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EVALUATION OF THE OUTCOMES OF PATIENTS COUNSELING IN DIABETES PATIENTS

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ABSTRACT

Background: Diabetes mellitus a disorder related with the metabolic impairment of carbohydrate metabolism (hyperglycemic condition) resulted from poor insulin secretion, as well as their action thereby resulting failure of maintaining consistent levels of sugar (glucose) in the blood. These studies evaluate the outcomes of Patients counseling in diabetes patients. **Aim & Objectives:** Study was focused on to assured and evaluated Demographic status of patients, Past and current medical records, Patients counseling status, Outcomes related to patient's query, Question based counselling. **Methodology:** This was an observational and prospective study was carried out in medicine ward and departments of a tertiary care hospital for a period of six months Patients Data was collected from the Patient counseling form, Patients consent form. **Result:** A total number of 130 subjects included from medicine ward and department to evaluate the study. In this study out 130 subjects, 68 (52.30%) subjects were male and 62 (47.69%) were female. All the subjects were found below the age of 75 years, Maximum subjects (60%) were found in age group of 18-36 years. In this study out of 130 subjects, 20 (29.01%) males and 01(1.61%) female was found alcoholic and 22 (32.35%) males and 05 (8.06%) females were found to smoke. 90 (60%) were found as diabetic, 25 (19%) subjects were suffering from hypertension, 36 (28%) subjects were suffering from Nephropathy, 21 (16%) suffering from retinopathy, 85 (85%) subjects were suffering from Neuropathy, 25

(19.23%) subjects were suffering from Asthma and 15 (11.53%) subjects were suffering from Erectile Dysfunction. 70 subjects were taking “Metformin alone”, 50 (38.46%) subjects were taking “Insulin alone”, 90 (69.23%) subjects were taking “sulfonyl urea and metformin combination”, 35 (26.92%) subjects were taking “thiazolidinedione/acarbose”, 110 (84.61%) subjects were taking “on the drugs for controlling HTN”, and 80 subjects were taking “Other combination” were observed these study. All the subjects were interviewing different kind of questions regarding diabetes and made them satisfy by answering all the questions and asked some questions to the subjects. The all questions were based on diabetes and related to the project. All the subjects were replied in “Yes” or “No. **Conclusion:** Patients education is of essence in achieving optimal treatment outcome in diabetes management. This is because education can influence knowledge that could empower patients to rise up and be effectively involved in the management of their health. Patients' understanding of their disease conditions, blood glucose monitoring and lifestyle modifications could be factored into patient education and counseling to optimize diabetes management outcomes.

KEYWORD: Patients Counseling, Diabetes, Comorbidities, Hyperglycemias.

INTRODUCTION

Diabetes mellitus, a disorder related with the metabolic impairment of carbohydrate metabolism (hyperglycaemic condition) resulted from poor insulin secretion, as well as their action thereby resulting failure of maintaining consistent levels of sugar (glucose)in the blood. The low level of insulin not efficiently reaches to the target site such as adipose tissues, skeletal tissues, at the receptor site of insulin and produce inadequate response to insulin resulting failure of signal transduction, genes, and effector enzyme associated these metabolic abnormalities.

In 2014, 8.5% of adults aged 18 years and older had diabetes. In 2016, diabetes was the direct cause of 1.6 million deaths and in 2012 high blood glucose was the cause of another 2.2 million deaths The chronic diabetes is the major cause of morbidity and mortality, in which large blood vessels linked with heart like coronary heart disease as well as small blood vessels such as retinal and renal vascular disease developed. https://www.who.int/news-room/fact-sheets/detail/diabetes].

1.1. Epidemiology of diabetes mellitus

1.1.1. Globally burden

Globally, an estimated 422 million adults were living with diabetes in 2014, compared to 108 million in 1980. The global prevalence (age-standardized) of diabetes has nearly doubled since 1980, rising from 4.7% to 8.5% in the adult population. This reflects an increase in associated risk factors such as being overweight or obese. Over the past decade, diabetes prevalence has risen faster in low- and middle-income countries than in high-income countries. Diabetes caused 1.5 million deaths in 2012. Higher-than-optimal blood glucose caused an additional 2.2 million deaths, by increasing the risks of cardiovascular and other diseases. Forty-three percent of these 3.7 million deaths occur before the age of 70 years. The percentage of deaths attributable to high blood glucose or diabetes that occurs prior to age 70 is higher in low- and middle-income countries than in high-income countries. [Vazquez G et al 2007]

1.1.2 Indian burden

Diabetes is fast gaining the status of a potential epidemic in India with more than 62 million diabetic individuals currently diagnosed with the disease. In 2000, India (31.7 million) topped the world with the highest number of people with diabetes mellitus followed by China (20.8 million) with the United States (17.7 million) in second and third place respectively. According to Wild et al. the prevalence of diabetes is predicted to double globally from 171 million in 2000 to 366 million in 2030 with a maximum increase in India. It is predicted that by 2030 diabetes mellitus may afflict up to 79.4 million individuals in India, while China (42.3 million) and the United States (30.3 million) will also see significant increases in those affected by the disease.India currently faces an uncertain future in relation to the potential burden that diabetes may impose upon the country. [Joshi SR et al 2007]

The prevalence of diabetes in rural populations is one-quarter that of urban population for India and other Indian sub-continent countries such as Bangladesh, Nepal, Bhutan, and Sri Lanka.[Wild S, Roglic et al 2004] Preliminary results from a large community study conducted by the Indian Council of Medical research (ICMR) revealed that a lower proportion of the population is affected in states of Northern India (Chandigarh 0.12 million, Jharkhand 0.96 million) as compared to Maharashtra (9.2 million) and Tamil Nadu (4.8 million). The National Urban Survey conducted across the metropolitan cities of India reported similar trend: 11.7 per cent in Kolkata (Eastern India), 6.1 per cent in Kashmir

Valley (Northern India), 11.6 per cent in New Delhi (Northern India), and 9.3 per cent in West India (Mumbai) compared with (13.5 per cent in Chennai (South India), 16.6 per cent in Hyderabad (south India), and 12.4 per cent Bangalore (South India). [Masoodi SR et al 2000]

1.2 Sign and symptoms

Symptoms of diabetes include [American Diabetes Association].

- increased thirst and urination,
- increased hunger,
- fatigue,
- blurred vision,
- numbness or tingling in the feet or hands,
- sores that do not heal,
- unexplained weight loss

1.3 Types of diabetes mellitus

The classification of diabetes was first proposed in 1997 by American Diabetes Association (ADA) as:

1.3.1 Type 1 diabetes mellitus: It is caused due to destruction of pancreatic β-cell resulting complete insulin deficiency. This type of diabetes constitutes 5 %-10 % of subjects diagnosed with diabetes The Type 1 diabetes is more prevalent in children and adolescent which is accounts for 80%-90% of diabetes [Craig ME Both Maahs DM et al., 2010].

