoking status or alcohol consumption.[14]

In our study population, 17.1% already had Diabetic Nephropathy when they were diagnosed with Diabetes or developed it within six months. This prevalence of Diabetic Nephropathy in our study on newly diagnosed diabetics is similar to that of a study by Agarwal et al published in JAPI in 2011, where the prevalence reported was 17.34%. There are various other studies with higher prevalences ranging from 37% to 56.2%.[15,16] In addition our study had a significant statistical correlation (P=0.002) between Diabetic Nephropathy and cognitive decline in Type2 diabetics as evidenced by MMSE scores. This is in accordance with the study by Barzilay et al where the presence of albuminuria in Diabetics correlated with greater declines in information processing speed and memory(P=0.001). Also the study by de bresser et al examining the relationship between microvascular complications and cognitive decline and the development of structural brain abnormalities in Diabetics, reported that presence of albuminuria predicted accelerated cognitive decline in Type2 diabetes (P<0.001).[17]

In our study, 10.7% already had Diabetic Retinopathy when they were diagnosed with Diabetes or developed it within six months. This prevalence of Diabetic Retinopathy in our study on newly diagnosed diabetics is somewhat similar to that of a study by Abdollahi et al in 2006, where the prevalence reported was 13.4%.[18] There are various other studies with higher prevalences ranging from 20% to 31.4%.[19] Our study demonstrated a significant statistical correlation (P=0.004) between Diabetic Retinopathy and cognitive decline in Type 2 diabetics as evidenced by MMSE scores. This was in accordance with the report by Crosby et al in 2011 which was a systematic review of studies reporting data on the relationship between diabetic eye disease and cognitive impairment in Type 2 diabetes. There were several studies that showed association between diabetic retinopathy and cognitive impairment, with a near threefold increased risk of cognitive impairment in patients with diabetic retinopathy compared to those without.[20]

In our study population, **o**ut of the 140 patients, 30% had MMSE scores <24, thus demented at baseline. The highest score was 29 and the lowest was 13. None had scores <10, hence no severe dementia cases. This is similar to the study conducted by Kataria et al in 2013 where the prevalence was 35% .[21] This was slightly lower than the study by Priyam et al done in Kolkata in 2011 where the prevalence stands at 42%. A Prevalence of 44% was found in a study conducted by Chukwuemeka O Eze et al in Nigeria in 2014.[22] This might be because this study represented all diabetic individuals irrespective of the time of onset of diabetes , though this study found no relation between duration of diabetes and cognitive impairment. Our study focused only on individuals with Type2 Diabetes of Maximum duration of only six months. In the Dutch prospective Doetinchem Cohort Study, people with recent onset of Type2 Diabetes had 2.5 times greater decline in cognitive function when compared to those without Diabetes. But this study showed that those with longer duration had 3.6times greater decline in cognition.[14]A study done by Tiwari et al in 2012 reported prevalence of 13.7%.[23] This study was conducted in individuals aged 55-59yrs whereas 35.7% of the population in our study were above the age of 60 years and it is a well known fact that age could be a confounding factor in the assessment of cognition which might be the reason for higher prevalence of our study.

Most of our cases had poor metabolic control with 75.7% having FBS of >130, 68.6% having PPBS >180 and 78.6% having HbA1c >7. In our study it was observed that there is statistically significant (p=0.013) difference in MMSE scores between Diabetics who had adequately controlled blood sugars as evidenced by PPBS ≤180 and those who were not. This is in accordance with the Hisayama study where the risk of developing All cause dementia and Alzheimer's dementia significantly increased in individuals with PPBS in the range of 140 to 200mg/dl compared to individuals with PPBS <120 and that of Vascular dementia increased with PPBS >200mg/dl. No such association was observed with Fasting blood glucose values.[24] In our study it was also noted that there is statistically significant difference in MMSE scores between those Diabetics who had HbA1c ≤7 and those above that. This is in accordance to the study by Cukierman et al where a 1% higher A1C value was associated with a significant 0.20-point lower MMSE score (P < 0.0001)[98]. Also in the study by Springer et al it was reported that an increase in HbA1C was an independent predictor for cognitive impairment.[25]

