ABSTRACT
Diabetes is the most common cause of chronic kidney disease (CKD) and end-stage renal disease (ESRD) in most parts of the world. 20 to 30% of diabetic patient have diabetic nephropathy in type 1 and type 2. Hyperglycemia is the key of nephropathy creation. Hyperglycemia also by production of toxic materials, advanced glycosylated end product (AGE), increased activity of aldose reductase has some role. Some metabolites of arachidonic acid, hemodynamic derangements and genetic factors have also some role. Although diabetic nephropathy is most common cause of nephropathy in these patients, but diabetic patients are also prone to other urinary tract and renal parenchymal disease and should not be confused with renal failure due to diabetic nephropathy. The principle of treatment of diabetic nephropathy is based on tight control of hyperglycemia, tight control of blood pressure and glomerular pressure, control of dyslipidemia, restriction of protein intake and smoking withdrawal.

KEYWORDS: Hyperglycemia, diabetes mellitus, diabetic nephropathy, Dyslipidemia, aldose reductase, prevalence, complications.

INTRODUCTION
Diabetes mellitus is a metabolic disorder that is characterized by elevated glucose levels.\(^1\) The Continuously elevated glucose levels may result in microvascular (involving the eyes, kidneys and nerves) and macrovascular (involving the heart, blood vessels) complications accelerating the risk of strokes and claudication (pain in extremities due to lack of blood supply)\(^2\) due to which there is a major increase in morbidity and mortality of diabetes and it is estimated to rise from 171 million to 366 million in 2030 worldwide.\(^3\)
Kidneys are one of the important organs that are involved in diabetes. Diabetes is the most common cause of endstage renal disease (ESRD) in most parts of the world. Involvement of Kidneys directly and indirectly, increase the involvement of other organs and increase morbidity and mortality in diabetic patients.[1]

Since with untreated diabetic nephropathy, a significant decrease in life expectancy of patients happens; therefore, prevention of this debilitating condition and if there is, early diagnosis and treatment is important.[2]

Due to the increasing industrialization of societies, increased immobility and changes in diet and lifestyle and increasing prevalence of obesity, insulin resistance and type 2 diabetes prevalence is increasing.[1]

Because many of the classic symptoms of diabetes in type 2 diabetes, unlike type 1 Diabetes, do not occur, the importance of screening methods for identifying these patients and screening them for kidney involvement is very important.[3]

One of the most frequently caused complication in diabetes case is chronic kidney failure in both developed and developing countries. Diabetic nephropathy, also known as Kimmelstiel-Wilson syndrome or nodular diabetic glomerulosclerosis or intercapillary glomerulonephritis, is a clinical syndrome characterized by albuminuria (>300 mg/day or >200 mcg/min) confirmed on at least two occasions 3-6 months apart, permanent and irreversible decrease in glomerular filtration rate (GFR), and arterial hypertension. The syndrome was first described by a British physician Clifford Wilson (1906-1997) and American physician Paul Kimmelstiel (1900-1970) in 1936.[34]

DIABETES

The word "diabetes" is derived from the Greek word meaning "a siphon". A Greek physician, Aretus the Cappadocian, of 2nd-century A.D. named the condition "diabetes." He explained that patients with it had polyuria and "passed water like a siphon." Diabetes mellitus and diabetes insipidus share the name "diabetes" because they are both conditions characterized by excessive urination (polyuria).[18]

DIABETES MELLITUS

Diabetes mellitus is a condition of hyperglycemia in blood. The glucose levels are tightly controlled by insulin, a hormone produced by the pancreas which lowers the elevated blood
glucose level. In patients with diabetes, the absence or insufficient production of insulin causes hyperglycemia which lead to spillage of glucose into the urine, hence the term sweet urine.\textsuperscript{[19]}

**TYPES**

There are of different types like Type 1, Type 2, Secondary diabetes and Gestational diabetes.

**TYPE 1 DIABETES**

Type 1 Diabetes was also called Insulin Dependent Diabetes Mellitus (IDDM) or juvenile onset diabetes mellitus is an autoimmune disease where antibodies are produced against the beta cells of the pancreas by the misdirected immune system or certain viral infections (mumps and Coxsackie viruses) or other environmental toxins. Therefore it can be said that the tendency to develop abnormal antibodies in type 1 diabetes in part is genetically inherited.

