"A CLINICAL PROFILE OF URINARY TRACT INFECTION IN TYPE 2 DIABETES MELLITUS WITH BACTERIAL CULTURE AND ANTIBIOTIC SENSITIVITY PATTERNS"

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ABSTRACT

Introduction

The present study of 100 type 2 diabetic patients with urinary tract infection admitted in Department of General Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre during the study period from January 2020 to December 2020 was undertaken to study the clinical patterns, risk factors, causative microorganisms and their antibiotic susceptibility of Urinary Tract Infections in Type 2 Diabetes Mellitus.

Materials and Methods

Diabetic patients admitted in the wards and intensive care units of General Medicine at KLES Dr. Prabhakar Kore Hospital, Belagavi.

Observation and Conclusion

We analyzed urinary tract infection whether symptomatic or asymptomatic was frequently encountered in diabetics with elevated HbA1c, poor glycemic control and female gender. In our study there was a *slight female preponderance*, accounting a ratio of M: F:: 1.2:1 (55 females and 45 males). The commonest organism isolated was Escherichia Coli. Urinary tract infection presented in mainly 3 forms namely asymptomatic bacteriuria, cystitis and pyelonephritis. Higher antibiotics mainly Carbapenems were required and administered in patients with Pyelonephritis.

Keywords:

Urinary tract infection, Diabetes mellitus

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INTRODUCTION

Diabetes mellitus is derived from the Greek term ‘diabetes’, translating to siphon – travel or to pass through and the Latin word ‘mellitus’ translating to sweet. History has it that the word ‘diabetes’ was initially used by Apollonius of Memphis around 250 to 300 BC. Prehistoric Greek, Indian, and Egyptian dynasties discovered the sweet quality of urine in this disease, and therefore coined the terms ‘Diabetes Mellitus’. In 1889, Mering and Minkowski postulated the endocrinological role of the pancreas in the development of diabetes. In 1922, Collip, Banting and Best genetically engineered the hormone insulin from the pancreas of ruminants at the Toronto University, leading to the accessibility of a breakthrough and effective treatment. Over the years, groundbreaking discoveries, as well as management plans, have been devised to tackle this exponentially growing cause of morbidity. Unfortunately, even in the present day, diabetes is one among the most common chronic health issues in the country and globally.(1)

According to the WHO, in 2014, 8.5% of population aged 18 years and above had diabetes. In 2019, diabetes caused 1.5 million deaths. In between 2000 and 2016, there was a 5% rise in premature mortality from diabetes. In high-income countries the premature mortality rate due to diabetes decreased from 2000 to 2010 but then increased in 2010-2016. In third world and developing countries, the premature mortality rate due to diabetes increased across both periods.

The prevalence of diabetes mellitus in the Indian subcontinent is showing an acute surge as is apparent from epidemiological trends from different parts of the Indian geography and studies of migrant Indians. (2) The World Health Organization claims around 19.4 million people in India were affected by Diabetes Mellitus in 1995 and these figures are charted to hike to 57.2 million by the end of the year 2025.(3)

Another study claims the estimated sufferers will be 80.9 million people by 2030.(4) According to recent epidemiological studies, migrant Indians residing in various parts of the world showed a higher population of diabetes mellitus than the birth citizen population of those countries. (5) This is ascribed to significant variations in environmental variables, such as better affluence, which unmasked an higher genetic or ethnic propensity for diabetes.(6) The basis of the noteworthy upsurge in diabetes in Indian population are attributed to mounting resistance to insulin, tougher genetic and environmental factors coinciding with urbanization.(7)

Apart from the mainstream complications, diabetes mellitus has been blamed for reduced activity of T cells, phagocytic function and disorders of humoral immunity. (8) Resultingly, diabetes mellitus increases the susceptibility to infections. Such infections, in addition to the consequences associated with infection, may trigger diabetes related complications such as hypoglycaemia and ketoacidosis.

Patients affected with diabetes mellitus are at increased hazard of infections, with the urinary tract being the most frequent site of infection. (9) Immunological impairments (10) along with poor glycemic control and partial bladder voiding(11) attributed to the complication of autonomic neuropathy may all promote the pathogenesis of UTIs in diabetics. Age, control of sugars, and long-term complications such as diabetic nephropathy and cystopathy were found to enhance the risk of urinary tract infections.(12)

The various forms of urinary tract infections in diabetics are asymptomatic bacteriuria (ASB), cystitis, pyelonephritis and urosepsis. Grave complications, such as emphysematous cystitis and pyelonephritis, renal abscesses and renal papillary necrosis, are all faced more often in diabetics than in the common population.(13) Diabetes mellitus is a serious and major risk factor for community-acquired urinary

tract infection and health care-associated urinary tract(14) infection namely catheter-associated urinary tract infection(15) and post-renal transplant recurrent urinary tract infection.(16)

Multi-drug resistant microbes are a frequent threat in urinary tract infections in diabetes, including multidrug resistant Enterobacteriaceae being extended-spectrum βlactamase positive(17), fluoroquinolone resistant(18), carbapenem resistant (19) and vancomycin resistant(20) uropathogens. Candida is also a very common troublesome cause of fungal urinary tract infections in diabetes mellitus.(21)

Patients with diabetes mellitus are increasingly susceptible to urinary tract infections and diabetes mellitus itself is on a rise globally, instigating a considerable burden on medical expenses(22) as it is linked with worse prognosis of urinary tract infections, including prolonged hospital inpatient care and significantly increasing mortality. Adding to the burden are the astronomical antibiotic prescribing rates and administration, including broad spectrum antibiotic drugs and unconventional higher antibiotics, ultimately giving rise to the menace of multidrug resistant microorganisms as a bitter consequence.(23)

Therefore, this study was strategized to comprehend the clinical and microbiological profile of urinary tract infections in diabetics exploring the spectrum of causative microorganisms and their antibiotic sensitivity patterns.

OBJECTIVES

I. To study the clinical patterns of Urinary Tract Infections in Type 2 Diabetes Mellitus

II. To study various risk factors associated with Urinary Tract Infections in Type 2 Diabetes Mellitus

III. To study Causative Microorganisms in Urinary Tract Infections with Type 2 Diabetes Mellitus and their Antibiotic sensitivity pattern.

REVIEW OF LITERATURE

INFECTIONS IN DIABETES MELLITUS

Diabetics are highly susceptible to infections and are often recognized to be frequent causes of morbidity and mortality. Infections are associated with impaired carbohydrate tolerance contributing to a significant percentage of deaths associated with ketoacidosis or inappropriate immunological function resulting in sepsis. Despite appropriate antimicrobial chemotherapy and surgical intervention, mortality rates are high with certain infections including Mucormycosis, necrotizing pneumonias, malignant external otitis, Gram negative or Staphylococcal septicemia, hepatobiliary infection, foot infection and urinary tract infections.(24)

WHY ARE DIABETICS PRONE TO INFECTIONS?

The human body makes use of its complex state of the art defence mechanisms to shield itself from invasion by the plethora of microbial pathogens.

In the presence of normal human metabolism and homeostasis, this defence system mounts an appropriate attack on most pathogens, arresting their growth and multiplication, hence reducing invasion and infection.

However there exist certain conditions where this defence system is attenuated by metabolic disorders including our focus of interest, diabetes mellitus.

Diabetes Mellitus disrupts the immunity of the human body in a multitude of ways mainly due to insulin deficiency and hyperglycaemia. Both humoral and cellular immunity are affected along with a threat to natural barrier as a result of diabetic neuropathy.

In accordance with the American Diabetes Association, infections are an imperative concern for individuals with diabetes due to the immunity's incompetence to ward off invading uropathogens.(25)

The mechanisms by which diabetes affects immune system include suppression of cytokine manufacture, phagocytotic defects, immune cells incompetence, and failure to terminate microbes.

Cytokine production Impairment

An in-vitro study by Mooradian AD et al, established that a smaller amount interleukin 1 beta (IL-1β) was secreted by humoral mononuclear cells and monocytic cells of diabetics as compared to non-diabetics after provocation with lipopolysaccharides.(26)

In another study by Ohno Y et al, monocytes of diabetics secreted smaller amounts of IL-1 and IL-6 compared to non-diabetic controls.(27)

Mononuclear cells collected from non-diabetics that were made to interact with anti-CD3 antibodies and exposed to hyperglycaemia showed suppression of IL-2, 6 and 10 productions.(28)

IL-6 is crucial for adaptive immunity by provoking antibody production and effector T-cell maturation(29), research have discovered that embarrassment of those cytokines in chronic elevation of blood sugars may weaken the immunological mediated response mounted against invasive pathogens.

A study piloted by Spindler et al claims mononuclear cells harvested from non-diabetic subjects and provoked with dextrose octreotide showed lowered IL-6 and 17A levels, especially in CD14+ and 16+ monocytes, indicating hampered immune mediated response due to hyperglycaemia.(30)

A study by Tan et al. showcased lower secretion of IL-12 along with IFNγ in mononuclear cells derived from diabetic subjects following Burkholderia pseudomallei infection compared non-diabetics. In addition to the above findings, intracellular bacterial counts were higher in mononuclear cells of diabetics linked to non-diabetics, implying hyperglycemia attenuates immunity against invading bacteria. Exogenous administration of recombinant IL-12 along with IFNγ remarkably dropped bacterial levels in mononuclear cells of diabetics, implying that hampered secretion of IL-12 along with IFNγ in diabetes handicaps capacity to limit bacterial proliferation during infection. Hence, chronic hyperglycemia associated with diabetics is believed to hamper macrophage and other leukocyte activity in eliminating pathogens.(31)

A study by Tessaro et al on the effect of insulin deficiency on immunological response verified that the insulin administration into bone marrow sourced macrophages isolated from mice with diabetes surged the production of TNF-α along with IL-6 associated with LPS interaction.(32)

Another research by Ferracini M et al making use of rats demonstrated that a short supply of insulin resulted in an impaired function of phagocytosis by alveolar macrophages as well as cytokine manufacture, both of which were reinstated following intervention with insulin.(33)

Leucocyte Recruitment Inhibition

An in vivo study by Martinez et al using streptozotocin induced diabetes in mice infected using Klebsiella pneumoniae, demonstrated impaired mobilization of CD45+ leukocytes and CD8+ T-cells with hampered expression of cell adhesion molecules for instance E-selectin and intracellular adhesion molecule 1. Lower levels of granulocytes were seen in the alveolar cavity of the mice with diabetes along with

hampered cytokine manufacture such as CXCL1, CXCL2, IL-1β, and TNF-α in lung parenchymal specimens.(34)

Defects in Pathogen Recognition

An in vivo study by Martinez et al using streptozotocin induced diabetes in mice infected using Klebsiella pneumoniae described that activation of Toll-like receptor 2 and Toll/IL-1R containing adaptor protein, which play a vital job in pathogen recognition, was hampered.(34)

A study by Gupta et al. showcased that TLR activation was hampered in diabetic subjects with complications and unsatisfactory glycemic control but higher in patients with satisfactorily controlled blood sugars without complications.(35)

Neutrophil Dysfunction

In a study by Chao WC, reactive oxygen species production of isolated neutrophils from diabetic tuberculosis patients following phorbol 12-myristate 13-acetate stimulation was impaired and was associated with increased levels of resistin in the serum of diabetics.(36)

Comparatively, in a study done by Perner et al. described hampering of superoxide in extracted neutrophils from non-diabetics when subjected to a medium with high sugar concentration. This impairment was a result of G6PD inhibition, which interfered with the synthesis of nicotinamide adenine dinucleotide phosphate.(37)

In a study by Stegenga et al. high blood sugars was induced in the serum of non-diabetic study population and then exposed to bacteria sourced cell wall components; the blood exhibited a significantly hampered neutrophil degranulation.(38)

Similarly, a study by Joshi et al. claimed that neutrophil performance to sufficient enough manufacture neutrophil extracellular traps was depressed as a result of hyperglycemia, leading to infection susceptibility. (39)

Macrophage Dysfunction

In a study by Restrepo et al. phagocytosis impairment was demonstrated due to defects in complement receptors and Fcγ receptors on isolated monocytes as a consequence of chronic hyperglycaemia.(40)

In an in-vitro by Pavlou S et al, using macrophages derived from mice bone marrow and peritoneum, exposed to high glucose showed diminished antibacterial activity and phagocytosis. This could be asserted to the reduced glycolytic capacity and reserve of macrophages following long-term sensitization to hyperglycaemia.(41)

Natural Killer Cell Dysfunction

In a study by Berrou et al, abnormalities were demonstrated in isolated natural killer cells from diabetics involving anomalies in Natural killer cell mobilizing receptors NKG2D and NKp46, resulting in impaired degranulation of natural killer cells.(42)

Antibody Inhibition and Complement Effector Impairment

In a rat-based animal study performed by Clifford et al, it was observed that chronic hyperglycaemia was associated with decreased C4-fragment opsonization responsible for inhibiting compliment activation by classic or lectin pathway, therefore resulting in dysfunctional complement activation.(43)

URINARY TRACT INFECTIONS IN DIABETICS

Diabetics are more inclined to all types of infection of the urinary tract.

In an observational study by Ishan Hirji et al of all diabetic patients in the United Kingdon research database observed that the incidence of urinary tract infection was 46.9 in 1,000 person-years among diabetics and 29.9 for non-diabetics.(44)

In a cohort study by Hammar N et al of more than six thousand patients recruited in 10 clinical trials showcased an incidence of 91.5 in 1,000 person-years in females and 28 in 1,000 person-years in males, and a combined incidence of 2% in 6 months.(45)

A study by Yu S et al involving more than 70,000 diabetics observed that 8.2% were detected to have urinary tract infection during 1 year (12.9% females and 3.9% males, with incidence rates surging with age in years).(22)

In a study by Alex Z Fu found that urinary tract infection was more observed in diabetic males and females than in non-diabetics (9.4% and 5.7% in males and females respectively) in the study population of 89,790 variable matched pairs of diabetics and non-diabetics.(46)

Asymptomatic bacteriuria is rampant in females, owing to a urethra that is shorter in length which is in close proximity to vulvar, and perianal regions that are warm, moist and densely colonized with enteric microbes. Asymptomatic bacteriuria surges with age, and is linked with abnormalities of urinary tract especially foreign objects like urinary catheters, stents, prostheses etc.(47)

A metanalytical compilation of twenty-two studies, discovered a point prevalence of 12.2% of Asymptomatic bacteriuria among diabetics and 4.5% in non-diabetics. Prevalence of asymptomatic bacteriuria was higher significantly in both diabetic females and males, higher significantly in diabetics with a prolonged diabetic

duration and not linked with glycemic status, represented by glycosylated hemoglobin.(48)

A prospective study by Srinivas M Aswani et al found a 30% prevalence of asymptomatic bacteriuria among diabetic inpatients.(49)

In a case control study by Delia Scholes et al, pyelonephritis was observed to be four times more common in diabetic females than in females without diabetes.(50)

In a study by L E Nicolle et al, women with diabetes mellitus were 6–15 times more often admitted for acute pyelonephritis than women without diabetes mellitus and diabetic males were admitted 3.4–17 times more than males without diabetes mellitus.(51)

In a study by T Benfield et al claimed diabetic patients were three times more probable to be admitted with pyelonephritis versus non-diabetes.(52)

Pathogenesis and risk factors of urinary tract infection in patients of diabetes mellitus

There are many reasons as to why diabetics are more susceptible to urinary tract infections.