In 2013, the number of youth diagnosed in the age of 0-14 diagnosed with type 1 diabetes was 497100 worldwide in 2013 was 497100 and their diagnosed number of every year is 78900 One report suggested that type 1 diabetes alone in US was reported 3 million in 2010. [Chiang JL, 2014].

Type 1 diabetes is related with autoimmune demolition of the pancreatic β cells through T-cell mediated inflammatory response along with a B cell (humoral) response [Devendra D, 2004].

The indication of autoantibodies against pancreatic islet cells is a distinct feature of type 1 diabetes. The autoimmune antibodies comprised of autoantibodies islet cell, autoantibodies to

insulin, protein tyrosine phosphatase, glutamic acid decarboxylase and these autoantibodies could be detected in the serum of the patient onset or long after the disease [Couper J, 2009]. This type 1 autoimmune diabetes is lack insulin secretion and is more prevalent in children and adolescents. Besides these predispositions genetic factor several environmental factors have been implicated in causing the disease. Certain viral factor such has congenital rubella, herpes virus, enterovirus, rotavirus, cytomegalovirus, endogenous retrovirus and Ljungan virus may be causative agent of diabetes [Stene LC, 2010; Yeung WC 2011].

In addition to these low vitamin D levels, exposure to pollutant in prenatal condition improved hygiene and living conditions reduced child age infections in developed with high socioeconomic status may prone to develop autoimmune diseases (hygiene hypothesis).

The indication of Type 1 diabetes often explicated and leads to certain symptoms such as polydipsia, enuresis, polyuria, heavy tiredness, lack of energy, polyphagia, slow-healing wounds, sudden weight loss, recurrent infections and finally blurred vision with severe dehydration and diabetic keto-acidosis (ketone bodies diposition) in children and adolescents. The appearance of symptoms is more severe in children compared to adults. These autoimmune type 1 diabetes patients are also prone to other autoimmune disorders such as Graves' disease, Hashimoto's thyroiditis, myasthenia gravis, vitiligo Addison' s disease, autoimmune hepatitis, and pernicious anemia. In some children, the requirement for insulin therapy may drop to a certain level and in this case the insulin therapy temporarily withdrawn without detecting hyperglycemia [American Diabetes Association].

1.3.2: Type 2 diabetes mellitus

The worldwide occurrence of diabetes in adults aged 20 to 79 as per the report published in 2013 by the International Diabetes Federation (IDF) was 8.3% (382 million people), which could be rise to beyond 592 million by 2035 with a 10.1% global prevalence. Insulin resistance in type 2 diabetes patients increases the demand for insulin in insulin-target tissues. In addition to insulin resistance, the increased demand for insulin could not be met by the pancreatic β cells due to defects in the function of these cells [Halban PA, 2014].

On the contrary, insulin secretion decreases with the increased demand for insulin by time due to the gradual destruction of β cells that could transform some of type 2 diabetes patients from being independent to become dependent on insulin [Druet C, 2006].

Most type 2 diabetes patients are not dependent on insulin where insulin secretion continues and insulin depletion rarely occurs. Dependence on insulin is one of the major differences from type 1 diabetes. Other differences include the absence of keto-acidosis in most patients of type 2 diabetes and autoimmune destruction of β cells does not occur. Both type 1 and type 2 diabetes have genetic predisposition, however, it is stronger in type 2 but the genes are more characterized in type 1 (the TCF7L2 gene is strongly associated with type 2 diabetes) [Saadi H, 2008].

Due to the mild symptoms of type 2 diabetes in the beginning, its diagnosis is usually delayed for years especially in countries where regular checkup without symptoms is not part of the culture. This delay in diagnosis could increase the incidence of long term complications in type 2 diabetes patients.

Apart from that in diabetes, insulin resistance has several manifestations which includes obesity, nephropathy, hypertension, dyslipidemia, low HDL, enhanced postprandial lipemia and remnant lipoprotein accumulation, ovarian hyperandrogenism, fatty liver disease (non-alcoholic) and systemic inflammation [Kraemer FB, 2014].

Some patients with features of type 2 diabetes have some type 1 characteristics including the presence of islet cell autoantibodies or autoantibodies to GAD65 are classified as a distinct type of diabetes which is called latent autoimmune diabetes in adults (LADA) [Pozzilli P, 2001].

People diagnosed with LADA do not require insulin treatment. In a recent study by Hawa et al [Hawa MI, 2009] showed that 7.1% of European patients with type 2 diabetes with a mean age of 62 years, tested positive for GAD autoantibodies and the prevalence of LADA was higher in patients diagnosed with diabetes at a younger age. This classification of LADA as a distinct type of diabetes is still controversial.

1.3.3: Gestational diabetes mellitus (GDM)

Gestational diabetes is a high blood sugar which develops in due course of pregnancy and often disappears after giving birth. It can occur at any stage of pregnancy but is more common in the second half.

It occurs if your body cannot produce enough insulin that has control over blood sugar levels to meet the extra needs in pregnancy. Gestational diabetes can cause problems both mother

and baby during and post birth. But the risk of these problems could be reduced and managed if it is well detected [https://www.nhs.uk/conditions/gestational-diabetes/].

Hyperglycemia in pregnancy whether in the form of type 2 diabetes diagnosed before or during pregnancy or in the form gestational diabetes has an increased risk of adverse maternal, fetal and neonatal outcome. Mothers with gestational diabetes and babies born to such mothers have increased risk of developing diabetes later in life. Hyperglycemia in pregnancy is responsible for the increased risk for macrosomia (birth weight ≥ 4.5 kg), large for gestational age births, preeclampsia, preterm birth and cesarean delivery due to large babies [Metzger BE et al 2007].

The risk factors for gestational diabetes include obesity, personal history of gestational diabetes, family history of diabetes, polycystic ovary syndrome, maternal age, sedentary life, and exposure to toxic factors. Diagnosis of type 2 diabetes before or during pregnancy is based on criteria mentioned before. Fasting plasma glucose ≥ 126 mg/dL (7.0 mmol/L) or 2-h plasma glucose ≥ 200 mg/dL (11.1 mmol/L) post 75 g oral glucose load [Galtier F et al 2010].

1.3.4 Other specific types of diabetes (due to other causes, e.g., genetic defects in β-cell function, genetic defects in insulin action, diseases of the exocrine pancreas, drug or chemical induced). [Metzger BE et al 2007].

1.5 Pathophysiology of diabetes mellitus

1.5.1 Pathophysiology involved in Type 1 diabetes mellitus

It is now well-recognized that Type 1 Diabetes mellitus is an autoimmune disorder characterized by the destruction of insulin-producing pancreatic β-cells.