Type 1 diabetes occurs in young, lean individuals, usually before 30 years of age. This subgroup is referred to as Latent Autoimmune Diabetes. In Adults (LADA), this is a slow progressive form of type 1 diabetes.\textsuperscript{[19]}

**TYPE 2 DIABETES**

It is also referred to as Non-Insulin Dependent Diabetes Mellitus (NIDDM), or Adult Onset Diabetes Mellitus (AODM). These patients can produce insulin, which is inadequate for their body's needs since they lack the sensitivity for insulin by the cells of the body (particularly fat and muscle cells).\textsuperscript{[19]}

This form of diabetes usually begins with insulin resistance, a condition where the body needs more insulin to help glucose enter the cells to generate energy. At first, the pancreas keeps up with the added demand by producing more insulin, but in time the pancreas loses its ability to secrete enough insulin in response to different stimuli. People can develop type 2 diabetes at any age, even during childhood. Now a days type 2 diabetes is more common in childhood than type 1 diabetes majorly due to sedenteric life style.

**SECONDARY DIABETES**

This is generally caused due to other hormonal disorders such as acromegaly and usage of drugs like glucocorticoids, it may often respond to oral antidiabetic agents.\textsuperscript{[20]}
GESTATIONAL DIABETES

Elevation of blood sugar levels during pregnancy is called gestational diabetes. Gestational diabetes usually resolves once the baby is born. However, 25 to 50% of women with gestational diabetes will eventually develop Type 2 diabetes later in life, especially in those who require insulin during pregnancy and those who remain overweight after their delivery.\cite{19}

DIABETES INSIPIDUS

Diabetes Insipidus (DI) is a rare disorder that can occur as a consequence of histiocytosis involving the pituitary gland. It should not be confused with the more common diabetes mellitus, also known as sugar diabetes, which results from too much sugar in the blood. Although both disorders have similar symptoms, in every other way including the cause and treatment, they are completely unrelated diseases.

PRE-DIABETES

The blood glucose levels are higher than normal but not high enough to diagnose diabetes. These people are prone to develop Type 2 diabetes within 10 years of Pre-diabetic condition. Pre-diabetic patients have an increased risk of heart disease and stroke. With weight loss and physical activity, people with pre-diabetes can prevent type 2 diabetes.\cite{21}

ETIOTOLOGY

- Insufficient production of insulin (either absolutely or relative to the body's needs) or Production of defective insulin (which is uncommon) or the inability of the cells to use insulin diabetes.\cite{19}
- Studies have shown that the appearance of islet cell antibodies often precedes the onset of clinical diabetes by as much as 3 years, especially Type 1.\cite{22}
- Other causes include
  - Genetic predisposition
  - Poor Diet (Malnutrition Related Diabetes)
  - Stress
  - Sedentary Lifestyle
  - Obesity and Fat Distribution
  - Drug Induced
  - Hypertension
MOST PREVALENT COMPLICATION IN DIABETES

The most frequent cause of chronic kidney failure in both developed and developing countries. Diabetic nephropathy, also known as Kimmelstiel-Wilson syndrome or nodular diabetic glomerulosclerosis or intercapillary glomerulonephritis, is a clinical syndrome characterized by albuminuria (>300 mg/day or >200 mcg/min) confirmed on at least two occasions 3-6 months apart, permanent and irreversible decrease in glomerular filtration rate (GFR), and arterial hypertension. The syndrome was first described by a British physician Clifford Wilson (1906-1997) and American physician Paul Kimmelstiel (1900-1970) in 1936. [34]

Diabetic nephropathy is a chronic complication of both type 1 DM (beta cell destruction absolute lack of insulin) and type 2 DM (insulin resistance and/or decreased secretion of insulin).

STAGES OF DIABETIC NEPHROPATHY

There are five stages in the development of diabetic nephropathy.

Stage I
Hypertrophic hyper filtration. In this stage, GFR is either normal or increased. Stage I lasts approximately five years from the onset of the disease. The size of the kidneys is increased by approximately 20% and renal plasma flow is increased by 10%-15%, while albuminuria and blood pressure remain within the normal range.

Stage II
The quiet stage. This stage starts approximately two years after the onset of the disease and is characterized by kidney damage with basement membrane thickening and mesangial proliferation. There are still no clinical signs of the disease. GFR returns to normal values. Many patients remain in this stage until the end of their life.