The growth of uropathogens may be promoted by the abnormally existing glucose concentration in urine.(53)

A study by Edward J Boyko et al did not find a correlation between level of HbA1c, and risk of infection of urinary tract among diabetics. Furthermore, SGLT2 inhibitor induced glycosuria, was not documented to increase the risk of infections of the urinary tract.(54)

Pyelonephritis and other kidney related complications such as emphysematous pyelonephritis, are frequently encountered in diabetics as the development and proliferation of uropathogens are favored by the environment of high renal

parenchymal glucose. Lesser urinary IL-6 and -8 levels were documented in diabetics with asymptomatic bacteriuria, as compared to non-diabetics with asymptomatic bacteriuria.(55)

In a study by S A Kaplan et al, dysfunctional voiding and urinary retention were observed as a result of autonomic neuropathy urinary bladder and outflow tract consequently leading to decreased physical bacterial drainage via micturition, thereby facilitating bacterial proliferation.(56)

A study by Khalid A Al-Rubeaan et al, observed female gender, hypertension, insulin therapy, body mass index >30 kg/m2 and nephropathy are risk factors associated with an increased risk of urinary tract infection in diabetics.(57)

SGLT2 inhibitors theoretically can lead to urinary tract infections due to iatrogenic glycosuria.(58) However, a study by Xu-Ping Yang et al observed similar incidences of urinary tract infection in diabetics treated with canagliflozin versus diabetics not on SGLT2 inhibitors.(59)

In a study by Agata Ptaszynska et al, urinary tract infection was observed slightly higher in diabetics taking Dapagliflozin.(60)

Uropathogens

The more prevalent uropathogens isolated from urine cultures of diabetics with urinary tract infections are Escherichia coli and other various Enterobacteriaceae for example Klebsiella spp., Proteus spp., Enterobacter spp., and Enterococci.(61) Multidrug resistant microbes are a frequent threat in infections of the urinary tract in diabetics, including multidrug resistant Enterobacteriaceae being extended spectrum β-lactamase positive(17), fluoroquinolone resistant(18), carbapenem resistant (19) and vancomycin resistant(20) uropathogens. Candida is also a very common troublesome cause of fungal infections of the urinary tract in diabetes mellitus.(21)

DIAGNOSIS

Any diabetic presenting with increased frequency, urgency, burning micturition and suprapubic pain should be a suspect for infection of the lower urinary tract and any diabetic presenting with the above symptoms accompanied by costovertebral angle pain or tenderness, fever, and chills are suspects for upper urinary tract infection. However, a handful of patients of diabetic neuropathy can have misleading clinical symptoms and signs.

A study by Yeonjae Kim et al, on patients diagnosed to have acute onset pyelonephritis observed that remarkably lesser of the diabetics presented with stereotypical symptoms and signs of upper urinary tract infection such as pain in the flank, tenderness in the costovertebral angle, and symptoms of infection of the lower urinary tract versus non-diabetics.(62)

Diabetics with urinary tract infection may present to the ER with hypoglycemia, hyperglycemia, hyperosmolar hyperglycemic state or diabetic ketoacidosis, warranting an urgent workup for source of infection.(63)

To rule in or rule out urinary tract infection in diabetics, a clean catch midstream urine sample must be obtained and subjected to microscopy to look for the presence of WBCs. A significant number of WBCs indicated urinary tract infection.(64)

The gold standard test to look for urinary tract infection is microscopy which is demarcated as greater than or equal to 10 leukocytes/mm3. Other tests include leukocyte esterase test by dipstick method (sensitivity of 75%–96% and specificity of 94%–98%). Presence of bacteria in the absence of WBCs indicated colonization not infection.(65)

Bacteria are visualized by microscopy. A dipstick can be utilized to detect existence of nitrates in urine sample. Positive dipstick demonstrates the existence of microbes in the urine sample. Negative on dipstick demonstrates a low count of bacteria or a species of bacterial unable to metabolize nitrate to nitrite. Hematuria and proteinuria can also be detected.(66)

Before administering antibiotics, a clean catch midstream urine culture should be sent in all diabetics with suspected urinary tract infection.(67)

A urine sample for culture can be gained through a sterile Foley's catheter introduced by strict aseptic method, or by aspirating urine suprapubically in patients with altered sensorium, neurological or urological deficits that renders them unable to void on their own. For patients having long standing indwelling catheters, the ideal method of procuring a urine sample for culture is changing the catheter and extracting urine from the newly inserted Foley's catheter, due to development of biofilm on the old Foley's catheter.(68)

In female population, a clean catch midstream urine sample with a uropathogens count $\geq 10^5$ cfu/mL is diagnostic of urinary tract infection such as cystitis or pyelonephritis. However, in female population with satisfactory glycemic control without diabetes related complications, diagnosed with acute cystitis which is uncomplicated, quantitative counts less than 10^5 cfu/mL are obtained from around 25% and 10% of premenopausal women and postmenopausal women respectively. Lower quantitative counts are seen in around 5% of patients with acute onset pyelonephritis.(67)

A clean catch midstream urine routine showing urine colony count of greater than or equal to 10^4 cfu/mL is diagnostic of urinary tract infection in male population.

Lower colony counts are acceptable as significant bacteriuria when coliform uropathogens are isolated.(69)

A quantitative growth of $\geq 10^2$ cfu/mL is indicative of urinary tract infection in an in-and-out catheter urine sample when the patient is symptomatic.(70) A quantitative growth of $\geq 10^3$ cfu/mL is indicative of urinary tract infection in patients with indwelling catheters or intermittent catheterization.(68) Establishing the presence of asymptomatic bacteriuria can be defined with the evidence of a growth of $\geq 10^5$ cfu/mL of the same uropathogenic microbe (up to 2) in two consecutive clean catch urine samples, or greater than or equal to 10^2 cfu/mL in a urine sample procured via a sterile Foley's catheter, even if signs or symptoms of infection of the urinary tract are absent.(71) Pyuria can be present in up to 70% of diabetic females with asymptomatic bacteriuria. Hence, the presence of pyuria cannot differentiate between symptomatic or asymptomatic urinary tract infection.(72)

OUTCOMES AND COMPLICATIONS

In a study by Pertel PE et al, urinary tract infection has a worse outcome in diabetics when compared to non-diabetics.(73)

In a study by Kim Y et al, Diabetes mellitus was observed to be a factor of risk for early clinical deterioration post 72 hours of intensive antibiotic therapy in female study population with acute onset pyelonephritis.(62)

In a study by Kofteridis D et al, infections of the urinary tract in diabetics are linked with prolonged inpatient care, bacteremia, azotemia and septic shock. As per the above-mentioned study population, mortality due to urinary tract infection was 5 times higher in diabetics above 65 years versus control population.(13)

In a study by Gorter KJ et al, observed higher recurrence of infection of the urinary tract in diabetics compared to patients without diabetes.(74)

Almost 90% cases of emphysematous pyelonephritis(55) and 67% cases of emphysematous cystitis(75) occur in patients with diabetes mellitus.

A study by Kofteridis D et al, shows diabetics are more prone to renal, peri nephric abscesses, urosepsis and bacteremia.(13)

MANAGEMENT

Treatment of urinary tract infections in diabetics depends on symptoms, site of involvement, presence of urological abnormalities, systemic severity, metabolic complications and renal function.(67)

Asymptomatic bacteriuria is not an indication for treatment.(47) Even though previous studies have implied that presence of significant levels of bacteria may be associated with progression of illness to symptom associated urinary tract infection and with a deterioration of kidney function in diabetics(76), advanced studies in the present found that female diabetics with asymptomatic bacteriuria don't have an accelerated deterioration of kidney function(77), and that there is no benefit in treating a non-pregnant diabetics for asymptomatic bacteriuria.(72)

Acute cystitis in female population with good glycemic control and absent diabetic complications can be considered to be uncomplicated lower urinary tract infection(67), and empirically treated with one among the following: nitrofurantoin 100 mg three times daily for 5 days, Fosfomycin 3 g single dose, or cotrimoxazole 960 mg twice daily for 3 days. For all patients a culture sensitivity must be sent, and antibiotic therapy tailored accordingly.

All other cases of infection of the lower urinary tract in diabetics are most often deemed to be complicated lower urinary tract infection and ought to be treated

with antibiotics. If a patient has an indwelling catheter, urinary tract infection warrants a catheter change.(68)

Empirical antibiotic treatment isn't easy in urinary tract infection in diabetics as there are a plethora of unconventional infecting organisms and probability of multi drug resistant uropathogens.(78)

Diabetics presenting with pyelonephritis can be treated with antibiotics orally if they present with mild–moderate symptoms,(79) provided they have no alterations or impairments in enteric luminal absorption, problems of gastric emptying or chronic diarrhea as a result of diabetic induced neuropathy.

However, diabetics presenting with severe debilitating symptoms, circulatory collapse, metabolic abnormalities, or symptoms that hamper patient's ability to ingest oral antibiotics such as nausea, vomiting etc. should be admitted for immediate parenteral antibiotics.(79)

Pharmacotherapy with empirical antibiotic chemotherapy, using broad spectrum cephalosporins, fluoroquinolones, aminoglycosides, piperacillin–tazobactam, or carbapenems ought to be introduced as hit hard and hit fast rule.(79)
Diabetics manifesting as urinary tract infection with morbid sepsis or those documented to harbor multiple drug resistant microbes or those who have received a plethora of antibiotics must receive broad spectrum treatment, directed by urinary cultures. As soon as the culture reports are available, antibiotics need to be changed accordingly. According to a study by Geerlings SE, the duration of antibiotic administration should be extended. However, there are no randomized control trials to support this claim. (80)

Nephrectomy or open drainage with a course of antibiotic chemotherapy was the treatment of choice for emphysematous pyelonephritis. However, in a recent

report by Lin WR et al, there have been good results with systemic antibiotics administered with extraction of purulent material along with gaseous contents with percutaneous catheter and relieving any urinary tract obstruction if present.(81)

There are certain antibiotics which impair glucose homeostasis and some have drug interactions with antidiabetic agents, this ought to be carefully looked for and appropriate antibiotic stewardship done.(82)

Renal dose adjustment is compulsory in diabetics with renal impairment for certain antibiotics. Aminoglycosides are to be used cautiously as they are nephrotoxic. Nitrofurantoin ought to be avoided in kidney failure because they lead to peripheral neuropathy due to accumulation.(83)

Recurrent episodes of infection of urinary tract in diabetics are managed quite in the same manner as to non-diabetics.(67)

In young diabetic women not having diabetic complications, prophylaxis with post sexual intercourse or regular low dose antibiotics may be prescribed.(79)

Intermittent catheterization is favored over a long-standing indwelling catheter in patients needing catheterization due to impaired voiding.(68)

METHODOLOGY

Study site: This study was conducted in Department of General Medicine at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre Belagavi.

Study population

Diabetic patients with urinary tract infection admitted in Department of General Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre.

Study design: Longitudinal study

Sample size: 100

Sampling method:

All the eligible subjects were recruited into the study consecutively by convenient sampling till the sample size was reached.

Study Duration

The data collection for the study was done between January 2020 to December 2020 for a period of one year.

Inclusion Criteria:

I. Patients of Type 2 Diabetes Mellitus, Adults above the age of 18 years, irrespective of Duration of Diabetes, Treatment Taken and Adherence

II. Patients with Clinical, Laboratory and Microbiological features of Urinary Tract Infection

Exclusion Criteria:

I. History of receiving antibiotics within two weeks prior to culture

II. Patients on continuous indwelling catheter

III. Menstruating women

Ethical Consideration

Study was approved by institutional human ethics committee. Informed written consent was obtained from all the study participants and only those participants who signed the informed consent were included in the study. The risks and benefits involved in the study and voluntary nature of participation were explained to the participants before obtaining consent. Confidentiality of study participants was maintained.

Data collection tool

All the relevant parameters were documented in a structured study proforma.

Methodology

All patients fulfilling the inclusion criteria and willing to participate were included in this study. A detailed history was taken after taking consent from the patient, with special reference to duration of diabetes, treatment taken and adherence, symptoms related to diabetes and its complications. History in relation to Urinary tract infection like burning micturition, frequency, urgency, dysuria, suprapubic pain, haematuria and any symptoms suggestive of acute pyelonephritis like fever, chills, nausea, vomiting was noted.

Past history of urinary tract instrumentation or catheterization was asked. A detailed examination of all systems with special emphasis on temperature, pulse rate, blood pressure, suprapubic tenderness and costovertebral angle tenderness was done. Blood samples were drawn for necessary investigations. Collection of mid-stream clean catch urine specimen was performed and urine was sent for routine and microscopic examination. Ultrasound abdomen and pelvis with special emphasis on kidney, ureter and bladder was performed on all patients to describe the type of urinary tract infection.

Statistical Methods

Descriptive analysis was carried out by mean and standard deviation for quantitative variables, frequency and proportion for categorical variables. Data was also represented using appropriate diagrams like bar diagram, pie diagram and box plots. Chis square test was used to assess the statistical significance of the associations. P value < 0.05 was considered statistically significant. IBM SPSS statistical software version 21 was used for data analysis.

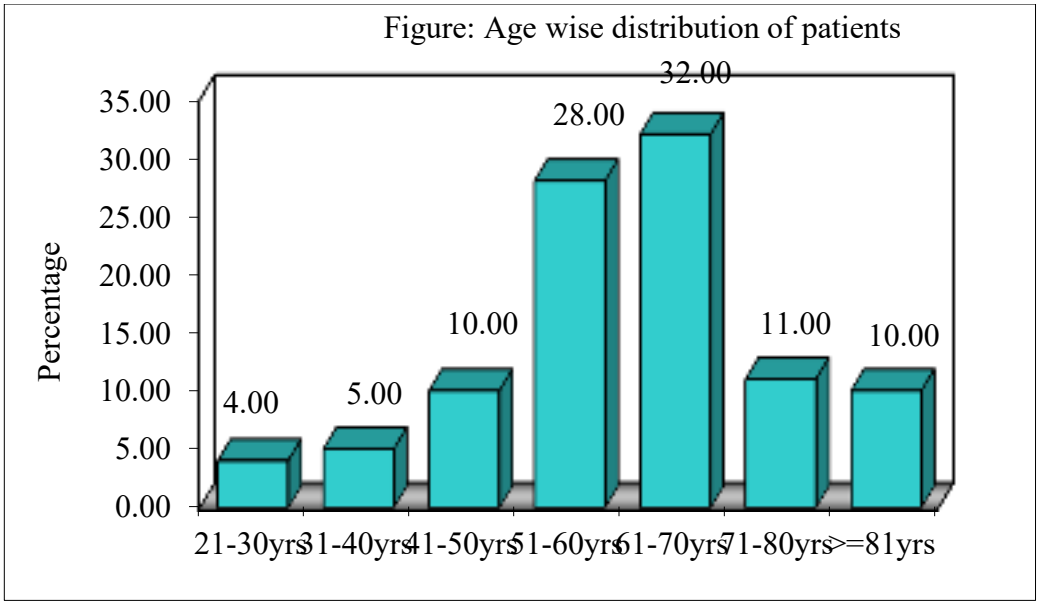
RESULTS

The present one-year longitudinal study titled **'A CLINICAL PROFILE OF URINARY TRACT INFECTION IN TYPE 2 DIABETES MELLITUS WITH BACTERIAL CULTURE AND ANTIBIOTIC SENSITIVITY PATTERNS'** was carried out in the Department of General Medicine, KLES Prabhakar Kore Hospital and Research Centre, Belagavi. During the study period from January 2020 to December 2020, a total of 100 patients of infection of the urinary tract in Type 2 diabetes mellitus were studied. The findings, observations and final results are tabulated below.

Table 1: Age wise distribution of patients

Age groups	No of patients (n=100)	% of patients
21-30yrs	4	4.00
31-40yrs	5	5.00
41-50yrs	10	10.00
51-60yrs	28	28.00
61-70yrs	32	32.00
71-80yrs	11	11.00
>=81yrs	10	10.00
Total	100	100.00
Mean ± SD	61.52±14.17	

Graph 1: Age wise distribution of patients

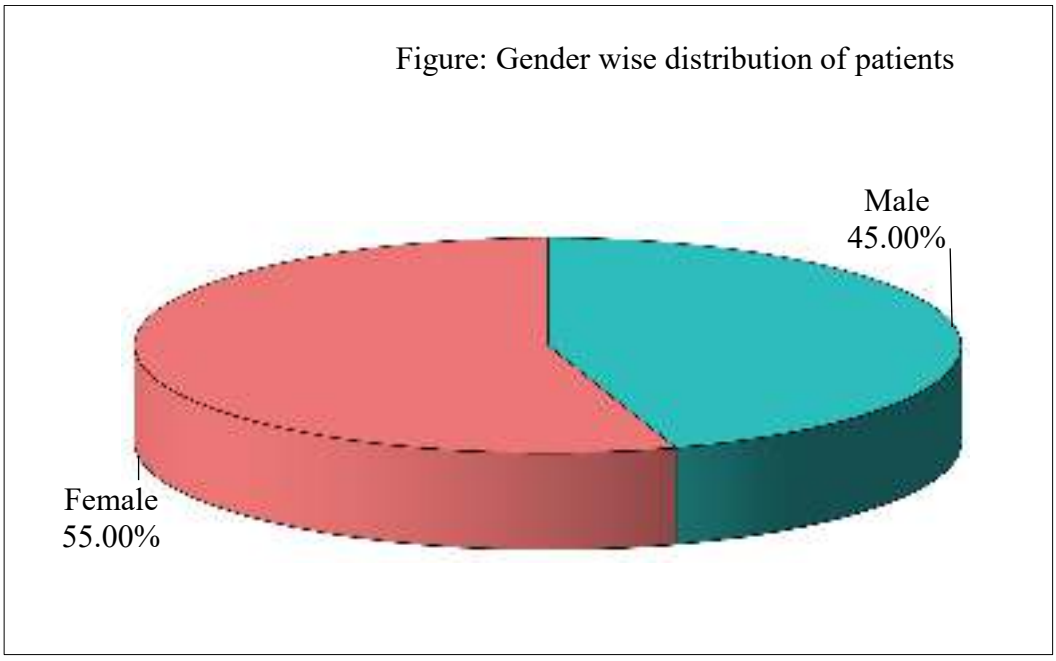


In our present study of 100 patients, patient's age ranges from 18-98 years, the youngest patient was 21 years and oldest was 98 years old. Maximum number of patients were in age range of 61- 70 years i.e., 32 patients (32%), 51-60 years- 28 patients (28%), 11 patients (11%) in 71-80%, 10 patients (10%) in > 81 years, 10 patients (10%) in 41-50 years and 9 patients (9%) between the age of 21-40 years.