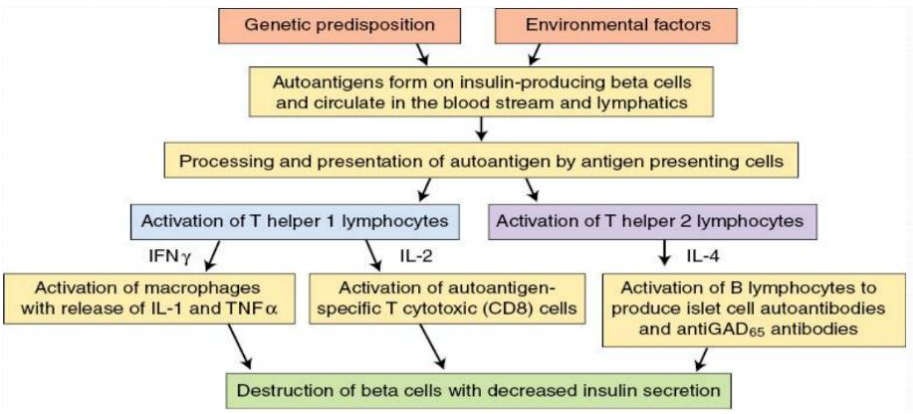

Fig.1.1: Pathophysiology of type 1 diabetes mellitus.

Progress in understanding the pathophysiology of Type 1 Diabetes mellitus cannot be separated from advances in the field of immunology. Like the vast majority of autoimmune disorders, the primary cause of Type 1 Diabetes mellitus is still unknown.Type 1 Diabetes mellitus is characterized by a selective and specific involvement of β-cells without apparent pathological alterations of other Langerhans cells, such as α- (secreting glucagon), δ- (somatostatin) and PP- (pancreatic polypeptide) cells [Willcox A et al 2009].

Both humoral and cellular immunity is involved in Type 1 Diabetes mellitus pathogenesis. T lymphocytes (which mature in the thymus gland and play aey role in cell-mediated immunity) are predominant in islet lesions, with lower concentrations of other immunological cells, such as macrophages, B lymphocytes and plasma cells [Willcox A et al 2009]. The presence of humoral immunity, on the other hand, was recognized over 40 years ago, when autoantibodies against pancreatic islets were detected in subjects with Type 1 Diabetes mellitus. From the 1980s, targets of autoantibodies have been discovered, and several auto antigens are currently widely used in clinical practice, such as insulin, pro insulin, glutamic acid decarboxylase (GAD65), glucose 6-phosphatase catalytic subunit-related protein (G6PC2, also known as IGRP), islet cell antibody (ICA) and zinc transporter 8 (ZnT8A) [Ziegler AG et al 2010].

1.5.2: Pathophysiology of Type 2 diabetes mellitus

While in recent years many major risk factors for the emergent Type 2 diabetes mellitus epidemic have been identified, the mechanisms linking them to the clinical manifestations of Type 2 diabetes mellitus and its complications are intensively investigated. The availability of radio immunoassays in the 1950s helped differentiate ‘insulin dependent’ from ‘non-insulin-dependent’ diabetes, and such differences were formally recognised in the 1979 classification of diabetes by the National Diabetes Data Group in type I (former ‘insulin-dependent’) and type II (‘non-insulin-dependent’) diabetes mellitus [National Diabetes Data Group].

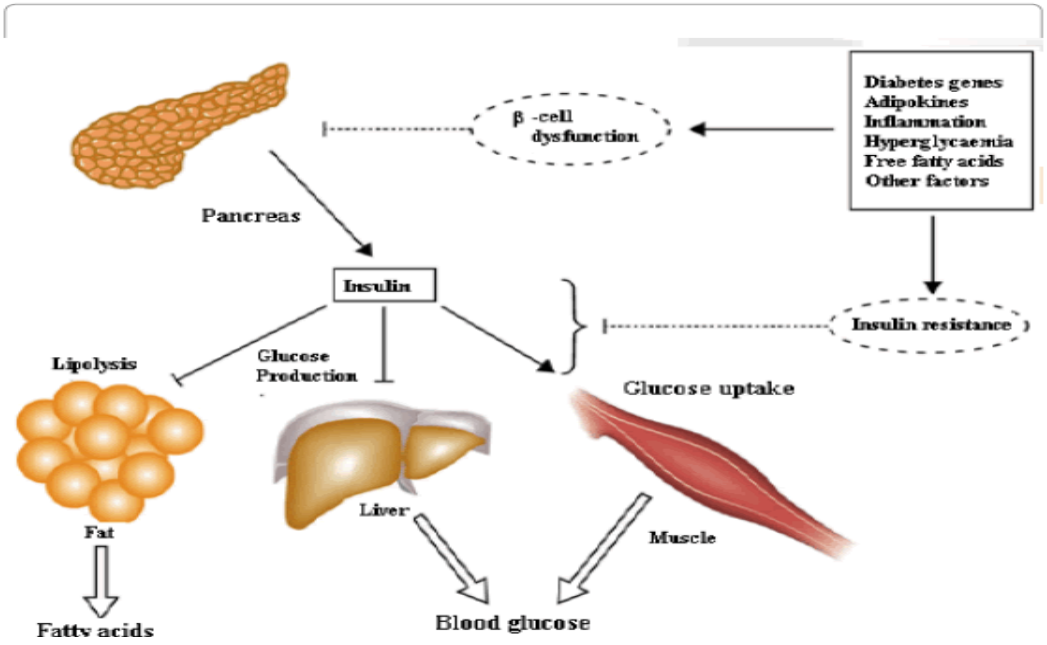

Fig. 1.2: Pathophyiology of type 2 diabetes mellitus.

Several pivotal pathophysiological studies covered the insulin resistance and secretion in the course of disease onset and progression.37–46 Subjects at risk of T2DM (obese subjects and first-degree relatives) display an initial state of insulin resistance compensated by β-cell hyper secretion of insulin (hyperinsulinaemia). Such pancreatic 'functional' reserve, however, unable to cope with the required insulin secretion. Compared with lean euglycaemic subjects, obese euglycaemic people have ~30% reduced insulin sensitivity and therefore show increased insulin secretion to maintain normal glucose tolerance (euglycaemichyperinsulinaemia). Over time, obese euglycaemic subjects experience a further reduction in insulin sensitivity that is no longer associated with compensatory hyperinsulinaemia, resulting in an increased blood glucose concentration (hyperglycaemichyperinsulinaemia). By the time diabetes is diagnosed, β-cells are no longer able to secrete enough insulin, with consequent manifestation of overt hyperglycemia (hyperglycemic hyperinsulinemia) [Allut D et al 1990].

