EUROPEAN JOURNAL OF BIOMEDICAL AND PHARMACEUTICAL SCIENCES
http://www.ejbps.com

ISSN 2349-8870
Volume: 4
Issue: 1
418-428
Year: 2017

ASSESSMENT OF PREVALENCE AND COMPLICATIONS OF TYPE-II DIABETES MELLITUS

Jaladi Himaja[1], Nancy Grace Daniel*[1], Linta Kurian[2] and Battu Rakesh[3]

*[1,1,2,3]Pharm.D Interns, Department of Pharmacy Practice, Bharathi College of Pharmacy, Bharathinagara, K.M. Doddi, Mandya, Karnataka, India-571422.

***Corresponding Author: Nancy Grace Daniel**
Pharm.D Interns, Department of Pharmacy Practice, Bharathi College of Pharmacy, Bharathinagara, K.M. Doddi, Mandya, Karnataka, India-571422.

Article Received on 16/11/2016 **Article Revised on 06/12/2016** **Article Accepted on 26/12/2016**

ABSTRACT
Background: Diabetes mellitus (DM) is an endocrinological and/or metabolic disorder with an accumulative global occurrence and incidence. Diabetes mellitus is a metabolic-cum-vascular syndrome of diverse etiologies characterised by prolonged hyperglycaemia with resultant disturbances of carbohydrate, fat and protein metabolism with subsequent defects in insulin secretion, insulin action or both. Recent guidelines for treating patients with type 2 diabetes mellitus are based on glycaemic standards derived from medical specialty data; nonetheless, the course of the disease, from prediabetes to end-stage complications, is not the similar in all patients. Microvascular complications, comprising nephropathy, retinopathy and neuropathy, are intensely related to haemoglobin A_{1c} (HbA_{1c}). However, vascular complications may headway in patients who have HbA_{1c} <7.0% and may appear even in undiagnosed patients owing to transitory increases in plasma glucose concentrations. Concomitant atherosclerosis and occult macro vascular disease may follow a hastened course in type 2 diabetes. Managing hyperglycaemia in the advanced stages of type 2 diabetes does not appear to be associated with value-added cardiovascular outcomes. The glucotoxicity and lipotoxicity that may precede sustained hyperglycaemia and β-cell dysfunction are initial, reversible pathophysiologic events. This suggests that quick management may modify the course of hyperglycaemia and avert or delay long-term complications. The challenge remains to identify patients with early type 2 diabetes who are at risk for hasty progression of β-cell failure and early development of microvascular complications. **Objective:** To assess the prevalence and complications of type-II diabetes mellitus. **Study Design:** A prospective observational study was carried out in the General Medicine Department, Mandya Institute of Medical Sciences and Teaching Hospital, Mandya, Karnataka, using a well-designed patient data collection form. **Results:** Out of 121 diabetic patients studied 56.20% were males, 43.80% were females. Most of the patients with diabetes fall in the age group of 61-70 years. Among the complications developed macrovascular complications (59%) were having more prevalence rate than microvascular complications (22%). **Conclusion:** Prevalence of DM was more in males than in females and macro vascular complications were having more prevalence rate than micro vascular complications.

KEYWORDS: Cardiovascular diseases, Multimorbidity, Cardiovascular Events and sedentary lifestyle.

INTRODUCTION
Diabetes mellitus is a metabolic-cum-vascular syndrome of numerous etiologies characterised by long-lasting hyperglycaemia with disturbances of carbohydrate, fat and protein metabolism with subsequent defects in insulin secretion, insulin action or both. This disorder is commonly associated with long term damage, which can lead to failure of organs like eyes, kidney, nerves and heart and blood vessels.[1]

Diabetes is linked with a number of complications. Acute metabolic complications associated with mortality comprise diabetic ketoacidosis from extremely high blood glucose concentrations (hyperglycaemia) and coma as the consequence of low blood glucose (hypoglycaemia). These complications are wide ranging and are due at minimum in part to chronic rise of blood glucose levels, which leads to damage of blood vessels (angiopathy) (Figure 1) (Figure 3). In diabetes, the resulting complications are grouped under "microvascular disease" (due to impairment to small blood vessels) and "macrovascular disease" (owing to damage to the arteries). Microvascular complications include eye disease or "retinopathy" kidney disease called "nephropathy" and neural damage or "neuropathy". The most important macrovascular complications embrace augmented cardiovascular disease ensuing in myocardial infarction and cerebrovascular disease exhibiting as strokes. Other

chronic complications of diabetes include depression, dementia and sexual dysfunction.[2]

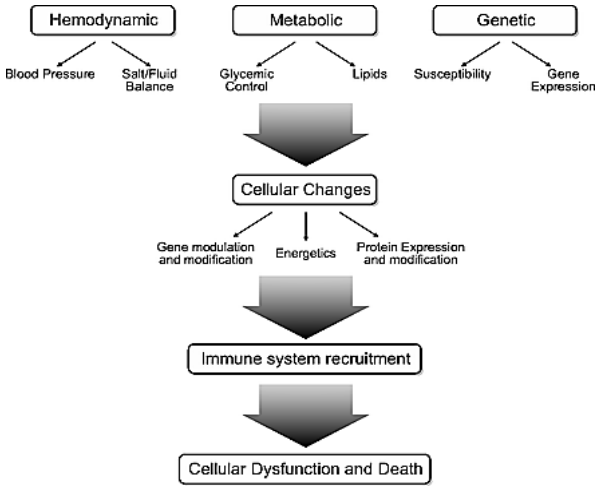

Fig.1: Schematic Overview of the Major Areas Contributing To Diabetic Complications.