Stage III
The microalbuminuria stage (albumin 30-300 mg/dU) or initial nephropathy. This is the first clinically detectable sign of glomerular damage. It usually occurs five to ten years after the
onset of the disease. Blood pressure may be increased or normal. Approximately 40% of patients reach this stage.

**Stage IV**
Chronic kidney failure (CKF) is the irreversible stage. Proteinuria develops (albumin > 300 mg/dL), GFR decreases below 60 mL/min/1.73 m², and blood pressure increases above normal values.

**Stage V**
Terminal kidney failure (TKF) (GFR < 15 mL/min/1.73 m²). Approximately 50% of the patients with TKF require kidney replacement therapy (peritoneal dialysis, hemodialysis, kidney transplantation)

In the initial stages of diabetic nephropathy, increased kidney size and changed Doppler indicators may be the early morphological signs of renal damage, while proteinuria and GFR are the best indicators of the degree of the damage.

In addition to diabetic nephropathy, glomerular sclerosis can also develop in other pathological conditions in patients with DM. These are:

a. dysproteinemia (amyloidosis and other deposit diseases)
b. conditions with chronic ischemia (cyanotic congenital heart disease)
c. chronic membranoproliferative glomerulonephritis
d. Idiopathic diseases mostly associated with smoking and increased blood pressure.

**PATHOGENESIS**
Pathogenesis of diabetic nephropathy is very complicated and results from the interaction of hemodynamic and metabolic factors.

**Glomerular Hyper Filtration**
Increased intraglomerular pressure and hyper filtration as early changes in the development of diabetic nephropathy were described by Stadler and Schmidt in 1959.[26] In the 1970's, Mogensen emphasized that as many as 40% newly found DM cases had increased glomerular filtration.
5. MATERIALS AND METHODOLOGY

5.1 Study Site
Department of Endocrinology, BBR hospitals, balanagar, Hyderabad, Telangana styate

Study Design
Prospective and Retrospective observational, Non-interventional study

Study Period: 6 Months.

5.2. PLAN OF WORK
• Literature Review was carried out
• Ethical committee approval
• Collection of data
• Compilation of various patient population with different types of complications in diabetes undergoing treatment, the dosage regimen used, and complications their management were studied.
• Analysis of data
• Report the data.

5.3. PATIENT CASE DETAILS
Total number of cases collected: 150
• INCLUSIVE
  ➢ Patients in the endocrinology department with diabetes and complications
  ➢ Patients who are willing to give Verbal informed consent for the study.

• EXCLUSIVE
  ➢ Patients in intensive care units, critical care units and other non selected departments
  ➢ Patients with previous history of any disorder or toxicity taking any other drug besides diabetes or its complications are not taken into consideration

5.4. PARAMETERS CONSIDERED FOR ANALYSIS OF DATA COLLECTION
✓ Age wise distribution of patients
✓ Sex wise distribution of patients
✓ Socio economic status wise distribution of patients
✓ Patients by body mass index
✓ Patients by type of diabetes
✓ Patients by blood glucose levels
✓ Diabetic patients by presence of micro & macro vascular complications
✓ Different category of drugs prescribed
✓ Patients by type of antibiotics prescribed
✓ Patient by serum creatinine levels
✓ Patients by changes in health related quality of life.

ANALYSIS OF DATA
The data collected from the patients is analysed statistically based upon the parameters presented.

RESULTS
Demographic analysis of data was revealed as follows.

I. AGE WISE DISTRIBUTION OF PATIENTS

Table 1: Age Wise Distribution of Cases

<table>
<thead>
<tr>
<th>S.No</th>
<th>Age</th>
<th>No of patients n=150</th>
<th>% of distribution</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>&lt;20</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>2</td>
<td>21-30</td>
<td>18</td>
<td>12</td>
</tr>
<tr>
<td>3</td>
<td>31-40</td>
<td>34</td>
<td>22.66</td>
</tr>
<tr>
<td>4</td>
<td>41-49</td>
<td>51</td>
<td>34</td>
</tr>
<tr>
<td>5</td>
<td>&gt;50</td>
<td>44</td>
<td>29.33</td>
</tr>
</tbody>
</table>

Fig 1: Graph On Age Wise Distribution Of Cases
II. GENDER WISE DISTRIBUTION OF PATIENTS

