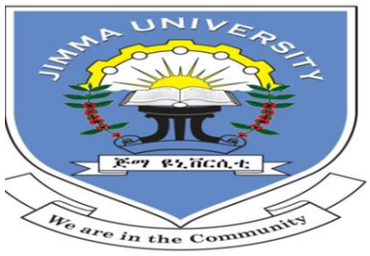

Jimma University

College of Health Sciences

Department of Biomedical Sciences

Cognitive Impairment among Type 2 Diabetes Mellitus Patients at Jimma University Specialized Hospital, Southwest Ethiopia; Comparative Cross Sectional Study

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A thesis to be submitted to Department of Biomedical Sciences, College of Health Sciences, Jimma University in partial fulfillment of the requirements for the Degree of Master of Science (MSc) in Medical Physiology

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Jimma, Ethiopia

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ABSTRACT

Background: *Diabetes mellitus (DM) is a chronic disorder caused by deficiency of insulin, which affects functions of the brain, kidneys, vision and other organs. Cognitive impairment is the major public health problem worldwide particularly in elderly people with type 2 DM. But, little is known about the net association between DM and cognitive impairment among adults.*

Objective: *The aim of the present study was to determine magnitude of cognitive impairment and associated factors among patients with T2DM in Jimma University Specialized Hospital, 2016.*

Methods: *Comparative cross sectional study was employed among 105 patients with T2DM, who were under follow-up in DM clinic and 105 age, sex and educational level matched healthy individuals coming to the clinic by using consecutive sampling technique. The tool contained 30-point standardized mini-mental state examination, sociodemographic, substance use and clinical archives. Descriptive statistics were done. Moreover, Chi-square test, independent t-test, and logistic regression were carried out and variables with $p<0.05$ were considered as significant.*

Results: *The prevalence of cognitive impairment in DM was 53.3% and in healthy controls it was 31.4%. Diabetics were 2.5 times more risky than healthy controls [OR=2.49, 95% CI (1.42, 4.38)] for cognitive impairment. DM patients who had fasting blood glucose (FBG) ≥126mg/dl were 4.4 times [AOR=4.43, 95% CI (1.14, 17.18)] more likely to have cognitive impairment than those who had FBG< 126 mg/dl. DM patients who relied on only oral hypoglycemic agents were 5.4 times higher than those using insulin only [AOR=5.39, 95% CI (1.37, 41.18)] to have cognitive impairment. DM patients aged ≥62 years had 7.5 times [AOR= 7.54, 95% CI (1.38, 41.38)] risk for cognitive impairment than those ≤ 45 years.*

Conclusion: *The prevalence of cognitive impairment among T2DM patients was significantly higher than healthy controls. Hyperglycemic state could lead to neuronal damage via direct toxic effect and/or free radical formation which might be worsened in the elderly and those who relied on oral hypoglycemic agents only. This emphasized the need to integrate screening and management options of cognitive impairment among T2DM patients as part of routine activity and awareness creation.*

Key words: *Cognitive impairment, Type 2 Diabetes Mellitus, FBG, MMSE, Ethiopia*

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ABBREVIATIONS

AGE	Advanced Glycation End Products
AOR	Adjusted Odds Ratio
CI	Confidence Interval
CKD	Chronic Kidney Disease
COR	Crude Odds Ratio
DM	Diabetes Mellitus
FBG	Fasting Blood Glucose
HIV-1	Human Immunodeficiency Virus 1
HTN	Hypertension
JUSH	Jimma University Specialized Hospital
MCI	Mild Cognitive Impairment
MMSE	Mini Mental State Examination
OR	Odds Ratio
SPSS	Statistical Package for Social Sciences
T2DM	Type 2 Diabetes Mellitus

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CHAPTER 1: INTRODUCTION

1.1. Background

Diabetes mellitus (DM) is a chronic endocrine disorder caused by partial or complete absence of insulin secretion from beta cells of pancreas or defect of insulin action with type 2 diabetes mellitus (T2DM) accounting 90-95% of all varieties of DM worldwide (1). The rise in blood glucose past the physiological limit results in acute or chronic complications to different body parts including central nervous system, particularly cognition (2,3)

Cognition refers to the processing of information, applying knowledge and changing predilection. Cognitive function comprises focused attention, executive function, recall, producing and understanding language, solving problem and making decisions (3).

Cognitive dysfunctions are commonly seen in DM patients for the most part T2DM (4). Patients with T2DM are at increased risk for cognitive decline than non-diabetes (5). Hyperglycemia, transient hypoglycemia and lack of insulin in the brain cells impart for cognitive impairment in DM patients (2). T2DM also leads to disturbance of brain metabolites which heightens cognitive impairment (6). Memory function appears to be chiefly affected amongst domains of cognitive function in patients with T2DM (7). Cerebrovascular disease is the commonest cause of acquired cognitive impairment and dementia and contributes to cognitive decline in the neurodegenerative dementias (8).

Cognitive impairment mainly ensues due to the disturbances of neurotransmitters like acetylcholine and dopamine which are responsible for cognition. Brain regions principally hippocampus, amygdala and prefrontal cortex are affected in the course of cognitive impairment (9). Reduced gray matter volume is linked with poorer performance on measures of general cognitive function, working memory, and executive function (10). Bilateral hippocampal atrophy occurs in T2DM patients that lead to impair hippocampus-mediated learning and aspects of memory function (11).

Cognitive impairment due to T2DM usually starts at the duration of disease of 1 year and above (12).

1.2. Statement of the problem

Diabetes mellitus affects 8.3% people of the globe, epidemic in developing nations, projected to be 8.8% by 2035. It was 5.7% in Africa in 2013 and projected to be 6% by 2035 (13). According to international diabetes federation 2012 report, prevalence of DM in Ethiopia was 3.32 % (14). Cerebrovascular derangement, as DM complication , through ischemia of microvascular system and endothelial damage leads to chronic cerebral hypoperfusion resulting in impairment of cerebral protein synthesis, a key factor for learning and memory (15). Large-scale epidemiological studies found that being diabetics is 1.3 times higher risk of developing cognitive impairment than non-diabetics population (16,17).

The global incidence rate of mild cognitive impairment (MCI) was 9.9/1,000 person–years. MCI was a good predictor of Alzheimer's disease with an annual conversion rate of 8.3% (18). Age-standardized prevalence for those aged 60 years and above varied in a narrow band, 5%–7% in most world regions, with higher prevalence in Latin America (8.5%), and distinctively lower in sub-Saharan Africa regions (2%–4%) (19) and higher in United Kingdom which is 18.3% (20). Prevalence of MCI in India (6%) and even higher in Malaysia (15.4%) and South Korea (9.7%) (21). A systematic review of Sub-Saharan nations showed ; Benin (10.4%), Botswana (9%), Central Africa republic (26%), Congo (18.8%) and Nigeria (11.8%) (22).

In Ethiopia, institution based cross sectional study conducted among T2DM in 2011 at Tikur Anbesa Hospital revealed 45% prevalence of cognitive impairment (29.6% mild and 15.4% moderate), 45.8% of impaired cases had cardiovascular problems of which 84.1% were hypertensive (23). Cross sectional study in Pakistan among 30 years and above revealed that T2DM patients experienced cognitive decline particularly attention and calculation, recall and language which is not associated with duration of disease (24). Comparative cross sectional study from Manipal indicated higher memory loss among T2DM patients than controls (25). In Jimma town, large proportions of individuals chew khat. Cross sectional study in Jimma town publicized that current khat chewing, cigarette smoking and alcohol drinking prevalence as 35.8%, 11.2% and 43.4% respectively (26).

Several studies probed cognitive impairment among T2DM patients worldwide but little is known about the net effect of T2DM for cognitive impairment. Above all the link of substance use with cognitive impairment in T2DM was not clearly studied.

1.3. Significance of the study

The universal growing of T2DM, with the utmost rate in developing nations including Ethiopia, leads to cognitive impairment which is a major public health concern. In Ethiopia there is no comparative cross sectional study done on cognitive impairment among T2DM patients. The results of this study will help health development planners to give special considerations for cognitive impairment in T2DM patients during designing diagnosis and management strategies particularly focusing on counseling in preventing risk factors. It will add additional knowledge besides the existing literatures for the scientific community. The last but not the least beneficiaries of this study are the diabetes mellitus population at large to seek medical advice, self-management and to know the burden of cognitive impairment to their life quality.

CHAPTER 2: LITERATURE REVIEW

2.1. Prevalence of Diabetes Mellitus

Amongst the top 10 world countries with the largest numbers of people foretold to have DM in 2030, five are in Asia of which India is at top with prevalence of 31.7% and to be projected by 79.4% in 2030 (27). Institution based study in Ayder referral hospital, Ethiopia revealed overall DM of 1.3% of which 82% had T2DM (28). Community based cross sectional study in Gilgel Gibe showed DM of 1.8% (29). Institution based cross sectional study in Jimma University Specialized Hospital in 2008 revealed 62% of DM patients were T2DM (30).