Table 2: Gender distribution of patients

Gender	No of patients(n=100)	% of patients
Male	45	45.00
Female	55	55.00
Total	100	100.00

Graph 2: Gender distribution of patients

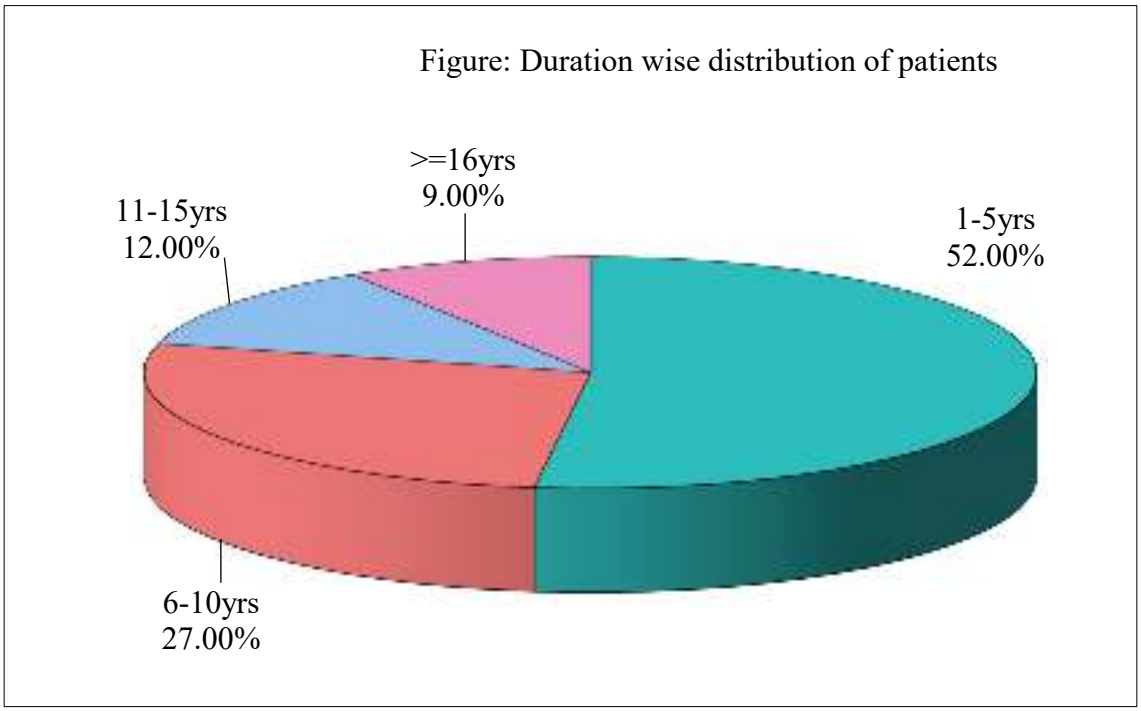


Out of 100 patients, females were 55 (55%) and males were 45 (45%) with a *slight female preponderance*, accounting a ratio of M:F :: 1.2:1.

Table 3: Duration of Type 2 Diabetes Mellitus

Duration (in yrs.)	No of patients (n=100)	% of patients
1-5yrs	52	52.00
6-10yrs	27	27.00
11-15yrs	12	12.00
>=16yrs	9	9.00
Total	100	100.00
Mean ± SD	24.89±7.02	

Graph 3: Duration of Type 2 Diabetes Mellitus

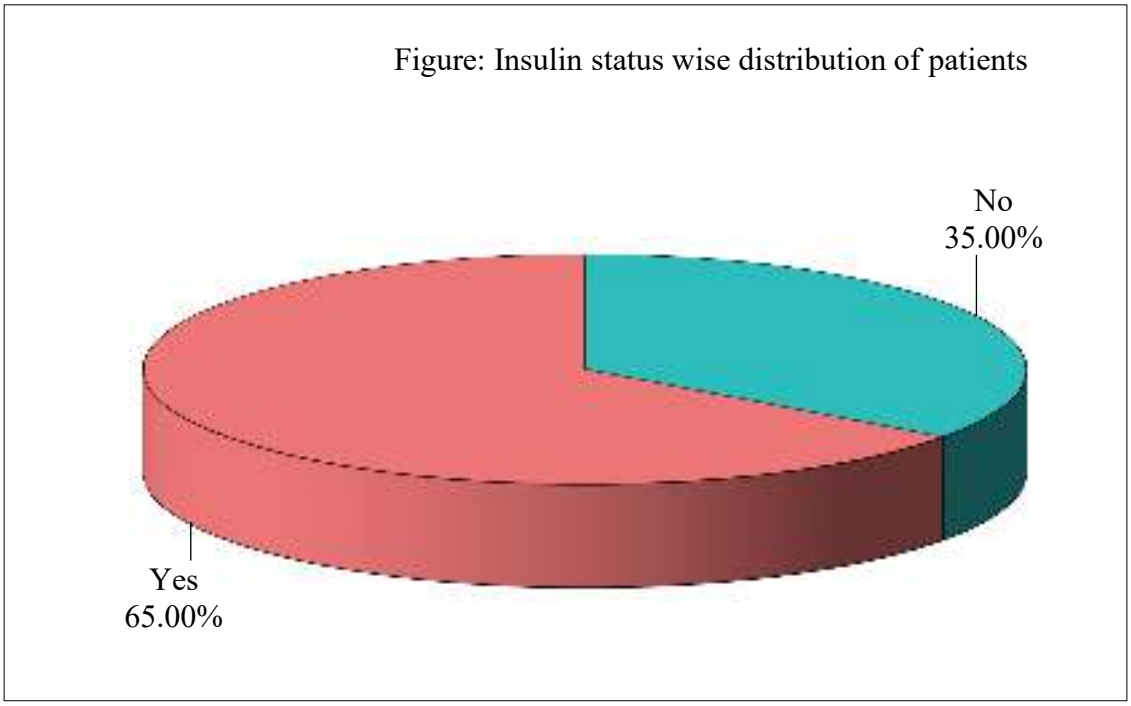


We observed maximum number of patients i.e., 52 patients (52%) had a type 2 diabetes mellitus duration of 1- 5 years, 27 patients (27%) duration of 6-10 years, 12 patients (12%) 11-15 years, and >15 years - 9 patients (9%) with a mean ± SD = 24.89 ± 7.02.

Table 4: Distribution of patients based on treatment with INSULIN.

Treatment with Insulin	No of patients (n=100)	% of patients
Yes	65	65.00
No	35	35.00
Total	100	100.00

Graph 4: Distribution of patients based on treatment with INSULIN.

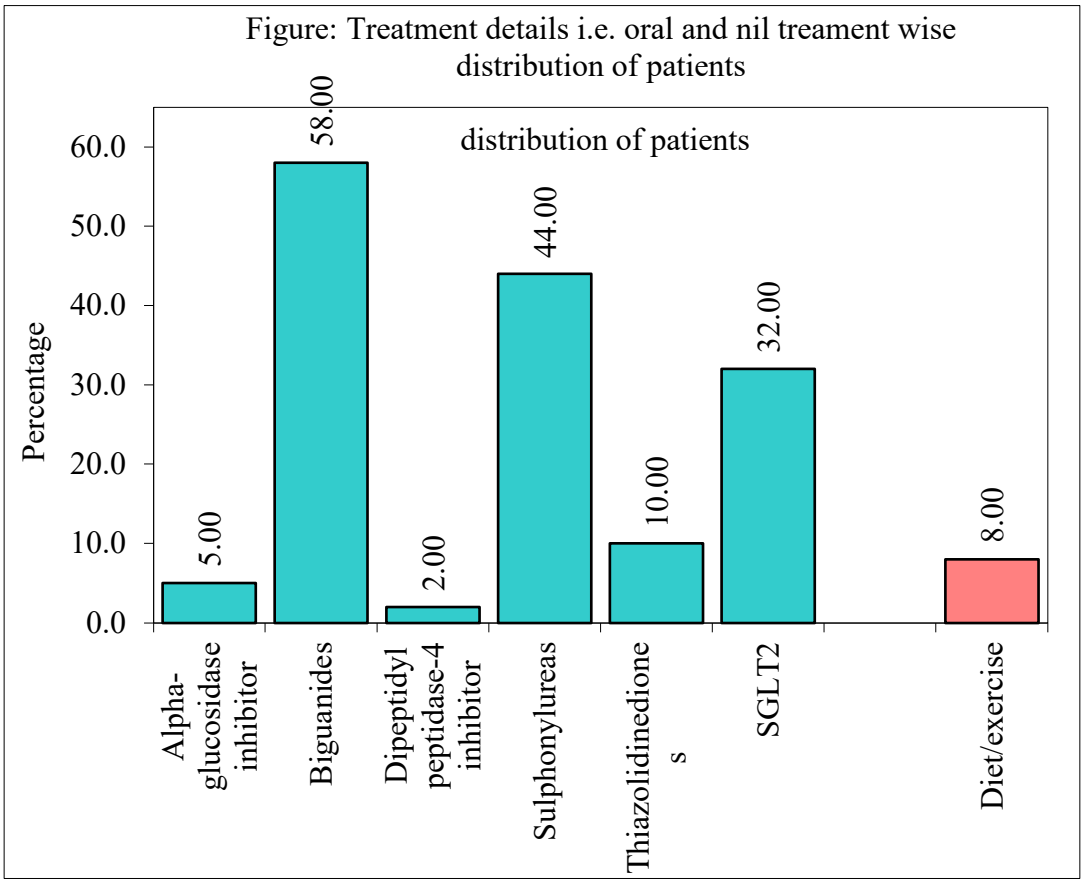


In our present study of 100 patients, 65 patients (65%) were on some or the form of Insulin treatment for their Type 2 diabetes mellitus status. Remaining 35 patients (35%), of which 8 patients (8%) were not on any treatment i.e., either Oral Hypoglycemic Agent or Insulin and 27 patients (27%) were on one of the other classes of Oral Hypoglycemic Agent for their diabetes mellitus treatment.

Table 5: Distribution of patients based on treatment with Oral Hypoglycemic Agents.

Treatment details	No of patients (n=100)	% of patients
Oral Hypoglycemic Agents		
Biguanides	58	58.00
Sulphonylureas	44	44.00
SGLT2 inhibitor	32	32.00
Thiazolidinediones	10	10.00
Alpha-glucosidase inhibitor	5	5.00
Dipeptidyl peptidase-4 inhibitor	2	2.00
Only Diet/exercise	8	8.00
Total	100	100.00

Graph 5: Distribution of patients based on treatment with Oral Hypoglycemic Agents.



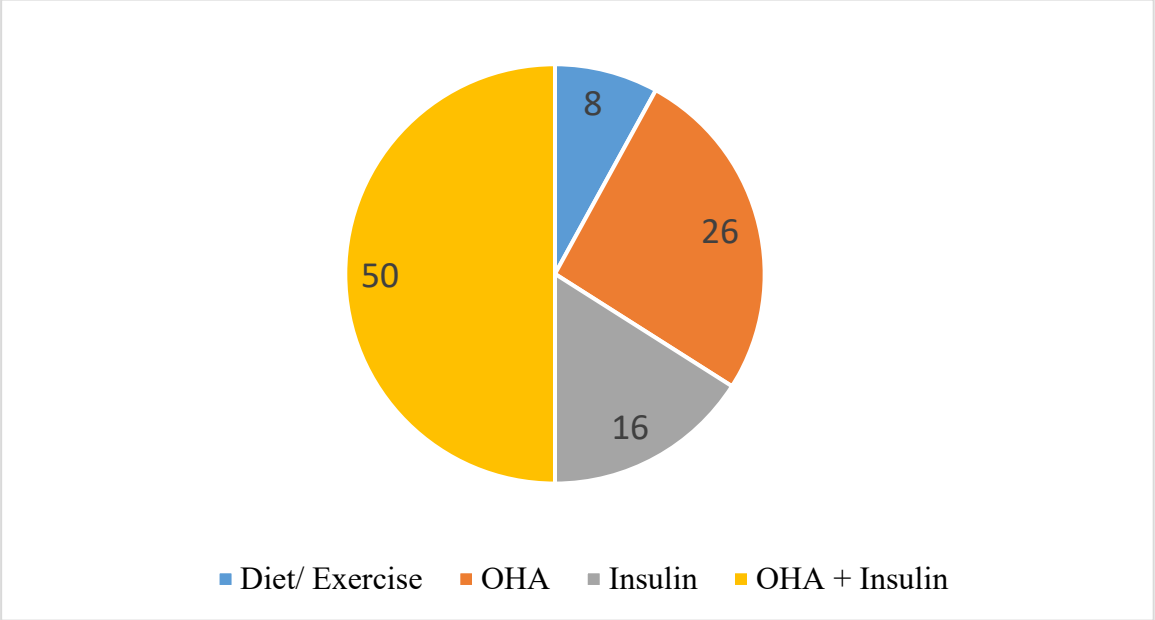
Majority of our patients were on Biguanides 58 patients (58%), Sulphonylureas 44 patients (44%), SGLT2 inhibitors 32 patients (32%), Thiazolidinediones 10 patients (10%), Alpha-glucosidase inhibitors 5 patients (5%) and Dipeptidyl peptidase-4 inhibitors 2 patients (2%).

However, 8 patients (8%) who were not on any treatment were following only diet and exercise for their management of diabetes mellitus.

Table 6: Distribution of patients based on type of treatment for diabetes mellitus.

Treatment details	Number of patients (n=100)	Percentage
OHA + Insulin	50	50.00
OHA only	26	26.00
Insulin only	16	16.00
Diet/ exercise	8	8.00

Graph 6: Distribution of patients based on type of treatment for diabetes mellitus.

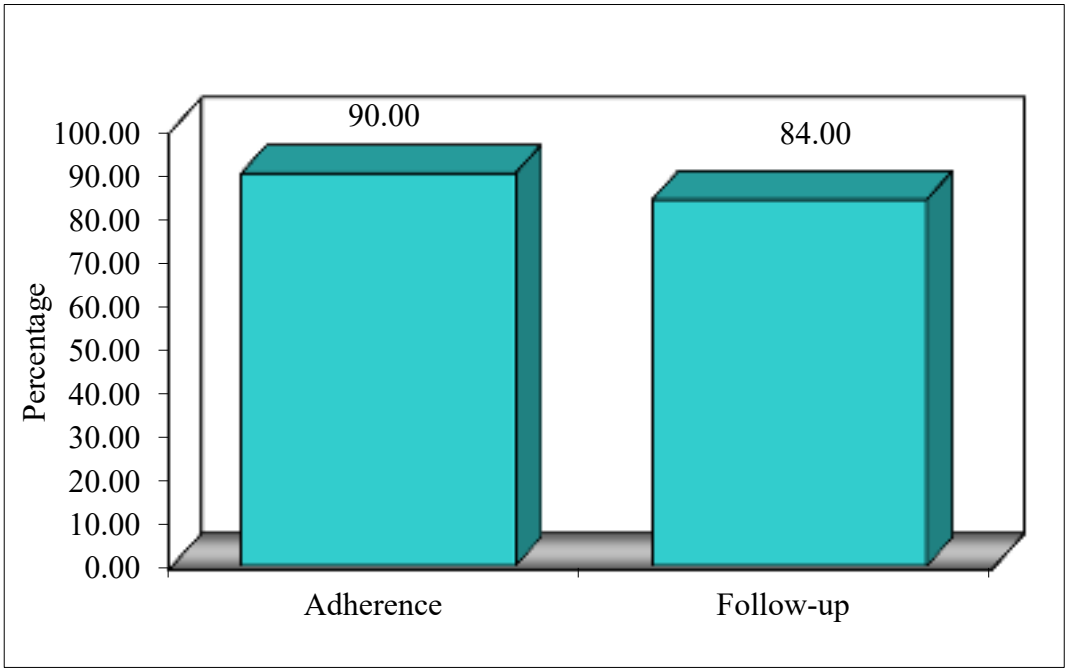


The Table 6 and Graph 6 depicts 50 patients (50%) were on treatment with both Insulin and Oral Hypoglycemic Agents, remaining 50 patients of which 42 patients (42%) were on either Insulin alone or Oral Hypoglycemic Agents alone, 8 patients (8%) were on diet plus exercise (No Insulin and No Oral Hypoglycemic Agent).