The results of these pathophysiological studies illustrate the failure of compensatory hyperinsulinaemia as the 'hallmark' of frank hyperglycaemia. Therefore, along with mechanistic studies investigating mechanisms forming the basis of insulin resistance, more recent research has also focused on the pathways leading to β-cell 'failure'.Liver and muscles have long been recognised as major contributors of systemic insulin resistance [De Fronzo RA et al 2004]. To ensure constant availability of a carbohydrate energy source during

fasting, the liver produces glucose from non-glucose substrates (gluconeogenesis) [Pilkis SJ et al 1992]. Several studies have shown increased gluconeogenesis in subjects with Type 2 diabetes mellitus, which occurs despite a state of hyperinsulinaemia, suggesting hepatic insulin resistance as a main determinant of fasting hyperglycaemia. The reasons behind reduced hepatic insulin sensitivity are poorly defined, but accumulation of fat within the liver (steatosis) is considered a major determinant. Interestingly, liver steatosis precedes overt Type 2 diabetes mellitus and is commonly associated with obesity, particularly visceral (android or abdominal) obesity [Birkenfeld AL, 2014, Taylor R, 2008, Sattar N et al 2014].

It is now well accepted that a continuous positive energy balance due to excess calories and a lack of physical activity leads initially to fat accumulation in the subcutaneous tissue. When this storage capacity is exceeded, fat is diverted to 'ectopic' compartments such as the liver, pancreas, and muscles. Hepatic and muscle fat accumulation results in impaired insulin mediated glucose uptake due to intracellular impairment of insulin signaling [DeFronzo RA et al 2004].

Fat accumulation within the pancreatic islets, on the other hand, determines β-cell dysfunction and increases in plasma glucose which, in turn, reduce insulin response to ingested glucose [Taylor R et al 2008]. Research performed in the last few years has attempted to clarify the mechanisms of β-cell failure. In genetically predisposed individuals, the increased demand of insulin synthesis and secretion eventually results in β-cell dysfunction [Ahlqvist E 2011 et al] Halban PA, 2014]. Yet, among the proposed potential mechanisms causing β-cell dysfunction (including direct effects of glucose and free fatty acids), their comparative and chronological role is unknown. It has been suggested that the 'stressed' β-cell may stimulate local inflammation and modify the balance between α- and β-cell mass and function within the Langerhans islets. Notably, insulin exerts negative paracrine (a signal inducing changes in nearby cells) action on α-cells, thus limiting the secretion of glucagon therefore, lack of insulin results in higher levels of glucagon, which further increase blood glucose concentration via hepatic gluconeogenesis [Xu E et al 2006].

1.6: Diagnostic aspect of diabetes mellitus

Diabetes mellitus is diagnosed using either the estimation of plasma glucose (FPG or OGTT) or HbA1c. Estimation of the cut off values for glucose and HbA1c is based on the association of FPG or HbA1c with retinopathy. Fasting plasma glucose of ≥ 126 mg/dL (7.0 mmol/L), plasma glucose after 2h OGTT ≥ 200 mg/dL (11.1 mmol/L), HbA1c ≥ 6.5% (48 mmol/mol)

or a random plasma glucose ≥ 200 mg/dL (11.1 mmol/L) along with symptoms of hyperglycemia is diagnostic of diabetes mellitus. In addition to monitor the treatment of diabetes, HbA1c has been recommended to diagnose diabetes by the International Expert Committee in 2009 [International Expert Committee], the WHO [World Health Organization] and many scientists and related organizations all over the world.

The advantages and limitation of the different tests used to diagnose diabetes have been reported by Sacks et al [Sacks DB et al 2011].

The advantages of using HbA1c over FPG to diagnose diabetes include greater convenience and preanalytical stability, lower CV (3.6 %) compared to FPG (5.7%) and 2h OGTT (16.6%), stronger correlation with microvascular complications especially retinopathy, and a marker for glycemic control has a direct link between diagnosis of diabetes and its complications [Shaw JE et al 2011]

It is recommended to repeat the HbA1c test in asymptomatic patients within two weeks to reaffirm a single apparently diagnostic result.

1. 7: Treatment of diabetes mellitus

the isolation of insulin in the 1920s, most patients died within a short time after onset. Untreated diabetes leads to ketoacidosis, the accumulation of ketones (products of fat breakdown).

The acid in the blood. Continued build up of these products of disordered carbohydrate and fat metabolism result in nausea and vomiting, and eventually the patient goes into a diabetic coma.

Treatment for diabetes mellitus is aimed at reducing blood glucose concentrations to normal levels. Measurements of HbA1c can be used to assess whether an individual's treatment for diabetes is effective. Target values of HbA1c levels should be close to normal.

1.7.1: Diet and exercise

All diabetes patients are put on diets designed to help them reach and maintain normal body weight, and they often are encouraged to exercise regularly, which enhances the movement of glucose into muscle cells and blunts the rise in blood glucose that follows carbohydrate ingestion. The Patients are encouraged to follow a diet that is relatively low in fat and

contains adequate amounts of protein. In practice about 40 percent of calories should come from fat, 20 percent from protein, and the remainder from carbohydrates, preferably from complex carbohydrates rather than simple sugars.

The total caloric content should be based on the patient's nutritional requirements for growth or for weight loss if the patient is obese. In overweight or obese patients with type 2 diabetes, caloric restriction for even just a few days may result in considerable improvement in hyperglycemia.

In recently weight loss, preferably combined with exercise, can lead to improved insulin sensitivity and even restoration of normal glucose metabolism.

1.7.2: Insulin therapies

Traditional insulin therapy entails regular injections of the hormone, which are often customized according to individual and variable requirements. The Beef or pork insulin, made from the pancreatic extracts of cattle or pigs, can be used to treat humans with diabetes. However, in the United States, beef and pork forms of insulin are no longer manufactured, having been discontinued in favour of human insulin production. Modern human insulin treatments are based on recombinant DNA technology. Human insulin may be given as a form that is identical to the natural form found in the body, which acts quickly but transiently (short-acting insulin), or as a form that has been biochemically modified so as to prolong its action for up to 24 hours (long-acting insulin). Another type of insulin acts rapidly, with the hormone beginning to lower blood glucose within 10 to 30 minutes of administration; such rapid-acting insulin was made available in an inhalable form in 2014.

The optimal regimen is one that most closely mimics the normal pattern of insulin secretion, which is a constant low level of insulin secretion plus a pulse of secretion after each meal. This can be achieved by administration of a long-acting insulin preparation once daily plus administration of a rapid-acting insulin preparation with or just before each meal. Patients also have the option of using an insulin pump, which allows them to control variations in the rate of insulin administration. A satisfactory compromise for some patients is twice-daily administration of mixtures of intermediate-acting and short-acting insulin. Patients taking insulin also may need to vary food intake from meal to meal, according to their level of activity; as exercise frequency and intensity increase, less insulin and more food intake may be necessary. The Research into other areas of insulin therapy include pancreas

transplantation, beta cell transplantation, implantable mechanical insulin infusion systems, and the generation of beta cells from existing exocrine cells in the pancreas. Patients with type 1 diabetes have been treated by transplantation of the pancreas or of the islets of Langerhans. However, limited quantities of pancreatic tissue are available for transplantation, prolonged immunosuppressive therapy is needed, and there is a high likelihood that the transplanted tissue will be rejected even when the patient is receiving immunosuppressive therapy. Attempts to improve the outcome of transplantation and to develop mechanical islets are ongoing.