2.2. Cognitive impairment among T2DM and general population

Cognitive impairment essentially affects the elderly people. Cross sectional study in Jamaica pointed out that the prevalence of mild and severe cognitive impairment among 60 years and above was 21.2% and 11% correspondingly (31). A relevant study done in India reported that cognitive impairment associated with age, sex, educational level, area of residence, subjective comorbid conditions like hypertension, DM, marital status, unemployment and poverty (32). Cognitive impairment was higher in rural (9.6%) population than urban (7.5%) as realized in India aged 60 years and above (17). Cognitive impairment is common among DM patients with longer disease duration but can occur in short disease duration. Cross sectional study among recently diagnosed Mexican T2DM patients (<3 years diagnosis, who were ≥18 years of age) reported 2.2% prevalence of cognitive impairment**.** Females and the elderly segment of the population were primarily affected by cognitive impairment (33). Study conducted in Korea stated higher cognitive impairment among elderly T2DM patients with 32.7% mild cognitive impairment which was associated with age, educational background and systolic blood pressure (34). Case control study in Poland revealed slight cognitive impairment in diabetes than controls which was significantly associated with the duration of DM (35). Similar study in Saudi Arabia showed 15.7% (mild) and 17.6 % (moderate) cognitive impairment among T2DM people (36). Comparative study in Iran among T2DM patients revealed 52% memory impairment (37). Systematic review study revealed that cognitive decline was observed among DM patients who relied on insulin therapy, and oral hypoglycemic agents (if used longer) have protective role particularly at longer disease duration (38).

One study in Australia point out that cognitive impairment is associated with metformin use in DM patients with twice higher among metformin users than nonusers; this could be due to the fact that

metformin has adverse effect of decreasing the serum level of neuro-vitamin vitamin B_{12} (39) Comparative cross sectional study conducted in Egypt hospitals revealed that DM patients had lower cognitive score than controls which was associated with age, duration of illness, blood glucose level and insulin resistance (40). Cross sectional study in Nigeria showed that the prevalence of cognitive impairment among T2DM of 30 years and above was 44% (41).

2.3. Pathogenesis of cognitive impairment in T2DM

Glucose is the only required source of energy for neurons and any disruption in glucose metabolism leads to compromised neuronal functions (42). The definite mechanisms of cognitive impairment in T2DM is not clearly established but hypothesized to be due to brain vasculature changes, disturbances of cerebral insulin signaling, glucose toxicity, accumulation of advanced glycation end products (AGE), hypoglycemic episodes and alterations in amyloid metabolism (43).

In T2DM, gradual decrement of beta cell function leads to increased hyperglycemia while the resistance to the action of insulin could lead to hyperinsulinaemia. This combination may lead to chronic hyperglycemia and glucose toxicity which have profound implications to the brain and so cognition (44). Poorly controlled blood glucose can damage nerve cells in the brain and lead to cognitive impairment (45). High glucose concentration may have effect on the neurons in the brain through oxidative stress and continued chronic hyperglycemia that leads to the formation of AGE, coupled with free radicals which can cause oxidative damage then neuronal injury (46).

Beta cell dysfunction disrupts insulin secretion; reduces insulin in the brain which results in increased amyloid-β and production of AGEs which contribute to the development of cognitive impairment (47). Reduced insulin and impaired insulin signaling impairs cerebral energy metabolism hence decreases the translocation of glucose transporter 4 causing instability of glucose metabolism which can impact on neuronal development, learning and memory (48). Chronic peripheral hyperinsulinaemia may lower brain insulin and thus reduce insulin degrading enzyme in the brain which in turn impairs amyloid-β clearance. Insulin regulates the central nervous system levels of acetylcholine and nor-epinephrine which influence cognitive function (49). Insulin also controls food intake and cognitive functioning; these are affected in insulin resistant states. Following insulin resistance and hyperinsulinaemia which are common features of T2DM, the transport of insulin into the brain across the blood brain barrier is reduced and this lowers the insulin levels in the brain (38).

Insulin regulates choline acetyl transferase, an enzyme responsible for acetylcholine production. Acetylcholine is responsible for cognition and memory formation, thus, a dysfunction in insulin production and insulin resistance could lead to a decrease in acetylcholine levels which may have upshots for cognitive impairment, particularly memory (50,51).

Long-term exposure to hyperglycemia is reflected by increased risk of vascular complications like neuropathy, nephropathy, retinopathy and stroke (52). In addition, diabetes is often accompanied by other vascular risk factors such as hypertension, dyslipidemia and obesity which are suggested to play important role for cognitive decline in T2DM (53).

Cognitive deficit in T2DM is typically associated with white matter lesion and lacunar infarcts leading to subtle reductions in mental speed, mental flexibility and verbal memory performance. Besides this comparative cross-sectional studies showed decline cognitive function on measures of verbal memory, information processing speed and attention and executive function for patients with T2DM compared to age, sex and education matched controls (54–56).

T2DM is associated with various risk factors that may influence cognitive functioning, including diabetes-specific factors (hyperglycemia and microvascular complications), risk factors that are not specific to the disease (hypertension, obesity) and genetic, demographic, and lifestyle factors (57).

2.4. Other Risk Factors for Cognitive Impairment

2.4.1. Chronic diseases as risk factors for cognitive impairment

Diseases other than T2DM may cause cognitive impairment. Longitudinal and, health and retirement survey in America revealed that the prevalence of mild cognitive impairment among chronic obstructive pulmonary disease patients as 17.5% (58).

Rheumatoid arthritis affects not only joints but also imposes extra articular complications like cognitive impairment which leads to 31% cognitive impairment (59). Cognitive impairment affects 50% HIV-1 patients (60). One study in Ethiopia revealed that epileptic patients scored lower MMSE; cognitive decline (61).

2.4.2. Substance use and cognitive impairment

Effect of chewing crude khat (*Catha Edulis Forsk*) on cognition still had contradictory findings across the globe. Even though the use of khat is widespread in east Africa, these regions are not suffering from

mental illness but study in Kenya showed khat chewing declines memory and learning (62,63). Chronic khat use can result in cognitive impairment and comparative study done in Netherlands revealed khat chewers scored significantly worse cognitive flexibility than controls (64). Amphetamine causes release of dopamine and norepinephrine from ventral tegmental area to the prefrontal cortex, which is known to play a role in a wide range of cognitive functions, including attention and working memory (65).

Chronic cigarette smoking appears to be associated with decline in cognitive performances from systemic review studies (66). Study conducted in Netherlands showed that mild cognitive decline among T2DM patients than controls which was strongly associated with cigarette smoking (67). However, there are incongruous studies elsewhere about cigarette smoking on cognition those showed improvement of cognitive performance among cognitively impaired patients than controls (68) and even not significantly associated with cognitive impairment among T2DM patients in Ethiopia (23). Nicotine, the ingredient of cigarette, has been shown to enhance memory function and increase the expression of nicotinic acetylcholine receptors and therefore, could have a promising therapeutic role in cognitive impairment. Nicotine has also been shown to exert positive effects on certain neurotrophins such as nerve growth factor (69).

Optimal alcohol use can result in memory improvement as explained in systemic review findings. However, chronic alcohol use can lead to memory loss even may result in amnesia (70). Alcohol acts as a general central nervous system depressant. It leads to distraction and inattention and significantly inhibits neuronal activity in the hippocampus, which impairs memory since hippocampus plays an important role in the formation of new declarative memories (71).