In our study, the mean MMSE score was 24.1 at baseline and 24.6 at one year follow up. We observed statistically significant difference (P<0.001) in MMSE scores at baseline and at one year follow up in our study. This can be due to the better glycemic status of our cases following treatment initiation as evidenced by 39.3% and 52.9% of our cases having adequately controlled HbA1C (≤7) and PPBS(≤180) respectively at one year follow up as compared to only 21.4% and 31.4% at baseline. There are various prospective studies reporting the beneficial effect of glycemic control on cognition. In a study by Meneilly et al conducted in elderly patients it was observed that selected areas of cognition showed significant improvement (p<0.05) following treatment initiation.[26] In the ACCORD MIND study after a 40 month follow up period , though there was no increased cognitive impairment in the standard treatment group when compared to the intensive treatment group, in MRI examination the overall brain volume was significantly higher in the intensive treatment group and it was suggested that there could already have been systemic changes in the brain before the cognitive impairment and therefore, there might be an interval between the protection of organic changes as a result of intensive glycemic control and suppression of cognitive decline.[27,28] But there are conflicting evidence for the same, with a recent Meta analysis by Richard H. Tuligenga reporting that intensive glucose control can lead to recurrent attacks of hypoglycaemia which might be detrimental to cognition.[29]

An assessment of the levels of cognitive impairment in patients with diabetes will help to understand the behavior of patients towards medication, treatment, nutrition and to their lifestyle. Type 2 diabetes is known to be associated with decrements in memory and executive functions but it is less clear, so far, as to at which stage of diabetes these cognitive decrements develop and how they progress over time. In this study, we investigated cognitive functioning of patients with recent screen-detected type 2 diabetes, thus providing insight into the nature and severity of cognitive decrements in the early stage of the disease. Strength of our study is the measurement of cognitive functions in the early stage of the disease. Previous studies focused mainly on patients with a longer diabetes duration. This study gives more information on early cognitive decrements.

Preliminary evidence from several small treatment intervention studies now suggests that Type-2 Diabetes-associated Cognitive deficits may be reversible. If Cognitive dysfunction arises in patients with Type-2 Diabetes largely because of their poor metabolic control, then one would expect that any intervention that improves control and reduces glycosylated hemoglobin levels should be associated with a parallel improvement in Cognitive function. So if one wishes to prevent diabetes-associated cognitive decrements,

interventions may need to be initiated at a very early stage.

The limitations of the present study were

1) This study was conducted in a small population in a tertiary care center and may not reflect the clear picture in the community. Further studies may have to be carried out in larger population to characterize the risk factors associated with accelerated cognitive decline in a better way to improve recognition of this treatable entity.
2) This study did not have a suitable control group hence we cannot comment on whether our patients had different outcomes from those with other diseases or from healthy general population.
3) The follow up period of one year of our study is a short term period for a chronic disease like Type2 diabetes. Hence long term follow up studies may be needed to accurately ascertain the progressive nature of this disease.
4) Moreover, we used MMSE in our study to assess dementia scores which is essentially a screening tool for assessment of global cognitive function and further comprehensive tests need to be done to ascertain the cognitive domains specifically affected in T2DM. Also MMSE is less sensitive to detect mild cognitive impairment and is known to produce false negative results.

Conclusions

A significant proportion (30%) of newly diagnosed Type2 Diabetes (within 6 months) patients had impairment of cognitive function. Cognitive dysfunction in patients with Type2 Diabetes was directly related to poor glycemic status. Among the glycemic targets, Post prandial blood glucose values and HbA1C had better correlation with cognitive decline than Fasting plasma glucose levels. Presence of microvascular complications of Type2 Diabetes such as Nephropathy and Retinopathy predicted cognitive decline. No definite association was seen between cognitive dysfunction and macrovascular disease especially Coronary artery disease in newly diagnosed Type 2 Diabetes. Cognitive dysfunction was independent of other vascular risk factors such as Hypertension, Dyslipidemia and Smoking. Adequate glycemic control in early stages of Type 2 Diabetes may result in improvement in cognitive function.