PATHOPHYSIOLOGICAL BASIS OF MICRO VERSUS MACROVASCULAR COMPLICATIONS

Patients with type-II Diabetes Mellitus and related micro vascular complications seem particularly at advanced risk of accelerated atherosclerosis which ultimately terminates in cerebrovascular and cardiovascular events and premature death.[3] Micro vessels are the basic functional unit of the cardiovascular system comprising of arterioles, capillaries and venules. They differ from macro vessels in both their architecture and cellular components. In contrast to macro vessels supplying blood to organs, micro vessels play important roles in maintaining blood pressure and proper nutrient delivery.[4] The microcirculation also has regulatory systems controlling vascular permeability and myogenic responses that can adapt blood flow according to local metabolic needs. Alteration in microvascular function may arise even before overt hyperglycaemia and vascular pathologic changes manifest. Diabetes induces pathognomonic changes in the microvasculature, affecting the vessel basement membrane comprising arterioles in the glomeruli, retina, myocardium, skin and muscle, by increasing their thickness, leading to the development of diabetic microangiopathy. This thickening ultimately leads to abnormality in vessel function, inducing numerous medical problems such as hypertension, delayed wound healing and tissue hypoxia. Similarly, neovascularization ascending from the vasa vasorum presumably can interconnect macro-and microangiopathy, envisage platelet rupture and stimulate atherosclerosis.[4]

ADVANCED GLYCATION PRODUCTS (AGEs)

Advanced glycation of free amino groups on proteins and amino acids is a non-enzymatic post translational modification, which begins with covalent attachment of heterogeneous sugar moieties. AGEs mount up in diverse forms of cells and affect their extracellular and intracellular organization and function by cross-linking not only with proteins but also lipids and nucleic acids backing to a diversity of diabetic complications.[5] Advanced glycation products crosslink with plasma membrane-localized receptors for AGEs (RAGE) leading to up-regulation of transcription factors such as nuclear factor-κB and its target genes, proclamation of pro-inflammatory molecules besides free radicals. Soluble AGEs activate monocytes and AGEs in the basement membrane impede monocyte migration. AGE-bound RAGE upsurges endothelial porosity to macromolecules (Figure 2). AGEs block nitric oxide action in the endothelium and cause the production of Reactive Oxygen Species (ROS).[6] Further, AGEs modify LDL particles and together with vascular damage hasten atherosclerosis.[7]

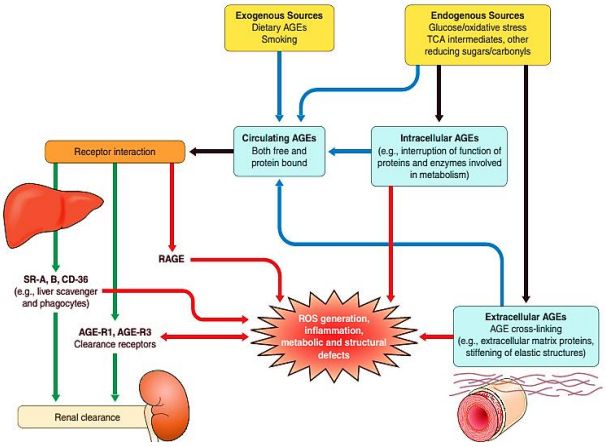

Fig.2: Advanced glycation end products and their trafficking. TCA, tricarboxylic acid cycle.

This reaction, first discovered one hundred years ago and termed the "Maillard reaction", is influenced by numerous aspects including intracellular glucose concentrations, pH and time.

MICROVASCULAR COMPLICATIONS OF DIABETES

Diabetic Retinopathy

Diabetic retinopathy might be the utmost common micro vascular complication of type-II diabetes. It is accountable for ~ 10,000 new cases of blindness each year in the United States only. The danger of mounting diabetic retinopathy or other micro vascular complications of DM rest on both the duration and the severity of hyperglycaemia. Advancement of diabetic retinopathy in patients with type 2 diabetes was found to be linked to both severity of hyperglycaemia and manifestation of hypertension. Retinopathy may originate to develop as early as 7 years before the diagnosis of diabetes in patients with type 2 diabetes.

There are numerous anticipated pathological mechanisms by which diabetes may lead to development of retinopathy:

- Aldose reductase may contribute in the development of diabetes complications. Aldose reductase is the preliminary enzyme in the intracellular polyol

pathway. This pathway comprises the transformation of glucose into glucose alcohol (sorbitol). High glucose levels rise the flux of sugar molecules through the polyol pathway, which causes sorbitol accumulation in cells. Osmotic stress from sorbitol build-up has been postulated as a fundamental mechanism in the development of diabetic microvascular complications, including diabetic retinopathy.

- Oxidative stress may also play an imperative role in cellular injury from hyperglycaemia. High glucose levels can stimulate free radical production and reactive oxygen species formation.
- Growth factors, comprising growth hormone, vascular endothelial growth factor (VEGF) and TGF (transforming growth factor) β, have also been assumed to play significant roles in the development of diabetic retinopathy. VEGF production is augmented in diabetic retinopathy, possibly in response to hypoxia.[8]