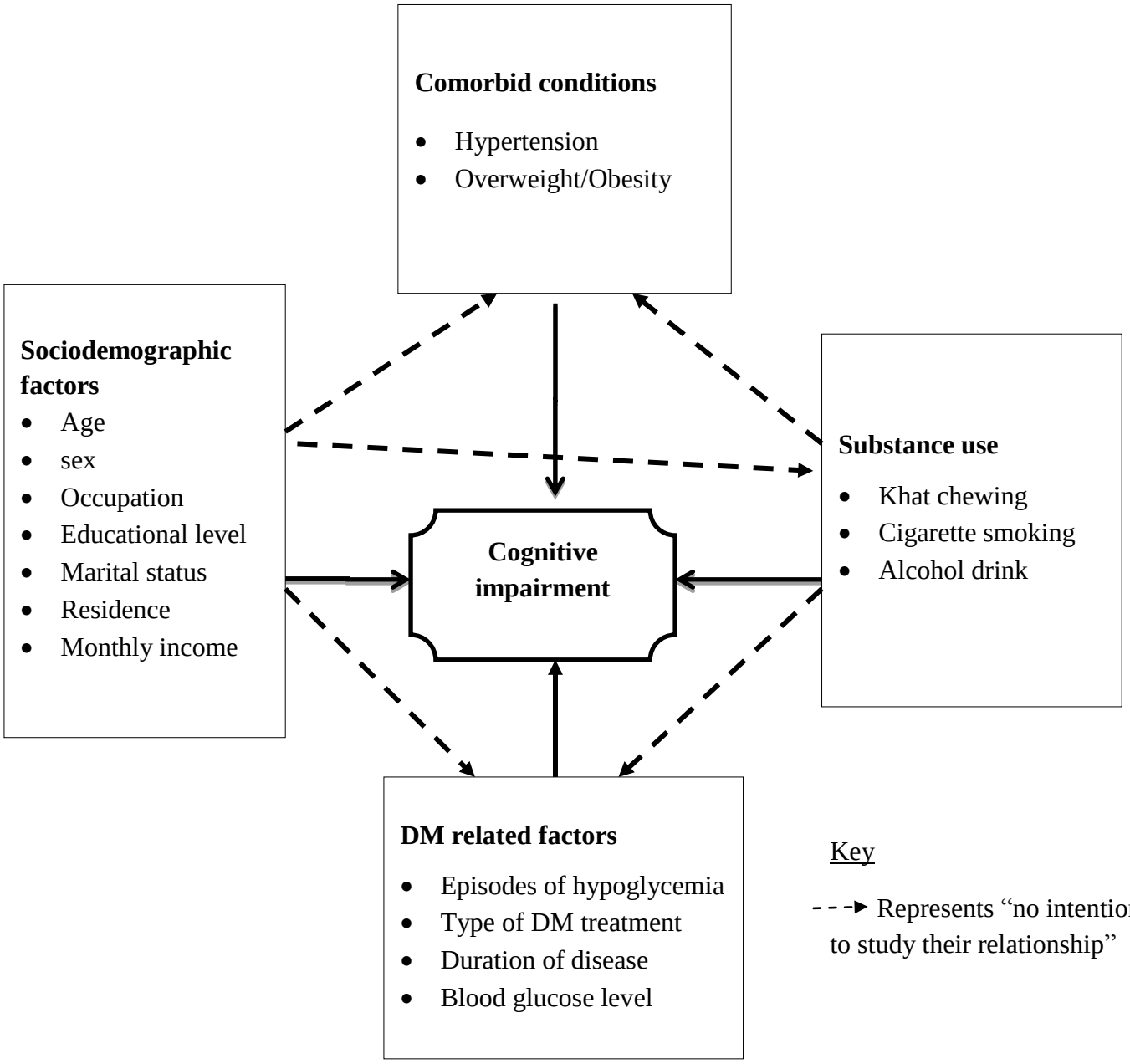

Figure 1: Adapted schematic presentation (conceptual framework) showing potential risk factors of cognitive impairment among type 2 DM patients at JUSH, Jimma, Ethiopia, 2016

CHAPTER 3: OBJECTIVES

3.1. General objective

The aim of the present study was to assess cognitive impairment and associated factors among T2DM patients at JUSH, Jimma, Ethiopia 2016

3.2. Specific objectives

- To determine magnitude of cognitive impairment among T2DM patients
- To verify association between blood glucose level and cognitive impairment among T2DM patients
- To evaluate the association of DM medication with cognitive impairment among T2DM patients
- To describe association of sociodemographic characteristics with cognitive impairment among T2DM patients
- To describe association between substance use and cognitive impairment among T2DM patients

CHAPTER 4: METHODS AND MATERIALS

4.1. Study area and period

The study was conducted at JUSH DM clinic, Jimma town, located 352 km Southwest of Addis Ababa, Ethiopia. JUSH is the only teaching and referral hospital in the Southwestern part of the country, providing services for approximately 15 million people in the catchment area including chronic care follow up for DM and other cases. Besides care provision it serves as training center for medical and health science students to develop their professional knowledge and skills. Data collection was carried out from March 25 to April 25, 2016.

4.2. Study design

Institution based comparative cross sectional study design was employed

4.3. Source population

All ≥30 years of age T2DM patients enrolled to JUSH DM clinic and healthy individuals who came to the hospital for routine purpose.

4.4. Study population

T2DM patients attending JUSH DM clinic whose age was ≥30 years and sex, age and educational level matched healthy individuals at the time of data collection.

4.5. Eligibility criteria

4.5.1. Inclusion criteria

For diabetes mellitus group: All T2DM patients aged 30 years and above attending JUSH Diabetic clinic, having duration of 1year and above from diagnosis.

For control group: All healthy individuals who came to JUSH DM clinic and matched for age, sex and educational level without DM were included

4.5.2. Exclusion criteria

Individuals with visual, hearing and speaking difficulty were excluded

4.6. Sample size determination and sampling technique

4.6.1. Sample size determination

Sample size was determined by using two population proportion formula with the assumption of P_1 = 45% (23) and P_2=26% (22), confidence level of 95% and power of 80%

$$n = \frac{(r+1)(Z\alpha/2+Z\beta)2\ P(1-P)}{r(p1-p2)2} = 100$$

For each group the sample size (n) was 100. By adding a non-response rate of 5% i.e. the total sample size for each group was 105. Therefore total sample size (T2DM and controls) was 210.

Where;

n = sample size, p1= proportion of T2DM with cognitive impairment, p2=proportion of non-diabetes people with cognitive impairment, Zβ= standard normal variate for power, Zα=standard normal variate for level of significance, p1-p_2= effect size, P= pooled proportion i.e. average proportion (p1+p2)/2, r = ratio of number of participants of cases to controls (1 in this case).

4.6.2. Sampling technique

Consecutive sampling technique was used for both T2DM patients and healthy control individuals. During the data collection period there were a total of 1853 DM patients registered for follow-up at JUSH DM clinic with 1378 T2DM (preliminary Hospital record overview). DM clinic had 2 consecutive days (every Monday and Tuesday) follow up per week for DM patients with regular appointment, usually 1month duration, for drug refill and further checkup and about 20-35 DM patients visited the DM clinic within a day.

4.7. Data collection procedure

Data were collected by using interviewer administered structured questionnaire which consisted of sociodemographic characteristics, substance use, physical measurements of height and weight, medical history and adapted standardized mini mental state examination for cognition assessment (23). An MMSE evaluates orientation (10 points), registration (3 points), attention and calculation (5 points), recall (3 points), language and praxis (9 points; naming, repetition, 3-stage command, reading, writing and copying) (72). Two data collectors (1 BSc Psychiatric Nurse and 1BSc Nurse) and one supervisor (BSc Psychiatric Nurse) were involved. Three days prior to the actual time of data collection pretest was done on 10 volunteers (5 from each group) at JUSH. Necessary corrections were taken concerning wording and contextual variations on the structured questionnaire.

Exit interview at separate (private) room was undertaken for questions related with sociodemography, substance use and cognition. Diabetes related questions were filled by looking the medical chart of the patients and FBG was taken from that day laboratory result of the patient. Weight and height were measured (combined height and weight scale of Seca gmbh co.Kg- Germany, Model-7862021994) to determine body mass index. Weight of the respondent was measured in kg that was recorded to the nearest 0.1kg and the scale was adjusted to zero level between individual measurements. In the meantime of measurement the participant stood with arms hanging at the sides, with bare foot and after taking off heavy wears. Height was measured in meter which was set from bottom up, the subject standing in anatomical position toward the examiner without any footwear or headgear and the records were taken to the nearest 0.5 centimeter.

4.8. Study variables

Dependent variable: - Cognitive impairment

Independent variables:

Sociodemographic variables: - Age, sex, monthly income, marital status, occupation, ethnicity, religion, educational status, residence.

Lifestyle and medical history related variables: - Substance use (Khat chewing, cigarette smoking, alcohol drink), body mass index, comorbid hypertension, type of DM medication, episodes of hypoglycemia, duration of disease, blood glucose level.

4.9. Operational (conceptual) definition

Cognitive impairment- A state of impairment in information processing and defined as the following; having scored (on MMSE)

- 21 or below for participants with educational level of 8^{th} grade or lower
- Below 23 for participants with educational level of high school to preparatory (9-12).
- 24 or below for participants with educational level of college and above

Mild cognitive impairment- A MMSE score of 20-24/30

Moderate cognitive impairment- A MMSE score of 10-19/30

Severe cognitive impairment- A MMSE score of 0-9/30

No cognitive impairment- A score of 25-30/30 on MMSE.

NB-the severity of cognitive impairment given above works for those individuals with educational level of college and above. For other categories first it should be adjusted

Substance use **–** Use at least one of the substances (alcohol, khat, cigarettes) in an individual's life time

Current user- Person who consumed any substance at least once within the last 30 days

Ever use- Use of any of the substances at least once in an individual's life time.

Habitual chat chewer- Frequent chewer of khat on a daily basis, otherwise referred as occasional user

Chronic khat chewer- A person who chew khat for more than 2 years duration

Underweight- A person having BMI of <18.5Kg/m^2

Overweight- A person having BMI of >24.9Kg/m^2

Obese- A person having BMI of $\geq$30Kg/m^2

Comorbid Hypertension- A person having Systolic blood pressure of 140mmHg and/or Diastolic blood pressure of 90mmHg and above that was recorded at the patient's medical chart besides T2DM.

4.10. Data analysis procedure

Data were checked for its completeness then entered to Epi data version 3.1 and exported to SPSS version 20.0 for windows. Descriptive statistics were done; frequency and percentage for categorical data while mean and standard deviation were used for continuous data. Independent t-test was used to compare the mean differences between the study groups whereas Chi-square(X^2) and Fisher exact test were used to compare the study groups regarding categorical variables. Binary logistic regression was done to entertain crude association between each exposure and outcome variable. Variables having p-value < 0.25 in the binary logistic regression were candidate for multiple logistic regression. Multiple logistic regression with backward likelihood ratio method was performed. From multiple logistic regression, exposure variables with p-value < 0.05 with 95% confidence interval were declared as significantly associated factors for cognitive impairment. Finally model fitness was checked by Hosmer and Lemshow test with final model having p< 0.621which indicated the model being good.