Acknowledgements: Nil

Funding; No funding sources

Ethical approval; The study was approved by Institutional Ethics Committee.

References

1. Mohan V, Sandeep S, Deepa R, Shah B, Varghese C. Epidemiology of type 2 diabetes: Indian scenario. Indian J Med Res. 2007 Mar;125(3):217–30.
2. Awad N, Gagnon M, Messier C. The relationship between impaired glucose tolerance, type 2 diabetes, and cognitive function. J Clin Exp Neuropsychol. 2004 Nov;26(8):1044–80.
3. Reis F, Soares, Nunes, Pereira. Diabetic encephalopathy: the role of oxidative stress and inflammation in type 2 diabetes. Int J Interferon Cytokine Mediat Res. 2012 Sep;75.
4. Draelos MT, Jacobson AM, Weinger K, Widom B, Ryan CM, Finkelstein DM, et al. Cognitive function in patients with insulin-dependent diabetes mellitus during hyperglycemia and hypoglycemia. Am J Med. 1995 Feb;98(2):135–44.
5. Rösen P, Nawroth PP, King G, Möller W, Tritschler HJ, Packer L. The role of oxidative stress in the onset and progression of diabetes and its complications: a summary of a Congress Series sponsored by UNESCO-MCBN, the American Diabetes Association and the German Diabetes Society. Diabetes Metab Res Rev. 2001 Jun;17(3):189–212.

6. Taylor VH, MacQueen GM. Cognitive dysfunction associated with metabolic syndrome. Obes Rev Off J Int Assoc Study Obes. 2007 Sep;8(5):409–18.
7. Perlmuter LC, Hakami MK, Hodgson-Harrington C, Ginsberg J, Katz J, Singer DE, et al. Decreased cognitive function in aging non-insulin-dependent diabetic patients. Am J Med. 1984 Dec;77(6): 1043–8.
8. Kent S. Is diabetes a form of accelerated aging? Geriatrics. 1976 Nov;31(11):140, 145, 149–51.
9. Kahle-Wrobleski K, Corrada MM, Li B, Kawas CH. Sensitivity and Specificity of the Mini-Mental State Examination for Identifying Dementia in the Oldest-Old: The 90+ Study. J Am Geriatr Soc. 2007 Feb;55(2):284–9.
10. Priyam M,Srijan M, Soumik G, Jayeeta B, Subhro C, Sumanto M ,et al. Cognitive dysfunction in diabetic patients with special reference to age of onset, duration and control of diabetes.
11. Reaven GM, Thompson LW, Nahum D, Haskins E. Relationship between hyperglycemia and cognitive function in older NIDDM patients. Diabetes Care. 1990 Jan;13(1):16–21.
12. Ott A, Stolk RP, van Harskamp F, Pols HA, Hofman A, Breteler MM. Diabetes mellitus and the risk of dementia: The Rotterdam Study. Neurology. 1999 Dec 10;53(9):1937–42.
13. Ruis C, Biessels GJ, Gorter KJ, Donk M van den, Kappelle LJ, Rutten GEHM. Cognition in the Early Stage of Type 2 Diabetes. Diabetes Care. 2009 Jul 1;32(7):1261–5.
14. Cohort Profile: The Doetinchem Cohort Study [Internet]. [cited 2015 Nov 14]. Available from: http://ije.oxfordjournals.org/content/37/6/1236.full
15. Agarwal N, Sengar NS, Jain PK, Khare R. Nephropathy in newly diagnosed type 2 diabetics with special stress on the role of hypertension. J Assoc Physicians India. 2011 Mar;59:145–7.
16. Barzilay JI, Lovato JF, Murray AM, Williamson J, Ismail-Beigi F, Karl D, et al. Albuminuria and cognitive decline in people with diabetes and normal renal function. Clin J Am Soc Nephrol CJASN. 2013 Nov;8(11):1907–14.
17. de Bresser J, Reijmer YD, van den Berg E, Breedijk MA, Kappelle LJ, Viergever MA, et al. Microvascular determinants of cognitive decline and brain volume change in elderly patients with type 2 diabetes. Dement Geriatr Cogn Disord. 2010;30(5):381–6.
18. A. Abdollahi MHM. Prevalence of diabetic retinopathy in patients with newly diagnosed type II diabetes mellitus. Acta Med Iran. 2006;44(6).
19. Lee KMC, Sum WMR. Prevalence of diabetic retinopathy in patients with recently diagnosed diabetes mellitus. Clin Exp Optom. 2011 Jul;94(4):371–5.
20. Crosby-Nwaobi RR, Sivaprasad S, Amiel S, Forbes A. The relationship between diabetic retinopathy and cognitive impairment. Diabetes Care. 2013 Oct;36(10):3177–86.
21. Prevalence and Pattern of Cognitive Dysfunction In Type 2 Diabetes Mellitus JuniorResident JuniorResident [Internet]. [cited 2015 Nov 14]. Available from: http://www.academia.edu/5260180/PREVALENCE_AND_PATTERN_OF_COGNITIVE_DYSFUNCTION_IN_TYPE_2_DIABETES_MELLITUS_JuniorResident_JuniorResident
22. O Eze C. The Prevalence of Cognitive Impairment amongst Type 2 Diabetes Mellitus Patients at Abakaliki South-East Nigeria. J Metab Syndr [Internet]. 2015 [cited 2015 Nov 14];04(01). Available from: http://www.omicsgroup.org/journals/the-prevalence-of-cognitive-impairment-amongst-type-2-diabetes-mellituspatients-at-abakaliki-southeast-nigeria-2167-0943-1000171.php?aid=44861