Diabetic Nephropathy

Diabetic nephropathy is one of the leading causes of end-stage renal disease in the western world. It is the major cause of morbidity and mortality in Type-I DM patients and is becoming a serious clinical problem in Type II DM patients. After presentation, the initial changes in kidney include increased renal blood flow, hypertrophy, glomerular hyper filtration and hyper perfusion. These early stage changes are reversible and are not considered as a reliable indicator for the development of diabetic nephropathy. Persistent hyperglycemia for numerous years persuades structural and cellular effects in the kidney. The substantial structural changes comprises thickening of the glomerular hypertrophy, increased extracellular matrix accumulation (tubule interstitial fibrosis) with mesangial expansion, glomerular basement membrane and modest enlargement of the tubule interstitium. High blood glucose encourages cellular changes in several kinds of cells existing in the kidney. The major cellular abnormalities progress in glomerular epithelial cells (podocytes), which embraces broadening of podocyte foot processes with advanced decline in their number and density per glomerulus.[9]

Clinically, there is a decline in glomerular-filtration rate (GFR) with progressive increase in urinary albumin secretion, and in connotation with a rise in blood pressure, it eventually leads to end-stage renal failure. The initial manifestation of diabetic kidney disease can be identified by the presence of micro albuminuria, a state known as *incipient diabetic nephropathy*, where there is presence of lesser amounts of albumin in the urine (30–300 mg/day).[9]

After the phase of micro albuminuria, there is a sustained rise in urinary protein excretion with declining GFR. This results in the development of Albustix-positive proteinuria and is known as *overt nephropathy or macro proteinuria*. In DM patients with >5 years of hyperglycemia, presence of determined albuminuria [albumin excretion rate (AER)>300 mg/24 hours] without any urinary tract infection (UTI), other renal diseases or heart diseases represents diabetic nephropathy. If left untreated, uremia will supervene and necessitate referral to end-stage renal failure programs, such as *dialysis or transplantation*.

Hypertension plays a critical role in the advancement of diabetic nephropathy. Controlling the blood pressure shows substantial Reno protective and antiproteinuric effects. In addition, lowering blood pressure diminishes albuminuria and weakens the rate of loss of GFR in both Type-I DM and Type-II 2 DM patients. The foremost strategies presently used to lessen the hazard of onset or progressions of diabetic nephropathy are glycemic control along with rigorous management of systemic blood pressure. The major approach used to manage blood pressure is modification in renin– angiotensin aldosterone system (RAAS) by means of angiotensin-converting enzyme (ACEI's) inhibitors and/or angiotensin II (AT-II) receptor antagonists.[9]

Diabetic Neuropathy

Diabetic peripheral neuropathy is one of the most frequent complications of DM. 59% of people with T2DM have objective evidence of peripheral neuropathy. The pathophysiology of diabetic neuropathy (DN) rests intricate and not entirely illuminated. The concerns of DN comprise reduced quality of life, pain, foot deformity, neuropathic ulceration, and amputation.[9]

Diabetic neuropathy is documented by the American Diabetes Association (ADA) as "the occurrence of symptoms and/or signs of peripheral nerve dysfunction in people with diabetes mellitus once the omission of other reasons". As with other microvascular complications, menace of mounting diabetic neuropathy is relative to both the magnitude and duration of hyperglycaemia and some individuals may possess genetic attributes that disturb their predisposition to mounting such complications.[8]

Diabetes Mellitus can affect the somatic and autonomic nervous system. The somatic neuropathies associated with diabetes fall into 2 comprehensive categories:
Focal neuropathies include mononeuropathies such as carpal tunnel syndrome, palsy of the peroneal nerve, palsy of the 3rd cranial nerve and proximal nerve conditions (e.g. diabetic amyotrophy). Diabetic sensorimotor polyneuropathy is the utmost common widespread neuropathy and, for this reason, the simplified term ''*diabetic neuropathy''* is frequently used. It is a polyneuropathy because of the turgid injury to all peripheral nerve fibres, motor, sensory and autonomic. Such damage occurs insidiously and increasingly and is regarded as at first by sensory loss and later by loss of motor function, in a stocking and glove distribution.

Autonomic neuropathy can cause impaired light reflex, bladder paresis, postural hypotension, vomiting, and diarrhoea, impotence, sweating abnormalities, impotence and reversing ejaculation. Clinical assessment comprises history taking, especially of numbness, persistent pain or paraesthesia; and ankle reflexes physical inspection, trembling and light touch sensation (by conventional neurological examination or by graduated monofilaments).[10]

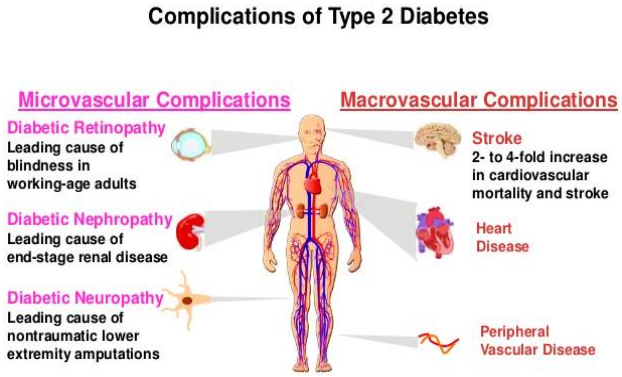

Fig.3: Complications of Type 2 Diabetes Mellitus

MACROVASCULAR COMPLICATIONS OF DIABETES

Although microvascular disease adds considerably to morbidity as well as to mortality in patients with diabetes, macrovascular complications are expected to be equally significant and determinants of life-span and quality of life. Macrovascular complications comprise coronary artery disease (CAD), cerebrovascular stroke and peripheral vascular disease (PVD). By the 1970s and 1980s, atherosclerotic disease had been recognized as one of the foremost complications of diabetes.[11]

Relation between Diabetes mellitus and Macro vascular disease

Diabetes increases the risk that an individual will develop cardiovascular disease (CVD). Although the exact mechanisms through which diabetes mellitus upsurges the probability of atherosclerotic plaque formation are not wholly demarcated, the association between the two is profound. CVD is the principal cause of death in people with type 2 diabetes.[12]