Table 7: Distribution of patients based on treatment compliance and follow up.

Treatment details	No of patients (n=100)	% of patients
Compliance		
Yes	90	90.00
No	10	10.00
Monthly Follow-up		
Yes	84	84.00
No	16	16.00
Total	100	100.00

Graph 7: Distribution of patients based on treatment compliance and follow up.



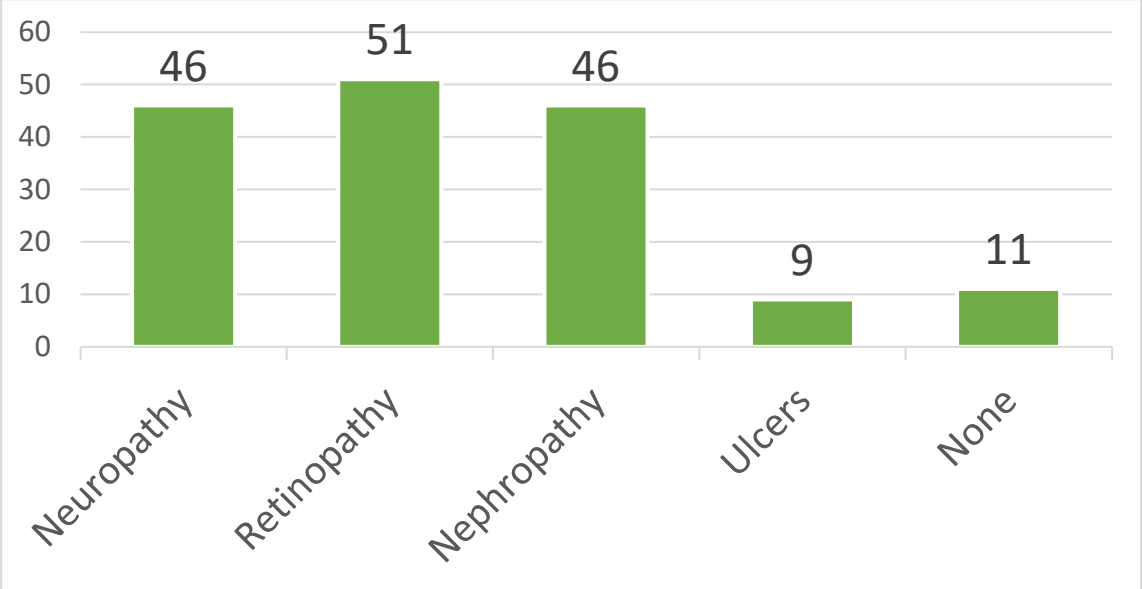
Amongst 100 patients, 90 patients (90%) were compliant to treatment (Insulin/ OHA). 10 patients (10%) were non-compliant.

84 patients (84%) were compliant to follow up, however 16 patients (16%) were non-compliant.

Table 8: Distribution of patients with complications of diabetes mellitus.

Complications	No of patients (n=100)	% of patients
Retinopathy	51	51.00
Neuropathy	46	46.00
Nephropathy	46	46.00
Diabetic Foot (Ulcers)	9	9.00

Graph 8: Distribution of patients with complications of diabetes mellitus.

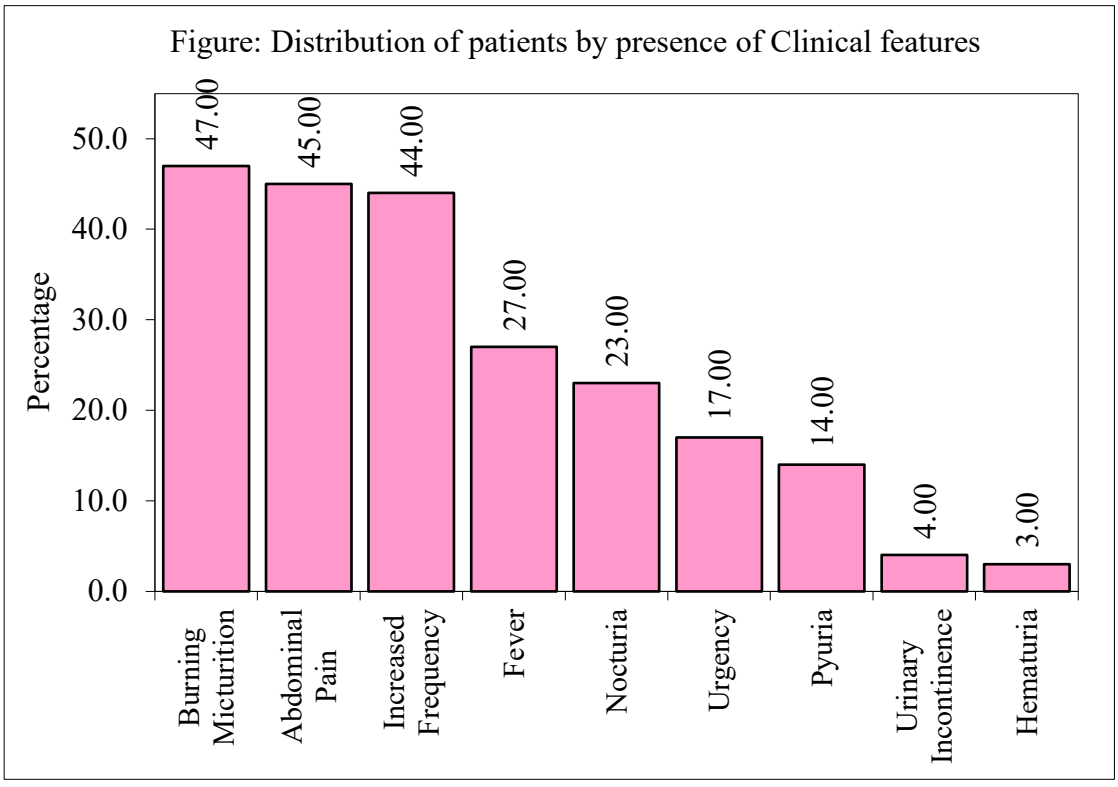


In our present study of 100 patients, majority (51 patients) had retinopathy as a complication of diabetes mellitus, neuropathy and nephropathy in 46 patients (46%) each, overlapping complications in 32 patients (32%) and diabetic foot in 9 patients (9%). Nearly 47 patients did not have any complications of diabetes mellitus.

Table 9: Clinical presentation of patients.

Clinical Features	No of patients (n=100)	% of patients
Burning Micturition	47	47.00
Abdominal Pain	45	45.00
Increased Frequency	44	44.00
Fever	27	27.00
Nocturia	23	23.00
Urgency	17	17.00
Pyuria	14	14.00
Urinary Incontinence	4	4.00
Haematuria	3	3.00
Asymptomatic	35	35.00

Graph 9: Clinical presentation of patients.



The clinical presentation of all 100 patients were observed and found the commonest symptom was burning micturition i.e., 47 patients (47%), abdominal pain 45 patients (45%), increased frequency 44 patients (44%), fever 27 patients (27%), nocturia 23 patients (23%), urgency 17 patients (17%), pyuria 14 patients (14%), urinary incontinence 4 patients (4%) and haematuria 3 (3%).
However, 35 patients (35%) were asymptomatic.

Table 10: Gender wise urinary tract infection.

Urinary Tract Infection	Male	Female	Total
Asymptomatic Bacteriuria	16	19	35
Cystitis	19	19	38
Pyelonephritis	10	17	27
	45	55	100

The above table depicts the rate of urinary tract infection was slightly more observed in female gender (n=36) and males (n=29), even asymptomatic infection was slightly more in female gender (n=19) as compared to their counterpart (n=16). Lab parameters

Table 11A: Urine Routine Analysis

	Present	Absent
Albumin	73	27
Sugar	86	14

Table 11B: Urine Microscopy

Number of Pus Cells	Number of patients (n=100)
<5	16
5-10	20
10-50	24
50-100	16
>100	20
Sheets	4
	100

All patients were subjected for urine routine analysis, Table 11A shows presence or absence of urine albumin and sugar and Table 11B shows microscopic findings.

Table 12A: Blood Glucose - FBS/PPBS and RBS.

RBS	>200	>300	>400
	25	27	48
FBS	<126	>126	
	54	46	
PPBS	<140	>140	
	15	85	

Table 12B: HbA1c

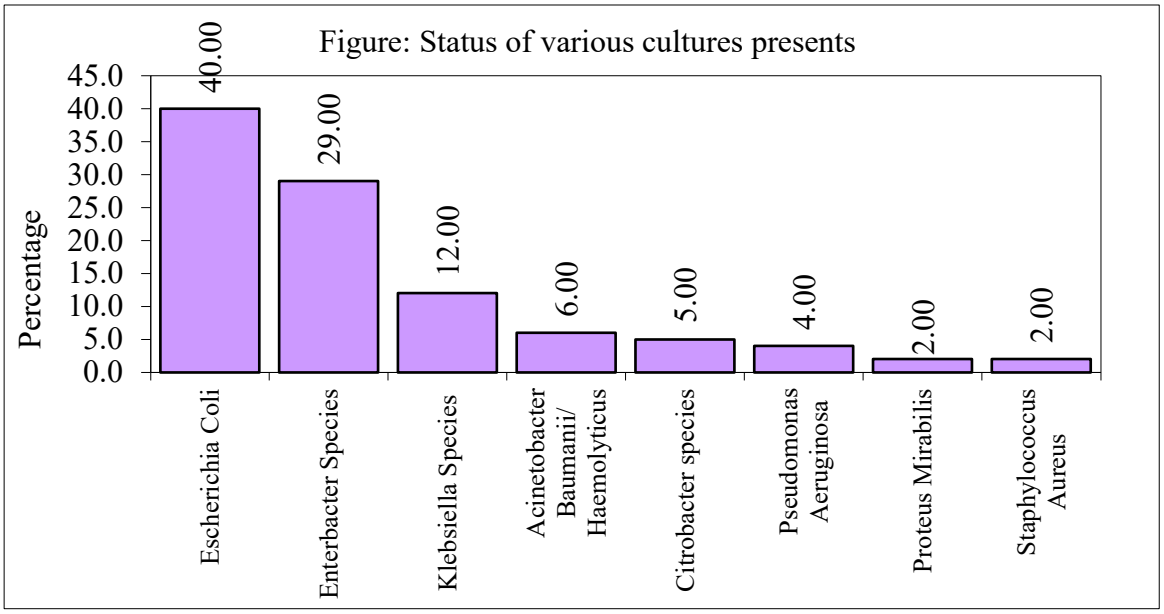
All patients were subjected to blood sugar estimation, at arrival RBS was done, later on, they were subjected to FBS/ PPBS on same day and HbA1c. The results obtained are shown in Table 12A and Table 12B.

Hba1c Levels	Number Of Patients (n=100)
6.5-7.5	41
7.5-8.5	20
>8.5	39

Table 13: Urine Culture

Culture	No of patients (n=100)	% of patients
Escherichia Coli	40	40.00
Enterobacter Species	29	5.00
Klebsiella Species	12	3.00
Acinetobacter Baumanii/ Haemolyticus	6	6.00
Citrobacter species	5	5.00
Pseudomonas Aeruginosa	4	4.00
Proteus Mirabilis	2	2.00
Staphylococcus Aureus	2	2.00
Total	100	100.00

Graph 10: Urine Culture

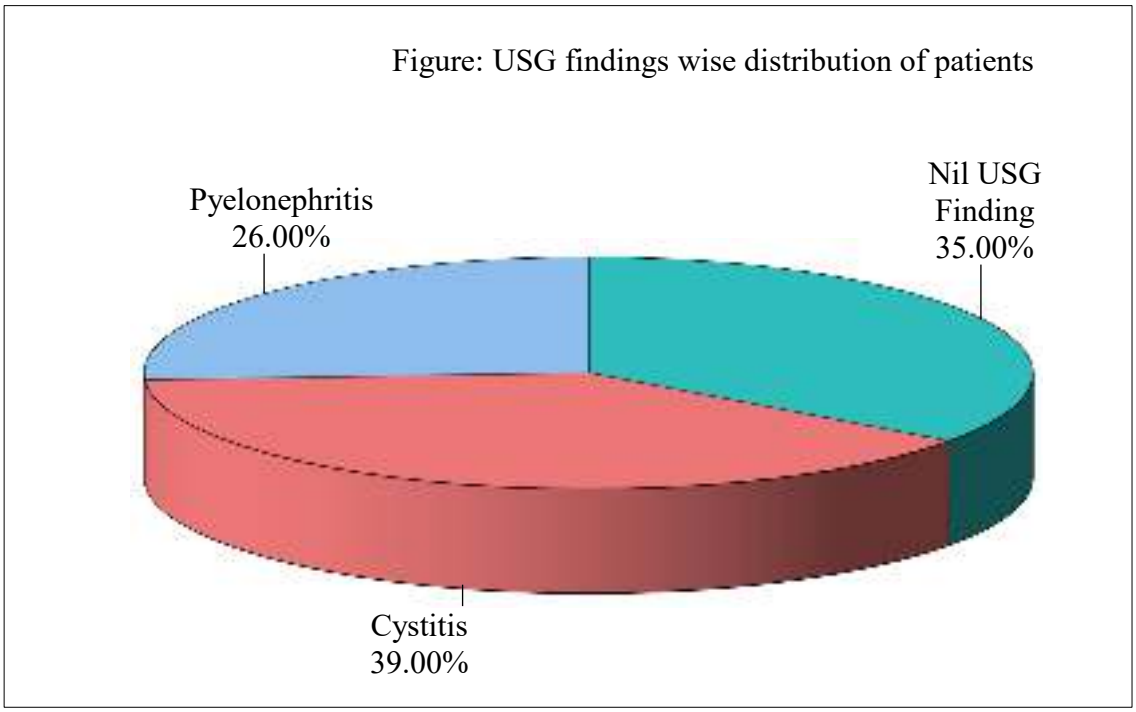


All our 100 patients were subjected to Urine Culture, most common organism isolated was *Escherichia Coli* in 40 patients (40%), 29 patients (29%) grew Enterobacter species including aerogenes, cloacae and faecalis. Klebsiella species including pneumoniae and oxytoca grew in the cultures of 12 patients (12%). Other isolated organisms are depicted in the above Table 13 and Graph 10.

Table 14: Ultrasound Abdomen and Pelvis including Kidney, Ureter and Bladder Study.

USG findings	No of patients (n=100)	% of patients
Nil USG finding	35	35.00
Cystitis	39	39.00
Pyelonephritis	26	26.00
Total	100	100.00

Graph 11: Ultrasound Abdomen and Pelvis including Kidney, Ureter and Bladder Study.



All patients were subjected to Ultrasound Abdomen and Pelvis with special reference to Kidney, Ureter and Bladder Study and were found to have cystitis in 39 patients (39%), pyelonephritis in 26 patients (26%) and remaining 35 patients (35%) scan was normal.