23. Tiwari SC, Tripathi RK, Farooqi SA, Kumar R, Srivastava G, Kumar A. Diabetes mellitus: A risk factor for cognitive impairment amongst urban older adults. Ind Psychiatry J. 2012;21(1):44–8.
24. Ohara T, Doi Y, Ninomiya T, Hirakawa Y, Hata J, Iwaki T, et al. Glucose tolerance status and risk of dementia in the community: the Hisayama study. Neurology. 2011 Sep 20;77(12):1126–34.
25. Ravona-Springer R, Moshier E, Schmeidler J, Godbold J, Akrivos J, Rapp M, et al. Changes in Glycemic Control are Associated with Changes in Cognition in Non-Diabetic Elderly. J Alzheimers Dis JAD. 2012;30(2):299–309.
26. Meneilly GS, Cheung E, Tessier D, Yakura C, Tuokko H. The effect of improved glycemic control on cognitive functions in the elderly patient with diabetes. J Gerontol. 1993 Jul;48(4): M117–21.
27. Launer LJ, Miller ME, Williamson JD, Lazar RM, Gerstein HC, Murray AM, et al. Effects of intensive glucose lowering on brain structure and function in people with type 2 diabetes (ACCORD MIND): a randomised open-label substudy. Lancet Neurol. 2011 Nov;10(11):969–77.
28. Cukierman-Yaffe T, Gerstein HC, Williamson JD, Lazar RM, Lovato L, Miller ME, et al. Relationship between baseline glycemic control and cognitive function in individuals with type 2 diabetes and other cardiovascular risk factors: the action to control cardiovascular risk in diabetes-memory in diabetes (ACCORD-MIND) trial. Diabetes Care. 2009 Feb;32(2):221–6.
29. Tuligenga RH. Intensive glycaemic control and cognitive decline in patients with type 2 diabetes: a meta-analysis. Endocr Connect. 2015 Jun 1;4(2):R16–24.