1.7.3: Drugs used to control blood glucose levels

There are several classes of oral drugs used to control blood glucose levels, including sulfonylureas, biguanides, and thiazolidinediones. Sulfonylureas, such as glipizide and glimepiride, are considered hypoglycemic agents because they stimulate the release of insulin from beta cells in the pancreas, thus reducing blood glucose levels. The most common side effect associated with sulfonylureas is hypoglycemia (abnormally low blood glucose levels), which occurs most often in elderly patients who have impaired liver or kidney function. [American diabetes association]

1.7.4.1. Biguanides: of which metformin is the primary member, are considered antihyperglycemic agents because they work by decreasing the production of glucose in the liver and by increasing the action of insulin on muscle and adipose tissues. A potentially fatal side effect of metformin is the accumulation of lactic acid in blood and tissues, often causing vague symptoms such as nausea and weakness**.** [American Diabetes Association]

1.7.4.2. Thiazolidinediones: such as rosiglitazone and pioglitazone, act by reducing insulin resistance of muscle and adipose cells and by increasing glucose transport into these tissues. These agents can cause edema (fluid accumulation in tissues), liver toxicity, and adverse cardiovascular events in certain patients. Furthermore, oral hypoglycemic agents lower mean blood glucose concentrations by only about 50–80 mg per 100 ml (2.8–4.4 mmol per litre), and sensitivity to these drugs tends to decrease with time.

There are several other agents that can be highly effective in the treatment of diabetes. Pramlintide is an injectable synthetic hormone (based on the human hormone amylin) that regulates blood glucose levels by slowing the absorption of food in the stomach and by inhibitingglucagon, which normally stimulates liver glucose production. Exenatide is an

injectable antihyperglycemic drug that works similarly to incretins, or gastrointestinal hormones, such as gastric inhibitory polypeptide, that stimulate insulin release from the pancreas. Exenatide has a longer duration of action than incretins produced by the body because it is less susceptible to degradation by an enzyme called dipeptidyl peptidase-4 (DPP-4). A drug called sitagliptin specifically inhibits DPP-4, thereby increasing levels of naturally produced incretins. Side effects associated with these drugs are often mild, although pramlintide can cause profound hypoglycemia in patients with type 1 diabetes [American diabetes association]

1.7.4.3. Secretogogues: The sulfonylureas (glyburide, glipizide) and the meglitinides (repaglinide, Nate glinide) stimulate the pancreas to secrete more insulin. The sulfonylureas also diminish hepatic glucose production and metabolism of insulin by the liver. The net effect is a normalization of insulin and glucose levels. [American diabetes association]

1.7.4.4. Alpha-glucosidase inhibitors: Acarbose and miglitol inhibit enzymes in the small intestine that metabolize complex carbohydrates. This slows the absorption of carbohydrates, reducing postprandial hyperglycemia. [American diabetes association]

1.7.4.5. Incretin-based therapy: It is thought that patients with type 2 diabetes have a suppressed incretin hormone system. The incretin hormones act in the gastrointestinal tract to control blood glucose levels by enhancing insulin secretion, suppressing glucagon secretion from the liver, suppressing glucose output from the liver, delaying gastric emptying (thus slowing carbohydrate and lipid absorption), reducing postprandial hyperglycemia, reducing appetite, and maintaining beta cell function. There are currently two classes of agents that increase incretin activity: (1) incretin mimetics (the glucagon-like peptide-1 [GLP-1] agonists exenatide and liraglutide) and (2) dipeptidyl peptidase-4 (DPP-4) inhibitors (sitagliptin, saxagliptin, and linagliptin). [American diabetes association]

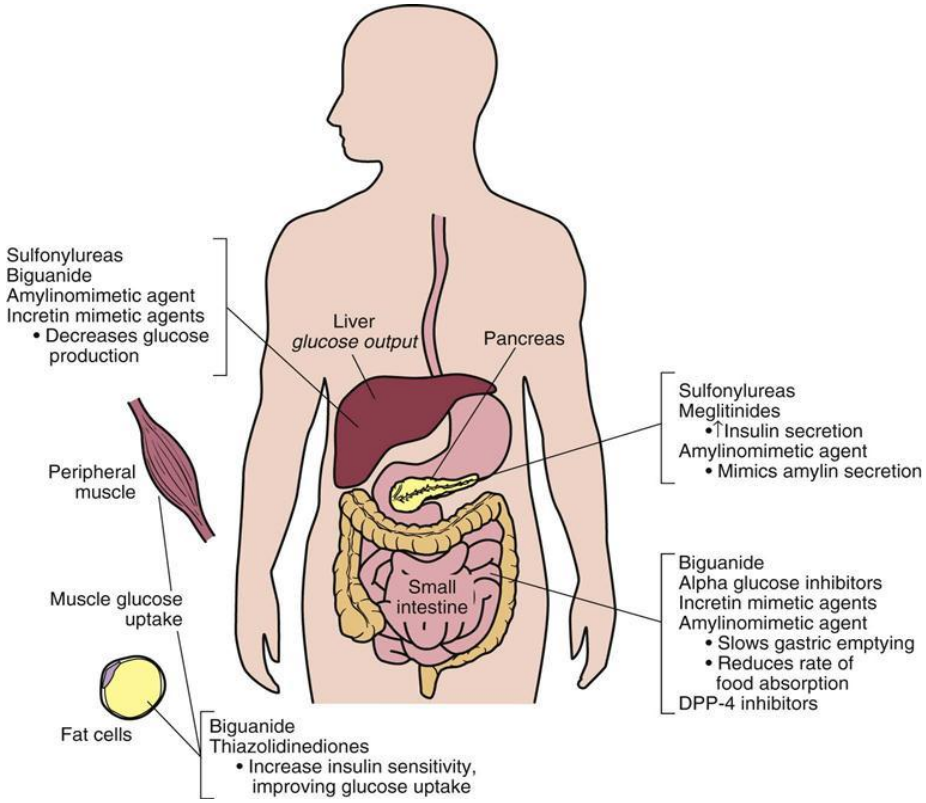

Figure 1.3: Sites and mechanisms action of Anti diabetic agents.

1. 8: Need of the study

For today, mostly disease are inherited, they vary from populations to populations. Lots of research and approached had done before. Diabetics is such disease in which patient will be aware of symptoms more. Therefore there is a requirement for patients counseling in the same and also need to evaluate the impact and severity of disease has reached in the current population.

Aim and Objectives

3. 1: AIM

To evaluate the outcomes of Patients counseling in diabetes patients.

3. 2: OBJECTIVES

The study will involve the following objectives, to be assured and evaluated:

(1) Demographic status of patients,

(2) Past and current medical records,

(3) Patients counseling status.

(a) Outcomes related to patients query

(b) Question based counseling

Study design and methodology

4. 1: Study design

This was an observational and prospective study conducted in a tertiary care hospital in city of Dehradun to evaluate the patient outcomes of patient counseling in diabetes patients.