The central pathological mechanism in macro vascular disease is the progression of atherosclerosis, which leads to thinning of arterial walls all over the body. Atherosclerosis is thought to result from chronic inflammation and injury to the arterial wall in the peripheral or coronary vascular system. Diabetes escalates the risk that an individual will develop cardiovascular disease (CVD).[13] Oxidized LDL produces numerous atypical biological responses, such as drawing leukocytes to the intima of the vessel, improving the ability of the leukocytes to ingest lipids and segregate into foam cells and encouraging the propagation of leukocytes, smooth muscle cells, endothelial cells and all of which are stages in the development of atherosclerotic plaque.[14]

Diabetes is also a robust self-governing predictor of risk of cerebrovascular disease, as in coronary artery disease. Patients with type 2 diabetes have a much higher risk of stroke, with an increased risk of 150–400%.[8]

The primary goals of DM management are to decrease the peril for microvascular and macrovascular complications, to better symptoms, to lessen mortality and to progress quality of life. Near-normal glycaemia will reduce the risk for development of microvascular disease complications, but aggressive management of traditional cardiac risk factors (i.e., smoking cessation, management of dyslipidaemia, rigorous blood pressure control and antiplatelet therapy) are required to reduce the likelihood of development of macrovascular disease. Hyperglycaemia not only increases the risk for microvascular disease, but adds to underprivileged wound healing, compromises white blood cell function and leads to typical symptoms of DM. Reducing the potential for microvascular complications is targeted at faithfulness to therapeutic lifestyle intervention (i.e., diet and exercise programs) and drug-therapy regimens, as well as at maintaining blood pressure as near normal as possible.[8]

The present Prospective Observational study was carried out to establish the prevalence of Type-II Diabetes Mellitus amongst various age groups and the various complications of diabetes mellitus in Mandya Institute of Medical Sciences and Teaching Hospital, Mandya, Karnataka, India. The results help in drawing out various strategic initiatives to decrease the incidence of diabetes mellitus and its complications, to improve quality of life of diabetes mellitus patients and to decrease the disease burden.

OBJECTIVE

- To assess the prevalence and complications of type-II diabetes mellitus.

MATERIALS AND METHODOLOGY

Ethical Clearance

The Ethical clearance for the study was obtained from the Institutional Ethics committee, Mandya Institute of Medical Sciences Teaching Hospital and Research Centre, Mandya.

Study Design

A Prospective observational study was carried out in various units Intensive Critical Care Unit (ICCU), Respiratory Intensive Care Unit (RICU), Medical Intensive Care Unit (MICU), Medical ward (Male and Female) of the department of general medicine, Mandya Institute of Medical Sciences and Teaching hospital, Mandya, Karnataka, India.

The essential data for Prospective observational study was collected using a well-designed patient data collection form. A total of 121 Type-II Diabetes Mellitus patient's files were screened and data was analysed. Both male and female patients with cardiovascular disease of age > 20 years admitted to hospital were selected.

INCLUSION AND EXCLUSION CRITERIA

Inclusion Criteria

- All adult patients of Type-II DM of age between 20-80 years.
- Individuals giving consent for study.

Exclusion Criteria

- Individuals who are not willing to be a part of the study.
- Pregnant women and lactating mothers.
- Paediatrics.
- Seriously and mentally ill patients.

STUDY PROCEDURE

Eligible patients were enrolled based on inclusion and exclusion criteria. The data collection form which was made by department of clinical pharmacy was used for collecting the details. Patient files were screened for the demographic information such as name, age, sex, date of admission, department and unit in which he/she was admitted and diagnosis. It also includes the present complaints, past medical history, past medication history, family history, social history (including diet, alcohol/smoking habits, sleep, bowel and bladder, appetite, exercise habit), physical examination and systemic examination were recorded in a suitably designed patient data collection form.

STATISTICAL ANALYSIS

Collected information was analysed using Microsoft Office (MS-Word and Excel) 2010. Descriptive data analysis has been performed in the form of percentage of demographic variables. For the analysis of the results, simple percentage calculations were used to arrive at a conclusion of our study.

RESULTS

A total number of 121 case sheets of 121 case sheets of diabetic patients admitted to MIMS teaching hospital were analyzed. Among these 121 diabetic patients, 68 patients were males (56.20%) and 53 were females (43.80%) (Figure 4).

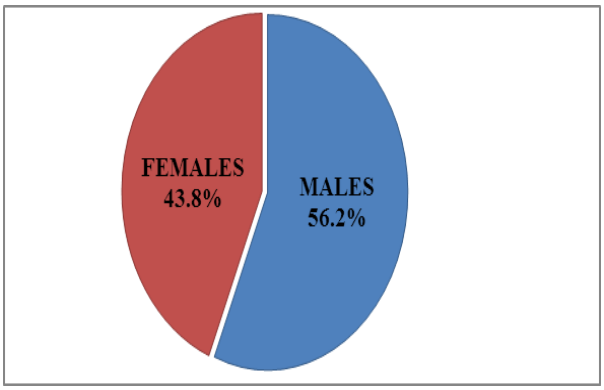

Fig.4: Percentage of males and females diabetic patients

All these patients are divided into 7 groups based on their age which are described below (Table 1, Figure 5):

1. 20 - 30 years of age.
2. 31- 40 years of age.
3. 41-50 years of age.
4. 51-60 years of age.
5. 61-70 years of age.
6. 71-80 years of age.
7. Above 80 years of age.