Table 2: gender Wise Distribution of Cases

<table>
<thead>
<tr>
<th>S.No</th>
<th>Age</th>
<th>Males</th>
<th>Females</th>
<th>% of males</th>
<th>% of females</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>&lt;20</td>
<td>1</td>
<td>2</td>
<td>0.67</td>
<td>1.30</td>
</tr>
<tr>
<td>2</td>
<td>21-30</td>
<td>13</td>
<td>5</td>
<td>8.00</td>
<td>3.00</td>
</tr>
<tr>
<td>3</td>
<td>31-40</td>
<td>23</td>
<td>11</td>
<td>15.33</td>
<td>7.00</td>
</tr>
<tr>
<td>4</td>
<td>41-49</td>
<td>32</td>
<td>19</td>
<td>21.33</td>
<td>12.66</td>
</tr>
<tr>
<td>5</td>
<td>≥50</td>
<td>31</td>
<td>13</td>
<td>20.66</td>
<td>8.66</td>
</tr>
</tbody>
</table>

Fig 2: Graph on gender Wise Distribution of Cases

III. SOCIO ECONOMIC STATUS WISE DISTRIBUTION OF PATIENTS

Table 3: Socio Economic Status Wise Distribution of Cases

<table>
<thead>
<tr>
<th>Sl. No</th>
<th>Status</th>
<th>No of patients n=150</th>
<th>% of distribution</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Govt. employee</td>
<td>28</td>
<td>18.66</td>
</tr>
<tr>
<td>2</td>
<td>Private employee</td>
<td>32</td>
<td>21.33</td>
</tr>
<tr>
<td>3</td>
<td>Business man</td>
<td>46</td>
<td>30.66</td>
</tr>
<tr>
<td>4</td>
<td>House Wife</td>
<td>26</td>
<td>17.33</td>
</tr>
<tr>
<td>5</td>
<td>Labour</td>
<td>18</td>
<td>12.00</td>
</tr>
</tbody>
</table>

Fig 3: Graph on Socio Economic Status Wise Distribution of Cases
IV. DISTRIBUTION OF PATIENTS BY BODY MASS INDEX

Table 4: Distribution of Cases Based On BMI

<table>
<thead>
<tr>
<th>S.No</th>
<th>Body mass index</th>
<th>Condition</th>
<th>No of patients n=150</th>
<th>% of distribution</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>&lt;18.5</td>
<td>Under weight</td>
<td>6</td>
<td>4.00</td>
</tr>
<tr>
<td>2</td>
<td>18.5-24.9</td>
<td>Normal weight</td>
<td>12</td>
<td>8.00</td>
</tr>
<tr>
<td>3</td>
<td>25.0-29.9</td>
<td>Over weight</td>
<td>42</td>
<td>28.00</td>
</tr>
<tr>
<td>4</td>
<td>30.0-34.9</td>
<td>Class I obesity</td>
<td>54</td>
<td>36.00</td>
</tr>
<tr>
<td>5</td>
<td>35.0-39.9</td>
<td>Class II obesity</td>
<td>12</td>
<td>8.00</td>
</tr>
<tr>
<td>6</td>
<td>&gt;40.0</td>
<td>Class III obesity</td>
<td>24</td>
<td>16.00</td>
</tr>
</tbody>
</table>

Fig 4: Distribution of Cases Based on BMI

V. DISTRIBUTION OF PATIENTS BY TYPE OF DIABETES

Table 5: Cases Based On Type of Diabetes

<table>
<thead>
<tr>
<th>Diabetes type</th>
<th>No. of patients n=150</th>
<th>% of distribution</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1 (IDDM)</td>
<td>3</td>
<td>2.00</td>
</tr>
<tr>
<td>Type 2 (NIDDM)</td>
<td>147</td>
<td>98.00</td>
</tr>
</tbody>
</table>

Fig 5: Chart of Cases Based on Type of Diabetes
VI. DISTRIBUTION OF PATIENTS BY BODY GLUCOSE LEVELS

Table No 6: Distribution of Cases Based on Body Glucose Levels of Patients

<table>
<thead>
<tr>
<th>Body glucose levels(Random) in mg/dl</th>
<th>No of patients</th>
<th>% of distribution</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;200</td>
<td>18</td>
<td>12</td>
</tr>
<tr>
<td>200-299</td>
<td>24</td>
<td>16</td>
</tr>
<tr>
<td>300-399</td>
<td>42</td>
<td>28</td>
</tr>
<tr>
<td>400-499</td>
<td>24</td>
<td>16</td>
</tr>
<tr>
<td>&gt;500</td>
<td>42</td>
<td>8</td>
</tr>
</tbody>
</table>