4. 2: Study site

The study was carried out in medicine ward and departments of a tertiary care hospital Shri Mahant Indresh Hospital, Patel Nagar, Dehradun.

4.3 :Study population

4.3.1 Inclusion criteria

i) Known or newly diagnosed diabetes mellitus patients

ii) Patients with 18- 80 year of age or older

iii) Patients with glycated hemoglobin 6.8-10.0 % (both incl.)

iv) Patient visiting inpatient and outpatient department

4.3.2 Exclusive criteria

i) Patients with glycated hemoglobin above 10.0%.

ii) Patients admitted in ICU with severe illness.

iii) Patients who refused to take part in the study.

4. 4: Sources of data

i) Patient counseling form.

ii) Patient consent form

4.4 :Duration of study

The duration of conducting the study was of six months after the approval from the Institutional ethical committee.

4. 6: Study document

The Patient counseling Performa was designed for data collection of patients including medical, medication history, present illness, current medication, demographic status, social history.

4. 7: Study procedure

The patient counseling Performa documents designed for data collection of patients demographic status, past and current medications records, social history and patients query and question based counseling.

RESULT AND DISCUSSION

A total number of 130 subjects included from Department of Medicine to evaluate the patient outcomes of patient counseling in diabetes patients at a tertiary care hospital. The data collection and reports were analyzed.

5. 1: Demographic profile of the patient included in the study

The demographic profile of patient is classified as gender wise distribution, age wise distribution.

5. 2: Gender wise distribution of subjects

In this study out of 130 subjects, 68 subjects were male and 62 were female. The percentage of male subjects and female subjects was calculated 52.30% and 47.69% respectively (Table No: 4.1)

Demographic status

Table 5. 1: gender wise distribution of patients

S No.	Gender	No. of Patients	Percentage (%)
1.	MALE	68	52.30%
2.	FEMALE	62	47.69%
TOTAL		130	100%

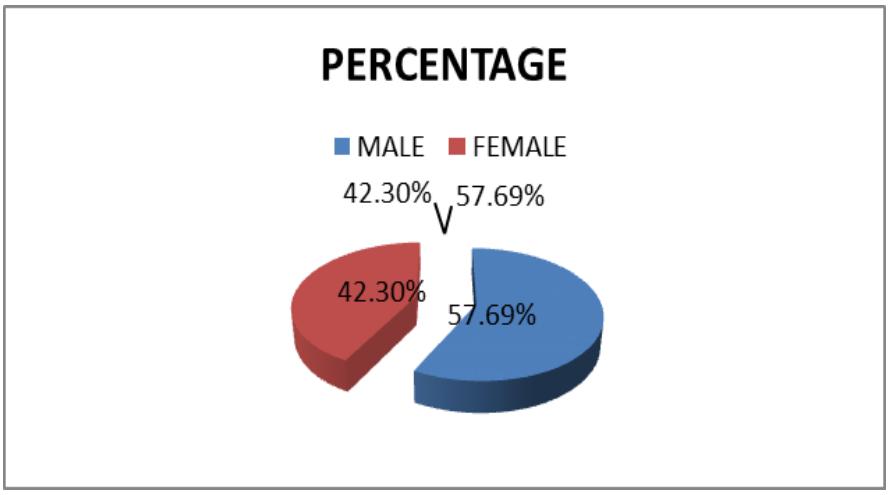

Figure 5. 1: Gender wise patients distribution.

5. 2: Age wise distribution

In present study, all the subjects were found below the age of 75 years. Subjects were categorised in four groups as per there age(less than 18, 18-36, 37-54, 55-72 more than 73 years). Maximum subjects (60%) were found in age group of 18-36 years.

Table 5. 2: Age wise distribution of patients.

S. no.	Age group	No. of patients	Male	Female	Percentage (%)
1.	18-36	50	30(60%)	20(40%)	38.46%
2.	37-54	30	18(30%)	12(40%)	23.07%
3.	55-72	40	18(45%)	22(55%)	30.76%
4.	>-73	10	02(20%)	08(80%)	7.69%
TOTAL		130	68	62	100%

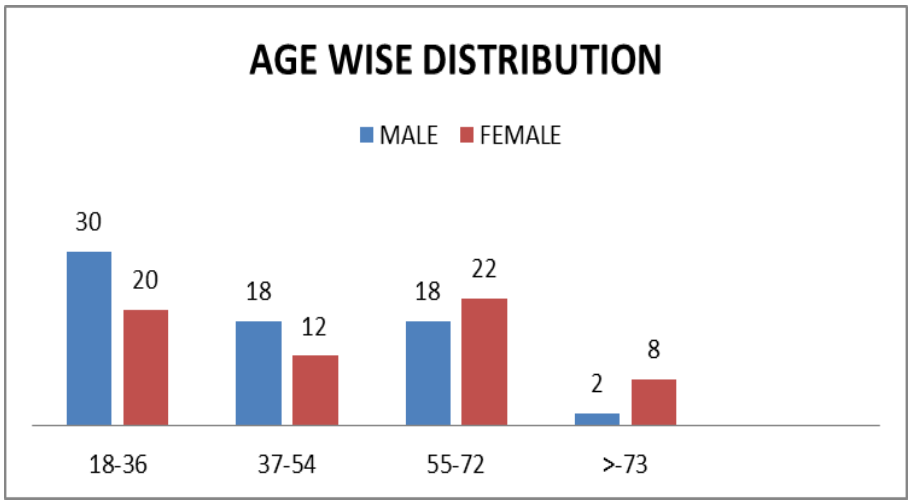

Figure 5. 2: Shows age wise distribution of patient.

5. 3: Alcoholic status of patients

In this study out of 130 subjects, 20 males and 01 females were found alcoholic and their percentage are 29.41 % and 1.61 % respectively. (Table No. 5.3)

Table 5. 3: Alcoholic status of patients.

S. no.	Total no. of patients		No. of patients taking alcohol		Percentage (%)	
	Male	Female	Male	Female	Male	Female
1.	68	62	20	01	29.41%	1.61%

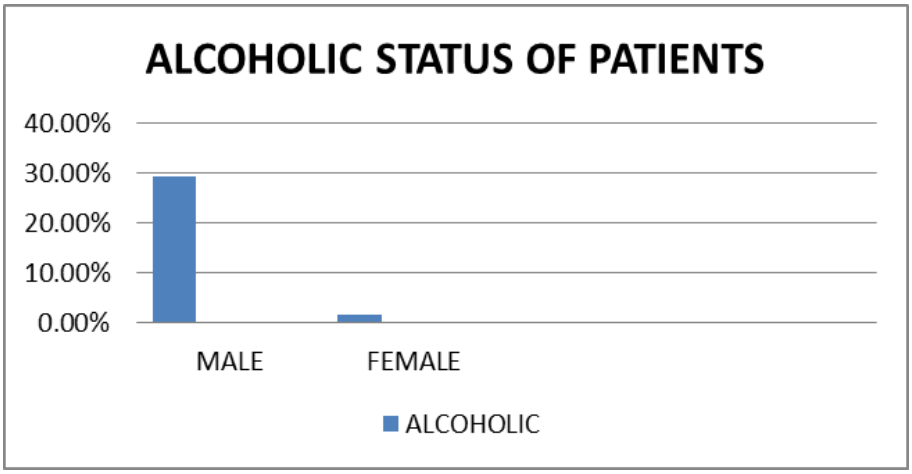

Figure 5. 3: Shows alcoholic status of patients.