4.11. Data quality management

Validated mini-mental state examination tool from other studies (23) with some modifications in local context was used for cognition assessment. Training for data collectors were given for 2 days regarding purpose of the study, interview, measurement techniques and ethical issues during data collection. Questionnaire was translated to Amharic language and then retranslated to English for its consistency

by another person and were checked daily for consistency and completeness by the supervisor and principal investigator with necessary corrective actions forwarded.

4.12. Ethical consideration

Ethical clearance was obtained from institution review board of Jimma University, College of Health Sciences and letter of cooperation was given from Jimma University and JUSH. Informed verbal consent was taken from the study participants to start data collection. Any identifiable issues were eliminated to ascertain confidentiality.

4.13. Dissemination plan of results

A document of results will be submitted to Jimma University Postgraduate School. The results will be communicated with the stakeholders through presentations on meeting, workshops and scientific panels. Attempts will be made to publish the thesis in peer reviewed reputable journals.

CHAPTER 5: RESULTS

5.1 Description of study participants

5.1.1. Sociodemographic and anthropometric characteristics

A total of 210 study participants with equal proportion of T2DM patients and healthy controls (105 each) were involved with 100% response rate. The mean age for DM group and healthy controls was 53.36 (SD±11.674) and 53.70 (SD±11.53) years respectively. Male to female ratio was 1.06 and majority of respondents 112(53.3%) were in the age range of 30-55 years. Ninety five (45%) respondents were Muslims and 116(55.2%) were Oromo ethnic. Primary education comprised 126(60%) followed by 56(26.7%) grade 9-12. One hundred thirty (66.2%) were married and 6(2.9%) single. Seventy six (36.2%) were wage employed and 36(17.1%) farmers. One hundred twenty (57.1%) respondents earned 1000 Ethiopian birr and lower per month. Almost three-fourth (74.3%) lived in urban area. One hundred forty two (67%) respondents had normal body mass index while 7(3.3%) were underweight and 61(29.1%) were overweight. Income had significant mean difference between the study groups; for DM mean=1680.33(SD±1093.274) and controls mean= 1213.01 (SD±1287.516), t-test= -2.835, p= 0.005) and the mean difference of body mass index was also significant; for DM group mean= 24.153 (SD± 4.1642) and controls mean=22.794 (SD± 2.4284), t-test=2.889, p=0.004). Besides this, the study groups had significant difference regarding marital status (Fischer exact test=20.0, p=0.00) and occupation (Fischer exact test= 25.894, p=0.00). However, there were no significant differences between the study groups for religion, ethnicity, residence and substance use (table 1).

Table 1: Description and comparisons of study participants at JUSH, Jimma, Ethiopia March to April 2016

Variable		Study groups (n=210)				
		Total N (%)	DM group (n=105) N (%)	Control group (n=105) N (%)	t/X^2	p-value
Age(years)	Mean±SD	53.53	53.36±11.674	53.70±11.53	-0.214[t]	0.831
	30-45	57(27.1%)	29(27.6)	28(26.7)		
	46-55	55(26.2%)	29(27.6)	26(24.8)	0.712	0.870
	56-61	49(23.3%)	22(21.0)	27(25.7)		
	≥62	49(23.3%)	25(23.8)	24(22.9)		
Sex	Male	108(51.4	54(51.4)	54(51.4)	0.000	1.000
	Female	102(48.6)	51(48.6)	51(48.6)		
Religion	Orthodox	85 (40.5%)	39(37.1)	46(43.8)	5.857[*]	0.115
	Muslim	95(45.2%)	55(52.4)	40(38.1)		
	Protestant	22(15.5%)	7(6.7)	15(14.3)		
	Catholic	8(3.8%)	4(3.8)	4(3.8)		
Ethnicity	Oromo	116(55.2%)	67(63.8)	49(46.7)	7.610[*]	0.103
	Amhara	50(23.8%)	21(20.0)	29(27.6)		
	Tigre	16(7.6%)	5(4.8)	11(10.5)		
	Guraghe	21(10.0%)	8(7.6)	13(12.4)		
	Other[***]	7(3.3%)	4(3.8)	3(2.9)		
	Grade 8 and lower	126(60.0%)	63(60)	63(60)	0.000	1.000
Education level	Grade 9-12	56(26.7%)	28(26.7)	28(26.7)		
	College and above	28(13.3%)	14(13.3)	14(13.3)		
Marital status	Single	6 (2.9%)	3(2.9)	3(2.9)	20.000[*]	**0.000****
	Married	139(66.2%)	84(80.0)	55(52.4)		
	Divorced	35(14.3%)	8(7.6)	27(19.0)		
	Widowed	30(16.7%)	10(9.5)	20(25.7)		
Occupation	Employed	76(36.2%)	33(31.4)	43(41.0)	13.306*	**0.016****
	Merchant	31(14.8%)	10(9.5)	21(20.0)		
	Farmer	36(17.1%)	26(24.8)	10(9.5)		
	Housewife	56(26.7%)	31(29.5)	25(23.8)		
	Daily laborer	4(1.9%)	2(1.9)	2(1.9)		
	Other	7(3.3%)	3(2.9)	4(3.8)		
Income (birr)	Mean±SD	1446.67	1213.01±1093.274	1680.33±1287.516	-2.835[t]	**0.005**
	≤500	57(27.1%)	32(30.5)	25(23.8)	7.583	0.055
	501-1000	63(30.0%)	37(35.2)	26(24.8)		
	1001-2000	46(21.9%)	21(20.0)	25(23.8)		
	≥2001	44(21.0%)	15(14.3)	29(27.6)		
Residence	Urban	156(74.3%)	74(70.5)	82(78.1)	1.222	0.269
	Rural	54(25.7%)	31(29.5)	23(21.9)		
BMI (Kg/m^2)	Mean ±SD	23.473±3.47	24.153±4.1642	22.794±2.4284	2.889[t]	**0.004**
	<18.5	7(3.3%)	5(4.8)	2(1.9)		
	18.5-24.9	142(67.6%)	61(58.1)	81(77.1)	14.647[*]	**0.001****
	25-29.9	48(22.9%)	27(25.7)	21(20.0)		
	≥30	13(6.2%)	12(11.4)	1(1.0)		

*- Fischer exact test, ** - significant ,***- Adere, Dawuro, Kulo, Kefa, X^2= chi-square, t= independent t-test, SD= standard deviation, BMI= body mass index

5.1.2. Substance use profiles of study participants

Out of the 210 study participants 98(53.3%) had lifetime history of khat chewing of which 57(58.2%) were currently chewing khat. Of the current chewers; 32(56.1%) were chewing khat on a daily basis. Seventy nine (37.6%) respondents had lifetime history of alcohol drink of which 29(36.7%) were currently drinking. With respect to cigarette smoking 16(7.6%) had been smoking at least once in their lifetime of which 4(25%) were currently smoking. Among T2DM patients, 55(52.4%) were chewing khat at least once in their life time of which 34(61.8%) were currently chewing. Thirty three (31.4%) T2DM patients had life time alcohol drink. Only 10(9.5%) T2DM participants had smoked cigarette at least once in their life time. However, there was no significant difference in respect to substance use between the study groups (Table 2).

Table 2: Substance use profiles of study participants at JUSH, Jimma, Ethiopia, March to April 2016

Variable	Study groups (n=210)				
	DM group (n=105)	Control group (n=105)	Total		
	N (%)	N (%)	N (%)	X^2	p-value
Life time khat chewing history					
Yes	55(52.4)	43(41.0)	98(53.3)	2.315	0.128
No	50(47.6)	62(59.0)	112(46.7)		
Current khat chewing					
Yes	34(61.8)	23(53.5)	57(58.2)	0.388	0.533
No	21(38.2)	20(46.5)	41(41.8)		
Khat chewing frequency					
Habitual	21(61.8)	11(47.8)	32(56.1)	0.590	0.442
Occasional	13(38.2)	12(52.2)	25(43.9)		
Life time alcohol drink					
Yes	33(31.4)	46(43.8)	79(37.6)	2.922	0.087
No	72(68.6)	59(56.2)	131(62.4)		
Current alcohol drink					
Yes	11(33.3)	18(39.1)	29(36.7)	0.084	0.771
No	22(66.7)	28(60.9)	50(63.3)		
Life time cigarette smoking					
Yes	10(9.5)	6(5.7)	16(7.6)	0.609	0.435
No	95(90.5)	99(94.3)	194(92.4)		
Current cigarette smoking					
Yes	2(20.0)	2(33.3)	4(25.0)	-	0.604
No	8(80.0)	4(66.7)	12(75.0)		

N(%)-number (percent)

5.1.3. Clinical archives of T2DM patients

The mean FBG among T2DM was 164.02mg/dl (SD±68.54). Seventy two (68.6%) had hyperglycemia (≥ 126mg/dl) at the time of data collection. The mean duration of disease was 6.9 years (SD±5.5) with 64 (61.9%) of respondents have been living with DM for ≤6 years. Twenty one (20%) T2DM participants had history of hypoglycemia and 43(41.0%) individuals had comorbid HTN. Regarding medication, 66 (62.9%) T2DM patients rely on oral hypoglycemic agents only whereas 25(23.8%) used both insulin and oral hypoglycemic agents (Table 3).