Out of 121 patients, 1 male patient (0.83%) in 20-30 years of age, 10 patients (8.26%) in 31- 40 years of age which includes 7 males and 3 females, 21 patients (17.35%) in 41-50 years of age which includes 9 males and 12 females, 28 patients (23.14%) in 51-60 years of age which includes 18 males and 10 females, 39 patients (32.23%) in 61-70 years of age which includes 22 males and 17 females, 17 patients (14.05%) in 71-80 years of age which includes 7 males and 10 females, 5 patients (4.14%) above 80 years of age which includes 4 males and 1 female.

Table 1: Age group distribution of Type-II Diabetes Mellitus patients

AGE GROUP (YEARS)	MALE	FEMALE	PERCENTAGE (%)
20-30	01	00	0.83%
31-40	07	03	8.26%
41-50	09	12	17.35%
51-60	18	10	23.14%
61-70	22	17	32.23%
71-80	07	10	14.05%
ABOVE 80	04	01	4.14%
TOTAL	**68**	**53**	**100%**

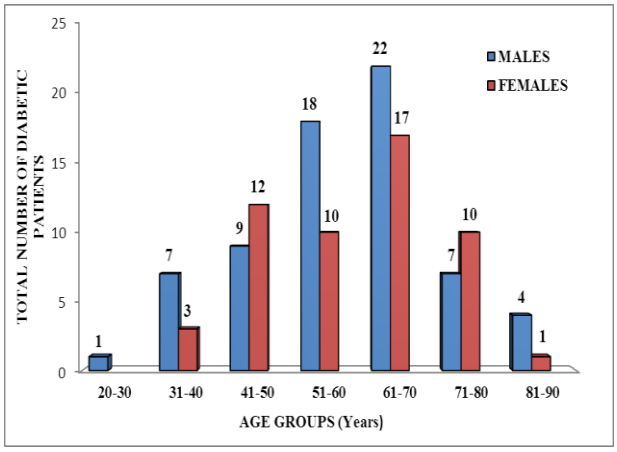

Fig.5: Age group distribution Age group distribution of Type-II Diabetes Mellitus patients

In 121 cases 116 patients (95.87%) are a known case of diabetes consists of 65 males (56%) and 51 females (44%). These includes 1 male patient within 20-30 years of age, 7 males and 3 females within 31-40 years of age, 9 males and 11 females within 41-50 years of age, 15 males and 10 females within 51-60 years of age, 22 males and 17 females within 61-70 years of age, 7 males and 9 females within 71-80 years of age, 4 males and 1 female patient above 80 years of age. Only 5 patients (4.13%) are newly detected cases of D.M. These includes 1 female patient within 41-50 years of age, 3 male patient within 51-60 years of age and 1 female patient within 71-80 years of age (Table 2, Figure 6).

Table 2: Unknown and Known cases of Diabetes Mellitus

PERCENTAGE OF DIABETIC PATIENTS		
	KNOWN CASE OF DIABETIC PATIENTS	NEWLY DETECTED DIABETIC PATIENTS
MALES	56%	44%
FEMALES	60%	40%

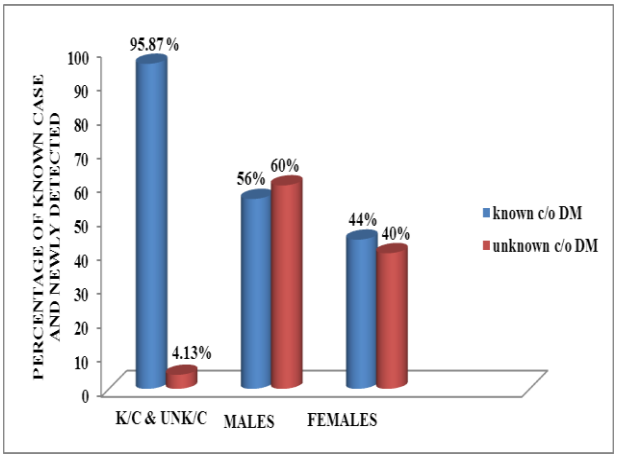

Fig.6: Unknown and Known cases of Diabetes Mellitus

In the total study population of 121 diabetic patients, 85 patients (70.25%) are found with complications associated with diabetes mellitus among which 46 are males (54.11%) and 39 are females (45.89%), the rest 35 patients (29.75%) are found without any complications among which 22 are males (61.11%) and 14 are females (38.89%) (Table 3, Figure 7).

Table 3: Complications of Diabetic Patients.

COMPLICATIONS OF TYPE-II DIABETES MELLITUS		
	% OF MALES	% OF FEMALES
WITH COMPLICATIONS (70.25%)	54.11%	45%
WITHOUT COMPLICATIONS (29.7%)	61.11%	38.89%

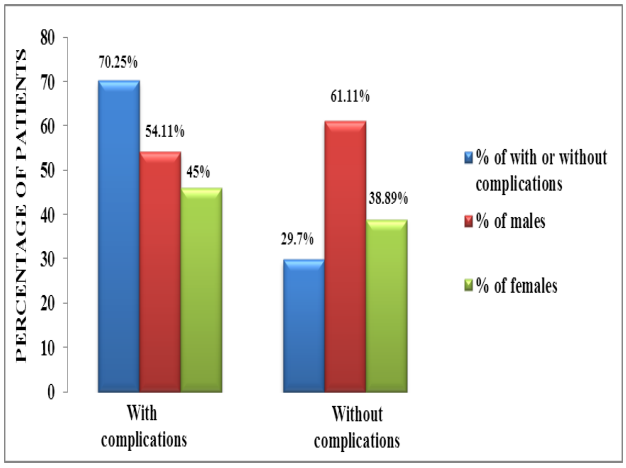

Fig.7: Complications in Diabetes Mellitus Patients.