5. 4: Smoking status of patients in diabetes.

In this study out of 130 subjects, 22 males and 05 females were found to smoke and their percentage was calculated 32.35 % and 8.06 % respectively. (Table No. 5.4)

Table 5. 4: Smoking status of patients in diabetes.

S no.	Total no. of patients		No. of patients taking smoker		Percentage (%)	
	Male	Female	Male	Female	Male	Female
1.	68	62	22	05	32.35%	8.06%

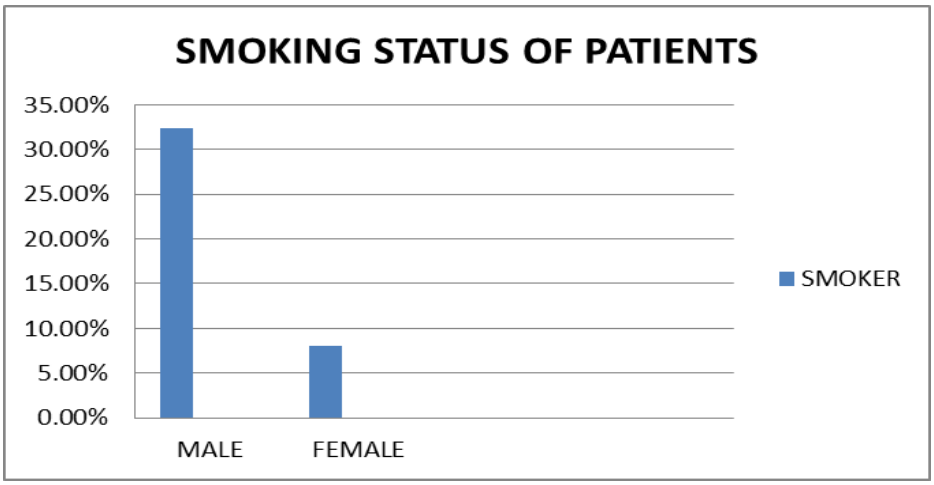

Figure 5. 4: Shows smoking status of parients.

5. 5: Past disease status

In this study out of 130 subjects, 90 were found as diabetic, 25 subjects were suffering from hypertension, 36 subjects were suffering from Nephropathy, 21suffering from retinopathy, 85 subjects were suffering from Neuropathy, 25 subjects were suffering from Asthma and 15 subjects were suffering from Erectile Dysfunction. The percentage was calculated 69%, 19%, 28%, 16%, 65%, 19.23% and 11.53% respectively.

Table 5. 5: Past disease status.

Sr no.	Past disease diagnosed	No.of patients	(%) of patients
1.	Diabetes	90	69%
2.	Hypertension	25	19%
3.	Nephropathy	36	28%
4.	Retinopathy	21	16%
5.	Neuropathy	85	65%
6.	Asthma	25	19.23%
7.	Erectile Dysfunction	15	11.53%

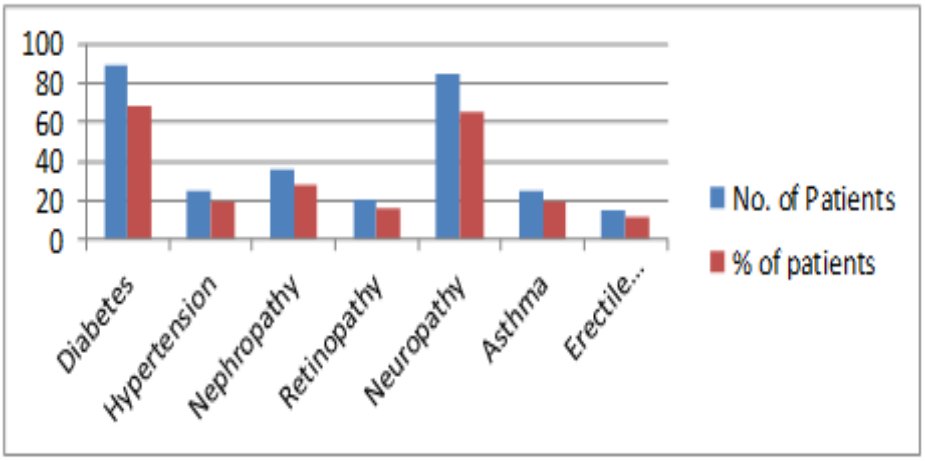

Figure 5.5: Shows past disease status of patients.

5. 6: Current medical records of diabetic patients

In this study out of 130 subjects, 70 subjects were taking "Metformin alone", 50 subjects were taking "Insulin alone", 90 subjects were taking "sulfonyl urea and metformin combination", 35 subjects were taking "thiazolidinedione/acarbose", 110 subjects were taking "on the drugs for controlling HTN", and 80 subjects were taking "Other combination". The percentage of subjects was found to be 53.84%, 38.46%, 69.23, 26.92%, 84.61%, 61.53 and 11.53% respectively.

Table 5. 6: Current medical records of diabetic patients.

S no.	Name of drugs	No of patients	(%) of patients
1.	Metformin Alone	70	53.84%
2.	Insulin Alone	50	38.46%
3.	Sulfonyl urea and metformin combination	90	69.23%
4.	Thiazolidinedione/acarbose	35	26.92%
5.	On the drugs for controlling HTN	110	84.61%
6.	Others combinations	80	61.53%
7.	Not on any drugs	15	11.53%

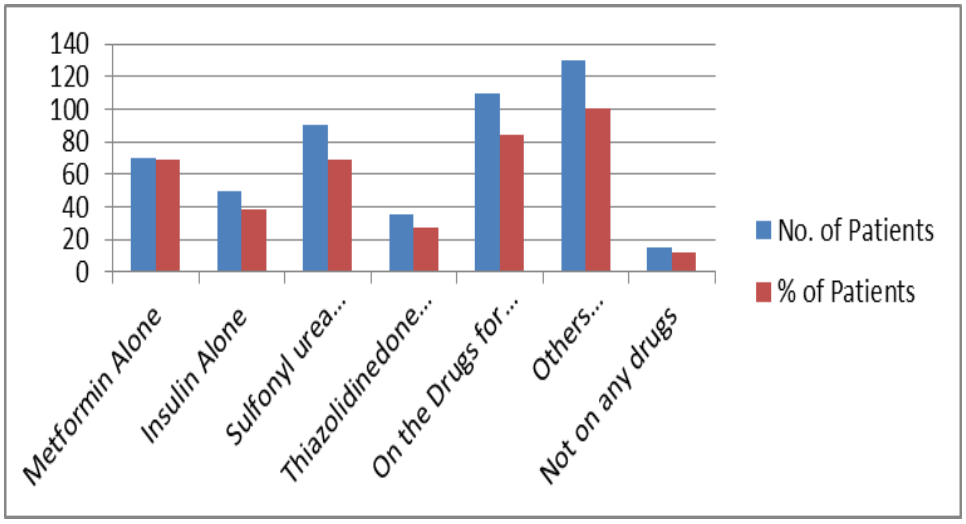

Figure 5. 6: Shows current medical records.