Drug Sensitivity Pattern of Organisms

Table 15A: Penicillin

Drugs	Resistance	Sensitive	Total
Amoxicillin/ Clavulanic Acid	72	28	100
Ampicillin	91	9	100
Piperacillin/ Tazobactam	73	27	100
Mezlocillin	97	3	100

Graph 12A: Penicillin

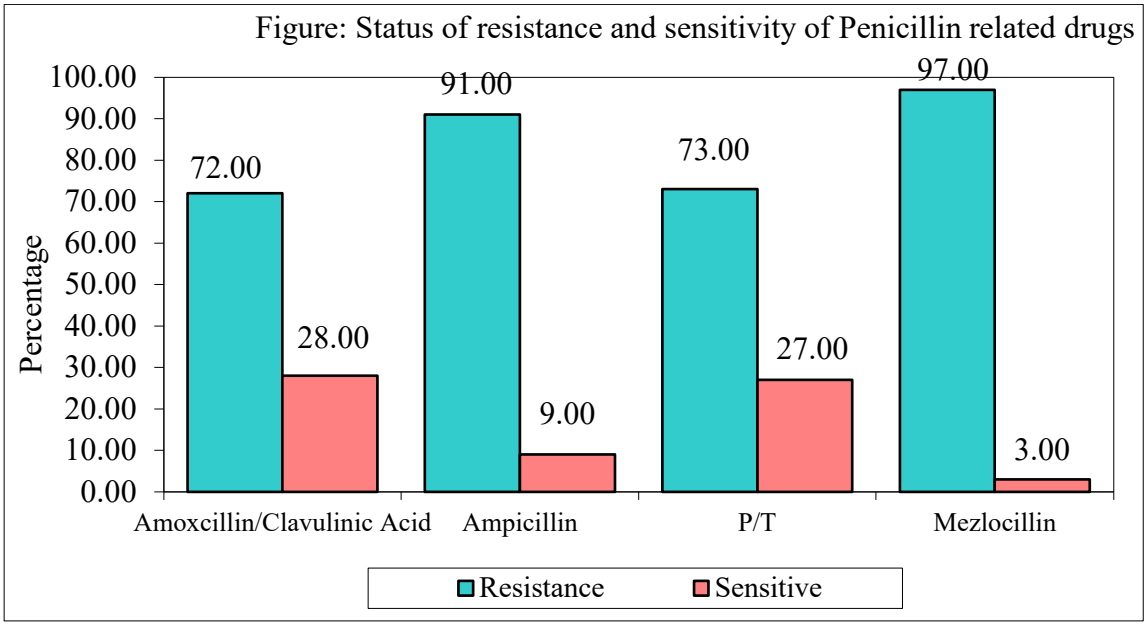


Table 15B: Carbapenem

Drugs	Resistance	Sensitive	Total
Ertapenem	71	29	100
Imipenem	63	37	100
Meropenem	67	33	100

Graph 12B: Carbapenem

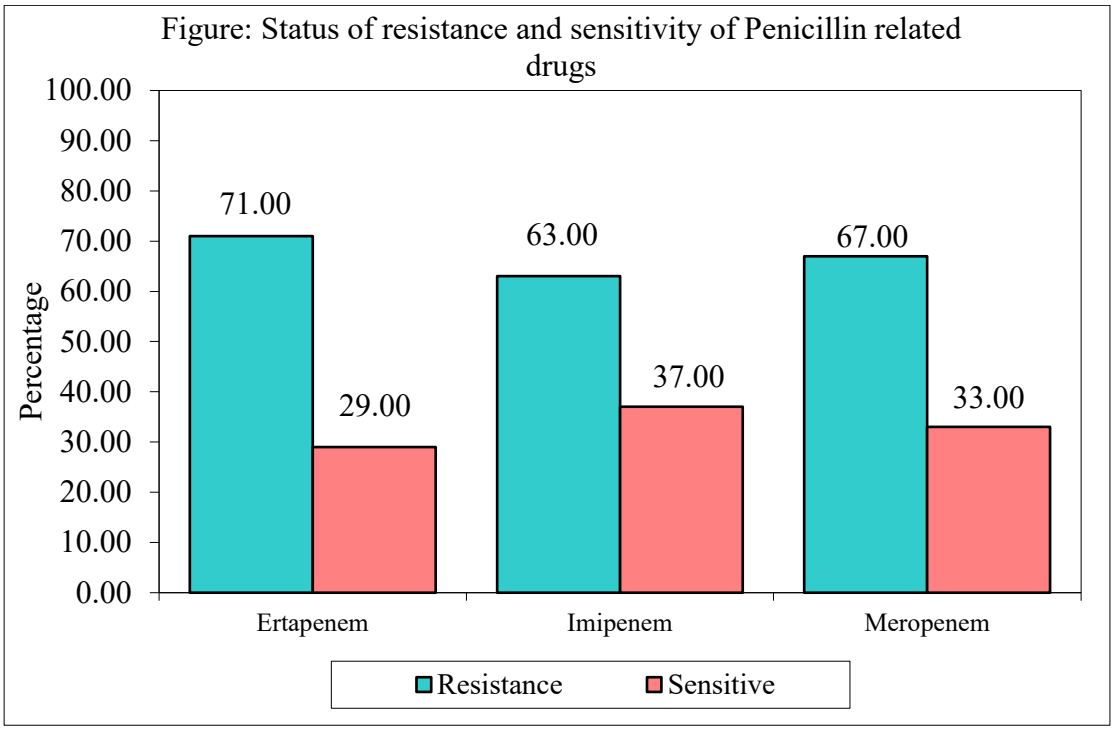


Table 15C: Cotrimoxazole

Drugs	Number	Percentage
Resistance	74	74
Sensitive	26	26
Total	100	100

Graph 12C: Cotrimoxazole

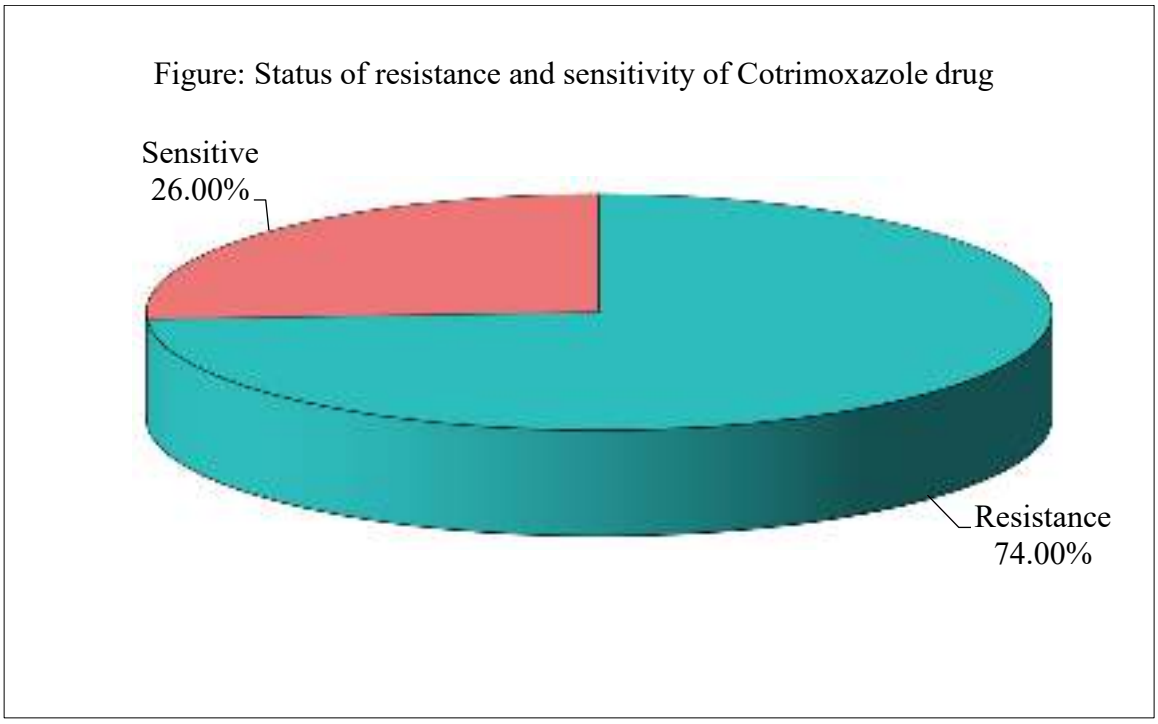


Table 15D: Macrolides

Drugs	Resistance	Sensitive	Total
Tetracycline	72	28	100
Tigecycline	46	54	100
Erythromycin	97	3	100

Graph 12D: Macrolides

Figure: Status of resistance and sensitivity of Macrolides related drugs

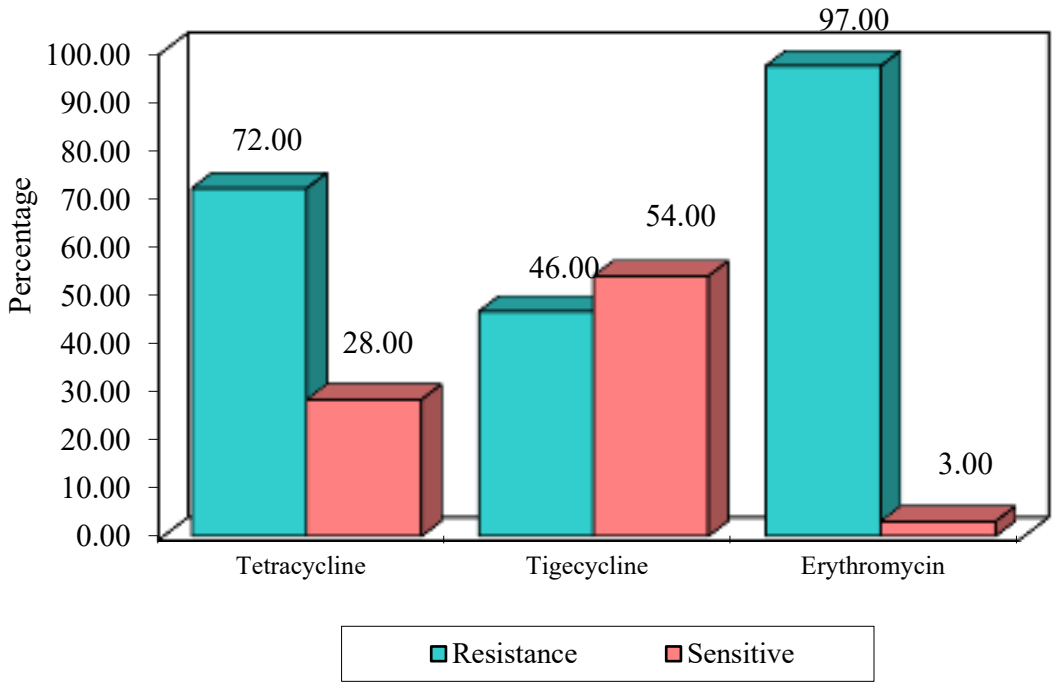


Table 15E: Aminoglycosides

Drugs	Resistance	Sensitive	Total
Gentamicin	67	33	100
Amikacin	65	35	100
Tobramycin	80	20	100

Graph 12E: Aminoglycosides

Figure: Status of resistance and sensitivity of Aminoglycosides related drugs

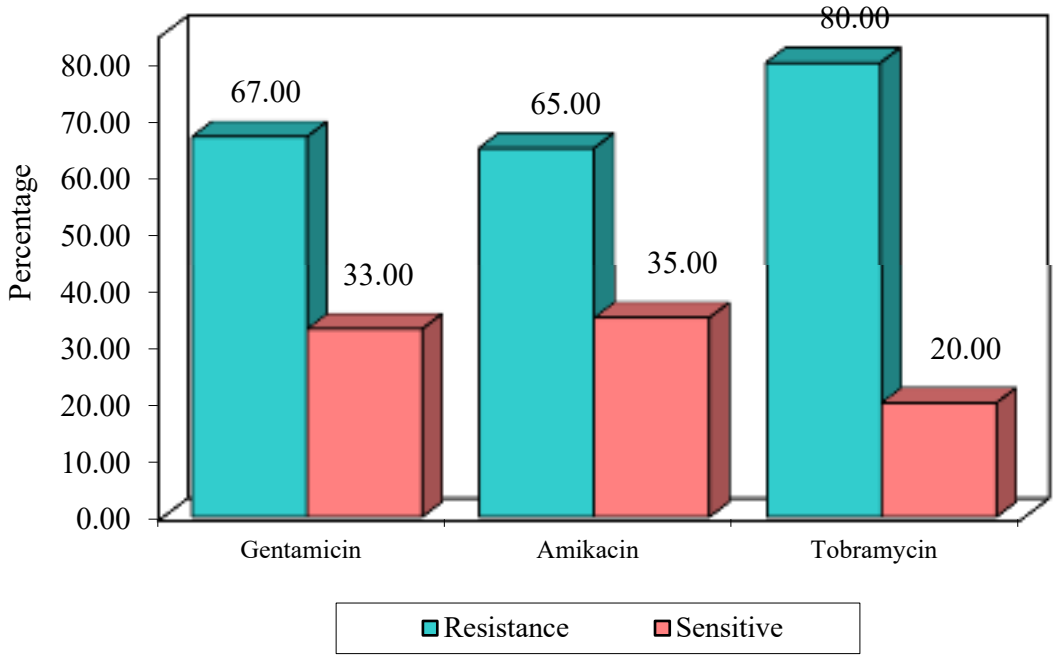


Table 15F: Cephalosporins

Drugs	Resistance	Sensitive	Total
Cefotaxime	83	17	100
Cefazolin	95	5	100
Cefuroxime	93	7	100
Ceftazidime	82	18	100
Cefoxitin	85	15	100
Cefepime	91	9	100

Graph 12F: Cephalosporins

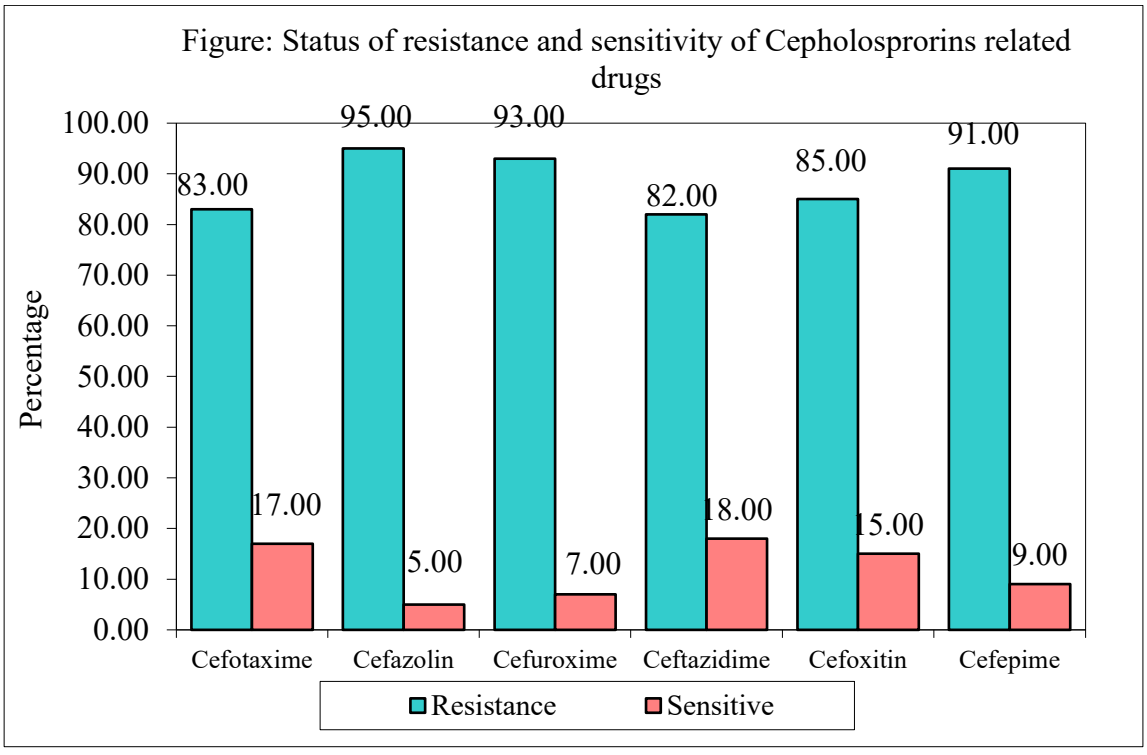


Table 15G: Fluoroquinolones

Drugs	Resistance	Sensitive	Total
Ciprofloxacin	81	19	100
Levofloxacin	79	21	100
Moxifloxacin	93	7	100
Norfloxacin	83	17	100

Table 15H: Other Individual Antibiotics

Drugs	Resistance	Sensitive	Total
Nitrofurantoin	60	40	100
Colistin	67	33	100
Fosfomycin	69	31	100
Chloramphenicol	93	7	100
Teicoplanin	92	8	100
Rifampin	95	5	100
Linezolid	90	10	100
Vancomycin	92	8	100
Aztreonam	98	2	100

Observing for sensitivity pattern of organism to various classes of drugs, the sensitivity and resistance pattern for Penicillin group is shown in Table 15A and Graph 12A, Table 15B and Graph 12B for Carbapenems, Table 15C and Graph 12C for Cotrimoxazole, Table 15D and Graph 12D for Macrolides, Table 15E and Graph 12E for Aminoglycosides, Table 15F and Graph 12F for Cephalosporins, Table 15G for Fluoroquinolones and Table 15H for other individual antibiotics.