These complications are categorized as follows:

- **Micro vascular Complications**
- **Macro vascular Complications**

In this study, 19 Patients (22.35%) are with micro vascular complications which consists of 13 males (68.42%) and 6 females (31.58%), 50 patients (58.83%) are with macro vascular complications which consists of 27 males (54%) and 23 females (46%) and 16 patients (18.82%) are found to have both micro vascular and macro vascular complications which consists of 8 males (50%) and 8 females (50%) (Table 4, Figure 8).

Table 4: Percentage of Complications in Diabetes Mellitus Patients

PERCENTAGE OF COMPLICATIONS IN DIABETIC PATIENTS	
MICROVASCULAR COMPLICATIONS	22%
MACROVASCULAR COMPLICATIONS	59%
MICRO + MACROVASCULAR COMPLICATIONS	19%

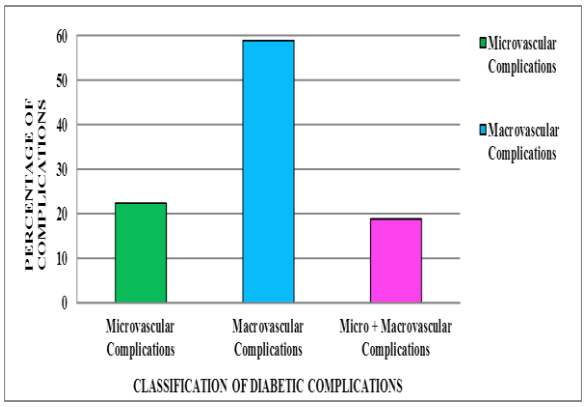

Fig.8: Percentage of Complications in Diabetes Mellitus Patients.

Among 19 patients (22.35%) with microvascular complications, 4 patients (21.05%) are found to have diabetic neuropathy in which 3 are males (75%) and 1 is female (25%), 3 patients (15.78%) are found to have diabetic nephropathy in which 2 are males (66.66%) and 1 is female (33.34%), 1 patient (5.27%) is found to have diabetic retinopathy who is a male patient (100%), 4 patients (21.05%) are found to have diabetic ketoacidosis in which 2 are males (50%) and 2 are females (50%), 5 patients (26.31%) are found to have diabetic foot in which 3 are males (60%) and 2 are females (40%), 1 patient (5.27%) is found to have neuropathy with diabetic ketoacidosis includes a male patient (100%), 1 patient (5.27%) is found to have nephropathy with diabetic foot who is a male patient (100%) (Table 5, Figure 9 and Figure 10).

The micro vascular complications found in this study includes:

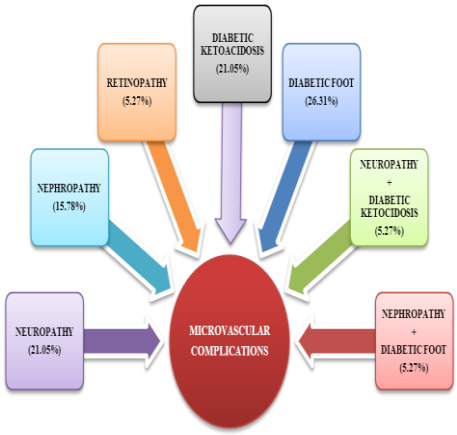

Fig.9: Microvascular Complications.

Table 5: Percentage of Microvascular Complications in Diabetic Patients.

MICROVASCULAR COMPLICATIONS	
COMPLICATION	**% OF PATIENTS**
DIABETIC FOOT	26.31
NEUROPATHY	21.05
NEPHROPATHY	15.78
DIABETIC KETOACIDOSIS	21.05
RETINOPATHY	5.27
NEUROPATHY + DIABETIC FOOT	5.27
NEUROPATHY + DIABTIC KETOACIDOSIS	5.27

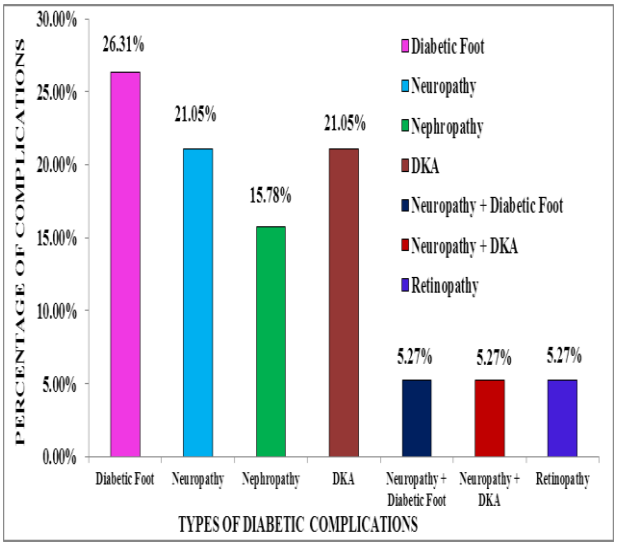

Fig.10: Percentage of Microvascular Complications in Diabetic Patients.