5. 7: Outcome of patients related query

In this study, all the subjects were asked different kind of questions regarding diabetes and made them satisfy by answering all the questions regarding diabetes. the questions that the subjects asked during this study are given in the table no. 5.7.

Table 5. 7: Outcome of patients related query.

S. no.	Patients query	No. of patients	% of patients
1.	What is diabetes?	130	100
2.	About diet in case of diabetes mellitus?	130	100
3.	Precaution and medication in diabetes mellitus?	130	100
4.	About causes of diabetes mellitus?	130	100
5.	What is the complication of diabetes mellitus?	121	93.07

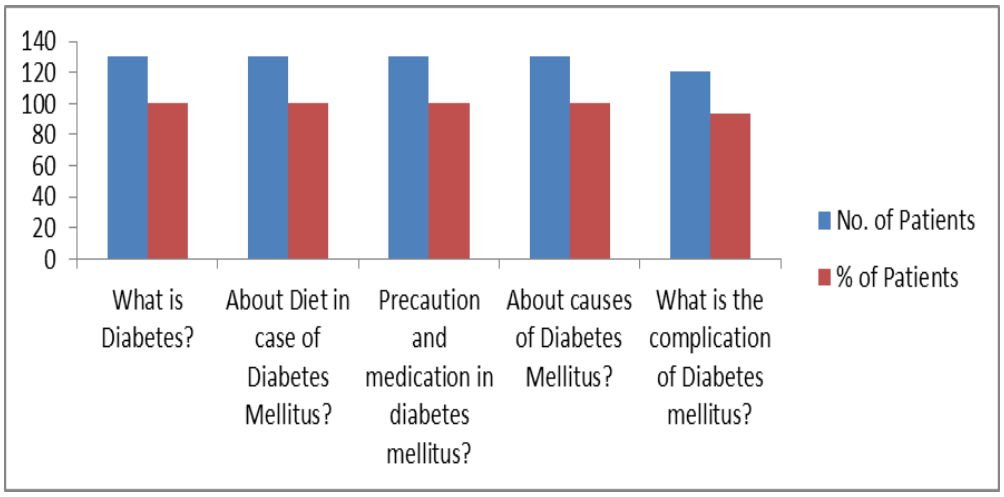

Figure 5.7: Shows outcome of patients related query.

5. 8: Question based councelling

During this study, asked some questions to the subjects. The all questions were based on diabetes and related to the project. All the subjects were replied in "Yes" or "No" type answers. The questions that were asked, given in the table No. 5.8.

Table 5. 8: Question based councelling.

S.no.	Question to be asked	No of patients	Answering the question (yes)	% of patients
1.	Do you understand about the disease diabetes?	130	61	46.92
2.	Do You know about the treatment of diabetes?	130	79	60.76
3.	Do you understand about the diabetic complications?	130	86	66.15
4.	Do you know about cause of Diabetes?	130	98	75.38
5.	Do you know the precautions to be taken in diabetes?	130	60	46.15

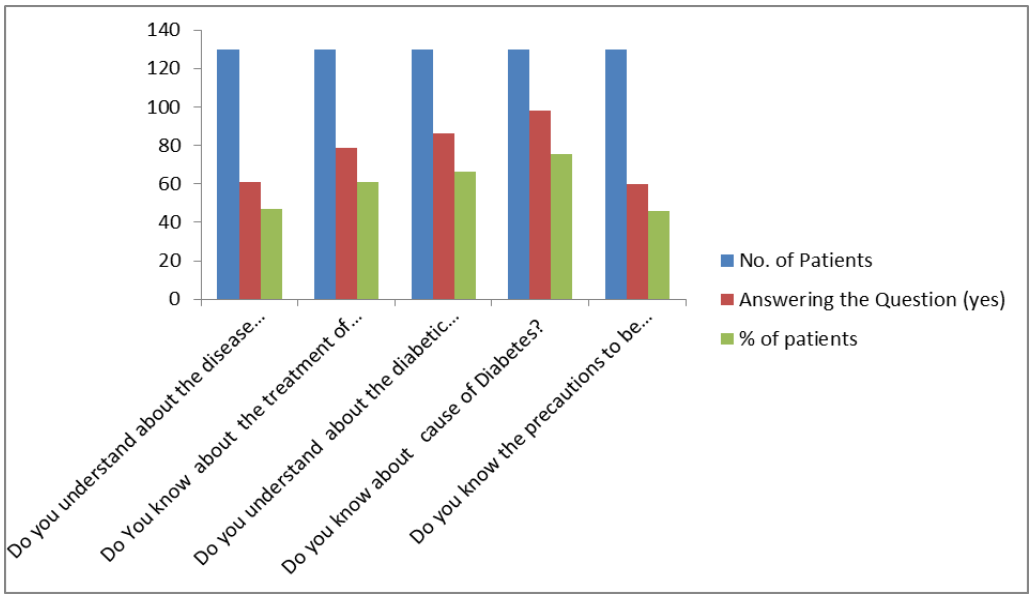

Figure 5. 8: Shows question based counceling status.

CONCLUSION

- In this study are confirms that improvement in knowledge of the disease and its management had positive impact on treatment outcomes and quality of life. Pharmacist provided patient counseling might be considered as an important element in implementing the disease management program.

- The pharmacists' expanded roles in the healthcare sectors are openings to perform relevant clinical functions for the patient's better outcomes. In chronic disease management such as in DM, patient education and counselling have become key tools in patients' self-successful management programmes because of their high effectiveness in achieving both glycaemic and BP controls including a reduction in their overall complications. Thus, the findings in this study are in agreement with earlier ones which demonstrated that clinical pharmacists are effective in the management of chronic diseases such as diabetes. This study encourages the inclusion of clinical pharmacists into multidisciplinary healthcare groups in hospital and clinic settings as well as incorporation of this type of intervention into diabetic management programmers for optimal patient's outcomes.

- Patients education is of essence in achieving optimal treatment outcome in diabetes management. This is because education can influence knowledge that could empower patients to rise up and be effectively involved in the management of their health. Patients' understanding of their disease conditions, blood glucose monitoring and lifestyle modifications could be factored into patient education and counseling to optimize diabetes management outcomes.

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