Comparison of Sensitivity Pattern of different organisms to different group of antibiotics.

Table 16A: Penicillin

Culture	Amoxicillin/ Clavulanic Acid		Ampicillin		Piperacillin/ Tazobactam		Mezlocillin	
	Resistance	Sensitive	Resistance	Sensitive	Resistance	Sensitive	Resistance	Sensitive
Acinetobacter Baumanii/ Haemolyticus	6	0	6	0	6	0	6	0
Citrobacter species	4	1	5	0	5	0	5	0
Enterobacter Aerogenes	4	1	4	1	3	2	4	1
Enterobacter Cloacae	10	2	12	0	10	2	12	0
Enterococcus Faecalis	12	0	8	4	12	0	12	0
Escherichia Coli	22	18	38	2	22	18	39	1
Klebsiella Oxytoca	3	0	3	0	3	0	3	0
Klebsiella Pneumoniae	4	5	8	1	6	3	9	0
Proteus Mirabilis	1	1	1	1	0	2	1	1
Pseudomonas Aeruginosa	4	0	4	0	4	0	4	0
Staphylococcus Aureus	2	0	2	0	2	0	2	0
Total	72	28	91	9	73	27	97	3

Table 16B: Carbapenems

Culture	Ertapenem		Imipenem		Meropenem	
	Resistance	Sensitive	Resistance	Sensitive	Resistance	Sensitive
Acinetobacter Baumanii/ Haemolyticus	6	0	6	0	6	0
Citrobacter species	4	1	4	1	4	1
Enterobacter Aerogenes	4	1	3	2	3	2
Enterobacter Cloacae	9	3	7	5	7	5
Enterococcus Faecalis	12	0	12	0	12	0
Escherichia Coli	21	19	18	22	20	20
Klebsiella Oxytoca	3	0	3	0	3	0
Klebsiella Pneumoniae	6	3	4	5	7	2
Proteus Mirabilis	0	2	1	1	0	2
Pseudomonas Aeruginosa	4	0	3	1	3	1
Staphylococcus Aureus	2	0	2	0	2	0
Total	71	29	63	37	67	33

Table 16C: Cotrimoxazole

Culture	Cotrimoxazole		
	Resistance	Sensitive	Total
Acinetobacter Baumanii/ Haemolyticus	6	0	6
Citrobacter species	5	0	5
Enterobacter Aerogenes	4	1	5
Enterobacter Cloacae	6	6	12
Enterococcus Faecalis	12	0	12
Escherichia Coli	23	17	40
Klebsiella Oxytoca	3	0	3
Klebsiella Pneumoniae	7	2	9
Proteus Mirabilis	2	0	2
Pseudomonas Aeruginosa	4	0	4
Staphylococcus Aureus	2	0	2
Total	74	26	100

Table 16D: Macrolides

Culture	Tetracycline		Tigecycline		Erythromycin	
	Resistance	Sensitive	Resistance	Sensitive	Resistance	Sensitive
Acinetobacter Baumanii/ Haemolyticus	5	1	6	0	6	0
Citrobacter species	4	1	3	2	5	0
Enterobacter Aerogenes	4	1	3	2	5	0
Enterobacter Cloacae	8	4	3	9	12	0
Enterococcus Faecalis	11	1	12	0	10	2
Escherichia Coli	26	14	8	32	39	1
Klebsiella Oxytoca	3	0	1	2	3	0
Klebsiella Pneumoniae	4	5	2	7	9	0
Proteus Mirabilis	2	0	2	0	2	0
Pseudomonas Aeruginosa	4	0	4	0	4	0
Staphylococcus Aureus	1	1	2	0	2	0
Total	72	28	46	54	97	3

Table 16E: Aminoglycosides

Culture	Gentamicin		Amikacin		Tobramycin	
	Resistance	Sensitive	Resistance	Sensitive	Resistance	Sensitive
Acinetobacter Baumanii/ Haemolyticus	6	0	5	1	6	0
Citrobacter species	4	1	3	2	4	1
Enterobacter Aerogenes	3	2	3	2	4	1
Enterobacter Cloacae	7	5	9	3	10	2
Enterococcus Faecalis	9	3	12	0	12	0
Escherichia Coli	23	17	18	22	29	11
Klebsiella Oxytoca	3	0	3	0	3	0
Klebsiella Pneumoniae	5	4	6	3	5	4
Proteus Mirabilis	2	0	0	2	1	1
Pseudomonas Aeruginosa	4	0	4	0	4	0
Staphylococcus Aureus	1	1	2	0	2	0
Total	67	33	65	35	80	20

Table 16F: Cephalosporins

Culture	Cefotaxime		Cefazolin		Cefuroxime	
	Resistance	Sensitive	Resistance	Sensitive	Resistance	Sensitive
Acinetobacter Baumanii/ Haemolyticus	6	0	6	0	6	0
Citrobacter species	5	0	4	1	5	0
Enterobacter Aerogenes	4	1	5	0	3	2
Enterobacter Cloacae	10	2	12	0	11	1
Enterococcus Faecalis	12	0	12	0	12	0
Escherichia Coli	31	9	38	2	38	2
Klebsiella Oxytoca	3	0	3	0	3	0
Klebsiella Pneumoniae	6	3	8	1	9	0
Proteus Mirabilis	0	2	1	1	0	2
Pseudomonas Aeruginosa	4	0	4	0	4	0
Staphylococcus Aureus	2	0	2	0	2	0
Total	83	17	95	5	93	7

Table 16G: Fluoroquinolones

Culture	Ciprofloxacin		Levofloxacin		Moxifloxacin		Norfloxacin	
	Resistance	Sensitive	Resistance	Sensitive	Resistance	Sensitive	Resistance	Sensitive
Acinetobacter Baumanii/ Haemolyticus	6	0	6	0	6	0	6	0
Citrobacter species	4	1	4	1	5	0	4	1
Enterobacter Aerogenes	3	2	3	2	4	1	3	2
Enterobacter Cloacae	10	2	10	2	11	1	10	2
Enterococcus Faecalis	12	0	12	0	12	0	10	2
Escherichia Coli	31	9	29	11	37	3	35	5
Klebsiella Oxytoca	3	0	3	0	3	0	3	0
Klebsiella Pneumoniae	6	3	6	3	8	1	6	3
Proteus Mirabilis	1	1	1	1	2	0	1	1
Pseudomonas Aeruginosa	4	0	4	0	4	0	4	0
Staphylococcus Aureus	1	1	1	1	1	1	1	1
Total	81	19	79	21	93	7	83	17

Table 16H: Other individual antibiotics

Culture	Nitrofurantoin		Colistin		Fosfomycin		Chloramphenicol	
	Resistance	Sensitive	Resistance	Sensitive	Resistance	Sensitive	Resistance	Sensitive
Acinetobacter Baumanii/ Haemolyticus	6	0	6	0	6	0	6	0
Citrobacter species	4	1	3	2	3	2	5	0
Enterobacter Aerogenes	3	2	4	1	3	2	5	0
Enterobacter Cloacae	10	2	10	2	12	0	12	0
Enterococcus Faecalis	6	6	12	0	11	1	7	5
Escherichia Coli	15	25	19	21	17	23	39	1
Klebsiella Oxytoca	3	0	3	0	3	0	3	0
Klebsiella Pneumoniae	6	3	3	6	8	1	9	0
Proteus Mirabilis	2	0	2	0	1	1	2	0
Pseudomonas Aeruginosa	4	0	3	1	4	0	4	0
Staphylococcus Aureus	1	1	2	0	1	1	1	1
Total	60	40	67	33	69	31	93	7

Culture	Teicoplanin		Rifampin		Linezolid		Vancomycin	
	Resistance	Sensitive	Resistance	Sensitive	Resistance	Sensitive	Resistance	Sensitive
Acinetobacter Baumanii/ Haemolyticus	6	0	6	0	6	0	6	0
Citrobacter species	5	0	5	0	5	0	5	0
Enterobacter Aerogenes	5	0	5	0	5	0	5	0
Enterobacter Cloacae	12	0	12	0	12	0	12	0
Enterococcus Faecalis	7	5	8	4	5	7	7	5
Escherichia Coli	39	1	39	1	39	1	39	1
Klebsiella Oxytoca	3	0	3	0	3	0	3	0
Klebsiella Pneumoniae	9	0	9	0	9	0	9	0
Proteus Mirabilis	2	0	2	0	2	0	2	0
Pseudomonas Aeruginosa	4	0	4	0	4	0	4	0
Staphylococcus Aureus	0	2	2	0	0	2	0	2
Total	92	8	95	5	90	10	92	8

Table 16A, 16B, 16C , 16D, 16E, 16F, 16G and 16H shows the sensitivity pattern of organisms to different classes of antibiotics.

Culture	Aztreonam		
	Resistance	Sensitive	Total
Acinetobacter Baumanii/ Haemolyticus	6	0	6
Citrobacter species	5	0	5
Enterobacter Aerogenes	5	0	5
Enterobacter Cloacae	12	0	12
Enterococcus Faecalis	12	0	12
Escherichia Coli	38	2	40
Klebsiella Oxytoca	3	0	3
Klebsiella Pneumoniae	9	0	9
Proteus Mirabilis	2	0	2
Pseudomonas Aeruginosa	4	0	4
Staphylococcus Aureus	2	0	2
Total	98	2	100

Table 17: Clinical response to treatment

Clinical Response	Number of patients (n=100)	Percentage
Responded	100	100
Not responded	0	0

Majority of our patients who were symptomatic i.e., 65 patients (65%), showed response within 48 hours of treatment of their urinary tract infection based on culture and sensitivity patterns.

Remaining 35 patients (35%), who were asymptomatic at presentation were also given treatment based on culture sensitivity patterns. However, we couldn't asses their response as they were asymptomatic at presentation. We have not followed up of these patients. (Repeat urine routine and culture was not done for these patients. Further, we observed patients with cystitis responded within 48 hours, patients with pyelonephritis took little longer time for clinical response i.e., roughly 5 days and above.

However, all symptomatic patients showed response to treatment.

DISCUSSION

In the present study of 100 patients with infection of urinary tract in type 2 diabetes mellitus, the urinary bacterial culture and antibiotic sensitivity pattern was studied. The following observations were made:

All 100 patients with type 2 diabetes mellitus presented with either asymptomatic or symptomatic urinary tract infection.

The age of the patients ranged from 18 years to 98 years (youngest was 21 years and eldest was 98 years). In our study, urinary tract infection was more observed in patients above 50 years of age (n=81) and only 19 patients had urinary tract infection below the age of 50 years (Table 1).

A study by Mario Bonadio et al, in their study population observed more than 70% patients of urinary tract infection in type 2 diabetes mellitus, the age was > 65 years of age.[84]

Similarly, the study by Captain Andrew Huvos et al observed evidence of urinary tract infection was more in the elderly patients in their study population.

A study by Aswani Srinivas et al, did not find any correlation between age of patients and incidence of urinary tract infection, with or without diabetes.(49)

The explanation for increased frequency for urinary tract infection in patients who are elderly with or without diabetes could be because of autonomic neuropathy leading on to incomplete bladder emptying in patients with type 2 diabetes mellitus.

Otherwise also in elderly, because of age, bladder emptying could be incomplete. In some of the studies, they have found the increased incidence of urinary tract infection due to indwelling catheters. Long standing diabetes mellitus may also predispose to urinary tract infection which was observed in some studies.

In our present study of 100 patients, the female patients (n=55) were slightly more than male patients (n=45) accounting for ratio of M: F::1.2:1. A slight female preponderance was observed in our study.

A study by Vijesh Kumar et al, have found a higher incidence of urinary tract infection in female gender.

A study by Orna Nitzan et al, showed the incidence of urinary tract infection in female gender was more as compared to their counterpart in their study population. The reason for urinary tract infection being more in female gender is because of anatomical reasons due to short urethra and proximity to the warm moist vulvar and perianal areas usually colonized with bacteria, traumatization of mucosal barrier during sexual act also may predispose to increased incidence of urinary tract infection due to diabetes mellitus itself which makes them more vulnerable for urinary tract infection as compared to non-diabetics.[85]

In patients with diabetes there is an increased chance of infection of kidney because of diabetes mellitus as well as impaired concentrating ability due to diabetes mellitus. (Due to variation in urinary pH i.e., urinary pH increases).

Other studies by D. J. Sullivan et al and GG Zhanel et al have also found increased incidence of urinary tract infection in female patients in their study population. [72]

We studied in all our 100 patients the duration of diabetes mellitus which revealed, 52 patients (52%) had diabetes ranging from 1- 5 years, between 5 to 10 years – 27 patients, 10 – 15 years – 12 patients and only 9 patients were more than 15 years with mean ± SD = 24.89 ± 7.02.

Studies by Khaled A et al, Dwijan Das et al, Vijesh Kumar et al and Aswani Srinivas et al had similar observations comparable to our study as far as duration of

diabetes was concerned, however they did not find any correlation with the rate of infection with increasing duration of diabetes mellitus in their studies.[86]

Similarly, we also did not find any correlation in our present study having significance with the duration of diabetes mellitus and urinary tract infection. P value being statistically insignificant.

But, one study by Hamdan et al in their study group found a correlation between diabetes mellitus duration with urinary tract infection i.e., more the duration of diabetes mellitus, they were more prone for urinary tract infection.

The probable explanation quoted by Bahl AL et al, as the duration of diabetes is more, they have increased prevalence of urinary tract infection, the increase is 1.9-fold for every one decade of diabetes mellitus status. Longstanding diabetes mellitus may attribute to autonomic dysfunction of bladder resulting in incomplete bladder evacuation leading to urinary tract infection.

The control of diabetes mellitus is also very important, with a good glycemic control, the chances of infection is less as compared to bad glycemic control. This was observed by Schimit JK et al, who found either symptomatic or asymptomatic bacteriuria was seen in patients with higher HbA1c levels and degree of glycemic control significantly associated with urinary tract infection.[87]

Many authors have not taken into consideration the treatment of patient either with insulin or oral hypoglycemic agents.

In our study, we segregated our patients based on their treatment strategies as shown in Table 4, Table 5 and Table 6.

But most of the authors found increased prevalence of bacteriuria in patients, with insulin treatment as compared to patient with oral hypoglycemic agent treatment.

The explanation for this offered by these authors is the patients on insulin reflects the bad control of their glycemia as a result they have increased frequency of bacteriuria.

Some authors have also found increased bacteriuria in patients on treatment with SGLT2 inhibitors. We did not find such correlation with the treatment (insulin, OHAs or both).

In our study, we attempted to look for compliance of patients with treatment and follow up. 90% of our patients were compliant with treatment, only 10% were non-complaint.

Similarly, monthly follow up, 84% were compliant with follow up and 16% weren't. (Table 7).

Going through different authors, nobody has studied the compliance of patients with treatment.

We observed various complications of diabetes mellitus, in our patients. The commonly observed complications were Retinopathy (n=51), Neuropathy (n=46), Nephropathy (n=46) and diabetic foot (n=9). 47 patients did not have any complications. Some patients had overlapping complications.

Studies by Dwijen Das et al, Suzanne Geerlings et al and Vijeth Kumar et al found varying degree of different complications like retinopathy, nephropathy and neuropathy in their study populations. None of the authors have found any correlation of complications of diabetes with bacteriuric urinary tract infection.[86]

We also observed no correlation between complications and bacteriuria.

Our patients presented with various permutations and combinations of symptoms as shown in table number 9. Many authors have found similar

presentations in their study populations. We did not observe any particular or atypical presentation in our study population. Same was observed by other authors.

We subjected all our 100 patients to routine estimation of RBS at arrival (Table 12A) followed by estimation of Fasting and Post Prandial sugars on the next day of admission (both FBS and PPBS on the same day).