The macro vascular complications found in this study includes

Among 50 patients (58.83%) with macrovascular complications, 27 patients (54%) are found to have hypertension in which 13 are males (48.14%) and 14 are females (51.86%), 4 patients (08%) are found to have Ischemic heart disease (IHD) in which 2 are males (50%) and 2 are females (50%), 1 patient (2%) is found to have myocardial infarction (MI) who is a male patient (100%), 1 patient (2%) is found to have left ventricular (LV) dysfunction who is a male patient (100%), 7 patients (14%) are found to have cerebrovascular accident with hypertension which includes 3 male patients (42.85%) and 4 female patients (57.15%), 4 patients (8%) are found to have angina pectoris with hypertension which includes 2 male patients (50%) and 2 female patients (50%), 2 patients (4%) are found to have IHD with LV dysfunction which includes 2 male patients (100%), 2 patients with MI with HTN in which 1 is a male patient (50%) and the other is a female patient (50%), 1 patient (2%) is found to have LV dysfunction with HTN who is a male patient (100%), 1 patient (2%) is found to have IHD with HTN with Angina pectoris who is a male patient (100%) (Figure 11, Table 5 and Figure 12).

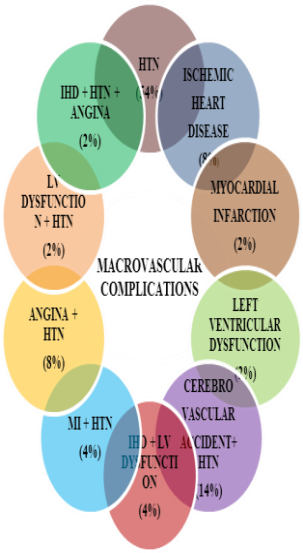

Fig.11: Macrovascular Complications

Table 5: Percentage of Macrovascular Complications in Diabetes Mellitus Patients.

MACROVASCULAR COMPLICATIONS	
COMPLICATION	**% OF PATIENTS**
HYPERTENSION	54%
ISCHEMIC HEART DISEASE	8%
MYOCARDIAL INFARCTION	2%
LEFT VENTRICULAR DYSFUNCTION	2%
CVA + HTN	14%
IHD + LV DYSFUNCTION	4%
ANGINA + HTN	8%
MI + HTN	4%
LV DYSFUNCTION + HTN	2%
IHD + HTN+ ANGINA	2%

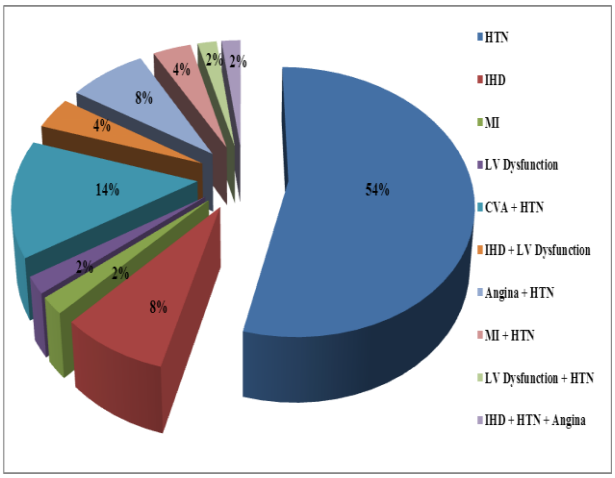

Fig.12: Percentage of Macrovascular Complications in Diabetes Mellitus Patients.

DISCUSSION

In recent years there has been increased emphasis on education of patients and their families in medical conditions. This has been driven in part by patients wishing to be better informed about their condition and in part by the recognition by health care professionals that self-management is important to patients. Most number of studies has shown that many hospital admissions and readmissions are due to co-morbid conditions. Study result shows that male patients admitted were more compared to female and may infer that male are more prone to Type-II Diabetes Mellitus compared to female gender. Average age of patients found was between 61-70 years, which indicates that the cardiac conditions found may be chronic in this age group. This increased prevalence is mainly due to the sedentary lifestyle and co-morbidities. Diabetes is associated with serious complications and further uncontrolled diabetes may lead to premature death, but people with diabetes can take steps to control the disease and lower the risk of complications by compliance to diet, regular exercise and medicines.[15]

In the analysis of complications, 70.25% of patients are found with complications, 29.37% patients are found without complications and 18.82% patients are found with both microvascular and macrovascular complications. The prevalence rate of both micro vascular and macro vascular complications are analysed in patients with complications of diabetes. Out of 121 patients, 72.94% of patients are found with macro vascular complications and 35.29% patients with micro vascular complications. 26.31% of patients are with diabetic foot which is found to be the most common microvascular complication in this study, 21.05% patients each with diabetic neuropathy and diabetic ketoacidosis, 15.78% patients are with diabetic nephropathy, 5.27% patients each with diabetic retinopathy, diabetic nephropathy with diabetic foot, diabetic neuropathy with diabetic ketoacidosis.

The most common macro vascular complication in this study is found to be hypertension i.e., 54%, 14% patients are found to have CVA with HTN, 8% patients each are found to have IHD and Angina with HTN, 4% patients each are found to have IHD with LV dysfunction and MI with HTN, 2% patients each are found to have LV dysfunction, MI, IHD with HTN with Angina and LV dysfunction with HTN.

Among all admitted patients in ICCU, RICU and MICU for cardiac problems, patients were suffering mostly

from co-morbid conditions and commonly found co-morbid condition was Hypertension and diabetes mellitus, which supports the study that Hypertensive and Diabetic patients are more prone to high risk of micro vascular and macro vascular complications.

CONCLUSION

This research highlights the necessity for implementing programs for early detection, screening, and awareness to mitigate the burden of managing the complications. Good blood glucose control recovers microvascular disease and must be executed initially and conserved for the ideal interval of time. Appropriate controls of blood pressure as well as dyslipidaemia are extremely important in macrovascular disease prevention besides glycaemic control. Patients with microvascular complications appear particularly prone to accelerated atherosclerosis and premature death. Neovascularization ascending from the vasa vasorum may connect macro- and microangiopathy.

A clear picture on differing responses to therapeutic interventions could lead to better management and improve T2DM outcomes not only concerning microvascular but also macrovascular complications as well. Further systematic research on the above interlinking hypothesis will help us get more clarity whether microvascular complications precede macrovascular complications or they are two ends of the same spectrum of disease existing in continuum. Uncontrolled Diabetes Mellitus, Hypertension, Smoking tobacco and Alcohol consumption are the foremost etiologies and risk factors for mounting diabetes related micro vascular and macro vascular complications. Given that the prevalence of diabetes is high at the population level, it imposes a financial burden on both our healthcare system and the individuals living with the disease. An attempt continues to be discussed; yet as the number of undiagnosed patients continues to grow, the prevalence and impact of the disease on patient quality of life and the overall cost of diabetes to healthcare is also important.

There are different approaches to diagnose diabetes among individuals, The 1997 American Diabetes Association recommendations for diagnosis of DM focus on fasting Plasma Glucose (FPG), while WHO focuses on Oral Glucose Tolerance Test (OGTT).[16] This is importance for regular follow-up of diabetic patients with the health care provider is of great significance in averting any long term complications. The impact of diabetes is reaching a crude state, it is essential to each country for implementation of preventive and curative measures. This may include restaurants to provide the caloric content of items on their menus; reduce the availability of high calories, high-fat foods in school cafeterias; Lifestyle modification will undoubtedly play a key role in the eventual resolution to the problem of diabetes, and more definitive solutions will depend on the ability of basic science to point prevention and treatment in new directions.

CONFLICT OF INTEREST

There is no conflict of interest in the study.

Financial support and sponsorship

Nil.

ACKNOWLEDGEMENT

All the authors would like to thank Bharathi College of Pharmacy, Bharathinagara, Mandya, Karnataka, India for continuous support and encouragement throughout this work. We are very grateful to our HOD, Department of Pharmacy Practice and guide for unreserved guidance, enormous support, mentoring that we have received and constructive suggestions and comments from the stage of proposal development to its completion. Finally our deepest gratitude goes to the doctors and staff workers of MIMS Teaching Hospital, Mandya, Karnataka, India who helped and allowed us in collecting and gathering data from the hospital.

REFERENCES

1. Guidelines for management of type-2 diabetes. Indian council of medical research. 2005; section-1: 1-6.
2. Josephine M Forbes and Mark E Cooper. Mechanisms Of Diabetic Complications. The American Physiological Society. 2013; 93: 137–188.
3. Orasanu G, Plutzky J. The pathologic continuum of diabetic vascular disease. J Am Coll Cardiol, 2009; 53-5 Suppl: S35-42.
4. Kalofoutis C, Piperi C, Kalofoutis A, Harris F, Phoenix D, Singh J. Type II diabetes mellitus and cardiovascular risk factors: Current therapeutic approaches. Exp Clin Cardiol, 2007; 12: 17-28.
5. Singh VP, Bali A, Singh N, Jaggi AS. Advanced glycation end products and diabetic complications. Korean J Physiol Pharmacol, 2014; 18: 1-14.
6. Schmidt AM, Hori O, Brett J, Yan SD, Wautier JL, Stern D. Cellular receptors for advanced glycation end products. Implications for induction of oxidant stress and cellular dysfunction in the pathogenesis of vascular lesions. Arterioscler Thromb, 1994; 14: 1521-8.
7. Goldin A, Beckman JA, Schmidt AM, Creager MA. Advanced glycation end products: Sparking the development of diabetic vascular injury. Circulation, 2006; 114: 597-605.
8. Michael J. Fowler, MD. Microvascular and Macrovascular Complications of Diabetes. Clinical Diabetes Journals. 2008; 26(2): 77-82.
9. Grace C Davey, Swapnil B Patil, Aonghus O Loughlin and Timothy O Brien. Mesenchymal stem cell-based treatment for microvascular and secondary complications of Diabetes mellitus. Frontiers In Endocrinology. 2014; 5(86): 1-16.
10. Donaghue KC, Chiarelli F, Trotta D, Allgrove J, Dahl-Jorgensen K. Microvascular and

macrovascular complications associated with diabetes in children and adolescents. Pediatric Diabetes. 2009; 10(12): 195–203.
11. Daniel J Moore, Justin M Gregory, Yaa A Kumah Crystal, Jill H Simmons. Mitigating micro- and macro-vascular complications of diabetes beginning in adolescence. Vascular Health and Risk Management. 2009; 5: 1015–1031.
12. Kohei KAKU. Pathophysiology of Type 2 Diabetes and Its Treatment Policy. Japan Medical Association-Journal. 2010; 53(1): 41-46.
13. Grace C Davey, Swapnil B Patil, Aonghus O Loughlin and Timothy O Brien. Mesenchymal stem cell-based treatment for micro vascular and secondary complications of Diabetes mellitus. Frontiers in Endocrinology. 2014; 5(86): 1- 16.
14. Michael J. Fowler, MD. Micro vascular and Macro vascular Complications of Diabetes. Clinical Diabetes Journals. 2008; 26(2): 77-82.
15. Mohammad Yaseen Abbasi, Md. Avez Ali1, Mohammed Alshammari. A Prospective Study On Prescribing Pattern Of Anti-Diabetic Drugs. World Journal of Pharmacy And Pharmaceutical Sciences. 2014; 3(5): 45-57.
16. Cox EM, Elelman D (2009) Test for screening and diagnosis of type 2 diabetes. Clin Diabetes, 4: 132-138.