![Fig 6: graph on distribution of cases based on body glucose levels of patients]

VII. DISTRIBUTION OF DIABETIC PATIENTS BY PRESENCE OF OTHER COMPLICATIONS

Table 7: Distribution Of Diabetic Patients By Presence Of Other Complications

<table>
<thead>
<tr>
<th>S.No</th>
<th>Complications</th>
<th>No. of patients n=150</th>
<th>% of distribution</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Hypertension(HT)</td>
<td>25</td>
<td>16.66</td>
</tr>
<tr>
<td>2</td>
<td>Obesity</td>
<td>31</td>
<td>20.66</td>
</tr>
<tr>
<td>3</td>
<td>Cancer</td>
<td>8</td>
<td>5</td>
</tr>
<tr>
<td>4</td>
<td>Angina</td>
<td>14</td>
<td>9.33</td>
</tr>
<tr>
<td>5</td>
<td>Asthma</td>
<td>11</td>
<td>7.33</td>
</tr>
<tr>
<td>6</td>
<td>Arthritis</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>7</td>
<td>Others</td>
<td>5</td>
<td>3</td>
</tr>
</tbody>
</table>
**VIII. DISTRIBUTION OF DIFFERENT CATEGORY OF DRUGS IN PRESCRIPTION**

**Table 8: Distribution of Different Category of Drugs In Prescription**

<table>
<thead>
<tr>
<th>S.No</th>
<th>Drugs category</th>
<th>No. of drugs prescribed n=150</th>
<th>% of distribution</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Anti diabetic (Insulin)</td>
<td>25</td>
<td>12.5</td>
</tr>
<tr>
<td>2</td>
<td>Oral anti diabetic drugs (ODDM)</td>
<td>30</td>
<td>17.5</td>
</tr>
<tr>
<td>3</td>
<td>Antibiotics (AB)</td>
<td>45</td>
<td>30</td>
</tr>
<tr>
<td>4</td>
<td>Analgesics (AG)</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>5</td>
<td>Antipyretic (APY)</td>
<td>5</td>
<td>2.5</td>
</tr>
<tr>
<td>6</td>
<td>Anti inflammatory (AI)</td>
<td>7</td>
<td>3.5</td>
</tr>
<tr>
<td>7</td>
<td>Anti platelets (AP)</td>
<td>5</td>
<td>7.5</td>
</tr>
<tr>
<td>8</td>
<td>Anti allergic (AA)</td>
<td>3</td>
<td>1.5</td>
</tr>
<tr>
<td>9</td>
<td>Anticoagulants (AC)</td>
<td>8</td>
<td>4</td>
</tr>
<tr>
<td>10</td>
<td>Steroids</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>11</td>
<td>Antihypertensive (AH)</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>12</td>
<td>B-complex and vitamins (B-C, Vit)</td>
<td>5</td>
<td>7.5</td>
</tr>
<tr>
<td>13</td>
<td>Others</td>
<td>3</td>
<td>1.5</td>
</tr>
</tbody>
</table>

**Fig 7: Graph on Distribution of Diabetic Patients By Presence Of Other Complications**

**Fig 8: Graph on Distribution of Different Category of Drugs in Prescription**
IX. DISTRIBUTION OF PATIENT BY SERUM CREATININE LEVELS

Table 9: Distribution of Patient by Serum Creatinine Levels

<table>
<thead>
<tr>
<th>S. No</th>
<th>Serum creatinine levels (mg/dl)</th>
<th>Condition</th>
<th>No of patients n=150</th>
<th>% of distribution</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>&lt;0.6</td>
<td>Below normal</td>
<td>24</td>
<td>16</td>
</tr>
<tr>
<td>2</td>
<td>0.6-1.2</td>
<td>Normal</td>
<td>42</td>
<td>28</td>
</tr>
<tr>
<td>3</td>
<td>&gt;1.2</td>
<td>Above normal</td>
<td>84</td>
<td>56</td>
</tr>
</tbody>
</table>

![Distribution of Patient by Serum Creatinine Levels](image)