Similarly, all patients were subjected to HbA1c estimation. (Table 12B).

Studies by Aswani Srinivas et al and Dwijen Das et al have subjected their patients to routine estimation of sugars and HbA1c in their study population and they observed patients with high HbA1c were more predisposed to urinary tract infections. The chances of urinary tract infection were more in patients with HbA1c greater or equal to 7%, however in our study we did not find such association as all our patients (n=100) had HbA1c > 6.5 % (ranging between 6.5 to greater than or equal to 8.5 (Table 12B).[49]

All our 100 patients were subjected to urine routine analysis and were found to have albumin present in 73 patients, absent in 27 patients. Urine sugar was present in 86 patients and absent in 14 patients. Microscopic finding revealed all had WBCs on microscopy. (Table 11A and 11B).

4 patients who has sheets of pus cells were incidentally cases of pyelonephritis.

Most authors have not revealed their patient's urine analysis and have directly discussed culture isolated of urinary specimen of their patients.

All our 100 patients, were subjected to urine and culture sensitivity patterns. The commonest organism isolated was Escherichia Coli. All the other isolates are shown in (Table 13).

Most authors like Aswani Srinivas et al and Hamdan Z Hamdan also found the commonest isolate was Escherichia Coli. The reason for this isolate (E. coli) as it is the commonest commensal found in the genitourinary tract making the patient vulnerable for urinary tract infection because of altered immunity as a result of diabetes mellitus.

All our patients were routinely subjected to USG Abdomen study with special reference to kidney- ureter and bladder and the findings were cystitis in 39 patients, pyelonephritis in 26 patients and 35 patients were asymptomatic.

A study by Aswani Srinivas et al has found cystitis as a more common finding on ultrasound abdomen and Pelvis.

The drug sensitivity pattern of different organism to different class of drugs is shown in (Table 15A to 15H).

Most of the patients in our study the sensitivity pattern was more observed with cephalosporin group and higher antibiotics like Piperacillin and Tazobactam combination.

Similar observation was made by Aswani Srinivas et al and Dwijen Das et al who found the response commonly with cephalosporin and higher antibiotics like Piperacillin and Tazobactam in their study.[49]

Most of our patients with evidence of Pyelonephritis responded to carbapenems group of antibiotics.

Some patients were treated as per culture and sensitivity reports with drugs like Colistin, Fosfomycin Teicoplanin as shown in the above tables.

Similarly, sensitivity patterns of organism to different class of drugs are depicted in Tables 16A to 16H.

Our study is also similar to the study by other authors as far as the response of organisms to different class of antibiotics.

Further we observed in all our 100 patients the clinical response which was seen in 65% within 48 hours of treatment who were symptomatic, remaining 35% who were asymptomatic assumed to have responded with the treatment based on culture sensitivity pattern as these patients were not subjected to repeat urine examination.

The observation by other authors such as Aswani Srinivas et al and Dwijen Das et al who found a good clinical response to treatment based on culture sensitivity report. They also observed patients requiring higher antibiotics, the response was slightly delayed and slow in their groups.[86]

The urinary tract infection in their populations with diabetes mellitus is a burden to patients and healthcare system because of costs involved in treatment, hospital stay and even complications associated with infections.

It was also observed in some patients the response was poor because of multidrug resistant strains. In our study we did not encounter such issues.

CONCLUSION

In our present study of 100 patients of Type 2 diabetes mellitus, we analyzed urinary tract infection whether symptomatic or asymptomatic was frequently encountered in diabetics with elevated HbA1c, poor glycemic control and female gender.

Pyelonephritis was more in patients who had elevated HbA1c and poor glycemic control, this was also observed by other authors, however we did not find the correlation with age, duration of diabetes mellitus, mode of treatment with or without complications, compliance treatment and follow up which did not influence the prevalence of urinary tract infection in these patients.

Patients with pyelonephritis required higher antibiotics for their treatment. The response was slow and delayed. However, they did respond to treatment.

35 patients who were asymptomatic in our present study were assumed to have responded to treatment as we did not subject them to repeat urine examination.

65 patients who were symptomatic, response to treatment was based on clinical grounds (symptoms disappeared with treatment).

SUMMARY

In our present study of 100 patients of type 2 diabetes mellitus with urinary tract infection admitted in Department of General Medicine KLE Prabhakar Kore Hospital in the study period of January 2020 to December 2020 was undertaken to find "A clinical profile of urinary tract infection in type 2 diabetes mellitus with bacterial culture and antibiotic sensitivity patterns".

The results observed were urinary tract infection was more in patients of female gender, elevated HbA1c and poor glycemic control. The commonest organism isolated was Escherichia Coli. Higher antibiotics mainly Carbapenems were required and administered in patients with Pyelonephritis.

We did not find any significant correlation to age, duration of diabetes mellitus, complications of diabetes mellitus and mode of treatment of diabetes mellitus.

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ANNEXURE I

INFORMED CONSENT

Title Of Research Study: A CLINICAL PROFILE OF URINARY TRACT INFECTION IN TYPE 2 DIABETES MELLITUS WITH BACTERIAL CULTURE AND ANTIBIOTIC SENSITIVITY PATTERNS

Principal Investigator: -

REGISTRATION NO: BG0119009

Post Graduate

Department of General Medicine,

JNM, Belagavi.

Guide: -

Dr _____

Professor

Department of General Medicine,

JNMC, Belgaum.

Introduction and Purpose:

- Type 2 diabetes mellitus is a heterogeneous group of disorders characterized by variable degrees of insulin resistance, impaired insulin secretion, and increased glucose production.
- Patients with type 2 Diabetes Mellitus are at increased risk of infections, with the urinary tract being the most frequent infection site
- Various impairments in the immune system, in addition to poor metabolic control of diabetes, and incomplete bladder emptying due to autonomic neuropathy, may all contribute in the pathogenesis of urinary tract infections (UTI) in diabetic patients.

- The spectrum of UTI in these patients ranges from asymptomatic bacteriuria, cystitis, pyelonephritis and severe urosepsis.
- Serious complications of UTI, such as emphysematous cystitis and pyelonephritis, renal abscesses and renal papillary necrosis, are all encountered more frequently in type 2 diabetes than in the general population.

Procedure:

If you agree to be part of the research study, you will be asked the relevant history and will be subjected to relevant clinical examination and investigations. You will also have to give blood and urine samples for the necessary investigations.

Risk and Benefits:

The only risk and possible discomfort you might get is while taking blood from your arm for the investigations. It may cause swelling, pain, redness (rarely happens) at the site from where the blood is drawn.

You may not be benefitted by these investigations but you will be part of this study which is going to be useful to others in the future.

Alternatives:

Taking part in this study is voluntary. You may choose not to take part in this study.

If you decide to take part you can later change your mind and withdraw from the study. Your decision will not change the present or future health care or other services that you receive. The study doctor or sponsor may stop your participation in this study at any time. If you choose not to take part in the study, you will receive the standard treatment for patients with your condition.

Privacy and Confidentiality: All information collected about you during the course of this study will be kept confidential to the extent permitted by law. The code numbers will identify you in this research record. Information from this study may be published but your identity will be confidential in any publication.

Institution / Sponsor's policy:

Does not apply to this research

Financial incentives for participation:

You will not be paid / offered any gifts /incentives for participating in the study.

Authorization to publish the results:

The results of the study would be forwarded to the KLE University, Belgaum as part of requirement towards the completion of MD degree, review and publishing.

In case of the queries during study or in future you may contact following persons,

3. REG. NO: BG0119009
Investigator,
PG in General Medicine,
JNMC, Belgaum.

2. Dr. _____
Professor
Dept of General Medicine,
JNMC, Belgaum.

1. Dr. Roopa M Bellad
Professor of Paediatrics,
J N Medical College, BELAGAVI, Chairman,
J.N.M.C Ethical Committee for Human Research
9480275601

CONSENT FORM

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicates that I have read this consent form, or it has Been read to me, this consent form and have had all the questions answered

Signature / Left Thumb print of the Participant or legally authorized representative

Participant's name ……...

Signature / Left thumb impression: ……...

of the participant

Name of the legally authorized ……...

representative / guardian

Signature / Left thumb impression ……...

Witness' name ……...

Signature / Left thumb impression ……...

Investigator's name and signature ……...

Date:

Place:

ತಿಳುವಳಿಕೆಯ ಸಮ್ಮತಿ

ಸಂಶೋಧನಾ ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ: ಟೈಪ್ 2 ಡಯಾಬಿಟಿಸ್ ಮೆಲ್ಲಿಟಸ್ನಲ್ಲಿ ಮೂತ್ರದ ಸೋಂಕಿನ ಕ್ಲಿನಿಕಲ್ ವಿವರ

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ: -

REGISTRATION NO: BG0119009

ಸ್ನಾತಕೋತ್ತರ ಪದವಿ
ಜನರಲ್ ಮೆಡಿಸಿನ್ ಇಲಾಖೆ,
ಜೆಎನ್ಎಂಸಿ, ಬೆಳಗಾವಿ.

ಮಾರ್ಗದರ್ಶಿ: -

ಡಾ.

ಪ್ರೊಫೆಸರ್
ಜನರಲ್ ಮೆಡಿಸಿನ್ ಇಲಾಖೆ,
ಜೆಎನ್ಎಂಸಿ, ಬೆಳಗಾವಿ.

ಪರಿಚಯ ಮತ್ತು ಉದ್ದೇಶ: -

•ಟೈಪ್ 2 ಡಯಾಬಿಟಿಸ್ ಮೆಲ್ಲಿಟಸ್ ಎನ್ನುವುದು ವೈವಿಧ್ಯಮಯ ಅಸ್ವಸ್ಥತೆಗಳ ಗುಂಪಾಗಿದ್ದು, ಇದು ಇನ್ಸುಲಿನ್ ಪ್ರತಿರೋಧದ ಅಸ್ಥಿರ ಡಿಗ್ರಿಗಳು, ದುರ್ಬಲಗೊಂಡ ಇನ್ಸುಲಿನ್ ಸ್ರವಿಸುವಿಕೆ ಮತ್ತು ಗ್ಲೂಕೋಸ್ ಉತ್ಪಾದನೆಯನ್ನು ಹೆಚ್ಚಿಸುತ್ತದೆ.

•ಟೈಪ್ 2 ಡಯಾಬಿಟಿಸ್ ಮೆಲ್ಲಿಟಸ್ ರೋಗಿಗಳು ಸೋಂಕಿನ ಅಪಾಯವನ್ನು ಹೆಚ್ಚಿಸುತ್ತಾರೆ, ಮೂತ್ರದ ಪ್ರದೇಶವು ಹೆಚ್ಚಾಗಿ ಸೋಂಕಿನ ತಾಣವಾಗಿದೆ

•ರೋಗನಿರೋಧಕ ವ್ಯವಸ್ಥೆಯಲ್ಲಿನ ವಿವಿಧ ದೌರ್ಬಲ್ಯಗಳು, ಮಧುಮೇಹದ ಕಳಪೆ ಚಯಾಪಚಯ ನಿಯಂತ್ರಣ ಮತ್ತು ಸ್ವನಿಯಂತ್ರಿತ ನರರೋಗದಿಂದಾಗಿ ಅಪೂರ್ಣ ಗಾಳಿಗುಳ್ಳೆಯ ಖಾಲಿಯಾಗುವುದು ಇವೆಲ್ಲವೂ ಮಧುಮೇಹ ರೋಗಿಗಳಲ್ಲಿ ಮೂತ್ರದ ಸೋಂಕಿನ (ಯುಟಿಐ) ರೋಗಕಾರಕ ಕ್ರಿಯೆಗೆ ಕಾರಣವಾಗಬಹುದು.

•ಈ ರೋಗಿಗಳಲ್ಲಿ ಯುಟಿಐನ ವರ್ಣಪಟಲವು ಲಕ್ಷಣರಹಿತ ಬ್ಯಾಕ್ಟೀರಿಯೂರಿಯಾ, ಸಿಸ್ಟೈಟಿಸ್, ಪೈಲೊನೆಫೆರಿಟಿಸ್ ಮತ್ತು ತೀವ್ರ ಯುರೋಸೆಪ್ಸಿಸ್ ನಿಂದ ಹಿಡಿದು ಇರುತ್ತದೆ.

•ಯುಟಿಐನ ಗಂಭೀರ ತೊಡಕುಗಳಾದ ಎಂಫಿಸೆಮಾಟಸ್ ಸಿಸ್ಟೈಟಿಸ್ ಮತ್ತು ಪೈಲೊನೆಫೆರಿಟಿಸ್, ಮೂತ್ರಪಿಂಡದ ಹುಣ್ಣುಗಳು ಮತ್ತು ಮೂತ್ರಪಿಂಡದ ಪ್ಯಾಪಿಲ್ಲರಿ ನೆಕ್ರೋಸಿಸ್ ಇವೆಲ್ಲವೂ ಸಾಮಾನ್ಯ ಜನಸಂಖ್ಯೆಗಿಂತ ಹೆಚ್ಚಾಗಿ ಟೈಪ್ 2 ಮಧುಮೇಹದಲ್ಲಿ ಹೆಚ್ಚಾಗಿ ಕಂಡುಬರುತ್ತವೆ.

ವಿಧಾನ :

ಸಂಶೋಧನಾ ಅಧ್ಯಯನದ ಭಾಗವಾಗಲು ನೀವು ಒಪ್ಪಿದರೆ, ನಿಮಗೆ ಸಂಬಂಧಿತ ಇತಿಹಾಸವನ್ನು ಕೇಳಲಾಗುತ್ತದೆ ಮತ್ತು ಸಂಬಂಧಿತ ಕ್ಲಿನಿಕಲ್ ಪರೀಕ್ಷೆ ಮತ್ತು ತನಿಖೆಗೆ ಒಳಪಡಿಸಲಾಗುತ್ತದೆ. ಅಗತ್ಯ ತನಿಖೆಗಾಗಿ ನೀವು ರಕ್ತ ಮತ್ತು ಮೂತ್ರದ ಮಾದರಿಗಳನ್ನು ಸಹ ನೀಡಬೇಕಾಗುತ್ತದೆ .

ಅಪಾಯ ಮತ್ತು ಪ್ರಯೋಜನಗಳು :

ತನಿಖೆಗಾಗಿ ನಿಮ್ಮ ತೋಳಿನಿಂದ ರಕ್ತವನ್ನು ತೆಗೆದುಕೊಳ್ಳುವಾಗ ನೀವು ಪಡೆಯುವ ಏಕೈಕ ಅಪಾಯ ಮತ್ತು ಸಂಭವನೀಯ ಅಸ್ವಸ್ಥತೆ. ಇದು ರಕ್ತವನ್ನು ಎಳೆಯುವ ಸ್ಥಳದಲ್ಲಿ ಬೆವರುವಿಕೆ, ನೋವು, ಕೆಂಪು (ವಿರಳವಾಗಿ ಸಂಭವಿಸುತ್ತದೆ) ಗೆ ಕಾರಣವಾಗಬಹುದು .
ಈ ತನಿಖೆಯಿಂದ ನಿಮಗೆ ಯಾವುದೇ ಪ್ರಯೋಜನವಾಗದಿರಬಹುದು ಆದರೆ ಭವಿಷ್ಯದಲ್ಲಿ ಇತರರಿಗೆ ಉಪಯುಕ್ತವಾಗಲಿರುವ ಈ ಅಧ್ಯಯನದ ಭಾಗವಾಗುತ್ತೀರಿ .

ಪರ್ಯಾಯಗಳು :

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವುದು ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸದಿರಲು ನೀವು ಆಯ್ಕೆ ಮಾಡಬಹುದು. ನೀವು ಭಾಗವಹಿಸಲು ನಿರ್ಧರಿಸಿದರೆ ನೀವು ನಂತರ ನಿಮ್ಮ ಮನಸ್ಸನ್ನು ಬದಲಾಯಿಸಬಹುದು ಮತ್ತು ಅಧ್ಯಯನದಿಂದ ಹಿಂದೆ ಸರಿಯಬಹುದು. ನಿಮ್ಮ ನಿರ್ಧಾರವು ಪ್ರಸ್ತುತ ಅಥವಾ ಭವಿಷ್ಯದ ಆರೋಗ್ಯ ರಕ್ಷಣೆ ಅಥವಾ ನೀವು ಸ್ವೀಕರಿಸುವ ಇತರ ಸೇವೆಗಳನ್ನು ಬದಲಾಯಿಸುವುದಿಲ್ಲ. ಅಧ್ಯಯನ ವೈದ್ಯರು ಅಥವಾ ಪ್ರಾಯೋಜಕರು ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮ ಭಾಗವಹಿಸುವಿಕೆಯನ್ನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ನಿಲ್ಲಿಸಬಹುದು. ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸದಿರಲು ನೀವು ಆರಿಸಿದರೆ, ನಿಮ್ಮ ಸ್ಥಿತಿಯ ರೋಗಿಗಳಿಗೆ ನೀವು ಪ್ರಮಾಣಿತ ಚಿಕಿತ್ಸೆಯನ್ನು ಸ್ವೀಕರಿಸುತ್ತೀರಿ.