5.2. Comparison of cognitive impairment among T2DM patients and healthy controls

The joint education adjusted MMSE score of the study participants was 24.55 with highly significant mean difference between the groups with mean of 23.41(SD±5.6) for DM and 25.7(SD±3.783) for controls (p= 0.001). Cognitive impairment had significant difference between the study groups (p= 0.002) (table 3).

Table 3: Comparison of cognitive status among study groups at JUSH, Jimma, Ethiopia 2016

Variable		Study groups (n=210)				
		Total N (%)	DM group (n=105) N (%)	Control group (n=105) N (%)	t/X^2	p-value
MMSE score	Mean±SD	24.55±4.9	23.41±5.60	25.70±3.783	-3.466^t	**0.001****
Cognitive impairment	Yes	89(42.4%)	56(53.3)	33(31.4)	9.438	**0.002****
	No	121(57.6%)	49(46.7)	72(68.6)		

** - significant , X^2= chi-square, t= independent t-test, SD= standard deviation, BMI= body mass index

Out of 105 DM patients, 31(29.5%) and 2(1.9%) had mild and severe cognitive impairment respectively whereas 26(24.8%) and 7(6.7%) healthy controls had mild and moderate cognitive impairment respectively (Figure 2). The overall cognitive impairment among DM was 53.3% [95% CI (43.8%, 62.8%)] and healthy controls it was 31.4% [95% CI (22.5%, 40.3%)]. DM patients had 2.5 times more risk of cognitive impairment than controls [OR= 2.49, 95% CI (1.42, 4.38), p=0.001)] (Table 4).

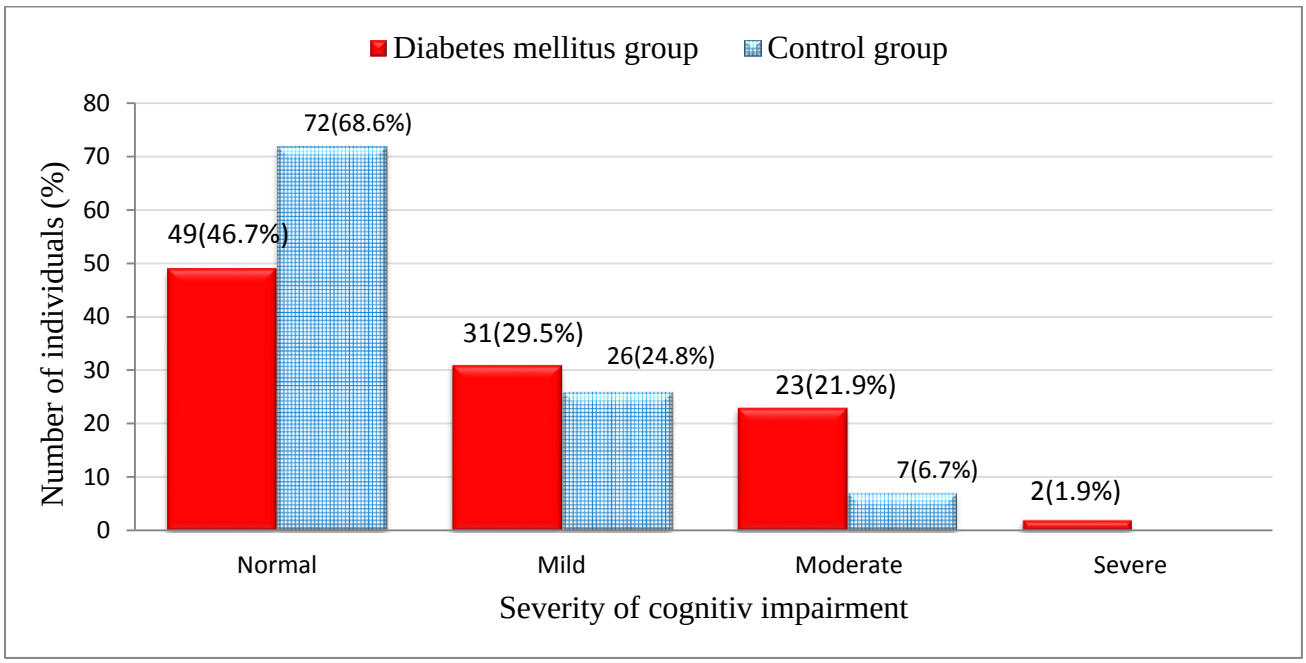

Figure 2: Levels of cognitive status among type 2 diabetes mellitus and healthy controls at JUSH, Jimma, Ethiopia March to April 2016

5.3. Predictors of cognitive impairment among T2DM patients

5.3.1. Association between clinical archives and cognitive impairment

From clinical variables of T2DM patients entered in binary logistic regression only duration of disease was associated with cognitive impairment. However, due to their clinical significance, all clinical variables were entered to multiple logistic regression using backward likelihood ratio method. Of all clinical variables; only FBG level and type of DM treatment were significantly associated with cognitive impairment in multiple logistic regression. T2DM patients with 126 mg/dl and above FBG were 4.4 times [AOR=4.43, 95% CI (1.14, 17.18)] more likely to develop cognitive impairment than those having FBG below 126mg/dl. The risk of having cognitive impairment among T2DM patients who used only oral hypoglycemic agents was nearly 5.4 times [AOR=5.39, 95% CI (1.37, 41.18)] higher than those who relied on insulin only (Table 4).

Table 4: Clinical variables and cognitive impairment in binary and multiple logistic regression among T2DM patients at JUSH, Jimma, Ethiopia, March to April 2016

Variable		Total N (%)	DM group (n=105)			
			Cognitive impairment			
			Yes N (%)	No N (%)	COR (95 % CI)	AOR (95 % CI)
FBG in mg/dl	Mean±SD		164.02±68.54			
	<126	33(31.4)	17(51.5)	16(48.5)	**1**	**1**
	≥126	72(68.6)	39(54.2)	33(45.8)	0.8(0.49-2.54)	**4.43(1.14-17.18)****
Disease duration(year)	Mean ±SD		6.883±5.5474			
	1-3	28(26.7)	13(46.4)	15(53.6)	**1**	**1**
	4-6	36(34.3)	18(50.0)	18(50.0)	1.15(0.43-3.10)	0.99(0.24-4.19)
	7-8	16(15.2)	11(68.8)	5(31.2)	**2.54(0.697-9.24)**	1.56(0.17-14.33)
	≥9	25(23.8)	14(56.0)	11(44.0)	1.47(0.497-4.34)	2.71(0.44-16.62)
Hypoglycemia episodes	Yes	21(20)	13(61.9)	8(38.1)	1.55(0.58-4.13)	3.02(0.78-11.72)
	No	84(80)	43(51.2)	41(48.8)	**1**	**1**
Comorbid HTN	Yes	43(41)	22(51.2)	21(48.8)	0.86(0.396-1.88)	1.05(0.33-3.35)
	No	62(59)	34(54.8)	28(45.2)	**1**	**1**
Type of DM medication	Insulin only	14(13.3)	6(42.9)	8(57.1)	**1**	**1**
	OHA only	66(62.9)	39(59.1)	27(40.9)	1.93(0.60-6.19)	**5.388(1.37-41.18)****
	Both	25(23.8)	11(44.0)	14(56.0)	1.048(0.28-3.92)	2.55(0.60-26.40)
Study group	DM group	105	56(53.3)	49(46.7)	**2.49(1.42,4.38)**	**p = 0.001**
	Control group	105	33 (31.4)	72(68.6)	**1**	

1-Reference,**-significant at p<0.05, OHA= Oral Hypoglycemic Agents, COR= Crude Odds Ratio, AOR= Adjusted Odds Ratio

5.3.2. Association between sociodemographic variables and cognitive impairment

Age, educational level, occupation, income and residence were associated with cognitive impairment among DM patients in binary logistic regression. Of all variables entered to multiple logistic regression only age and occupation along with FBG level and type of DM medication were significantly associated with cognitive impairment. T2DM patients aged ≥62 years were 7.5 times [AOR= 7.54, 95% CI (1.38, 41.38)] more risky for cognitive impairment than those ≤45 years (Table 5).

Table 5: Sociodemographic covariates and cognitive impairment in binary and multiple logistic regression analysis among DM patients at JUSH, Jimma, Ethiopia March to April 2016

Variables		DM group (n=105)			
		Cognitive impairment			
		Yes N (%)	No N (%)	COR (95 % CI)	AOR ((95 % CI))
Age (year)	30-45	11(37.9)	18(62.1)	**1**	**1**
	46-55	14(48.3)	15(51.7)	1.53(0.54-4.35)	1.59(0.39-6.54)
	56-61	13(59.1)	9(40.9)	**2.36(0.76-7.34)**	4.6(0.89-23.81)
	≥62	18(72.0)	7(28.0)	4.208(1.33-13.30)	**7.54(1.38-41.38)****
Sex N	Male	26(48.1)	28(51.9)	**1**	-
	Female	30(58.8)	21(41.2)	1.54(0. 71- 3.33)	
Educational level	Grade 8 and lower	47(74.6)	16(25.4)	**7.34(2.02-26.70)**	2.51(0.39-16.23)
	Grade 9-12	5(17.9)	23(82.1)	0.543(0.12-2.46)	0.27(0.04-1.71)
	college and above	4(28.6)	10(71.4)	**1**	**1**
Marital Status N	Single	0(0.0)	3(100)	0.0	
	Married	46(54.8)	38(45.2)	**1**	-
	Divorced	4(50.0)	4(50)	0.83(0.19-3.53)	
	Widowed	6(60.0)	4(40.0)	1.24(0.33-4.71)	
Occupation	Employed	9(27.30)	24(72.7)	**1**	**1**
	Merchant	4(40.0)	6(60)	1.78(0.41-7.80)	1.01(0.16-6.32)
	Farmer	22(84.6)	4(15.4)	**14.67(3.95-54.48)**	**7.38(1.26-43.15)****
	Housewife	20(64.5)	11(35.5)	**4.85(1.68-14.03)**	2.72(0.54-13.77)
	Daily laborer	0(0.0)	2(100)	0.0(-)	0.0(-)
	Other	1(33.3)	2(66.7)	1.33(0.11-16.57)	0.70(0.03-19.9)
Monthly income	≤500	21(65.6)	11(34.4)	**5.25(1.35-20.40)**	2.28(0.09-57.91)
	501-1000	23(62.2)	14(37.8)	**4.518(1.20-16.97)**	2.99(0.14-66.10)
	1001-2000	8(38.1)	13(61.9)	1.69(0.40-7.17)	2.61(0.15-46.29)
	≥2001	4(26.7)	11(73.3)	**1**	1
Residence	Urban	32(43.2)	42(56.8)	**1**	1
	Rural	24(77.4)	7(22.6)	**4.50(1.72-11.75)**	0.79(0.14-4.45)
BMI (Kg/m^2) N	<18.5	2(40.0)	3(60.0)	0.49(0.08-3.18)	
	18.5-24.9	35(57.4)	26(42.6)	**1**	-
	25-29.9	14(51.9)	13(48.1)	0.80(0.32-1.99)	
	≥30	5(41.7)	7(58.3)	0.53(0.15-1.86)	

N-Variable not candidate for multiple logistic regression, **- significant at p<0.05

5.3.3. Substance use and cognitive impairment among T2DM patients and healthy controls

Substance use related variables (life time and current khat chewing, alcohol drink, cigarette smoking and frequency of khat chewing) were tested for crude association with cognitive impairment in binary logistic regression. Nonetheless, there was no substance related variable with p <0.25 hence nothing was entered to multiple logistic regression analysis. So there was no association between substance use and cognitive impairment in both ` DM patients and healthy controls (Table 6).

Table 6: Substance use and cognitive impairment in binary logistic regression among type 2 DM and healthy controls in JUSTH, Jimma, Ethiopia, 2016

Variables		Study groups (n=210)					
		DM group (n=105)			Control group (n=105)		
		Cognitive impairment			Cognitive impairment		
		Yes N (%)	No N (%)	COR(95 % CI)	Yes N (%)	No N (%)	COR(95 % CI)
Life time khat	Yes	30(54.5)	25(45.5)	1.11(0.51-2.39)	14(32.6)	29(67.4)	1.09(0.47-2.52)
chewing	No	26(52.0)	24(48.0)	**1**	19(30.6)	43(69.4)	**1**
Current khat	Yes	20(58.8)	14(41.2)	0.90(0.39-2.05)	7(30.4)	16(69.6)	0.81(.23-2.92)
chewing	No	10(47.6)	11(52.4)	**1**	7(35.0)	13(65.0)	**1**
Khat chewing	Habitual	13(61.9)	8(38.1)	1.39(0.34-5.66)	4(36.4)	7(63.6)	1.71(0.29-10.3)
Frequency	Occasional	7(53.8)	6(46.2)	**1**	3(25.0)	9(75.0)	**1**
Life time alcohol drink	Yes	17(51.5)	16(48.5)	0.90(0.39-2.05)	16(34.8)	30(65.2)	1.32(0.58-3.02)
	No	39(54.2)	33(45.8)	**1**	17(28.8)	42(71.2)	**1**
Current alcohol drink	Yes	5(45.5)	6(54.5)	0.69(0.16-2.97)	5(27.8)	13(72.2)	0.59(0.17-2.14)
	No	12(54.5)	10(45.5)	**1**	11(39.3)	17(60.7)	**1**
Life time cigarette	Yes	4(40.0)	6(60.0)	0.55(0.15-2.08)	3(50.0)	3(50.0)	2.30(0.44-12.1)
	No	52(54.7)	43(45.3)	**1**	30(30.3)	69(69.7)	**1**
Current cigarette	Yes	1(50.0)	1(50.0)	1.67(0.07-37.73)	1(50)	1(50)	1.0(0.03-29.8)
	No	3(37.5)	5(62.5)	**1**	2(50)	2(50)	**1**

N- number,% percentage

CHAPTER 6: DISCUSSION

Characteristics of study participants

This research, the first in its kind (comparative study) in Ethiopia, tried to offer insight on magnitude of cognitive impairment and its significant predictors among T2DM patients in comparison with healthy individuals. In this study equal number of DM and healthy control groups were involved. Group matching was done for age, sex and educational status between groups which were expected to be major predictors of cognitive impairment. There was no significant difference between the study groups concerning religion, ethnicity, income, residence and substance use. However, body mass index, occupation and marital status showed significant difference between the groups of which occupation was predictor of cognitive impairment among DM patients.

Prevalence of cognitive impairment

Cognitive impairment is the foremost neurophysiologic disturbance which would be caused due to neuronal damage and /or functional defect of neurotransmitters (44,46). In this study, there was higher prevalence of cognitive impairment (53.3%) among T2DM patients which was nearly similar with the findings of study conducted in Tikur Anbesa referral Hospital, Ethiopia with similar instrument (MMSE) (23) and Nigeria (41). The possible reason for higher prevalence of cognitive impairment among DM individuals could be due to lack of insulin or signaling disturbance in the brain and/or effect of hyperglycemia in brain regions particularly in those involved in cognitive activities. However, a cross sectional study conducted in Saudi Arabia reported a lower prevalence of cognitive impairment than the findings of this research (36). This difference might be due to differences in sample size and sociodemographic characteristics among the study participants.

Even though the study instruments may differ; the prevalence of cognitive impairment among DM was higher than those with chronic obstructive pulmonary disease and Rheumatoid arthritis which was 17.5% and 31% respectively and almost similar with cognitive impairment due to HIV/AIDS which was 50% (58–60).

The prevalence of cognitive impairment among healthy controls in this study was 31.4% which was similar with the report in Central Africa republic (22). But findings from India, South Korea and Malaysia (21) as well as systematic review of African nations among the general population reported a lower prevalence of cognitive impairment (22) than our finding from healthy individuals.

The possible reason for this difference might be due to variations in sociodemographic characteristics, level of study and sample size.

This study indicated a significantly higher prevalence of cognitive impairment among DM patients than healthy controls; with 2.5 times risk among DM patients than controls [OR= 2.49, 95% CI (1.42, 4.38)]. It might be due to the effect of rise in blood glucose level and type of medication used for DM. Other studies braced this finding in that DM patients were 1.3 times more likely (16,17) to develop cognitive impairment than healthy controls (35,54–56). The mean MMSE score of DM patients was significantly lower than healthy controls ($p = 0.001$) in that 5.5% [eta-squared= $0.055= t^2/ [t2+ (n_1+n_2)-2]$] MMSE score variation (effect size) was explained by the presence of T2DM. This was comparable with the results of study conducted in Egypt (40). This would be an alarming phenomenon for people to consider type 2 DM as more dreadful than other diseases as risk for cognitive impairment.

Predictors of cognitive impairment

Uncontrolled blood glucose level was statistically significant predictor of cognitive impairment in T2DM patients which was similar with other studies (40) which could be due to the effect of hyperglycemia in neuronal toxicity and accumulation of AGE and free radical formation those lead to oxidative stress and enhance cognitive impairment.

Use of only oral hypoglycemic agents as a treatment option was significantly associated with cognitive impairment. The possible reason for this could be due to effect of oral hypoglycemic agents on neurovitamins or use of oral hypoglycemic agents without combining with insulin might indirectly bared the additional neuroprotective role of insulin in the brain hence increase the risk of cognitive impairment. But systematic review study disclosed contrary finding to this study in that oral hypoglycemic agent prevents cognitive impairment (38). The possible reason for the contradiction might be due the level of study, study design and also the review study might dealt on oral hypoglycemic agents combing with insulin but this study solely reported oral hypoglycemic agents only.

Cognitive impairment was significantly associated with age (particularly elderly) which was consistent with other studies (32,34,40). This might be due to the neurotransmitter derangement during ageing in either quantity or quality which might result in defect of cognitive components like memory.

Occupation was also significant predictor of cognitive impairment in DM patients in that farmers were 7.5 times risky to have cognitive impairment than wage employed individuals which was in line with a study in India (32). The possible reason might be due to educational (lack of intellectual stimulation) and life style differences within the participants that could contribute to poor glycemic control.

Substance use and cognitive impairment

The results of this study disclosed that any of the substances used were not associated with cognitive impairment, neither protective nor risk. This was supported by one study in Ethiopia in that cigarette smoking was not associated with cognitive impairment in T2DM patients (23). Contrary to this in Kenya and Netherlands khat chewing was associated with impaired cognition (59,60) and also one study pointed out that cigarette smoking would improve cognitive performance (68). The possible reason for the difference might be due to sample size, sociodemographic characteristics and techniques used.

Limitations of the study

There were difficulties in the process of matching. However, the sociodemographic data showed that the groups were not significantly different in respect to key characteristics which might ascertain the less likely occurrence of important bias due to matching. Lead time bias, recall and social desirability bias were the problems of the design itself. Due to small sample size, there might be difficult for generalization. The MMSE is only screening test hence could not diagnose real cognitively impaired cases. Failure to do biochemical tests other than blood glucose level those might contribute for cognitive impairment was also another limitation.

CHAPTER 7: CONCLUSION AND RECOMMENDATION

7.1. Conclusion

The findings of this study bared the preponderant prevalence of cognitive impairment among T2DM patients than healthy controls. The independent predictors of cognitive impairment among DM patients were blood glucose level, type of DM medication used, age and occupation. DM patients with ≥FBG level were 4.4 times more probable to acquire cognitive impairment than those with < 126mg/dl. The likelihood of cognitively impaired was nearly 5.4 times higher among DM patients who relied on only oral hypoglycemic agents than those using insulin only. DM patients aged ≥62 years were 7.5 times risky for cognitive impairment than those ≤45 years. Occupation was also significantly associated with cognitive impairment. Despite the higher proportion of substance use, no substance use related variable was significantly associated with cognitive impairment among DM patients.

7.2. Recommendation

Federal ministry of health of Ethiopia

- To integrate screening strategies for cognitive impairment among DM patients as part of diagnostic modality

Jimma Zone Diabetes Mellitus Association

- To organize regular panel discussions for DM patients to create awareness about compilations of DM particularly on cognition

For researchers

- To undertake further study concerning the issue with relatively strong study designs like cohort and longitudinal.

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ANNEX I- ENGLISH VERSION QUESTIONNAIRE

Jimma University College of Health Sciences

Department of Biomedical Sciences (Medical Physiology)

Informed consent form

Hello dear, my name is---------- I come here as data collector to assess cognitive impairment among T2DM patients in JUSH DM clinic. On this questionnaire your name will not be written and I am going to ask some questions related to sociodemographic, health related issues and cognition as well as I will examine your blood pressure, weight and height. You may end this interview any time you want. However, your honest answer to these questions will help to focus on how to care on this problem in order to develop better strategies and solve the problems for the future. We would greatly appreciate your truthful and active participation in responding to this questionnaire.

Are you willing to participate? A. Yes B. No

If yes continue the data collection process

Date of interview…….

Interviewer name…….

Signature ----------

Part 1- Sociodemographic characteristics Code number of participant _____

S/N	Variables	
101	Age	_____ years
10	Sex	0. Male 1. Female
103	Religion	0. Orthodox 1.Muslim 2.Protestant 3.Catholic 4. Others(specify)
104	Ethnicity	0.Oromo 1.Amhara 2.Tigre 3.Guraghe 4 .others(specify)
105	Educational status	0. Grade 8 and lower1. Grade 9-122. 2. >grade 12(college and & above)
106	Marital status	0. Single 1. married 2.divorced 3.widowed
107	Occupation	0. Government employee 1. Private employee 2. Merchant 3. Farmer 4. House wife 5. Daily laborers 6. Others specify--
108	Monthly income	_____Ethiopian birr
109	Residence	0. Urban 1. Rural

Part 2.Medical history - see chart of the patient

201	Episodes of hypoglycemia	0. Yes 1. No
202	Presence of co morbid HTN	0. Yes 1. No
203	Type of DM treatment the patient rely on	0. Insulin 1.Oral hypoglycemic drugs 2.Both
204	Duration of disease since diagnosis	_____ Months
205. FBS _____mg/dl (fasting blood glucose level of the patient at time of data collection)		

Part 3- Substance use assessment (alcohol intake, khat chewing, cigarette smoking)

301	Have you ever chewed khat in your life time?	0. Yes 1. No
302	If yes to Q301, for how many years have you chewed khat?	_____months
303	If yes to Q301, have you chewed khat within the last 30days?	0. Yes 1. No
304	If yes to Q303, how often you chew khat? Specify (daily, weekly,	_____
305	If yes to Q303, what amount of khat you chew per day?	_____grams
306	Have you ever drunk alcohol in your life time?	0. Yes 1. No
307	If yes to Q306, for how long have you been drinking alcohol?	_____
308	If yes to Q306, have you drink alcohol within the last 30days?	0. Yes 1.No
309	If yes to Q308, what type of alcohol do you drink? Specify	_____
310	If yes to Q308, how much liter of alcohol you drink per week?	_____L
311	Have you ever smoked cigarette in your life time?	0. Yes 1. No
312	If yes to Q311, have you smoked within the last 30days?	0. Yes 1.No
313	If yes to Q312, how many cigarettes you smoke daily (in pcs)	_____

Part 4- Physical examination (measurement)

401.	Body mass index	Weight ____Kg Height ___meter

Part 5- Standardized mini-mental state examination for cognitive assessment (write the score)

501	Types of questions	score	T/s	Types of questions	Score	T/s
	What **year** is this?	__/1	10	What **country** are we in	__/1	10
	What **season** is this	__/1	10	What **region** are we in	__/1	10
	What **date** is this	__/1	10	What **town** are we in?	__/1	10
	What **day** is this?	__/1	10	What is the name of this **hospita**l?	__/1	10
	What **month** is this?	__/1	10	What **floor of the building** are we on?	__/1	10
502	I am going to name three objects. When I have finished, I want you to repeat them. Remember what they are because I am going to ask you to name them again later : **Bag / key/ arm**[score out of three]				_/3	20
503	Please count from 5 backwards (5,4,3,2,1)				__/5	30
504	What were the three objects I asked you to remember? (score one point for each correct answer regardless of order)				__/ 3	10
505	Show **wrist watch.** What is this called?				__/1	10
	Show **pencil**. What is this called?				__/1	10
506	I would like you to repeat a phrase after me: **No ifs, ands or buts**?				__/1	10
507	Read this and then do what it says. Then, hands the person the sheet with **CLOSE YOUR EYES** on it. If the participant just reads and does not close eyes, you may repeat to a maximum of three times. Score one point only if the subject closes eyes.				__/1	10
508	Hand the person a pencil and paper Say: Write any complete sentence on that piece of paper. The sentence must make sense. Ignore spelling errors.				_/1	30
509	Place design, eraser and pencil in front of the person. Say: Copy this design please. Allow multiple tries. Wait until the person is finished and hands it back. Score one point for a correctly copied diagram. The person must have drawn a four-sided figure between two five-sided figures				___/1	60
510	Ask the person if he is right or left handed. Take this paper in your right/left hand (whichever is non-dominant), fold the paper in half once with both hands and put the paper down on the floor.			Take paper in correct hand	__/1	30
				Folds it in half	__/1	
				Puts it on the floor	__/1	
				Total score	**__/30**	**5m,10s**

NB- T=time, S=seconds, m= minutes

ANNEX II- AMHARIC VERSION QUESTIONNAIRE

ጅማ ዩኒቨርሲቲ ጤና ሳይንስ ኮሌጅ

ባዮሜዲካል (ፊዚዮሎጂ) ትምህርት ክፍል

የፈቃደኝነት ማረጋገጫ

ጤና ይስጥልኝ _____ እባላለሁ፤ እዚህ የመጣሁት በጅማ ዩኒቨርሲቲ ስፔሻላይዝድ ሆስፒታል በዓይነት 2 የስኳር ህሙማን ላይ የሚደርሰውን የአእምሮ ሁነት ጉዳትን ለማጥናት መረጃ ሰብሳቢ ሁኝ ነው፡፡ በዚህ መጠይቅ ላይ ስምዎ የማይጻፍ ሲሆን ማህበራዊ እና ኢኮኖሚያዊ፣ አእምሮአዊ ሁነት፣ ንጥረ ነገርን (ጫት፣ አልኮሆል እና ሲጋራን) በተመለከተ እና አካላዊ ልኬት አደርጋለሁ፡፡ የእርስዎ ተሳትፎ ለዚህ ጥናት ከፍተኛ ዋጋ ያለው ሲሆን በህሙማኑ የሚደርሰውን ችግር ለመከላከል ከፍተኛ ሚና አለው፡፡ እርስዎ በማንኛዉም ሰአት የቃለ መጠይቁን ሂደት ማቋረጥ ይችላሉ፡፡

ለመሳተፍ ፈቃደኛ ነዎት?

ሀ. አዎ ለ. አይደለሁም

መልሱ አዎ ከሆነ መጠይቁን ይጀምሩ፡፡ አይደለሁም ከሆነ ግን አመስግነው ይሰናበቱ

ቃለ መጠይቁ የተካሄደበት ቀን…….

ቃለ መጠይቁን የጠየቀው መረጃ ሰብሰቢ ስም……

ፊርማ ----------

ክፍል I.ማህበራዊ እና ኢኮኖሚያዊ ሁኔታን የሚመለከቱ ጥያቄዎች የጥናቱ ተሳታፊ መለያ ቁጥር_____

S/N	ጥያቄዎች	ምላሾች
101	እድሜ	_____ዓመት
102	ፆታ	0. ወንድ 1. ሴት
103	ሃይማኖት	o. ኦርቶዶክስ 1.ሙስሊም 3.ፕሮቴስታንት 4. ካቶሊክ 5. ሌላካለ
104	ብሄር	0. ኦሮሞ 2. አማራ 3. ትግሬ 4. ጉራጌ 5. ሌላ ካለ ጥቀስ----------
105	የትምህርት ደረጃ	o. የመጀመሪያ ደረጃ (≤8) 1. ሁለተኛ ደረጃ (9-12) 2. ኮሌጅ ወይም ዩኒቨርሲቲ
106	የጋብቻዎ ሁኔታ	o. ያላገባ/ች 1. ያገባ/ች 2. አግብቶ/ታ የፈታ/ች 3. የትዳር አጋር በሞት ያጣ/ች
107	ስራ	0. የመንግስት ሰራተኛ 1. የግል ሰራተኛ 2. ነጋዴ 3. አርሶ አደር 4. የቤት እመቤት5. የቀን ሰራተኛ 6. ሌላካለ___
108	ወርሃዊ ገቢ	_____ ብር
109	በቋሚነት የሚኖሩበት ቦታ የት ነው?	o. ከተማ 1. ገጠር

ክፍል II. የህክምና ታሪክ (የታካሚውን የህክምና ቻርት ተመልከት)

201	በደም የስኳር መጠን መቀነስ አጋጥሟቸው ያዉቀል?	0. አዎ 1. የለም
202	ከስኳር በተጨማሪ የደም ግፊት መጨመር አለባቸው?	0. አዎ1. የለም
203	ለስኳር ህመሙ የሚጠቀሙት የመድሀኒት ዓይነት	0.ኢንሱሊን ብቻ 1.በአፍ የሚወሰድ ክኒን ብቻ 2.ሁለቱንም
204	የስኳር ህመሙ ከታወቀ ጀመሮ ያለው የቆይታ ጊዜ (በወር)?	_____ዓመት ከ_____ ወር

205 FBS_____mg/dl

ክፍል III. የንጥረ ነገር ሁኔታ ጥያቄዎች (አልኮሆል መጠጥ፣ጫት መቃም፣ ሲገራ ማጨስ)

301	በሂዎትዎ ጫት ቅመው ያውቀሉ	0. አዎ1. አላውቅም
302	ለጥየቄ 301 መልሱ አዎ ከሆነ, ለስንት ጊዜ ያህል ቅመወል? (በወር)	_____ዓመት ከ_____ ወር
303	ለጥየቄ 301 መልሱ አዎ ከሆነ, ባለፉት 30 ቀናት ዉስጥ ቅመወል?	0. አዎ1. የለም
304	ለጥየቄ 303 መልሱ አዎ ከሆነ, ምን ያህል ጊዜ ቅማሉ?(በየቀኑ,በሳምንት)	_____
305	ለጥየቄ 303 መልሱ አዎ ከሆነ, በቀን ምን ያህል መጠን ጫት ይቅማሉ ?	_____ግራም ወይም_____ ዙርባ) (እስር)
306	በሂዎትዎ አልኮሆል ያለበት መጠጥ ጠጥተው ያውቃሉ?	0. አዎ1. አላውቅም
307	ለጥየቄ 306 መልሱ አዎ ከሆነ, ለስንት ጊዜ አልኮሆል ጠቅመዋል?	_____
308	ለጥየቄ 306 መልሱ አዎ ከሆነ, ባለፉት 30 ቀናት አልኮሆል ጠቅመወል?	0. አዎ 1.የለም
309	ለጥየቄ 308 መልሱ አዎ ከሆነ, ምን አይነት አልኮሆል ተጠቀሙ? (ዘርዝር)	_____፣_____፣_____፣_____
310	ለጥየቄ 308 መልሱ አዎ ከሆነ, ምን ያህል አልኮሆል በሳምንት ይጠቀማሉ?	_____ሊ
311	በሂዎትዎ ሲጋራ አጭሰው ያውቃሉ?	0. አዎ1. አላውቅም
312	ለጥየቄ 311 መልሱ አዎ ከሆነ, ባለፉት 30 ቀናት አጭሰዋል?	0. አዎ 1.የለም
313	ለጥየቄ 312 መልሱ አዎ ከሆነ, በቀን ምን ያህል ሲጋራ ያጨሳሉ ? (ቁጥር)	_____

ክፍል IV- የአካል ልኬት

401	የሰውነት ክብደት ጠቋሚ: ክብደት _____ኪ.ግ ቁመት_____ሜ

ክፍል V፡የአእምሮ ስራን ሁኔታ በተመለከተ

	ጥየቄ	ስኮር	ጊዜ/ሴ		ስኮር	ጊዜ./ሴ
501	ዘንድሮ ዓመቱ ማን ነው?	__/1	10	አሁን በየትኛው ሃገር ነው የምንኖረው?	___/1	10
	ይህ ወቅት ምንድን ነው?	__/1	10	በየትኛው ክልል ነው አሁን የምንገኘው?	___/1	10
	ዛሬ ቀኑ ስንት ነው?	__/1	10	አሁን የምንገኝበት ከተማ ማን ነው?	___/1	10
	የዛሬው እለት ማን ነው?	__/1	10	ያለንበት ሆስፒተል ስም ማን ይባላል?	___/1	10
	ይህ ወር ማን ነው?	__/1	10	ያለንበት ቤት ወለል የተሰራው ከምንድን ነው?	___/1	10
502	የምጠራልህን ቃላት አድምጥና ደግመህንገረኝ ፡፡ከትንሽ ቆይታ በኋላ በድጋሜ እንድትነግረኝ ስለምፈልግ ለማስታወስ ሞክር፡፡ቃሎቹም፡- ቦርሳ፣ ቁልፍ፣ ክንድ (አንድ ቃል ለመጥራት 1 ሴኮንድ ተጠቀም፣ ቅደም ተከተሉን ጠብቆ ካልመለሰ ‹0› ይሰጠዋል)				___/3	20
503	“ከ5 ጀምሮ ወደ ኋላ ይቁጠሩ (5፣4፣3፣2፣1)				___/5	30
504	ቅድም እንድታስታውስ የነገረኩህን ቃላት አሁን ድገምልኝ? (ቅደም ተከተል በይጠብቅም በጠራው ልክ ስጥ)				__/ 3	10
505	ይህ ምን ይባላል?(የእጅ ሰዓት አሳየው)				___/1	10
	ይህ ምን ይባላል? (እርሳስ አሳየው)				___/1	10
506	የሚከተሉትን ሀረጎች ደጋግመህ ጥራልኝ፤ አይሆንም/እናስ/ ነገርግን (በትክክል ከደገመ 1 ስጥ)				___/1	10
507	በካርድ ላይ የሚነበብ ነገር ጻፍ “ አይንህን ጨፍን” የሚል:: ይህን አንብበው ያነበቡትን በተግባር ያሳዩ (አንበቦ በተግበር ከልመለሰ ቢበዘ ለ3 ጊዜ ደግሞ ይሞክር፡፡ ዐይኑን ከጨፈነ ብቻ 1 ይሰጠው.)				___/1	10
508	ለግለሰቡ ወረቀት እና እርሳስ ስጠው:: ሰዋስዋዊ ስርዓቱን ያሟላ ዐረፍተ ነገር እንዲጽፍ ጠይቀው። ዐረፍተ ነገሩን መፃፍ ያለበት በራሱ ምርጫና ፍላጎት በፍጥነት መሆን ይኖርበታ. የፊደል ስህተት ችግር የለም.				__/1	30
509	ለግለሰቡ ቀጥሎ ያለውን ስዕል፣ ወረቀት እና እርሳስ ስጠውና በሁለቱ ስዕሎች መካከል ያለውን አራት ጎን የለው ስዕል እንዲስል ጠይቀው፡፡የሁለቱም ስዕሎች ሁሉም አንግሎች መኖር አለባቸው። በትክክል ከሳለ 1 ስጠው፡፡ (ይህን ምስል አስመስሎ ይሳል)				___/1	60
510	ተሳታፊው በግራ ወይም በቀኝ እጅ የሚጠቀም መሆኑን ከጠየከ በኋላ፤ ቁራጭ ወረቀት ይዘህ ይህን ትእዘዝስጥ፤ ይህን ወረቀት በቀኝህ/በግረህ (በብዛት በምትጠቀምበት እጅ) ዉስድ፣ አንድ ጊዜ በሁለት እጅህ አኩል እጠፈው፣ ከዚያም የታጠፈውን ወረቀት በወሰድከበት እጅ ወለል ላይ አስቀምጥ	በትክክለኛው እጅ ወስዷል	___/1	30	
		እኩል አጥፏል	___/1		
		ወረቀቱን በትክክለኛው እጅ ወለል ላይ አስቀምጧል	___/1		
	አጠቃላይ ስኮር (ድምር)				__/30	5ደ ከ10ሴ

DECLARATION

I, the undersigned, declare that this thesis is my original work, has not been presented for a degree in this or any other university and that all sources of materials used for the thesis have been fully acknowledged.

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This thesis has been submitted for examination with my approval as University advisor

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