Fig 9 Distribution of Patient by Serum Creatinine Levels

X. DISTRIBUTION OF PATIENTS BY CHANGES IN HEALTH RELATED QUALITY OF LIFE

Table 10: Distribution of Patients by Changes In Health Related Quality of Life

<table>
<thead>
<tr>
<th>S. No</th>
<th>Physical functionality</th>
<th>Poor</th>
<th>Good</th>
<th>Excellent</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Role limitation</td>
<td>12</td>
<td>9</td>
<td>4</td>
</tr>
<tr>
<td>2</td>
<td>Mental health</td>
<td>2</td>
<td>16</td>
<td>8</td>
</tr>
<tr>
<td>3</td>
<td>General health</td>
<td>12</td>
<td>8</td>
<td>5</td>
</tr>
<tr>
<td>4</td>
<td>Social functioning</td>
<td>13</td>
<td>8</td>
<td>4</td>
</tr>
<tr>
<td>5</td>
<td>Body pain</td>
<td>9</td>
<td>12</td>
<td>4</td>
</tr>
</tbody>
</table>

![Distribution by health related quality of life](image)

Fig 10 Distribution of patients by changes in health related quality of life
DISCUSSION
Our study was completely an observational study regarding diabetes and its complications prevailing in the present society, because of lack of knowledge among the people, about how it occur and type of diet to be taken.

Most of the diseases like kidney diseases, visual impairment, neurological disorders, heart diseases, occur due to increase in glucose levels in the body (i.e) diabetes. Present survey in American research institute says that cancer also occur due to diabetes which has the capability to change the DNA pattern.

Still in India 40% people are un-aware of diabetes and do not take proper medication or the treatment which inturn leading to major complications like nephropathy.

Therefore, by considering all the factors which contributed to diabetic nephropathy, it was proposed to study the management in diabetic patients like patient counselling, treatment with drugs, duration of treatment and little focus on other complications in diabetes, drug utilization studies.

The prevalence of diabetic complications is reported from the observational study of rural diabetic subjects in BBR hospital. Logical observational analysis has been used to discovering potential risk factors associated with each complication.

A distinction has been made between time-related variables (age at diagnosis, duration of diabetes, diet related problems, stress related, gender etc..,) and other risk variables. We have attempted to identify the major diet-related and complication related risk variables for each complication and then examined the effect of other risk variables after accounting for the major complication-related variables.

The important diet-related variables was found to be duration of diabetes for nephropathy, age for macrovascular disease, duration and age at diagnosis of diabetes for kidney related problems, and age for renal impairment. When matched on these important age-related variables, the overall prevalences of complications for insulin-dependent (IDDM) compared with non-insulin-dependent (NIDDM) diabetic patients and it was found to be more in case of NIDDM compared with that of IDDM. An exception is retinopathy, for which IDDM patients had a higher prevalence than did NIDDM patients of the same age.
After allowing for complication-related variables, the analysis also demonstrates positive independent associations between diabetic control (glycosylated hemoglobin) and nephropathy and between diabetic control and macrovascular disease. Plasma cholesterol (positively) and high-density lipoprotein cholesterol (negatively) were related independently to both macrovascular disease and renal impairment.

CONCLUSION
In the last several years, we have witnessed an enormous progress made not only in our understanding of the risk factors and mechanism of the development of diabetic nephropathy, but also in the treatment possibilities aimed at preventing the progression of diabetic nephropathy.

Early detection of this chronic DM complication along with the treatment of main risk factors (hyperglycemia, hypertension, and dyslipidemia) and use of renoprotective drugs (ACEI and ARB) may decrease the progression of this kidney disease. The treatment of increased blood pressure is a priority. All listed measures lead to a decrease in the overall and cardiovascular mortality in patients with DM.

The principle treatment of diabetic nephropathy is based on tight control of hyperglycemia, tight control of blood pressure and glomerular pressure, control of dyslipidemia, restriction of protein intake and smoking withdrawal and the main important is to follow the good diet and proper exercise daily.

AKNOWLEDGEMENT
We thank all doctors, patients and staff in BBR hospital, balanagar, Hyderabad, telangana state, india for their Fellowship grant support to Dr.Kiran for our work carried for 6 month It is a great pleasure to express our deep sense of gratitude and sincere thanks to our Research Co-coordinator, Dr.Sambasivaraao Professor in Pharmaceutical chemistry, Principal of Sri Indu Institute of Pharmacy, Sheriguda, for his valuable suggestions and providing all facilities to carry out our research work successfully.

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