ಗೌಪ್ಯತೆ ಮತ್ತು ಗೌಪ್ಯತೆ :

ಈ ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ನಿಮ್ಮ ಬಗ್ಗೆ ಸಂಗ್ರಹಿಸಲಾದ ಎಲ್ಲಾ ಮಾಹಿತಿಯನ್ನು ಕಾನೂನಿನಿಂದ ಅನುಮತಿಸುವ ಮಟ್ಟಿಗೆ ಗೌಪ್ಯವಾಗಿಡಲಾಗುತ್ತದೆ. ಈ ಸಂಶೋಧನಾ ದಾಖಲೆಯಲ್ಲಿ ಕೋಡ್ ಸಂಖ್ಯೆಗಳು ನಿಮ್ಮನ್ನು ಗುರುತಿಸುತ್ತವೆ. ಈ ಅಧ್ಯಯನದ ಮಾಹಿತಿಯನ್ನು ಪ್ರಕಟಿಸಬಹುದು ಆದರೆ ಯಾವುದೇ ಪ್ರಕಟಣೆಯಲ್ಲಿ ನಿಮ್ಮ ಗುರುತು ಗೌಪ್ಯವಾಗಿರುತ್ತದೆ .

ಸಂಸ್ಥೆ / ಪ್ರಾಯೋಜಕರ ನೀತಿ: ಈ ಸಂಶೋಧನೆಗೆ ಅನ್ವಯಿಸುವುದಿಲ್ಲ

ಭಾಗವಹಿಸುವಿಕೆಗೆ ಆರ್ಥಿಕ ಪ್ರೋತ್ಸಾಹ :

ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನಿಮಗೆ ಯಾವುದೇ ಉಡುಗೊರೆಗಳನ್ನು / ಪ್ರೋತ್ಸಾಹಕಗಳನ್ನು ನೀಡಲಾಗುವುದಿಲ್ಲ / ನೀಡಲಾಗುವುದಿಲ್ಲ .

ಫಲಿತಾಂಶಗಳನ್ನು ಪ್ರಕಟಿಸಲು ಅಧಿಕಾರ:

ಅಧ್ಯಯನದ ಫಲಿತಾಂಶಗಳನ್ನು ಭಾಗವಾಗಿ ಬೆಳಗಾವಿಯ ಕೆಎಲ್ಇ ವಿಶ್ವವಿದ್ಯಾಲಯಕ್ಕೆ ರವಾನಿಸಲಾಗುತ್ತದೆ
ಎಂಡಿ ಪದವಿ, ಎಮರ್ಜೆ ಮತ್ತು ಪ್ರಕಟಣೆಯ ಪೂರ್ಣಗೊಳಿಸುವಿಕೆಯ ಅವಶ್ಯಕತೆ .

ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಅಥವಾ ಭವಿಷ್ಯದಲ್ಲಿ ನೀವು ಈ ಕೆಳಗಿನ ವ್ಯಕ್ತಿಗಳನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು,

ಡಾ.ರೂಪಾ ಎಂ ಬೆಲ್ಲಾದ, ಎಂಡಿ

ಅಧ್ಯಕ್ಷ. ಕಾಲೇಜು ನೈತಿಕ ಪ್ರಬಂಧ

ಸಂಶೋಧನಾ ಸಮಿತಿ ಜೆ.ಎನ್. ವೈದ್ಯಕೀಯ. ಕಾಲೇಜು

ನೆಹರೂ ನಗರ, ಬೆಳಗಾವಿ - 590010

ಡಾ.

ಪ್ರೊಫೆಸರ್

ಜನರಲ್ ಮೆಡಿಸಿನ್ ಇಲಾಖೆ,

ಜೆಎನ್ಎಂಸಿ, ಬೆಳಗಾವಿ. 9845710945

REGISTRATION NO: BG0119009

ತನಿಖಾಧಿಕಾರಿ,

ಸ್ನಾತಕೋತ್ತರ ಪದವಿ

ಜನರಲ್ ಮೆಡಿಸಿನ್ ಇಲಾಖೆ,

ಜೆಎನ್ಎಂಸಿ, ಬೆಳಗಾವಿ. 8496040850

ಒಪ್ಪಿಗೆ ಪತ್ರ

ಕೆಳಗೆ ಸಹಿ ಮಾಡುವ ಮೂಲಕ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನಾನು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪುತ್ತೇನೆ. ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಹಿಂತೆಗೆದುಕೊಳ್ಳಬಹುದು. ಈ ಫಾರ್ಮ್ ಸಹಿ ಮಾಡುವ ಮೂಲಕ ನಾನು ನನ್ನ ಯಾವುದೇ ಕಾನೂನು ಹಕ್ಕುಗಳನ್ನು ಬಿಟ್ಟುಕೊಡುತ್ತಿಲ್ಲ. ಕೆಳಗಿನ ನನ್ನ ಸಹಿ ನಾನು ಈ ಒಪ್ಪಿಗೆಯ ಫಾರ್ಮ್ ಅನ್ನು ಓದಿದ್ದೇನೆ ಅಥವಾ ಈ ಸಮ್ಮತಿಯ ಫಾರ್ಮ್ ಅನ್ನು ನನಗೆ ಓದಿದ್ದೇನೆ ಮತ್ತು ಎಲ್ಲಾ ಪ್ರಶ್ನೆಗಳಿಗೆ ಉತ್ತರಿಸಿದೆ ಎಂದು ಸೂಚಿಸುತ್ತದೆ

ಭಾಗವಹಿಸುವವರ ಅಥವಾ ಕಾನೂನುಬದ್ಧವಾಗಿ ಅಧಿಕೃತ ಪ್ರತಿನಿಧಿಯ ಸಹಿ / ಎಡ ಹೆಬ್ಬೆರಳು ಮುದ್ರಣ
ಭಾಗವಹಿಸುವವರ ಹೆಸರು: .....

ಭಾಗವಹಿಸುವವರ ಸಹಿ / ಎಡ ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆ: .....

ಕಾನೂನುಬದ್ಧವಾಗಿ ಅಧಿಕೃತ ಪ್ರತಿನಿಧಿ / ರಕ್ಷಕರ ಹೆಸರು: ....

ಸಹಿ / ಎಡ ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆ: ....

ಸಾಕ್ಷಿಯ ಹೆಸರು: ....

ಸಹಿ / ಎಡ ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆ: ....

ದಿನಾಂಕ:

ಸ್ಥಳ

माहितीपूर्ण संमती

संशोधन अभ्यासाचे शीर्षक: टाइप 2 मधुमेह इन्शूलिनच्या कमतरतेमुळे रक्तामध्ये व लघवीमध्ये साखर आढळणे मूत्रमार्गात मुलूख संक्रमण एक क्लिनिकल प्रोफाइल

प्रधान अन्वेषक: -

REGISTRATION NO: BG0119009

पदव्युत्तर
सामान्य औषध विभाग,
जेएनएम, बेलागावी.

मार्गदर्शन:-

डॉ.

प्राध्यापक
सामान्य औषध विभाग,
जेएनएमसी, बेळगावी.

परिचय आणि उद्देश: -

टाइप २ मधुमेह इन्शूलिनच्या कमतरतेमुळे रक्तामध्ये व लघवीमध्ये साखर आढळणे आणि मूत्रमार्गात मुलूख सर्वात वारंवार संसर्ग होणारी साइट असल्याने संसर्ग होण्याचा धोका जास्त असतो.

मधुमेहावरील खराब चयापचय नियंत्रणाव्यतिरिक्त आणि स्वायत्त न्यूरोपॅथीमुळे अपूर्ण मूत्राशय रिक्त होणे याव्यतिरिक्त, प्रतिरक्षा प्रणालीतील विविध कमजोरी मधुमेहाच्या रुग्णांमध्ये मूत्रमार्गाच्या संसर्गाच्या (यूटीआय) रोगजनकांच्या कार्यात योगदान देऊ शकतात.

टाइप २ मधुमेह इन्शूलिनच्या कमतरतेमुळे रक्तामध्ये व लघवीमध्ये साखर आढळणे आणि मूत्रमार्गात मुलूख सर्वात वारंवार संसर्ग होणारी साइट असल्याने संसर्ग होण्याचा धोका जास्त असतो.

या रुग्णांमधील यूटीआयच्या स्पेक्ट्रममध्ये एसीम्प्टोमॅटिक बॅक्टेरियूरिया, सिस्टिटिस, पायलोनेफ्रायटिस आणि गंभीर युरोपेसिसचा समावेश आहे.

यूटीआयच्या गंभीर गुंतागुंत, जसे की एम्फिसेमॅटस सिस्टिटिस आणि पायलोनेफ्रायटिस, रेनल फोसे आणि रेनल पॅपिलरी नेक्रोसिस, सर्व सामान्य लोकांपेक्षा टाइप 2 मधुमेहामध्ये वारंवार आढळतात.

प्रक्रिया:

आपण संशोधन अभ्यासाचा भाग होण्यास सहमत असल्यास, आपणास संबंधित इतिहास विचारला जाईल आणि संबंधित क्लिनिकल परीक्षा आणि तपासणीस पात्र केले जाईल. आवश्यक तपासणीसाठी आपल्याला रक्त आणि लघवीचे नमुने देखील द्यावे लागतील.

जोखीम आणि फायदे :

तपासणीसाठी आपल्या बाहेरून रक्त घेत असताना आपल्याला फक्त धोका आणि संभाव्य असुविधाची समस्या उद्भवू शकते. ज्या स्थानावरून रक्त ओढले आहे त्या साइटवर स्फुल्लिंग, वेदना, लालसरपणा (क्वचितच घडते) होऊ शकते. या तपासणीमुळे आपल्याला फायदा होणार नाही परंतु आपण या अभ्यासाचा भाग व्हाल जे भविष्यात इतरांना उपयुक्त ठरेल.

विकल्प:

या अभ्यासामध्ये भाग घेणे ऐच्छिक आहे. आपण या अभ्यासामध्ये भाग न घेणे निवडू शकता.
आपण भाग घेण्याचा निर्णय घेतल्यास आपण नंतर आपले मत बदलू आणि अभ्यासापासून दूर जाऊ शकता. आपल्या निर्णयामुळे आपल्याला प्राप्त झालेल्या वर्तमान किंवा भविष्यातील आरोग्य सेवा किंवा इतर सेवा बदलणार नाहीत. अभ्यास डॉक्टर किंवा प्रायोजक या अभ्यासात आपला सहभाग कधीही थांबवू शकतात. आपण अभ्यासामध्ये भाग न घेणे निवडल्यास, आपल्या आट असलेल्या रुग्णांसाठी तुम्हाला प्रमाणित उपचार मिळेल.

गोपनीयता आणि गोपनीयता :

या अभ्यासाच्या दरम्यान आपल्याबद्दल संकलित केलेली सर्व माहिती कायद्याद्वारे परवानगी असलेल्या मर्यादेपर्यंत गोपनीय ठेवली जाईल. कोड नंबर आपल्याला या संशोधन रेकॉर्डमध्ये ओळखतील. या अभ्यासाची माहिती प्रकाशित केली जाऊ शकते परंतु आपली ओळख कोणत्याही प्रकाशनात गोपनीय असेल.

संस्था / प्रायोजक यांचे धोरण:

या संशोधनास लागू होत नाही

सहभागासाठी आर्थिक प्रोत्साहन:

अभ्यासामध्ये भाग घेण्यासाठी आपल्याला कोणत्याही भेटवस्तू / प्रोत्साहन दिले जाणार नाहीत.

परिणाम प्रकाशित करण्यासाठी अधिकृतताः

अभ्यासाचा निकाल भाग म्हणून केएलई विद्यापीठ, बेळगाव येथे पाठविला जाईल एमडी पदवी, पुनरावलोकन आणि प्रकाशन पूर्ण करण्यासाठी आवश्यक

अभ्यासाच्या वेळी किंवा भविष्यातील प्रश्नांच्या बाबतीत आपण खालील व्यक्तींशी संपर्क साधू शकता,

REGISTRATION NO: BG0119009

सहयोगी प्राध्यापक
सामान्य औषध विभाग,
जेएनएमसी, बेळगावी. 9845710945

डॉ.
अन्वेषक,
पदव्युत्तर विद्यार्थी,
सामान्य औषध विभाग,
जेएनएमसी, बेळगावी. 8886355338

डॉ. रूपा एम बेलाड एमडी
अध्यक्ष, महाविद्यालयीन नैतिक प्रबंध
संशोधन समिती जे एन एन मेडिकल. कॉलेज
नेहरू नगर, बेलागवी - 590010

संमती फॉर्म

मी खाली स्वाक्षरी करून या अभ्यासात भाग घेण्यास स्वेच्छेने सहमत आहे. मी कधीही माघार घेऊ शकतो. या फॉर्मवर सही करून मी माझा कोणताही कायदेशीर हक्क सोडत नाही. खाली माझी स्वाक्षरी सूचित करते की मी हा संमती फॉर्म वाचला आहे किंवा हा संमती फॉर्म मला वाचला आहे आणि मला सर्व प्रश्नांची उत्तरे दिली आहेत.

सहभागी किंवा कायदेशीररित्या अधिकृत प्रतिनिधीची सही / डावा अंगठा प्रिंट

सहभागीचे नाव: .....

स्वाक्षरी / डावा अंगठा ठसा: _____ ...
सहभागीचा

कायदेशीररीत्या अधिकृत नाव: _____
प्रतिनिधी / पालक

स्वाक्षरी / डावा अंगठा ठसा: _____

साक्षीचे नाव: _____ ...

स्वाक्षरी / डावा अंगठा ठसा: _____ ...

अन्वेषकांचे नाव आणि स्वाक्षरी: _____

तारीख:

ठिकाण:

ANNEXURE II. ETHICAL CLEARANCE.

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed to be University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (Govt)

JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)

Website: http://www.jnmc.edu
E-Mail : dome@jnmc.edu

Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 315. **Date: 24/12/2019**

To,

REG.NO: BG0119009

PG student in Medicine,
J. N. Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"A CLINICAL PROFILE OF URINARY TRACT INFECTION IN TYPE 2 DIABETES MELLITUS WITH BACTERIAL CULTURE AND ANTIBIOTIC SENSITIVITY PATTERNS"**, is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Anita Dalal)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

ANNEXURE III

PROFORMA

Name: Age: Sex: M / F Hsp No:

Symptoms of UTI: (+/-)

1. No Complaints:
2. Dysuria
3. Suprapubic tenderness
4. Increased frequency
5. Urgency
6. Nocturia
7. Pyuria
8. Hematuria
9. Urinary incontinence
10. Others: - fever

Other Diabetic complications

1) Peripheral Neuropathy (Y / N Specify):
2) Retinopathy (Y / N Specify):
3) Nephropathy (Y / N Specify):
4) Diabetic Ulcers (Y / N Specify):
5) Others (Specify):

History of Diabetes Mellitus:

No: of years since diagnosed:

Treatment: Diet/ Exercise Only:

OHA:

Insulin:

Adherent to medications: Yes/ No

Routine follow up: Yes / No

Blood Sugar levels on previous follow ups: Normal/ elevated

Previously treated for/diagnosed to have UTI:

Personal History:

Married/ Unmarried:

Alcoholic/Smoker/any habits:

Bowel habits

Clinical Findings:

BP- (Supine- Standing:) Pulse: Temp: RR:

Wt: Ht: BMI

Pallor Icterus Cyanosis Oedema Clubbing

P/A:

Suprapubic tenderness:

Costovertebral angle tenderness

Mass on palpation

Others

CVS:

RS:

CNS:

Investigations:

Hb: ESR: TC : DC: N- L- E- M- B- Band:

Blood Sugars RBS: FBS: PPBS: HbA1C:

Urine RE: Specific gravity: pH: Albumin: Sugar:
. Microscopy: