A Dissertation on

"CLINICAL AND LABORATORY PROFILE OF TYPE 2 DIABETIC PATIENTS WITH ASYMPTOMATIC BACTERIURIA"

For M.D. DEGREE EXAMINATION BRANCH 1

GENERAL MEDICINE

APRIL 2017

Submitted to

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI

KILPAUK MEDICAL COLLEGE

CHENNAI

CERTIFICATE

This is to certify that this dissertation entitled "**CLINICAL AND LABORATORY PROFILE OF TYPE 2 DIABETIC PATIENTS WITH ASYMPTOMATIC BACTERIURIA**" submitted by **Dr.S.ARUNA,** Postgraduate Student in the department of General Medicine, to The Tamil Nadu Dr.M.G.R Medical University, Chennai in partial fulfilment of the requirement for the award of M.D degree Branch-1 (General Medicine) is a bonafide research work carried out by her under my direct supervision and guidance.

GUIDE:
Prof.Dr.S. MAYILVAHANAN M.D.,
Chief Medical Unit-1,
Department of Medicine,
Govt. Royapettah Hospital,
Chennai 14.

HOD:
Prof.Dr.S. USHALAKSHMI M.D.,
Department of Internal Medicine
Govt. Kilpauk Medical College & Hospital
Chennai

PROF.DR.R. NARAYANA BABU, M.D,
THE DEAN.
KILPAUK MEDICAL COLLEGE AND HOSPITAL, CHENNAI -10

DECLARATION

I, Dr.S. ARUNA declare that I carried out this work on "**CLINICAL AND LABORATORY PROFILE OF TYPE 2 DIABETIC PATIENTS WITH ASYMPTOMATIC BACTERIURIA**" at Department of Medicine, Government Royapettah Hospital during the period of April 2016 to September 2016. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, and diploma to any other university, board either in India or abroad.

This is submitted to The Tamilnadu Dr.M.G.R. Medical University, Chennai in partial fulfilment of the rules and regulation for the M.D. Degree examination in General Medicine.

DR.S. ARUNA

ACKNOWLEDGEMENT

I am extremely thankful to our beloved Dean Prof.

Dr.R. NARAYANA BABU M.D., Govt. Kilpauk Medical College, for having granted permission to do this dissertation in Government Royapettah Hospital, Chennai.

I am very grateful to our Professor and Head of the Department of Medicine Prof. & HOD **Dr.S. USHALAKSHMI M.D.,** for acceptance to do this dissertation.

I am extremely grateful to my unit chief **Dr.S. MAYILVAHANAN M.D**., who taught me the basic aspects and clinical skills in internal medicine which is an essential pre requisite for pursuing any dissertation work. The guidance and encouragement they provided need a special mention

I recall with gratitude the other unit chiefs of Department of Medicine, Prof. Dr.RAJALAKSHMI, M.D., Prof. Dr.S.SHANTHI, M.D.,

Prof. Dr.P.PARANTHAMAN DTCD, M.D., for their valuable guidance.

I am extremely thankful to our Registrar Dr.Kalaiselvi DGO,M.D., and our unit Asst.Professor Dr.S.MALATHY M.D., Dr.V.MADHAVAN M.D.,Dr.VIJAI USHA RAJ Dch, M.D., for their invaluable suggestions, guidance and support.

I thank Dr. THAMAYANTHI M.D., Assistant in Department of Microbiology for providing me with a precise Urine culture sensitivity with colony count and Microbiology work up without which this study would have not been a reality.

I thank Dr. MEERA M.D., Department of Biochemistry for providing me with facilities for accurate measurement of the biochemical parameters involved in the thesis work which was very crucial for the study.

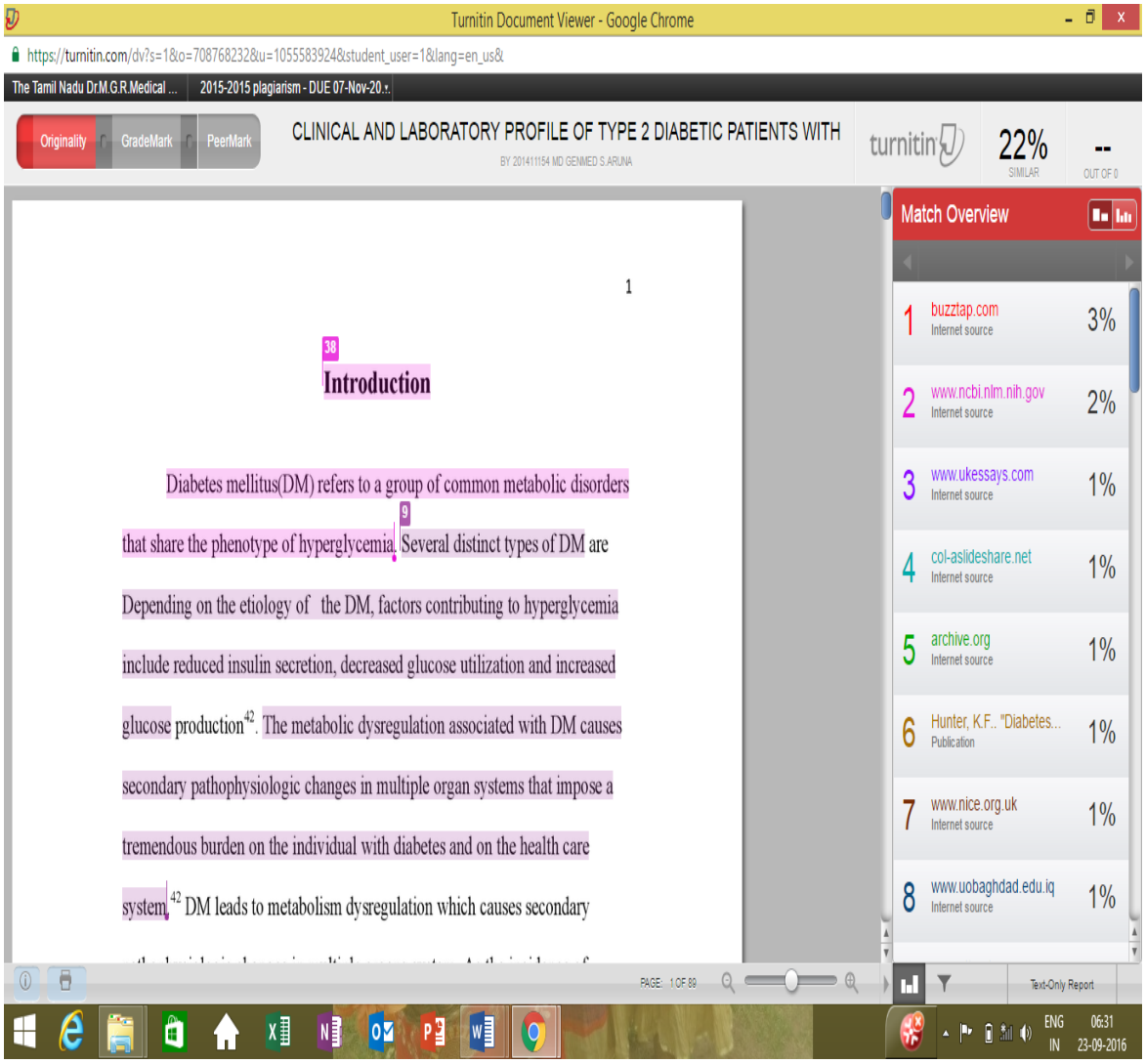
I sincerely thank the members of institutional ethical committee, Kilpauk medical college for approving my Dissertation topic. I thank Dr. Arun Murugan MD., professor (Community Medicine) for his guidance in analysing this study.

I also thank our patients without whom the study would not be possible.

I extend my love and gratitude to my family and friends for their immense help for this study.

I owe my thanks to almighty for successful completion of this study

.


Turnitin Document Viewer - Google Chrome
https://turnitin.com/dv?s=1&o=708768232&u=1055583924&student_user=1&lang=en_us&
The Tamil Nadu Dr.M.G.R.Medical ...
2015-2015 plagiarism - DUE 07-Nov-20...
Originality
GradeMark
PeerMark
CLINICAL AND LABORATORY PROFILE OF TYPE 2 DIABETIC PATIENTS WITH
BY 201411154 MD GENMED S.ARUNA
turnitin
22%
SIMILAR
--
OUT OF 0
1
Introduction
Diabetes mellitus(DM) refers to a group of common metabolic disorders that share the phenotype of hyperglycemia. Several distinct types of DM are Depending on the etiology of the DM, factors contributing to hyperglycemia include reduced insulin secretion, decreased glucose utilization and increased glucose production[42]. The metabolic dysregulation associated with DM causes secondary pathophysiologic changes in multiple organ systems that impose a tremendous burden on the individual with diabetes and on the health care system.[42] DM leads to metabolism dysregulation which causes secondary
Match Overview
1 buzztap.com Internet source 3%
2 www.ncbi.nlm.nih.gov Internet source 2%
3 www.ukessays.com Internet source 1%
4 col-aslideshare.net Internet source 1%
5 archive.org Internet source 1%
6 Hunter, K.F.. "Diabetes... Publication 1%
7 www.nice.org.uk Internet source 1%
8 www.uobaghdad.edu.iq Internet source 1%
PAGE: 1 OF 89
Text-Only Report
ENG IN
06:31
23-09-2016

INSTITUTIONAL ETHICS COMMITTEE
GOVT.KILPAUK MEDICAL COLLEGE,
CHENNAI-10
Protocol ID. No. 12/2016 Dt: 04.04.2016
CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval " **Clinical and laboratory profile of type 2 diabetic patients with asymptomatic bacteriuria** " - For Project Work submitted by **Dr.S.Aruna**, MD General Medicine, Govt. Kilpauk Medical College/GRH, Chennai – 10.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

DEAN
Govt.Kilpauk Medical College,
Chennai – 10.

CONTENTS

Introduction

Diabetes mellitus(DM) refers to a group of common metabolic disorders that share the phenotype of hyperglycemia. Several distinct types of DM are Depending on the etiology of the DM, factors contributing to hyperglycemia include reduced insulin secretion, decreased glucose utilization and increased glucose production[42]. The metabolic dysregulation associated with DM causes secondary pathophysiologic changes in multiple organ systems that impose a tremendous burden on the individual with diabetes and on the health care system.[42] DM leads to metabolism dysregulation which causes secondary pathophysiologic changes in multiple organs system. As the incidence of diabetes increasing worldwide it will continue to be a leading cause of morbidity mortality.

The complication related to diabetes can produce wide range of symptoms and signs ranging from secondary to acute hyperglycaemia to those related to chronic complications that begin to appear usually during the second decade of hyperglycaemia. Diabetes leads to several abnormalities of the host defence system that may result in higher risk of certain infection including UTI[43].These include immunologic impairment such as impaired migration, intra cellular killing, phagocytosis, chemotaxis of polymorphonuclear leukocytes from diabetic patients[44] and neuropathic complications such as impaired bladder emptying[45]. The increased glucose concentration in urine may serve as culture medium for various pathogenic microorganisms[1]

. Individuals with DM have a greater frequency and severity of infecton[42]. Urinary tract is the most common site for infection. Many common infections are more severe in the diabetic population, whereas several rare infections are seen almost exclusively in the diabetic population.[42]

Lower genitourinary tract disease in diabetic patients is of particular concern because of the perception that these patients tend to have more complicated infection of upper urinary tract. Development of ASB is much more common in diabetic women than no diabetic women. ASB is much more common in diabetic women [46]compared to diabetic men. Various risk factors for ASB in women with diabetes have been suggested including sexual intercourse, age, and duration of diabetes and metabolic control[47-53]. Also anatomic factors such as short urethra may be responsible for higher susceptibility of females to these infections.

The incidence of bacteremia due to the Enterobacteriaceae also is increased in patients with diabetes presumably because of increased incidents of urinary tract infection. Infection may be both cause and an effect of renal papillary necrosis. Renal abscess occurs with twice the frequency in persons with diabetes as in persons without diabetes. Diabetes is routinely mentioned as important risk factor in the development of perinephric abscess.

Urinary tract infections usually present with lower abdomen pain, bladder discomfort, bladder spasm, nocturia, dysuria, urgency and frequency of micturition. These symptoms may or may not be associated with constitutional symptoms like fever, fatigue and malaise. The term asymptomatic bacteriuria refers to the presence of positive urine culture in an asymptomatic person. ASB is common in neonates, pre-school children, pregnant women, elderly people and diabetics. Various studies have been conducted to analyse the risk factors for ASB in diabetic patients. Many studies have been conducted to estimate the frequency of asymptomatic bacteriuria in diabetic men and women. There have been studies which have recommended screening of patients with diabetes to detect and treat diabetes with ASB because of increased frequency and severity of upper urinary tract infections in such patients. Most of the studies done on this condition have been in Europe and North America[6]. There are hardly any reports from south India, hence this study was done in our hospital which is a tertiary care centre in south India with a view of looking into potentially modifiable risk factors like diabetic cystopathy, glycemic control, weight reduction etc.

AIMS AND OBJECTIVES OF THE STUDY

AIM

To study the prevalence and the clinical and laboratory parameters of asymptomatic bacteriuria in Type 2 diabetic patients less than 45 years.

OBJECTIVES

- To determine if there is any correlation of asymptomatic bacteriuria with age, BMI, duration of diabetes and metabolic control.
- To access the prevalence of microvascular and micro vascular complications in patients with asymptomatic bacteriuria.

Review of literature

The term diabetes mellitus describes several diseases of abnormal carbohydrate metabolism that are characterised by hyperglycemia. It is associated with a relative or absolute impairment in insulin secretion along with varying degrees of peripheral resistance to the action of insulin. Diabetes is fast gaining the status of the potential epidemic in India with more than 62 million diabetic individuals currently diagnosed with the disease[54.55]. In 2000 India topped the word with the highest no of people with diabetes mellitus followed by china and US[56]. It is predicted that by 2030 diabetes mellitus may afflict up to 79.4 million individuals in India[56,57].

The etiology of the diabetes in India is multifactorial and includes genetic factors coupled with environmental influences such as obesity, raising living standard, steady urban migration and life style changes[7]. The national urban survey conducted across the metropolitan cities of India reported prevalence of diabetes as below[58]

Metro cities	% Prevalence of Diabetes
Kolkata	11.7
Mumbai	9.3
Northern India	6.1
New Delhi	11.6
Chennai	13.5
Bangalore	12.4
Hyderabad	16.6

An upsurge in number of early onset diabetes cases is also responsible for the development of various diabetic complications due to longer disease duration[59,60]. A recent international study that diabetes control in individuals worsened with longer duration of disease[59] with neuropathy being the most common complication (24.6%) followed by cardiovascular complications (23.6%) renal issues (21.1%) retinopathy (16.6%)[58]. These results were closely in line with other results from south Indian population. Poor glycemic control observed in Indian diabetic population[61] is responsible for micro and macrovascular changes that present with diabetes and predispose to other complications such as diabetic myonecrosis[62] and muscle infarction[63]

The access to screening methods for diabetes and availability of anti-diabetic treatment is not an issue to our urban population whereas this is not the scenario in rural population. In addition to this the increased incidence of infections, improper sanitation, improper diet, inadequate screening methods, poor literacy, inadequate allotment of health resources to rural area may result in defective management of diabetes and its attendant complication in rural areas compared to urban area.

Type 1 diabetes is easily recognised. Recognition of new cases of type2 diabetes depends on severity of symptoms, diagnostic activity of medical care system and choice of diagnostic criteria.

Type 1 diabetes is a chronic autoimmune disease characterised by immune mediated destruction of the β cells of the pancreas by T cells leading to absolute deficiency of insulin. Type 1 diabetes rarely occurs during first year of life. The incidence rises sharply from age 12 to 14 then declines. Multiple susceptibility genes that confer disease risk include HLA at the MHC loci and several non-HLA loci. Evidences favour a complex interaction between genetic components and several environmental insults (virus, cow milk protein etc.) in the disease causation. Exposure to viruses such as coxsackie, cytomegalovirus, rubella, mumps in genetically susceptible individuals or dietary protein initiates a cascade of autoimmune events that leads to selective destruction of pancreatic β cells. Autoantibodies to GAD65, IA-2 and IAA appear in serum of subjects as a response to underlying destructive process. This is followed by cellular infiltration of pancreas by monocytes macrophages and T cells which initiate immune destruction. This is called 'insulitis'. Subsequent chronic destruction of β cells brings about a stage of absolute insulin deficiency and at clinical presentation almost 60-70% of pancreatic β cells are destroyed. Candidate genes associated with Type 1 DM are INS VNTR(11p), CTLA4(2q), PTPN22(1p), CD4(12p), IRS-1 (2q), VDR(12q).

T2DM is due to imbalance between energy intake and expenditure and is regulated by complex interaction between multiple genes and environmental factors. The heritability of T2DM is high. The major risk factor is high calorie food intake with sedentary lifestyle which is linked to development of obesity and T2DM. Micronutrient imbalances such as Vitamin D. Vitamin B12 and

increased iron stores have been implicated in the pathogenesis of T2DM. Intrauterine growth retardation low birth weight by multiple mechanisms may lead to greater propensity of T2DM in adult life.

DIAGNOSTIC CRITERIA FOR DIABETES

WHO criteria[8]**:**

2006 WHO criteria defines diabetes as an

FPG ≥126 mg/dl

2 hrs glucose challenge value ≥ 200mg/dl

HbA1C>6.5% and

Impaired glucose tolerance test (IGT):

FBS< 126mg/dl

Two hr glucose ≥140 mg/dl but less than 200 mg/dl

Impaired fasting glucose is defined as FBS of 110-125mg/dl

ADA criteria[9]:

In 2003 ADA defines

Normal- FPG <100 mg/dl, two-hour glucose during OGTT< 140 mg/dl

IFG: FPG between 100-125 mg/dl

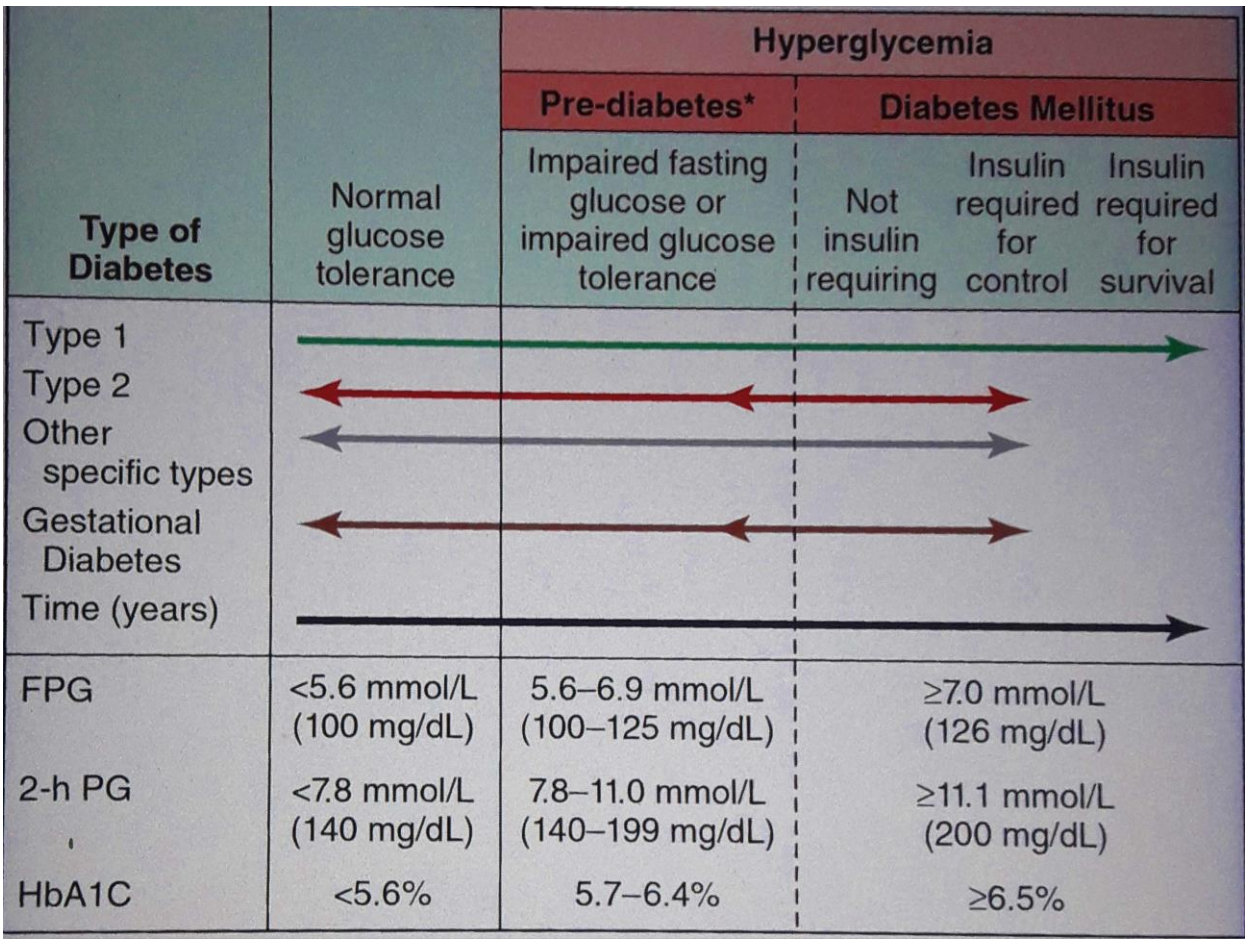
IGT- Two-hour plasma glucose value during a 75 gm OGT between 140-199 mg/dl

Diabetes mellitus:

FPG≥126 mg/dl and two-hour value in a OGTT≥200mg/dl and HbA1C ≥ 6.5%in a patient with classical symptoms

Or

Symptoms of hyperglycemia and a casual blood sugar of ≥200 mg/dl. Casual is defined as any time of the day without regards to time of last meal.[64]



INFECTIONS IN DIABETES MELLITUS

There are host and organism specific factors that may explain why people with diabetes are more susceptible to infections

HOST FACTORS

1)Impairment of immune response: Hyperglycemia impairs neutrophil chemotaxis and adherence to vascular endothelium. Opsonophagocytosis is impaired because NAPH is diverted from superoxide production into the polyol pathway. Intracellular bactericidal activity and cell mediated immunity are also depressed in diabetic patients.

2)Vascular insufficiency resulting in local tissue ischemia enhance the growth of microaerophilic and anaerobic organism. Vascular disease related to diabetes may also impair the local inflammatory response and the absorption of antibiotics.

3)Sensory neuropathy: Unnoticed local trauma in patients with diabetes associated peripheral neuropathy may result in skin ulcer resulting in diabetic foot infections.

4)Autonomic neuropathy: Bladder involvement leads to urinary retention and stasis which in turn leads to development of urinary tract infection.

5)Increased skin and mucosal colonisation: patients with diabetes have asymptomatic nasal and skin colonisation with S.aureus which are more likely to be methicillin resistant. This colonisation leads to transient bacteremia resulting in infections at distant site. Mucosal colonisation with candida albicans is also common.

ORGANISM SPECIFIC FACTORS

1)E.coli that cause invasive symptomatic infections of the urinary tract in otherwise normal hosts often possess and express surface adhesins that mediate binding to specific receptors on surface of uroepithelial cells. The best studied adhesin are p fimbriae and are important in the pathogenesis of pyelonephritis.

Another adhesion is the type 1 pilus which all E.coli strains possess but not all E.coli express. They play a key role in initiating E.coli bladder infection.

They mediate binding to uroplakins on the luminal surface of bladder uroepithelial cells.

2)Candida albicans has glucose inducible proteins which promotes adhesion and this impairs phagocytosis.

3)Rhizopus has ketone reductases which help them thrive in high glucose acidic conditions.

INFECTIONS WITH AN INCREASED PREVELANCE IN DIABETICS

HEAD AND NECK:

Oral and esophageal candidiasis.

GENITOURINARY:

Bacteriuria and cystitis in women

Pyelonephritis and perinephric abscess

SKIN AND SOFT TISSUE:

Surgical site infection

Cellulitis and osteomyelitis of the extremities, pyomyositis

PULMONARY:

Tuberculosis, staphylococcal and gram negative pneumonia.

ABDOMINAL:

Emphysematous cholecystitis

Infections with salmonella enteritidis, campylobacter jejuni, listeria monocytogenes.[65]

INFECTIONS UNIQUE TO PATIENTS WITH DIABETES

HEAD AND NECK:

Rhinocerebral mucormycosis, malignant otitis externa[65].

URINARY TRACT:

Emphysematous cystitis, emphysematous pyelitis, pyelonephritis[65]

SKIN AND SOFT TISSUE:[65]

Synergistic necrotising cellulitis, fourniers gangrene.

URINARY TRACT INFECTIONS IN DIABETES MELLITUS

There is roughly a fivefold greater propensity towards urinary tract infections in diabetic women. Urinary tract infections tend to be more severe in diabetics than non-diabetics. There is an increased risk of complications involved.

ASYMPTOMATIC BACTERIURIA

Approximately half decade ago quantitative culture for etiological diagnosis of urinary tract infections was done by kass and other investigators[10,11,12]. Extensive use of urine culture identified several patients who were clinically asymptomatic but had a high prevalence of positive urine culture[13]. These included antenatal women and persons with urological abnormalities, patients with indwelling urinary catheters, elderly individuals and diabetics. Increased prevalence of asymptomatic bacteriuria (ASB) was generally found in older individuals and also non ambulant patients. The prevalence of ASB in various groups of patients is shown in the following table.

Prevalence of Asymptomatic bacteriuria in different populations

Population	Prevalence (%)
Healthy premenopausal women[14]	1.0 to 5.0
Pregnant women[14]	1.9 to 9.5
Post-menopausal women[14] (50-70 yrs)	2.8 to 8.6
Patients with Diabetes	
Women[15]	9.0 to 27.0
Men[15]	0.7 to 1.0
Older communities dwelling patients	
Women[14] (>70 yrs.)	>15
Men[15]	3.6 to 19.0
Older long term care patients	
Women[15]	25 to 50
Men[15]	15 to 40
Patients with spinal cord injuries[16]	
Intermittent catheter	23 to 89
Sphincterotomy and condom catheter[17]	57
Patients undergoing haemodialysis[18]	28
Patients with indwelling catheter	
Short term[19]	9 to 23
Long term[19]	100

DEFINITION OF ASYMPTOMATIC BACTERIURIA

Asymptomatic bacteriuria refers to the presence of high quantities of uropathogen in the urine of a asymptomatic person.

The 2005 IDSA guidelines[20] recommend the following guidelines for diagnosis of ASB in adults:

1)For asymptomatic women bacteriuria is defined as two consecutive clean-catch voided urine specimens with isolation of the same bacterial strain in counts$\geq 10^5$ cfu/ml.[67]

2) For men, a single voided specimen with a quantitative count of a potential uropathogen of ≥105 CFU/mL is sufficient to diagnose bacteriuria[66].

3)For any asymptomatic patient's bacteriuria is defined as a single catheterised urine specimen with bacterial isolates in count$\geq 10^2$cfu/ml[67].

Studies that define a bacteriuria with a single voided specimen have reported a higher prevalence of ASB than those requiring persistent bacteriuria of two or more specimens.

ASB often precedes symptomatic urinary tract infection in diabetes and the risk of progression to upper urinary tract infection is higher. The overall prevalence of ASB is about 26% in diabetic women. Routine screening of women with diabetes is not considered cost effective. E.coli

and other gram negative organism are the usual causative organisms. Antibiotic therapy doesn't affect the frequency or time to symptomatic UTI including pyelonephritis. Reinfection/ recurrence is common once antibiotic is discontinued. Long term prognosis is not affected. More over persistent ASB is not associated with detoriation of renal functions.

PATHOPHYSIOLOGY OF ASB

The absence of symptoms in ASB can be due to specific characteristics of bacteria, host or both. Fimbrial adhesion of bacteria is very important in causation of symptomatic infection. Some bacterial strains with reduced capability for fimbriae expression have capacity for relatively rapid growth and thus may cause ASB.

Alternatively, the strains implicated in the causation of ASB may be less virulent. E.coli strains recovered from spinal cord injury patients with asymptomatic bacteriuria demonstrated diminished capacity for RBC hemagglutination and hemolysis than strains implicated in symptomatic urinary tract infections. Such strains even on persistence are unlikely to cause serious infections.

The absence of symptoms in patient with ASB can also reflect differences in host response. A study of children with ASB demonstrated lower levels of neutrophil Toll-like receptor 4(TLR4) expression compared to age matched controls.

BACTERIOLOGY OF ASB

The bacteriology of ASB in diabetics and nondiabetics are similar with the preponderance being caused by Escherichia coli and other gram negative organisms.

SCREENING FOR ASYMPTOMATIC BACTERIURIA

Screening for ASB is not recommended except in second trimester of pregnancy or before an urological procedure with risk of mucosal injury[20].

IDSA RECOMMENDTAIONS FOR SCREENING & TREATMENT OF ASB[20]

Recommendation[20]	**Grade**
Pregnant women should be screened for bacteriuria by urine culture atleast once in early pregnancy and they should be treated if results are positive[20]	A-1
Screening for and treatment of ASB before transurethral resection of the prostate is recommended[20]	A-1
Screening for and treatment of ASB is recommended before other urological procedures for which mucosal bleed is anticipated[20]	A-III

IDSA RECOMMENDATIONS AGAINST SCREENING FOR & TREATMENT OF ASB[20]

Recommendation[20]	**Level[20]**
Premenopausal, non-pregnant women	A-I
Diabetic women	A-II
Older people living in the community	A-I
Elderly institutionalised people	A-I
People with spinal cord injury	A-I
People with indwelling catheter	A-I

Strength of recommendation[20]

A: Good evidence to support a recommendation; recommendation always valid.

B: Moderate evidence to support a recommendation; it is valid in most cases.

C: Poor evidence to support a recommendation; optional

D: Moderate evidence against a recommendation; generally, not offered.

E: Good evidence against a recommendation; never be offered.

Quality of evidence[20]

I: Evidence from ≥ 1 properly randomised control trial

II: From cohort or case control analytic studies.

III: Opinions of experts, clinical experience, descriptive studies in absence of appropriate clinical trials.

CONSEQUENCES OF ASYMPTOMATIC BACTERIURIA IN DIABETES MELLITUS

ASB identifies a high risk group of diabetics for subsequent severe urinary tract infections. In one study a large cohort of diabetic women in Netherlands was studied to determine the incidence of symptomatic UTIs. In women with type 2 diabetes the presence of ASB increased the risk of subsequent UTI in 18 month follow up period from 19-34 percent. The rate of ASB in this population was approximately 28 percent. Incidence of ASB was only 6 % in non-diabetics.[68]

SYMPTOMATIC UTI IN DIABETICS

CYSTITIS

Cystitis is diagnosed by the triad of suprapubic pain frequency and urgency of micturition. The presence of fever, flank pain, costovertebral angle tenderness and nausea or vomiting suggests an upper urinary tract infection and warrant

more aggressive diagnostic and therapeutic measures. The bacteriology is similar to ASB.

ACUTE PYELONEPHRITIS

Pyelonephritis is more common with diabetics and is more likely to involve both kidneys. There is increased risk of complications like perinephric abscess, emphysematous pyelonephritis and renal papillary necrosis. ESBL are emerging pathogens making them resistant to third generation cephalosporins like cefotaxime, ceftazidime. Carbapenems are most effective as they are highly resistant to hydrolytic activity of all ESBL enzymes.

RENAL PAPILLARY NECROSIS

When renal pyramids get infected due to vascular disease of the kidney or urinary tract obstruction it results in renal papillary necrosis. This type of infection is more common with diabetics. Renal papillary necrosis occurs bilaterally. If there is sudden detoriation of renal functions in a diabetic the diagnosis of renal papillary necrosis should be sought even in the absence of pain or fever.

PERINEPHRIC AND RENAL ABSCESS

Before the era of antibiotics most renal and perirenal abscess were hematogenous in origin. Staphylococcus aureus was most commonly isolated

organism. Nowadays >75% of these abscesses arises from an ascending infection of urinary. Many of these patients may have associated nephrolithiasis. Organism encountered now a days are E coli, Proteus, Klebsiella species. The virulence properties of E.coli promotes adherence to uroepithelial cells. Bacterial proliferation is also enhanced by the urease splitting action of proteus thus making urine more alkaline

EMPHYSEMATOUS PYELONEPHRITIS

This severe form of acute multifocal bacterial nephritis occurs almost exclusively in diabetics. E.coli and gram negative coliforms are the usual causative organism[69]. In addition to features of acute pyelonephritis patient may present with flank mass and sometimes crepitus may be elicited. Gas can be demonstrated on x ray plain film but CT scan is better at localising the extent of involvement.

ASB identifies a group of diabetic patients who are at significantly increased risk of urosepsis and requiring hospitalisation. However there is consensus proving in the absence of anatomical and/or functional abnormalities of the urinary tract ASB does not lead to hypertension, renal scarring or renal dysfunction. A 14 year follow up of asymptomatic and symptomatic diabetic patients was done to look at natural history of untreated asymptomatic bacteriuria. On observation it was found that there was no significant difference

in the major clinical symptoms of acute pyelonephritis and there was no resultant detoriation of renal function. In addition it was also found that the occurrence of arterial hypertension also did not differ in both groups.[49]

RISK FACTORS FOR ASYMPTOMATIC BACTERIURIA

A prospective study in which 796 sexually active nonpregnant women from 18 through 40 years were evaluated for a period of over six months for the occurrence of ASB concluded that ASB was associated with similar risk factors as for symptomatic urinary tract infection[22]

The factors that have been correlated with ASB in Type 2 Diabetes are female sex preponderence , urinary incontinence, leucocyturia and in addition elevated C reactive protein.[23,24]

A prospective cohort study in which 218 diabetics and 799 non diabetics post menopausal women were examined for risk factors for ASB and UTI. Increased risk occurs in women taking insulin(relative risk 3.7) and those with a longer duration of diabetes (> 10 years, relative risk 2.6)[70]. Women with diabetes and concomitant bacteriuria are characterised by longer duration of diabetes and associated long term complications such as diabetic neuropathy but not with abnormal parameters of diabetic control[25]

There have been studies that has refuted the concept that ASB is associated with increased risk. In another study done which compared the incidence of symptomatic UTI in patients with diabetes with or without ASB to identify

other associated risk factors for these infections, totally 289 females and 168 males were studied in a period of 12 month.[26]

Symptomatic UTI developed in 69.2% of patients with ASB (67.6% were female and 76.5% were males) versus 9.8% without ASB (14.9% were female and 2.6% were male). There was increased incidence of urinary incontinence in both patients with ASB and UTI. Other risk factors which were studied were previous antimicrobial therapy and the presence of macrovascular complications. The presence of ASB in diabetics was found to be important risk factor for the development of symptomatic urinary tract infection.

GUIDELINES FOR TREATMENT OF ASYMPTOMATIC BACTERIURIA

Whether or not treatment for ASB is needed is a dilemma. There have been several studies which show that the benefit of eliminating ASB doesn't persist. Treatment of ASB doesn't decrease subsequent infection[27]. There is also a increased risk of emergence of resistant infection which can have detrimental effect. Hence treatment of ASB in diabetic patients is not recommended based on several studies which have not shown improved outcome with therapy.

A prospective trial conducted with 105 diabetic women with ASB. They were randomly assigned to either antibiotic or placebo for 14 days[28]. It was found that at the end of 4 weeks increased proportion of patients in antibiotic group were free of bacteriuria. These patients were followed for next 27 months.

They were tested for ASB every three months for nearly 27 months. These patients were also evaluated for symptomatic UTI. There was no significant difference between development of symptomatic UTI and other associated parameters.

In addition, patients in the antibiotic group had nearly five additional days of antibiotic therapy in addition to control group. Hence it was concluded that antibiotic therapy for ASB in diabetic patients doesn't affect the development of symptomatic infection or reinfection.

POST VOID RESIDUAL URINE

PVR (post void residual urine) is defined as volume of urine remaining in the bladder at end of micturition. Usually a volume of less than 50 ml is considered normal. A PVR of more than 200 ml is usually considered abnormal. When the PVR is between 50- 200ml it has to correlated in context with clinical; scenario[29]

Estimation of residual urine is usually done by the following method:

1)Transurethral or suprapubic catheter

2)radiography

3)ultrasound.

4)radioisotopes

The use of diuretics, the presence of vesicourethral reflex may elevate the residual urine volume. Bladder diverticulum poses problems with interpretation since diverticulum can be regarded either as a part of bladder cavity or outside functioning bladder. Each method of estimation of residual volume has its own limitations. The mere absence of PVR doesn't rule out bladder dysfunction or infravesical obstruction. The presence of significant PVR warrants further testing.

BLADDER DYSFUNCTION

Bladder dysfunction is considered a common complication of diabetes and is characterized by impaired bladder sensation, decreased detrusor contractility and increased bladder capacity. This results in increased residual volume, retention of urine and overflow incontinence.[30]

Several clinical studies have demonstrated that diabetic neuropathy such a as gastroparesis, orthostatic hypotension and abnormal sweating were frequently found in patients with diabetic cystopathy[31]. It has been usually agreed that peripheral neuropathy plays an important role in the development of diabetic cystopathy.

The bladder capacity values varied among various studies. In 53 patients investigated for bladder dysfunction 36% had increase in bladder capacity of more than 400ml with a flat trace on cystometrography according to the criteria proposed by Kahan et al[32] In other clinical studies the rates of bladder

dysfunction varied from 30-80%[33]. Bladder dysfunction was observed in patients without voiding symptoms, early stage of diabetes like with patients with diabetes of duration of less than 12 months. It was also observed in patients without diabetic retinopathy and on treatment with diet only.[34]

Diabetic retinopathy is considered as a chronic complication of diabetes with insidious onset and increased length of time between voiding and it affects the quality of day to day living. Prevalence of diabetic cystopathy is estimated to be between 32% to 45%[35,36] Diabetic cystopathy is assosciated with classic symptoms including decreased bladder sensation, impaired detrusor contractility and increased bladder capacity[37]. All these together result in increased PVR[38]. Increased PVR makes the patient more prone for UTIs.

Diabetic cystopathy is usually described as a manifestation of autonomic neuropathy[36,39] in addition to cardiovascular and gastrointestinal neuropathies. Autonomic neuropathy are considered as serious and irreversible complications of diabetes.

Axonal degeneration, demyelination and fibre loss occurs in diabetic autonomic neuropathy. Hyperglycemia is indicated as the main contributor for development of neuropathic complications. Tight blood glucose control can decrease long term microvascular and neurological complication of diabetes.[40]

The increased polyol pathway activity due to glucotoxicity in nervous tissue leads to accumulation of sorbitol and fructose which induces damage. The exact mechanism of such damage has not been elucidated.

Since diabetic cystopathy has been considered as a complication of autonomic neuropathy screening of other signs of autonomic neuropathy has to be included particularly heart rate variability and orthostatic hypotension. Since the hall mark of diabetic cystopathy has been considered the presence of increased PVR, the residual urine by ultrasound within minutes of voiding.one study has suggested the normal range of PVR as between 50ml-150ml.[41]

Best of all intervention for diabetic cystopathy is glycemic control, preventing further progression of diabetic neuropathy, relief of symptoms, prevention of infections and adequate bladder emptying.

MANAGEMENT OF DIABETIC CYSTOPATHY

Glycemic control	Diet, lifestyle modifications, OHA, Insulin
Voiding strategies	Scheduled toileting, Double voiding, Bladder expression, follow up PVR to assess bladder emptying.
Catheterization	Intermittent catheterization, Indwelling catheterization(as last resort)
Nocturnal polyuria	Fluid restriction, avoidance of caffeine, bladder emptying before going to bed.

Encouraging the patient to void every two to four hours during day along with double voiding technique improves bladder emptying, decreases PVR and prevents infection. No effective medications are available for management of diabetic cystopathy. Bethanechol a parasympathomimetic agent has given inconsistent results. Other drugs such as aldolase reductase inhibitors (Eprilistat, Fidarestat) which inhibits accumulation of fructose and sorbitol and thus potentially improves neuropathy.

TESTS FOR DETECTING URINARY INFECTION

1)Grams staining: It is least expensive and very reliable for screening. Presence of one or more bacteria per oil immersion field correlates with $\geq 10^5$/ml on culture with a sensitivity and specificity of over 90%.[71]

2)Griess Nitrite test: Calorimetric detection of nitrite provides a simple and rapid method for detecting bacteriuria. The major disadvantage of this test is lack of sensitivity. False negative tests can occur with urobilinogen, ascorbic acid low Ph[71]

3)Leucocyte esterase test: This test detects the esterases which are releases from degraded white blood cells. It is a very rapid test and doesn't require much technical expertise.

4)Catalase test: Most uropathogens generate catalase. If hydrogen peroxidase is added to infected urine bubbles of oxygen are then released. [71]

5)Glucose oxidase test: This test is based on the fact that bacteria metabolises glucose normally present in urine. So in the presence of infection glucose is not detected in urine.

QUANTITATIVE URINE CULTURE:

Conventional microbiological quantitation of bacteriuria is performed by streaking urine from a calibrated platinum loop onto agar plates, incubating at 37°C for 24 hours, and then counting the number of bacterial colonies[72]. Other methods include the accurate but time consuming pour plate method and the dip inoculum method.[71]

MATERIALS AND METHODS

Setting: 90 diabetic patients <45 years attending the diabetic opd in Government Royapettah Hospital to satisfy the inclusion criteria were recruited for the study.

Duration of study: 6 months

Inclusion criteria: Patients with Type 2 diabetes age <45 years and gave written voluntary consent were recruited for the study

Exclusion criteria:

1.Patients with features of lower urinary tract infection (dysuria with frequency or urgency)

2.Patients who had taken antibiotics in the previous two weeks.

3.Women with history of sexual intercourse one week prior.

4.Pregnancy.

5.Instrumentation of the urogenital tract in the previous two months

6.Recent hospitalisation or surgery in past 4 months.

7.Gynaecological infections.

8.Patients with history of uretric/renal calculus.

9.Subjects unwilling to participate are voluntarily excluded.

Ethical approval:

The study had the approval of the medical and ethical committee for research.

Study design:

The study was a cross-sectional comparative study on asymptomatic bacteriuria in Type 2 Diabetic patients < 45 years attending the diabetology opd of our centre. Non diabetic patients < 45 years were taken as control.

Sample size:

The study was conducted on 90 diabetic patients age <45 years and 90 controls of <45 years.

Study protocol:

During initial visit relevant details and history regarding the patients are collected like age, duration of diabetes, medications , pregnancy , history of hospitalisation , catheterization , surgery, history pertaining to urinary symptoms and gynaecological infections like dysuria , frequency of micturition, history of white discharge of pruritis vulva.

Relevant investigations like fasting and post prandial sugar , urine analysis, urine culture and sensitivity , renal function tests, ultra sound for PVR, urine

microalbuminuria, screening for diabetic retinopathy and neuropathy to be carried out (Questionnaire attached)

Urine Culture:

Urine was read for growth post incubation at 32°C for 24 hours on blood and McConkey agar. The urine cultures that showed growth of more than 10^5 colonies / ml were considered as asymptomatic bacteriuria.

Definitions:

1.Diabetic mellitus: Any patient who is on hypoglycaemic agents / insulin was considered to be diabetic or any subject fulfilling the ADA criteria for diabetes.

Fasting ≥ 126 mg/decilitre or

2 hours post prandial sugars ≥ 200 mg / decilitre or

Symptoms of diabetic plus random blood sugar ≥ 200 mg / decilitre.

2. Body Mass Index :

a. Underweight if BMI < 18 kg/m^2

b. If BMI was between 19 & 24 kg/m^2

c. Overweight BMI was between 25 & 29 kg/m^2

d. Obese if BMI was more than 30 kg/m^2

3. Peripheral Neuropathy:

Presence of atleast 4 of the following symptoms: Pain , burning, pricking , numbness, or tingling sensations in the feet, disturbances in pinprick or light touch sense of foot abnormalities.

4. Nephropathy[73]:

Microalbuminuria was present if the urine microalbumin was between 30 and 300 microgm/mg of creatinine or 30- 300 mg/day in a 24 hour urine collection. -macroproteinuria. If urine microalbumin >300 microgm/mg of creatinine or 24 hour urine protein was more than 500mg/24 hours.

5. Retinopathy:- Present or absent as confirmed by an Ophthalmologist .The diagnosis was made in the presence of microaneurysms, dot and blot hemorrhages and evidence of clinically significant macular edema, or any patient who LASER/ intervention for retinal detachment/ vitreous hemorrhage.

6. Cardiovascular disease: - Any of the following features were taken:

a) Past history of acute coronary syndrome

b) Stable angina

c) History of PTCA/ Coronary artery bypass grafting

d) Tread Mill Test (TMT) positivity

7. Cerebrovascular disease: - Any of the following features:

a) History of transient ischemic attack/ stroke

b) Carotid stenosis- either carotid bruit or Doppler proven

8. Peripheral vascular disease: - Any of the following features:

a) Absent peripheral pulses

b) Claudication pain

c) History of gangrene/ amputation

9. Renovascular disease: - Any of the following features:

a) Renal bruit

b) Doppler evidence of renal artery stenosis

10. Obstructive uropathy /cystopathy:-This was defined as per the standard urologic terminology of the International Continence Society guidelines .Abnormal post void residual urine was defined as PVR more than 10% of the voided volume measured by ultrasound.

.11. Asymptomatic bacteriuria:-Defined as the presence of at least 105 colony forming units /ml of 1 or 2 of the same microorganism in a culture of clean voided midstream urine from a patient without fever or a symptoms of a urinary tract infection

STATISTICAL ANALYSIS OF DATA

Groups – Cases Vs Controls

Groups	Definition	Number
Cases	Type 2 Diabetes patients age<45	90
Controls	Non Diabetics patients age<45	90

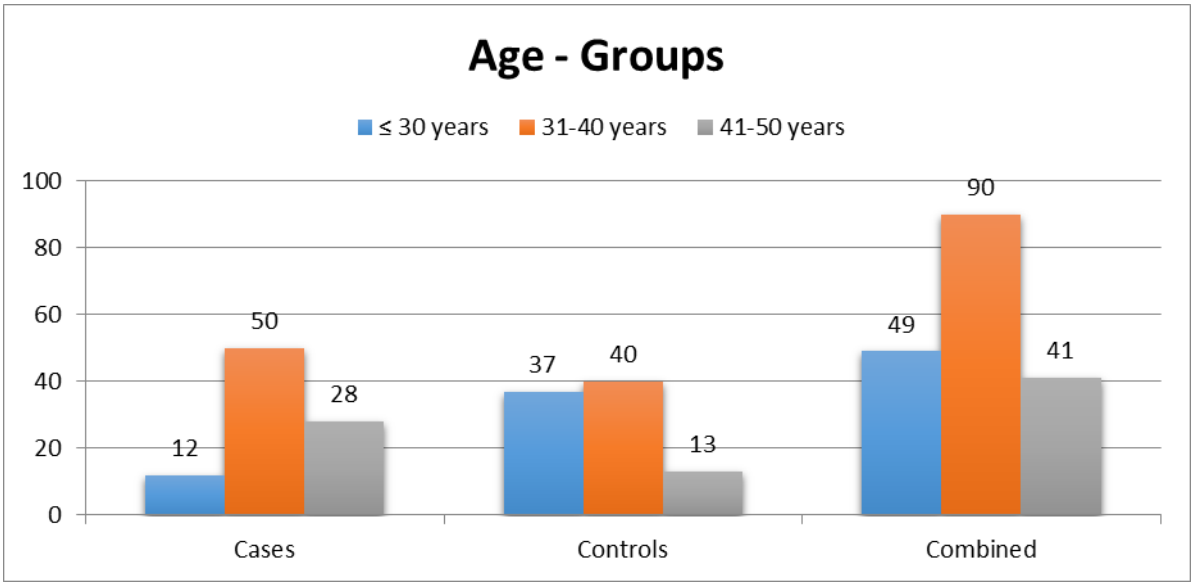
Null Hypothesis

Null Hypothesis : H0	Cases equal in effect compared to controls in relation to ASB
Alternate Hypothesis : H1	Cases have more hazardous effect compared to controls in relation to ASB

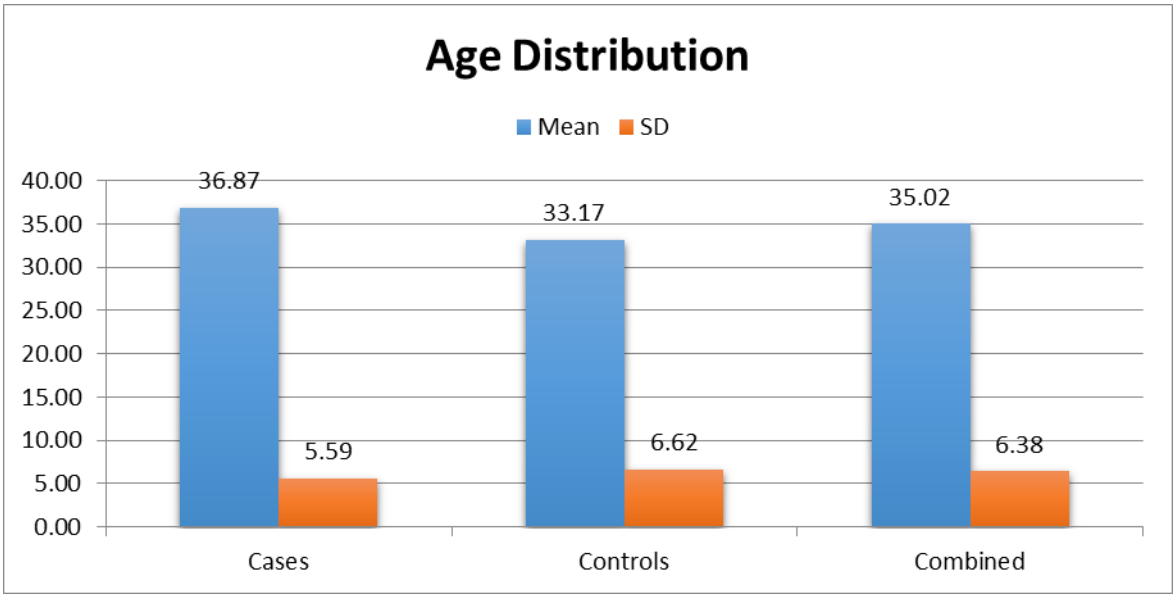
Data Analysis

Descriptive statistics was done for all data and were reported in terms of mean values and percentages. Suitable statistical tests of comparison were done. Continuous variables were analysed with the unpaired t test.. Categorical variables were analysed with the Chi-Square Test and Fisher Exact Test. Statistical significance was taken as $P < 0.05$. The data was analysed using SPSS version 16 and Microsoft Excel 2007.

Age



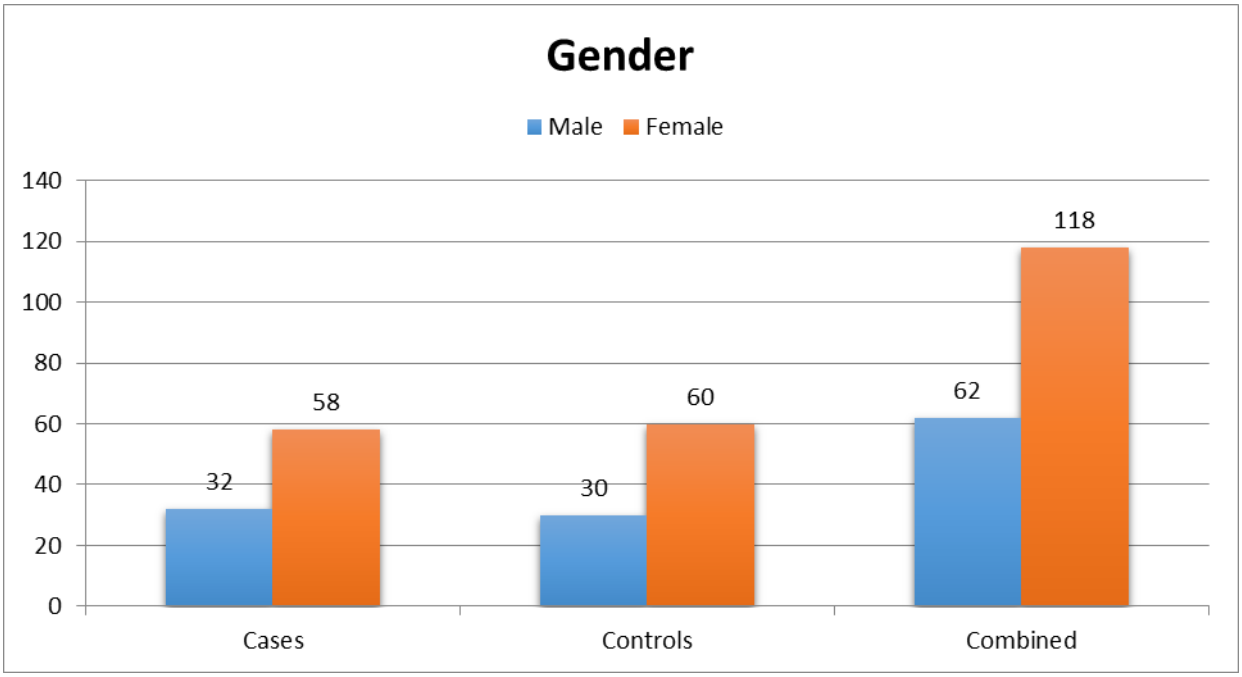
Age – Groups	Cases	Controls	Combined	Cases %	Controls %	Combined %
≤ 30 years	12	37	49	13.33	41.11	27.22
31-40 years	50	40	90	55.56	44.44	50.00
41-50 years	28	13	41	31.11	14.44	22.78
Total	90	90	180	100	100	100



Age Distribution	Cases	Controls	Combined
Mean	36.87	33.17	35.02
SD	5.59	6.62	6.38
P value Unpaired t Test			0.1123

Among the study patients, there was no statistically significant difference in relation to age distribution between cases group (mean=36.87, SD=5.59) and control group (mean=33.17, SD=6.62) with a p value of <0.05 as per unpaired t test. Therefore we fail to reject the null hypothesis that there is no difference in age distribution between the study groups.

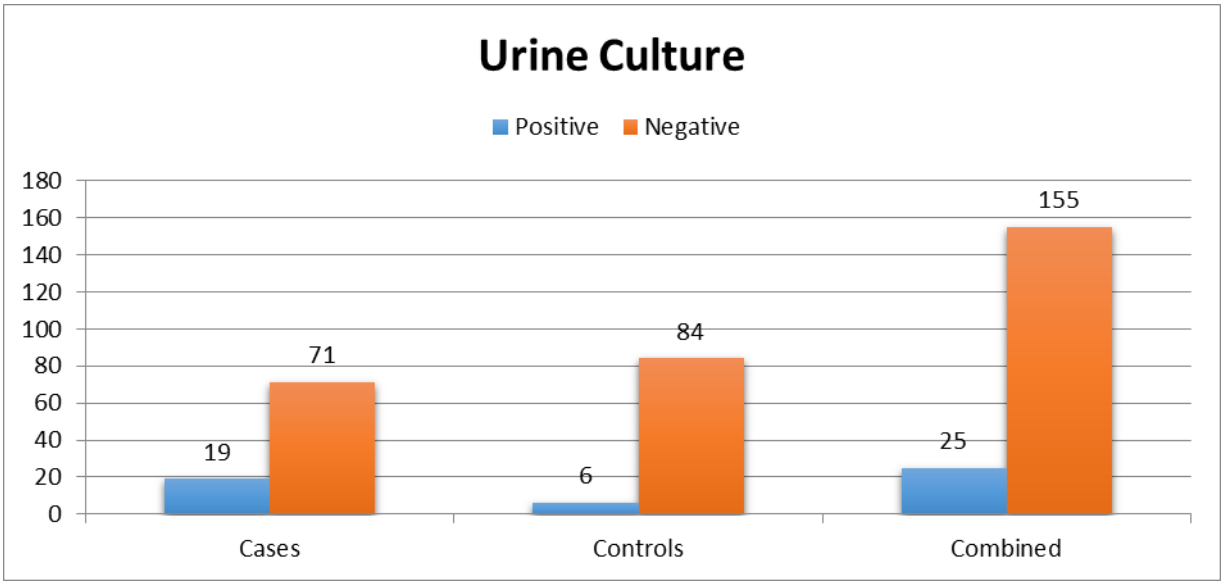
Gender



Gender	Cases	Controls	Combined	Cases %	Controls %	Combined %
Male	32	30	62	35.56	33.33	34.44
Female	58	60	118	64.44	66.67	65.56
Total	90	90	180	100	100	100
P value Chi Squared Test				0.7537		

Among the study patients, there was no statistically significant difference in relation to gender status between cases group (majority are females – 64.44%) and control group (majority are females – 60%) with a p value of <0.05 as per chi squared test. Therefore we fail to reject the null hypothesis that there is no difference in gender status between the study groups.

Urine Culture



Urine Culture	Cases	Controls	Combined	Cases %	Controls %	Combined %
Positive	19	6	25	21.11	6.67	13.89
Negative	71	84	155	78.89	93.33	86.11
Total	90	90	180	100	100	100
P value Chi Squared Test				0.0051		

Among the study patients, there was a statistically significant difference in relation to urine culture status between cases group (majority are negative – 78.89%) and control group (majority are negative – 93.33%) with a p value of <0.05 as per chi squared test. Therefore we reject the null hypothesis that there is no difference in urine culture status between the study groups.

Discussion

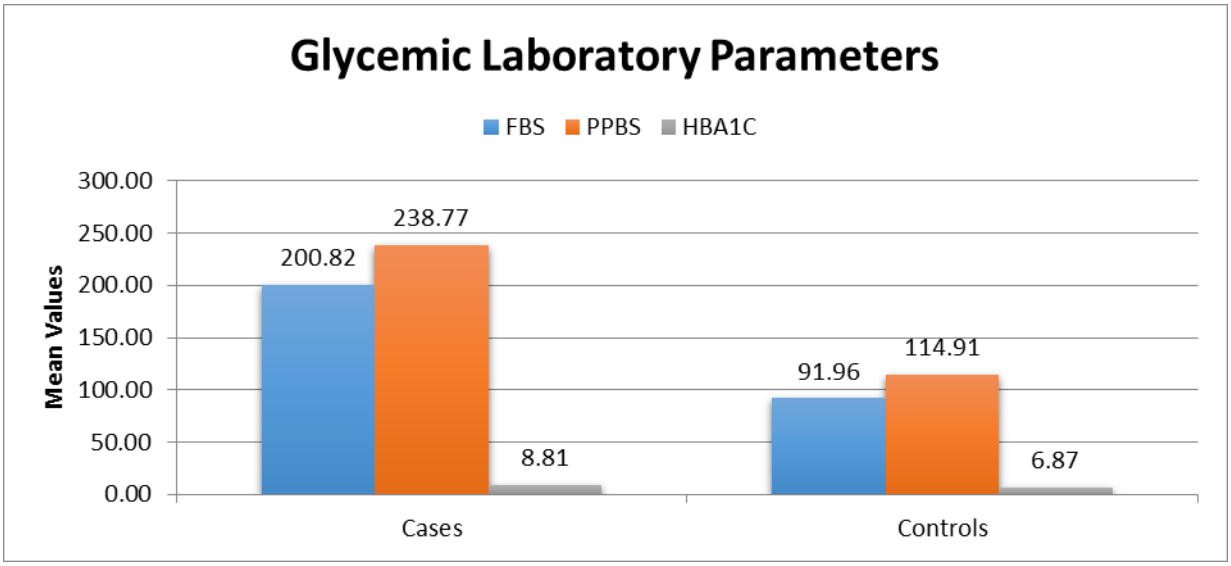
The incidence of urine culture positivity was significantly more in cases group compared to control group by a percentage difference of 14.44 percentage points (68% higher). This difference is significant with a p-value of 0.0051 as per chi squared test.

Conclusion

In this study we can safely conclude that a significant increase in urine culture positivity is associated with type 2 diabetics below age 45 compared to non diabetics below age 45. . Geerling et all in 2000 reported a prevalence of 26%[74]. Our study was similar to earlier studies with prevalence of ASB being 21%.

In other words urine culture positivity was 3.17 times more common in type 2 diabetics below age 45 compared to non diabetics below age 45

Glycemic Laboratory Parameters



Glycemic Laboratory Parameters		FBS	PPBS	HBA1C
Cases	Mean	200.82	238.77	8.81
	SD	61.94	55.89	1.20
Controls	Mean	91.96	114.91	6.87
	SD	10.09	11.90	0.26
P value Unpaired t Test		<0.0001	<0.0001	<0.0001

FBS

Among the study patients, there was a statistically significant difference in relation to fasting blood sugar distribution between cases group (mean=200.82, SD=61.94) and control group (mean=36.87, SD=10.09) with a p value of <0.05 as per unpaired t test. Therefore we reject the null hypothesis that there is no difference in fasting blood sugar distribution between the study groups.

Discussion

The mean FBS was significantly more in cases group compared to control group by a mean difference of 108.86 mg/dl (44% higher). This difference is significant with a p-value of <0.0001 as per unpaired t test.

Conclusion

In this study we can safely conclude that a significant increase in fasting blood sugar is associated with type 2 diabetics below age 45 compared to non diabetics below age 45 among our study subjects without features of lower UTI

In other words elevated fasting blood sugar levels was 2.18 times more common in type 2 diabetics below age 45 compared to non diabetics below age 45 among our study subjects without features of lower UTI

PPBS

Among the study patients, there was a statistically significant difference in relation to post prandial blood sugar distribution between cases group (mean=238.77, SD=55.89) and control group (mean=114.91, SD=11.90) with a p value of <0.05 as per unpaired t test. Therefore we reject the null hypothesis that there is no difference in post prandial blood sugar distribution between the study groups.

Discussion

The mean PPBS was significantly more in cases group compared to control group by a mean difference of 123.86 mg/dl (42% higher). This difference is significant with a p-value of <0.0001 as per unpaired t test.

Conclusion

In this study we can safely conclude that a significant increase in post prandial blood sugar is associated with type 2 diabetics below age 45 compared to non diabetics below age 45 among our study subjects without features of lower UTI

In other words elevated post prandial blood sugar levels was 2.08 times more common in type 2 diabetics below age 45 compared to non diabetics below age 45 among our study subjects without features of lower UTI

HBA1C

Among the study patients, there was a statistically significant difference in relation to HBA1C distribution between cases group (mean=8.81, SD=1.20) and control group (mean=6.87, SD=0.26) with a p value of <0.05 as per unpaired t test. Therefore we reject the null hypothesis that there is no difference in HBA1C distribution between the study groups.

Discussion

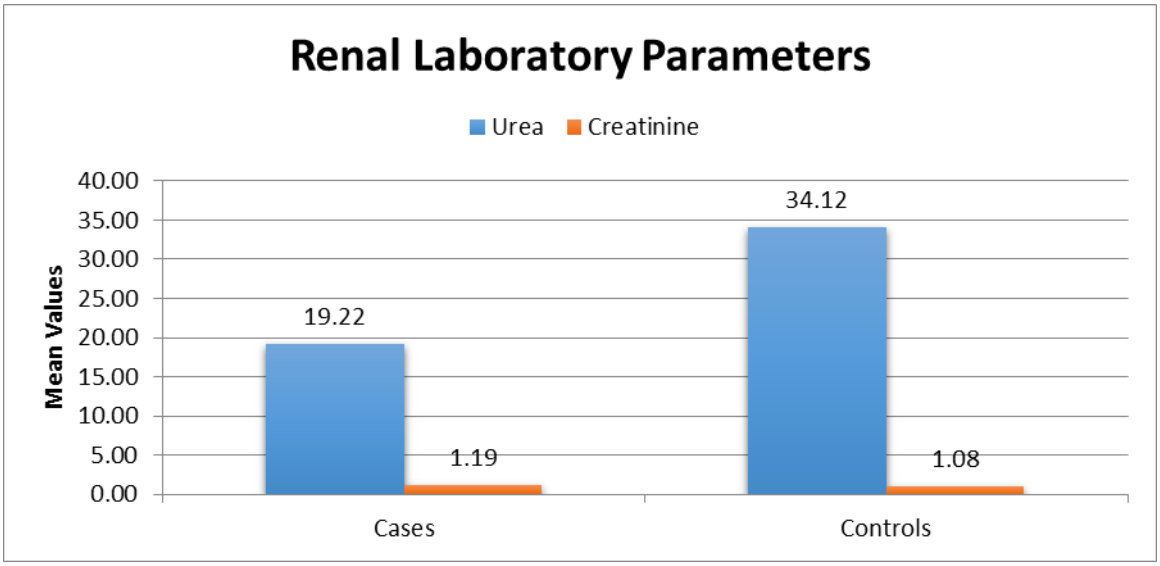
The mean HBA1C was significantly more in cases group compared to control group by a mean difference of 1.93% (22% higher). This difference is significant with a p-value of <0.0001 as per unpaired t test.

Conclusion

In this study we can safely conclude that a significant increase in HBA1C is associated with type 2 diabetics below age 45 compared to non diabetics below age 45 among our study subjects without features of lower UTI..

In other words elevated HBA1C levels was 1.28 times more common in type 2 diabetics below age 45 compared to non diabetics below age 45 among our study subjects without features of lower UTI

Renal Laboratory Parameters



Renal Laboratory Parameters		Urea	Creatinine
Cases	Mean	19.22	1.19
	SD	18.33	0.19
Controls	Mean	34.12	1.08
	SD	3.25	0.14
P value Unpaired t Test		<0.0001	<0.0001

Blood Urea

Among the study patients, there was a statistically significant difference in relation to blood urea distribution between cases group (mean19.22, SD=18.33) and control group (mean=34.12, SD=3.25) with a p value of <0.05 as per unpaired t test. Therefore we reject the null hypothesis that there is no difference in blood urea distribution between the study groups.

Discussion

The mean blood urea was significantly less in cases group compared to control group by a mean difference of 14.91 mg/dl (44% lower). This difference is significant with a p-value of <0.0001 as per unpaired t test.

Conclusion

In this study we can safely conclude that a significant decrease in blood urea is associated with type 2 diabetics below age 45 compared to non diabetics below age 45 among our study subjects without features of lower UTI..

In other words lowered blood urea levels was 1.78 times more common in type 2 diabetics below age 45 compared to non diabetics below age 45 among our study subjects without features of lower UTI

Serum Creatinine

Among the study patients, there was a statistically significant difference in relation to serum creatinine distribution between cases group (mean=1.19, SD=0.19) and control group (mean=1.08, SD=0.14) with a p value of <0.05 as per unpaired t test. Therefore we reject the null hypothesis that there is no difference in serum creatinine distribution between the study groups.

Discussion

The mean serum creatinine was significantly less in cases group compared to control group by a mean difference of 0.11 mg/dl (9% lower). This difference is significant with a p-value of <0.0001 as per unpaired t test.

Conclusion

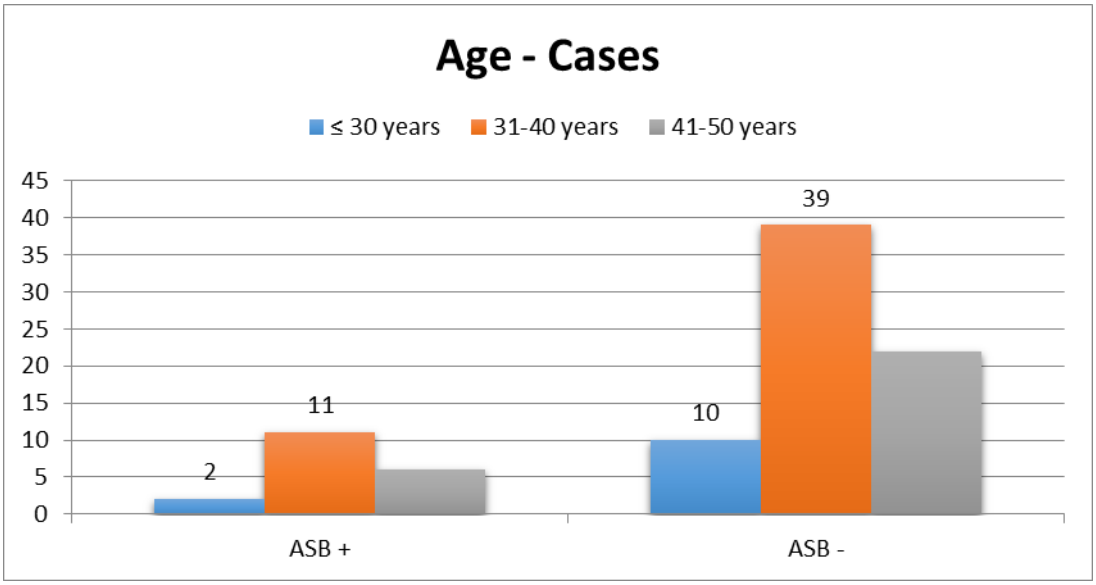
In this study we can safely conclude that a significant increase in serum creatinine is associated with type 2 diabetics below age 45 compared to non diabetics below age 45 among our study subjects without features of lower UTI..

In other words elevated serum creatinine levels was 1.10 times more common in type 2 diabetics below age 45 compared to non diabetics below age 45 among our study subjects without features of lower UTI

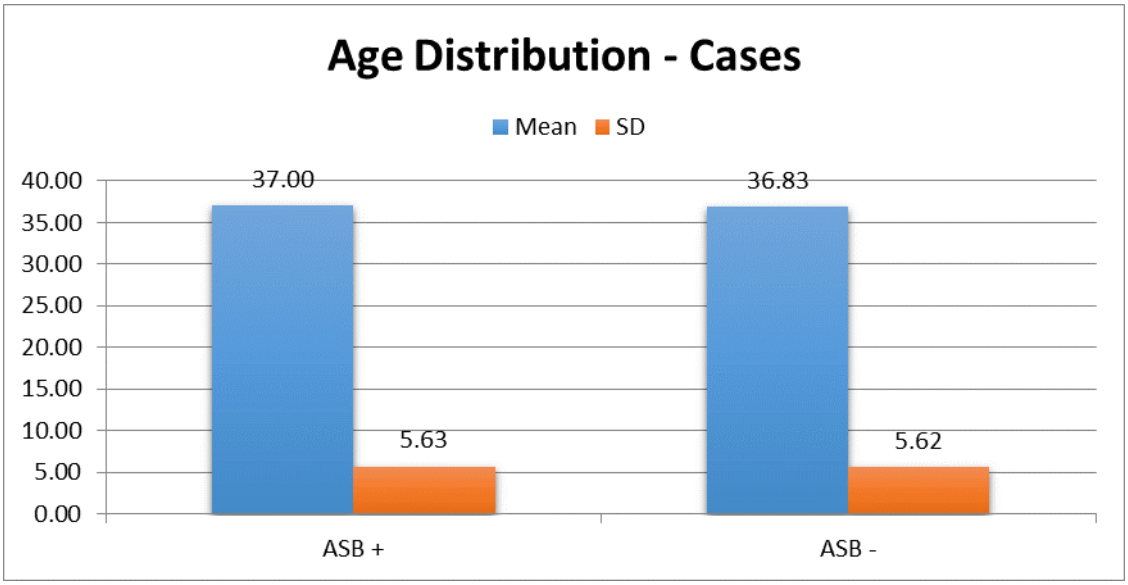
Groups – ASB +ve Vs ASB – ve in Cases

Groups	Definition
ASB +ve	Type 2 Diabetes patients with urine culture +ve
ASB -ve	Type 2 Diabetes patients with urine culture -ve

Age - Cases



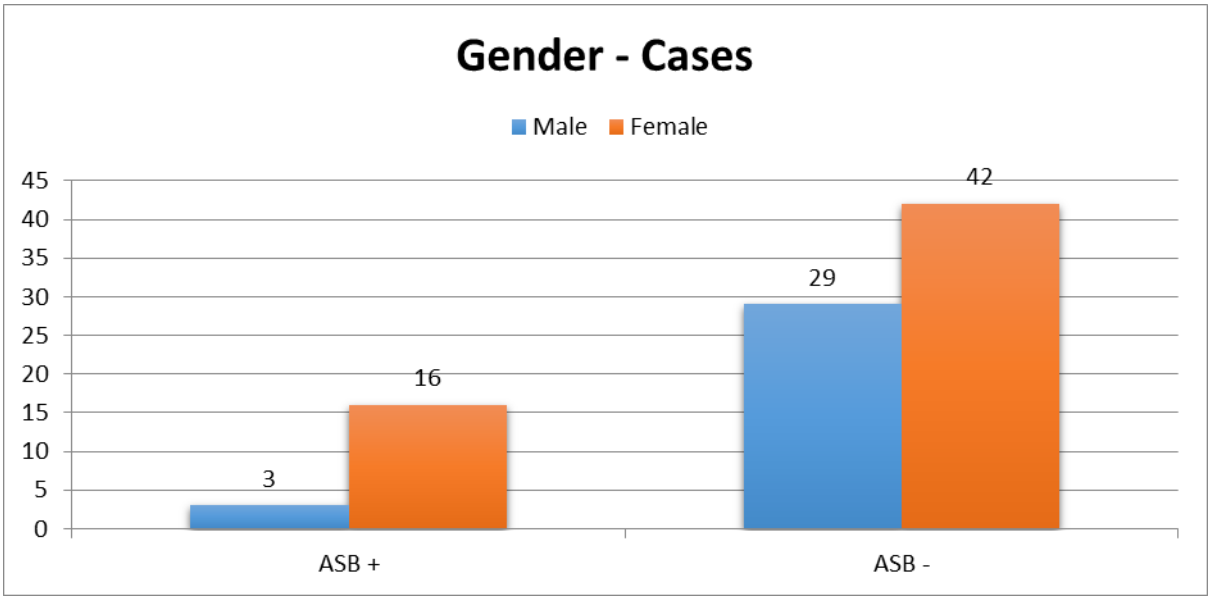
Age - Cases	ASB +	ASB -	ASB + %	ASB - %
≤ 30 years	2	10	10.53	14.08
31-40 years	11	39	57.89	54.93
41-50 years	6	22	31.58	30.99
Total	19	71	100	100



Age Distribution - Cases	ASB +	ASB -
Mean	37.00	36.83
SD	5.63	5.62
P value Unpaired t Test		0.9076

Among the study patients, there was no statistically significant difference in relation to age distribution between ASB +ve group (mean=37.00, SD=5.63) and ASB -ve group (mean=36.83, SD=5.62) with a p value of <0.05 as per unpaired t test. Therefore we fail to reject the null hypothesis that there is no difference in age distribution between the study groups.

Gender - Cases



Gender - Cases	ASB +	ASB -	ASB + %	ASB - %
Male	3	29	15.79	40.85
Female	16	42	84.21	59.15
Total	19	0	100	100
P value Chi Squared Test			0.0427	

Among the study patients, there was a statistically significant difference in relation to gender status between ASB +ve group (majority are females – 84.21%) and ASB -ve group (majority are females – 59.15%) with a p value of <0.05 as per chi squared test. Therefore we reject the null hypothesis that there is no difference in gender status between the study groups.

Discussion

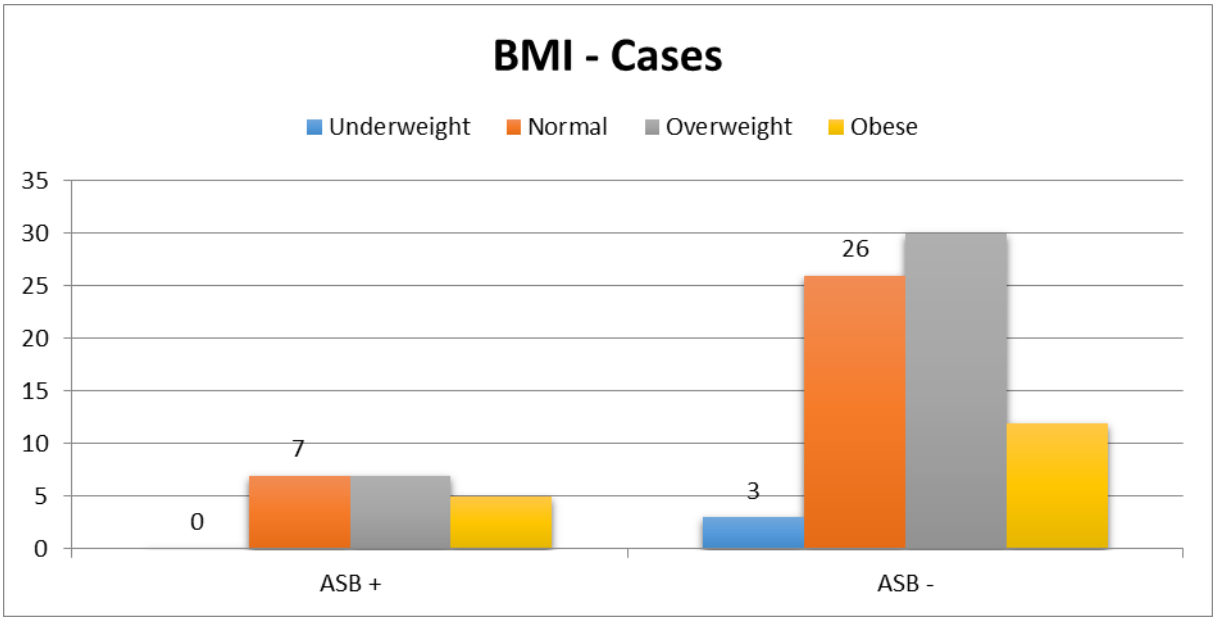
The incidence of female gender was significantly more in ASB +ve group compared to ASB -ve group by a percentage difference of 25.06 percentage points (30% higher). This difference is significant with a p-value of 0.0427 as per chi squared test.

Conclusion

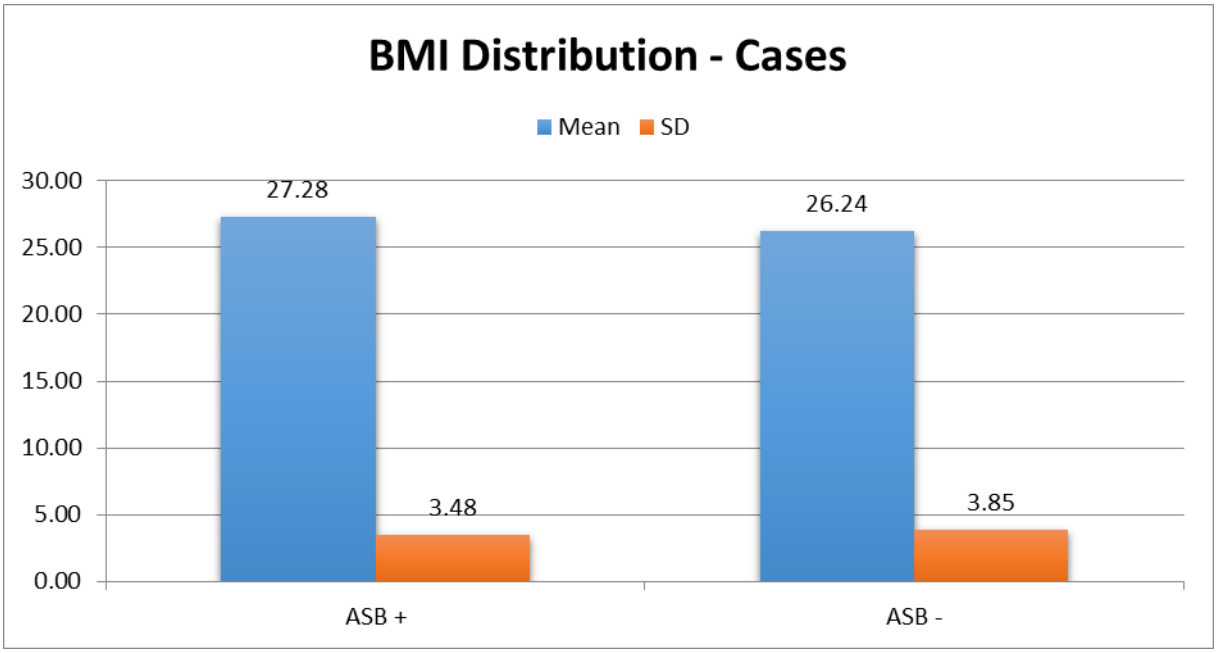
In this study we can safely conclude that a significant increase in female gender status is associated with ASB positivity compared to ASB negativity among our study subjects belonging to cases group without features of lower UTI. .

In other words ASB positive patients had 1.42 times more female representation compared to ASB negative patients among our study subjects belonging to cases group without features of lower UTI

BMI - Cases



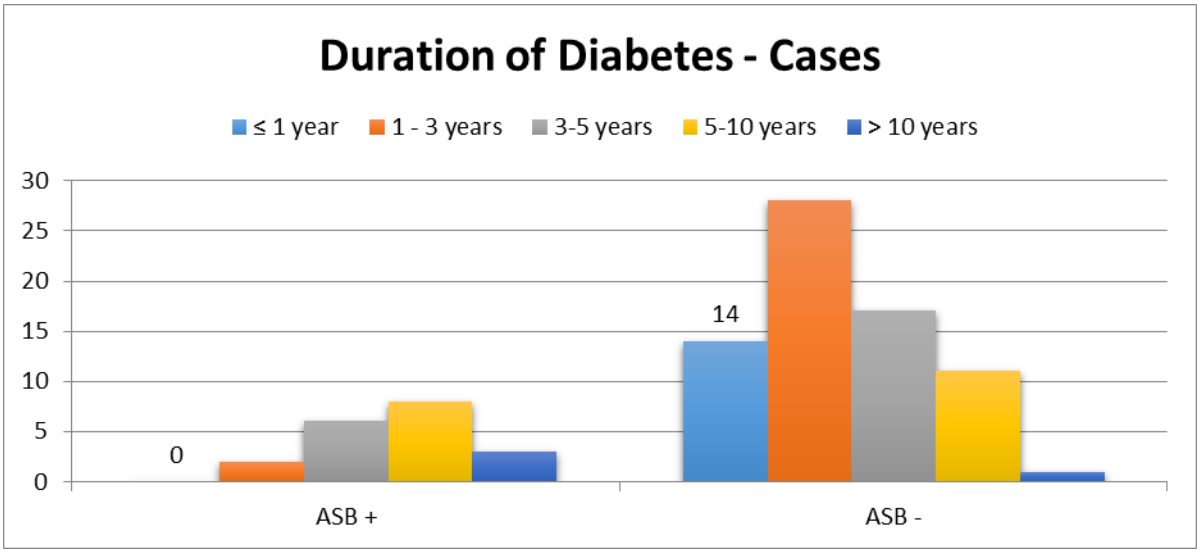
BMI - Cases	ASB +	ASB -	ASB + %	ASB - %
Underweight	0	3	0.00	4.23
Normal	7	26	36.84	36.62
Overweight	7	30	36.84	42.25
Obese	5	12	26.32	16.90
Total	19	71	100	100



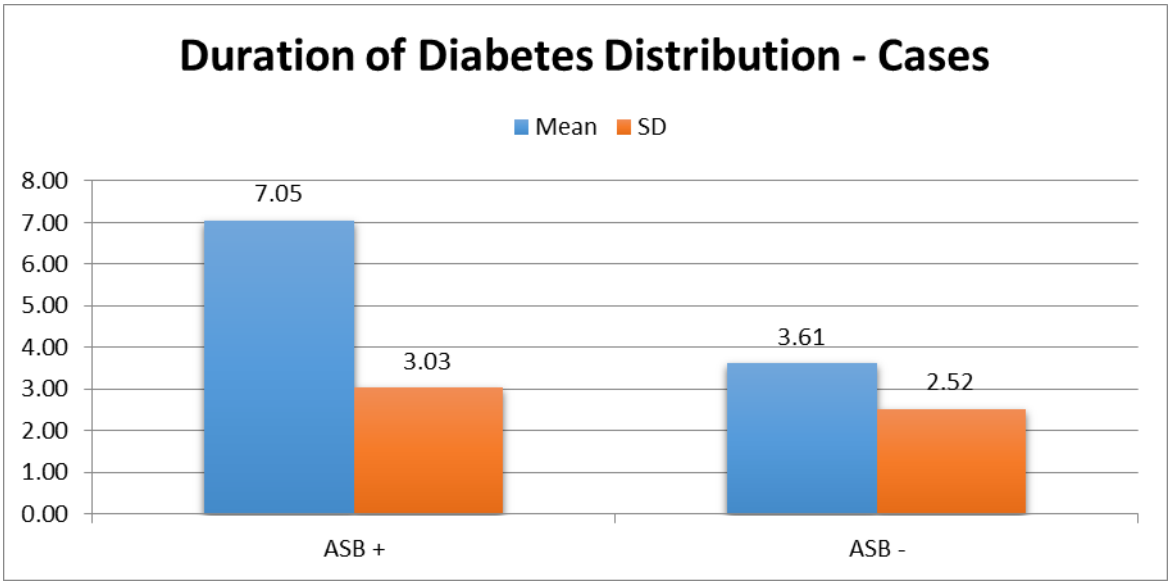
BMI Distribution - Cases	ASB +	ASB -
Mean	27.28	26.24
SD	3.48	3.85
P value Unpaired t Test		0.2855

Among the study patients, there was no statistically significant difference in relation to BMI distribution between ASB +ve group (mean=27.28, SD=3.48) and ASB -ve group (mean=26.24, SD=3.85) with a p value of <0.05 as per unpaired t test. Therefore we fail to reject the null hypothesis that there is no difference in age distribution between the study groups.

Duration of Diabetes - Cases



Duration of Diabetes - Cases	ASB +	ASB -	ASB + %	ASB - %
≤ 1 year	0	14	0.00	15.38
1 - 3 years	2	28	10.53	30.77
3-5 years	6	17	31.58	18.68
5-10 years	8	11	42.11	12.09
> 10 years	3	1	15.79	1.10
Total	19	71	100.00	78.02



Duration of Diabetes Distribution – Cases	ASB +	ASB -
Mean	7.05	3.61
SD	3.03	2.52
P value Unpaired t Test		<0.0001

Among the study patients, there was a statistically significant difference in relation to duration of diabetes distribution between ASB +ve group (mean=7.05, SD=3.03) and ASB -ve group (mean=3.61, SD=2.52) with a p value of <0.05 as per unpaired t test. Therefore we reject the null hypothesis that there is no difference in duration of diabetes distribution between the study groups.

Discussion

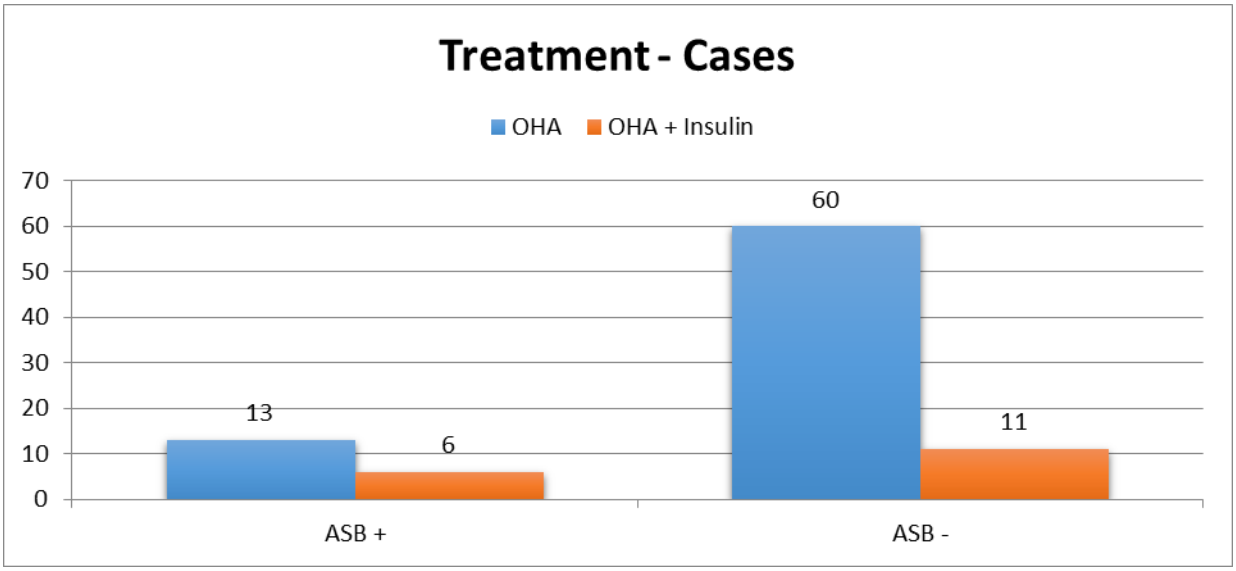
The mean duration of diabetes was significantly more in ASB +ve group compared to ASB -ve group by a mean difference of 3.44 years (49% higher). This difference is significant with a p-value of <0.0001 as per unpaired t test.

Conclusion

In this study we can safely conclude that a significant increase in duration of diabetes is associated with ASB positivity to ASB negativity among our study subjects belonging to cases group without features of lower UTI. .

In other words ASB positive patients had 1.95 more times duration of diabetes compared to ASB negative patients among our study subjects belonging to cases group without features of lower UTI

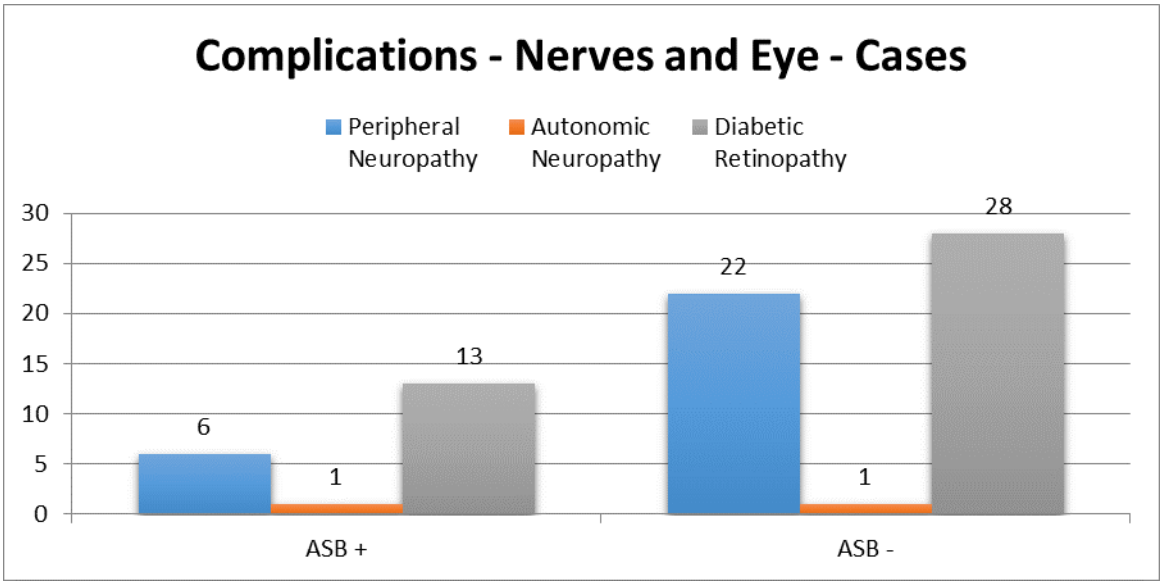
Treatment – Cases



Treatment - Cases	ASB +	ASB -	ASB + %	ASB - %
OHA	13	60	68.42	84.51
OHA + Insulin	6	11	31.58	15.49
Total	19	71	100.00	100.00
P value Chi Squared Test			0.1116	

Among the study patients, there was no statistically significant difference in relation to treatment status between ASB +ve group (majority were on OHA – 68.42%)) and ASB -ve group (majority were on OHA – 84.51%) with a p value of <0.05 as per chi squared test. Therefore we fail to reject the null hypothesis that there is no difference in treatment status between the study groups.

Complications - Nerves and Eye - Cases



Complications - Nerves and Eye – Cases	ASB + (n=19)	ASB – (n=71)	ASB + %	ASB - %	P Value Chi squared Test
Peripheral Neuropathy	6	22	31.58	30.99	0.9604
Autonomic Neuropathy	1	1	5.26	1.41	0.3113
Diabetic Retinopathy	13	28	68.42	39.44	0.0204

Among the study patients, there was no statistically significant difference in relation to peripheral neuropathy and autonomic neuropathy between ASB +ve group and ASB -ve group with a p value of <0.05 as per chi squared test.

Therefore we fail to reject the null hypothesis that there is no difference in peripheral neuropathy and autonomic neuropathy status between the study groups.

Among the study patients, there was a statistically significant difference in relation to diabetic retinopathy status between ASB +ve group (majority had diabetic retinopathy – 68.42%) and ASB -ve group (majority had no diabetic retinopathy – 60.56%) with a p value of <0.05 as per chi squared test. Therefore we reject the null hypothesis that there is no difference in diabetic retinopathy status between the study groups.

Discussion

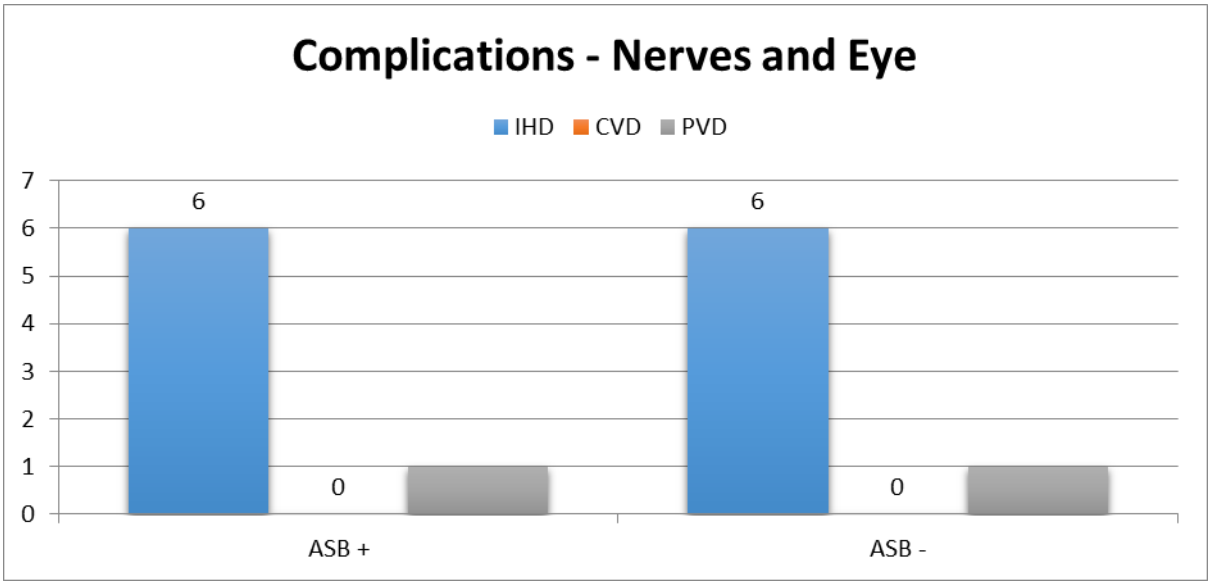
The incidence of diabetic retinopathy was significantly more in ASB +ve group compared to ASB -ve group by a percentage difference of 28.98percentage points (42% higher). This difference is significant with a p-value of 0.0204 as per chi squared test.

Conclusion

In this study we can safely conclude that a significant increase in diabetic retinopathy status is associated with ASB positivity compared to ASB negativity among our study subjects belonging to cases group without features of lower UTI. .

In other words ASB positive patients had 1.73 times more diabetic retinopathy compared to ASB negative patients among our study subjects belonging to cases group without features of lower UTI

Complications - Nerves and Eye - Cases

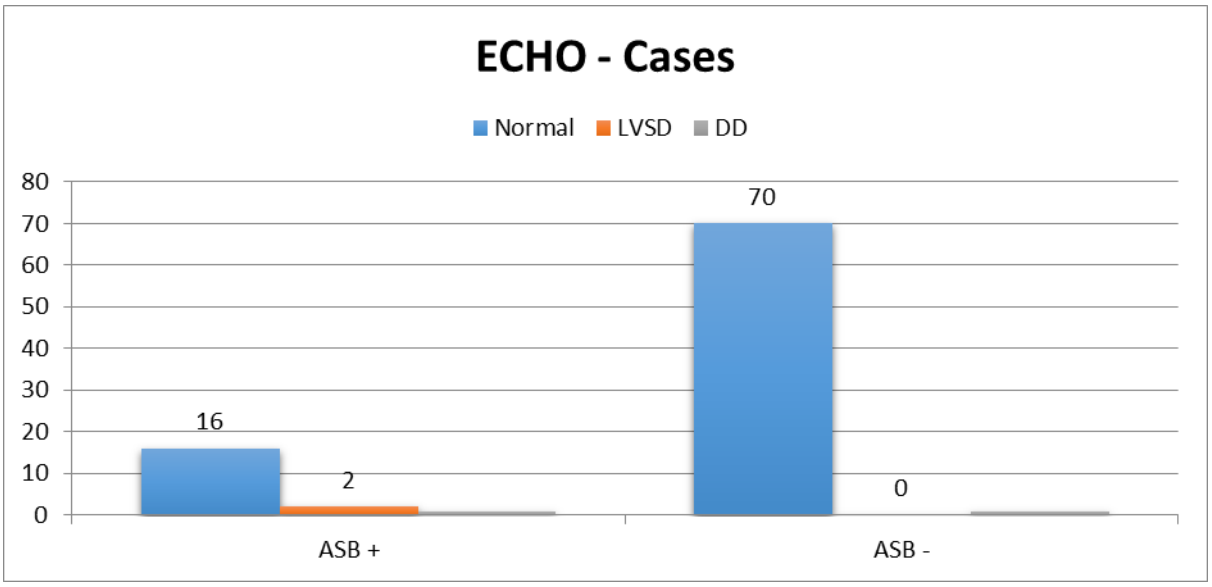


Complications - Nerves and Eye - Cases	ASB +	ASB -	ASB + %	ASB - %	P Value Chi squared Test
IHD	6	6	31.58	8.45	0.1284
CVD	0	0	0.00	0.00	>0.9999
PVD	1	1	5.26	1.41	0.3113

Among the study patients, there was no statistically significant difference in relation to IHD, CVD and PVD between ASB +ve group and ASB -ve group with a p value of <0.05 as per chi squared test. Therefore we fail to reject the null

hypothesis that there is no difference in IHD, CVD and PVD status between the study groups.

ECHO - Cases

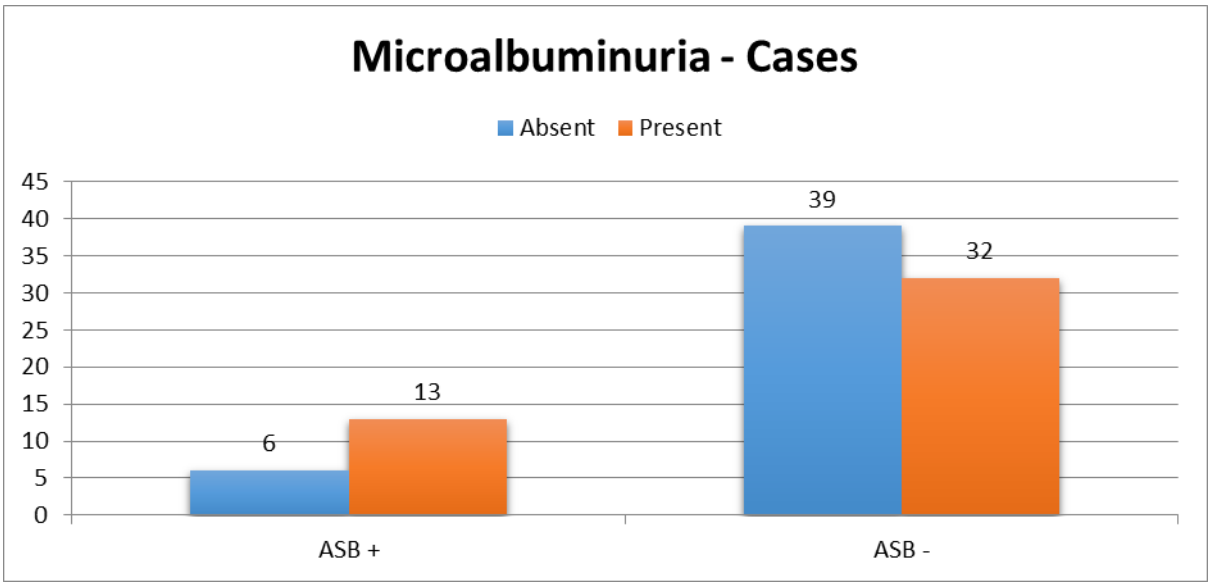


ECHO - Cases	ASB +	ASB -	ASB + %	ASB - %
Normal	16	70	84.21	98.59
LVSD	2	0	10.53	0.00
DD	1	1	5.26	1.41
Total	19	71	100.00	100.00
P Value Chi squared Test			0.1269	

Among the study patients, there was no statistically significant difference in relation to ECHO findings between ASB +ve group and ASB -ve group with a p value of <0.05 as per chi squared test. Therefore we fail to reject the null

hypothesis that there is no difference in ECHO findings status between the study groups.

Microalbuminuria - Cases

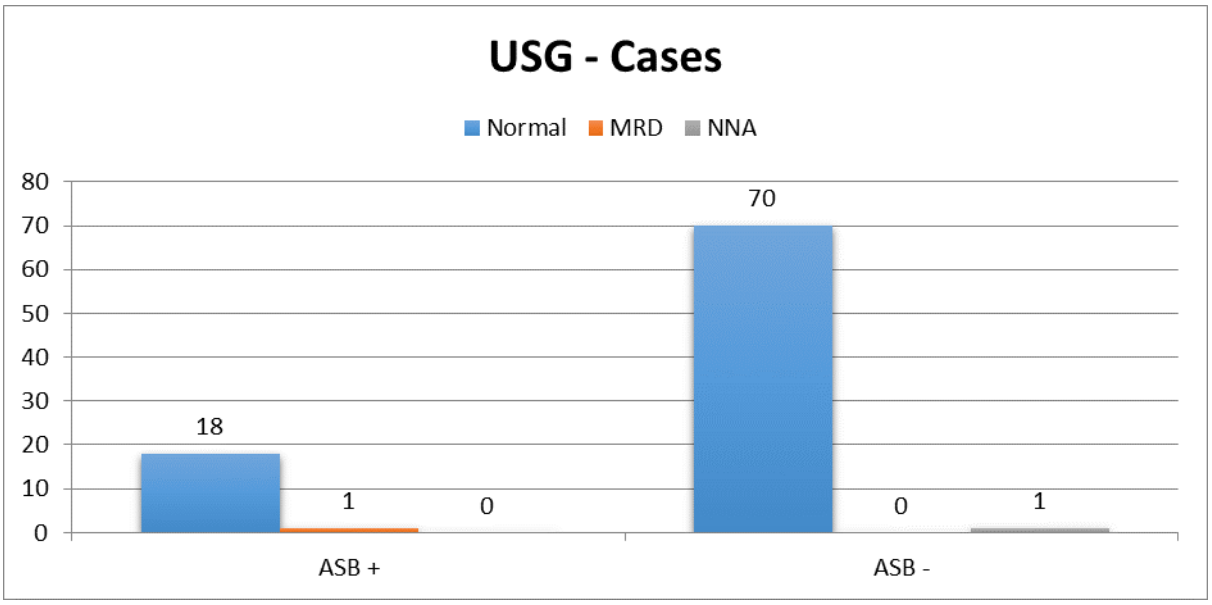


Microalbuminuria - Cases	ASB +	ASB -	ASB + %	ASB - %
Absent	6	39	31.58	54.93
Present	13	32	68.42	45.07
Total	19	71	100	100
P Value Chi squared Test			0.0706	

Among the study patients, there was no statistically significant difference in relation to microalbuminuria status between ASB +ve group and ASB -ve group with a p value of <0.05 as per chi squared test. Therefore we fail to reject the

null hypothesis that there is no difference in microalbuminuria status between the study groups.

USG - Cases

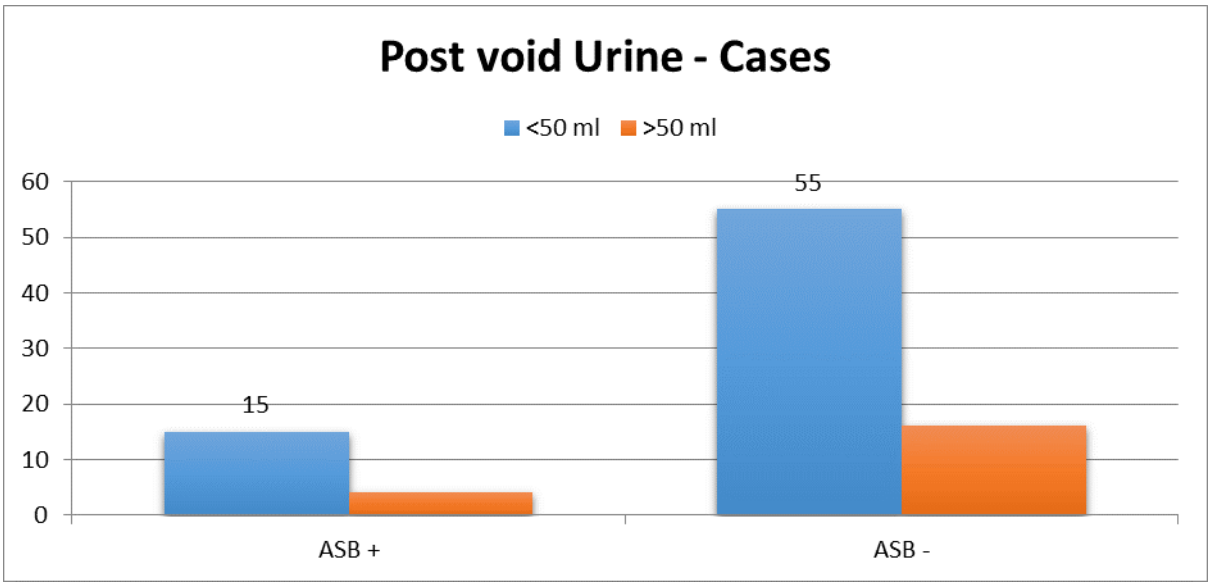


USG - Cases	ASB +	ASB -	ASB + %	ASB - %
Normal	18	70	94.74	98.59
MRD	1	0	5.26	0.00
NNA	0	1	0.00	1.41
Total	19	71	100	100
P Value Chi squared Test			0.3113	

Among the study patients, there was no statistically significant difference in relation to USG diagnosis status between ASB +ve group and ASB -ve group with a p value of <0.05 as per chi squared test. Therefore we fail to reject the

null hypothesis that there is no difference in USG diagnosis status between the study groups.

Post void Urine - Cases

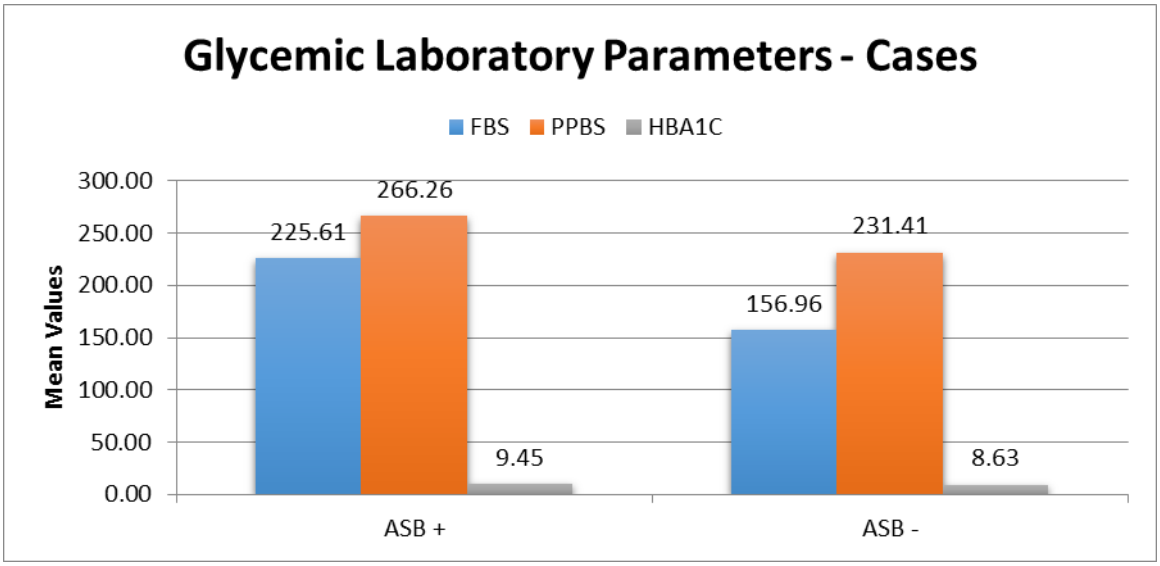


Post void Urine – Cases	ASB +	ASB -	ASB + %	ASB - %
<50 ml	15	55	78.95	77.46
>50 ml	4	16	21.05	22.54
Total	19	71	100	100
P Value Chi squared Test			0.8904	

Among the study patients, there was no statistically significant difference in relation to post void urine status between ASB +ve group and ASB -ve group with a p value of <0.05 as per chi squared test. Therefore we fail to reject the

null hypothesis that there is no difference in post void urine status between the study groups.

Glycemic Laboratory Parameters – Cases



Glycemic Laboratory Parameters - Cases		FBS	PPBS	HBA1C
ASB +	Mean	225.61	266.26	9.45
	SD	53.46	42.90	1.01
ASB -	Mean	156.96	231.41	8.63
	SD	42.75	56.90	1.19
P value Unpaired t Test		0.0075	0.0149	0.0075

FBS

Among the study patients, there was a statistically significant difference in relation to fasting blood sugar distribution between ASB +ve group (mean=225.61, SD=53.46) and ASB -ve group (mean=156.96, SD=42.75) with a p value of <0.05 as per unpaired t test. Therefore we reject the null hypothesis that there is no difference in fasting blood sugar distribution between the study groups.

Discussion

The mean fasting blood sugar was significantly more in ASB +ve group compared to ASB -ve group by a mean difference of 68.65 mg/dl (30% higher). This difference is significant with a p-value of 0.0075 as per unpaired t test.

Conclusion

In this study we can safely conclude that a significant increase in fasting blood sugar is associated with ASB positivity to ASB negativity among our study subjects belonging to cases group without features of lower UTI. .

In other words ASB positive patients had 1.44 more times fasting blood sugar levels compared to ASB negative patients among our study subjects belonging to cases group without features of lower UTI

PPBS

Among the study patients, there was a statistically significant difference in relation to post prandial blood sugar distribution between ASB +ve group (mean=226.26, SD=42.90) and ASB -ve group (mean=231.41, SD=56.90) with a p value of <0.05 as per unpaired t test. Therefore we reject the null hypothesis that there is no difference in post prandial blood sugar distribution between the study groups.

Discussion

The mean post prandial blood sugar was significantly more in ASB +ve group compared to ASB -ve group by a mean difference of 34.85 mg/dl (13% higher). This difference is significant with a p-value of 0.0149 as per unpaired t test.

Conclusion

In this study we can safely conclude that a significant increase in post prandial blood sugar is associated with ASB positivity to ASB negativity among our study subjects belonging to cases group without features of lower UTI. .

In other words ASB positive patients had 1.15 more times post prandial blood sugar levels compared to ASB negative patients among our study subjects belonging to cases group without features of lower UTI

HBA1C

Among the study patients, there was a statistically significant difference in relation to HBA1Cdistribution between ASB +ve group (mean=9.45, SD=1.01) and ASB -ve group (mean=8.63, SD=1.19) with a p value of <0.05 as per unpaired t test. Therefore we reject the null hypothesis that there is no difference in HBA1C distribution between the study groups.

Discussion

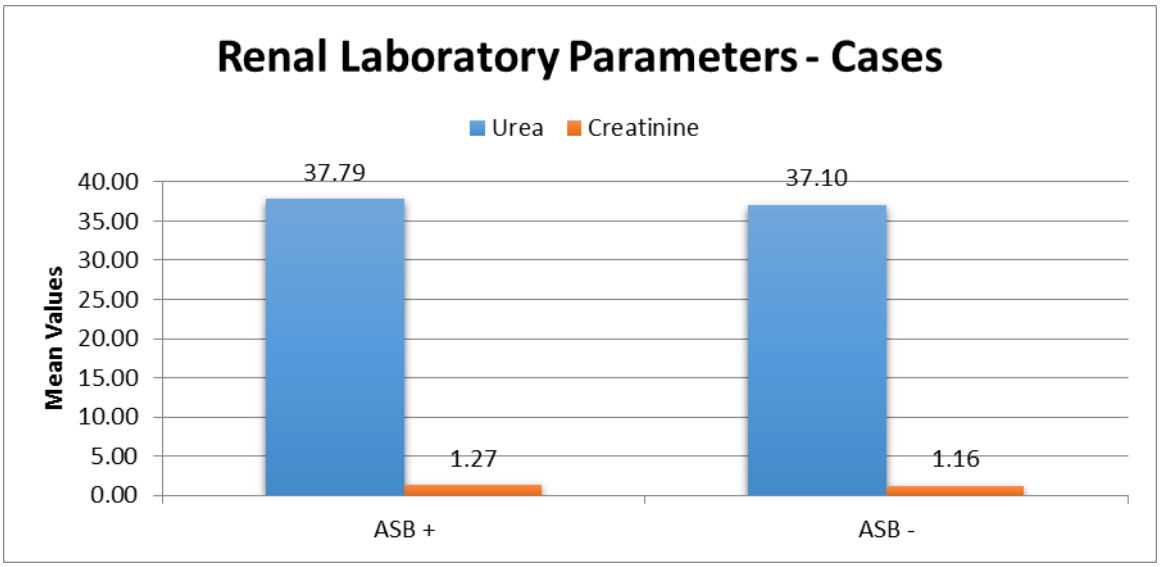
The mean HBA1Cwas significantly more in ASB +ve group compared to ASB -ve group by a mean difference of 0.82% (9% higher). This difference is significant with a p-value of 0.0075as per unpaired t test.

Conclusion

In this study we can safely conclude that a significant increase in HBA1C is associated with ASB positivity to ASB negativity among our study subjects belonging to cases group without features of lower UTI.

In other words ASB positive patients had 1.09 more times HBA1C levels compared to ASB negative patients among our study subjects belonging to cases group without features of lower UTI

Renal Laboratory Parameters - Cases



Renal Laboratory Parameters - Cases		Urea	Creatinine
ASB +	Mean	37.79	1.27
	SD	5.05	0.27
ASB -	Mean	37.10	1.16
	SD	4.03	0.16
P value Unpaired t Test		0.5313	0.1224

Among the study patients, there was no statistically significant difference in relation to blood urea and serum creatinine distribution between ASB +ve group and ASB -ve group with a p value of <0.05 as per chi squared test. Therefore, we fail to reject the null hypothesis that there is no difference in blood urea and serum creatinine distribution between the study groups.

CONCLUSION

1)Asymptomatic bacteriuria is a common finding in adults and diabetic patients especially women have more prevalence than men.

2)In our study the prevalence of Asymptomatic bacteriuria in diabetic patients ≤45 years is 21.1%

3)There was no significant correlation between Asymptomatic bacteriuria and post void residual urine.

4)There was significant association between Asymptomatic bacteriuria and retinopathy.

5)There was no association between Asymptomatic bacteriuria and peripheral neuropathy, IHD, CAD, peripheral vascular disease and nephropathy.

6)There was significant association between poor glycemic control, duration of diabetes and Asymptomatic bacteriuria signifying the importance of metabolic control.

QUESTIONNAIRE

S no			
1	Name		
2	Age		
3	Address		
4	Telephone no		
5	Hospital no		
6	Weight in Kg		
7	Height in meters		
8	Body mass index		
		YES	NO
9	Urinary tract infection in past		
10	Urinary catherisation in past		
11	Renal stones in the past		
12	History of urinary incontinence		
13	Recent hospitilisation / surgery		
		Type 1	Type 2
14	Type of Diabetes		
15	Duration of Diabetes		

Circle the appropriate responses
Microvascular & Macrovascular complications

S No					
16	Medication	OHA	Insulin		
17	Peripheral Neuropathy	Pain	Burning sensation	Numbness	Tingling sensation
		Disturbance in pinprick	Absence of Ankle jerk	Disturbance in light touch	Foot Abnormalities
18	Autonomic Neuropathy	Postural drop in BP	YES		
			NO		
19	Diabetic Retinopathy	Non proliferative DR	Proliferative DR	Laser treatment	Vitreous haemorrhage / Retinal detachment
20	IHD	Stable angina	Old MI	TMT positive	PTCA / CABG
21	Cerebrovascular disease	TIA	CVA	Carotid bruit/ doppler abnormality	
22	Peripheral vascular disease	Absent pulses	Claudication or pain	Gangrene	Amputation
23	Dyslipidemia		YES		
			NO		

INVESTIGATIONS

25	FBS			
26	PPBS			
27	HbA1c			
28	Blood urea			
29	Serum creatinine			
30	Microalbuminuria		YES	
			NO	
31	Urine Culture with colony count			
32	Ultra sound			
33	Post void Urine			

Bibliography

[1] Risk of Urinary Tract Infection and Asymptomatic Bacteriuria among Diabetic and Nondiabetic Postmenopausal Women – Edward J. Boyko1,2, Stephan D. Fihn2,3, Delia Scholes4,5, Linn Abraham5, and Barbara Monsey5 – Feburary 2, 2004

[2] Geerlings, SE, Stolk, RP, Camps, MJ, et al. Risk factors for symptomatic urinary tract infection in women with diabetes. Diabetes Care 2000; 23:1737-41.

[3] Vejlsgaard R:Studies on urinary infection in diabetics. I.Bacteriuria in patients with diabetes mellitus and in control subjects. ActaMedScand 179:173-182,1966

[4] OsterbyHanston R: Bacteriuria in diabetic and non-diabetic out-patients. Acta Med Scand 176:721–730, 1964

[5] Asymptomatic bacteriuria in type 2 Iranian diabetic women: a cross sectional study Mohammad Ali Boroumand Email author, Leila Sam, Seyed Hesameddin Abbasi, Mojtaba Salarifar,Ebrahim Kassaian and Saeedeh Forghani Published**:** 23 February 2006.

[6]Geerlings, SE, Stolk, RP, Camps, MJ, et al. Asymptomatic bacteriuria may be considered a complication in women with diabetes. Diabetes Care 2000; 23:744- 49

[7] Kaveeshwar SA, Cornwall J. The current state of diabetes mellitus in India, AMJ 2014, 7, 1, 45-48. http//dx.doi.org/10.4066/AMJ.2014.1979

[8] Definition and diagnosis of diabetes mellitus and intermediate hyperglycemia, report WHO,IDF consultation appendix 2.

[9] Association AD. Standards of Medical care in Diabetes- 2007. Diabetes Care.[Review]. 2007 Jan 2007;30(Supplement 1)

[10] Kass EH. Bacteriuria and the diagnosis of infections of the urinary tract. Arch Intern Med 1957;100:709–14

[11] Kass EH. Pyelonephritis and bacteriuria: a major problem in preventive medicine. Ann Intern Med 1962;56:46–53.

[12] The nitrite and leukocyte esterase tests for the evaluation of asymptomatic bacteriuria in obstetric patients. Robertson AW, Duff P. Obstet Gynecol.1988 Jun; 71(6 Pt 1):878-81.

[13] Kunin CM. Asymptomatic bacteriuria. Ann Rev Med 1966;17: 383–406

[14] Nicolle LE.Asymptomatic bacteriuria : when to screen and when to treat. Infect Dis Clinic North Am 2003;17:367-94

[15] Zhanel, GG, Harding, GKM, Nicolle, LE. Asymptomatic bacteriuria in patients with diabetes mellitus. Rev Infect Dis 1991; 13:150-4.

[16] Nicolle LE. Asymptomatic bacteriuria in the elderly. Infect Dis Clin North Am 1997;11:647–62.N Engl J Med 1940; 223:224

[17] Waites KB,Canupp KC,De Vivo MJ.Epidemiology and risk factors for urinary tract infection following spinal cord injury .Arch Phys Med Rehabil 1993;74:691-5

[18] Bakke A,Digranes A .Bacteriuria in patients treated with clean intermittent catheterization.Scand J Infect Dis 1991;23:577-82.

[19] Choudhary A,Stone WJ,Breyer JA. Occurence of pyuria and bacteriuria in asymptomatic haemodialysis patients.Am J Kidney Dis 1993; 21: 180-3

[20] [O]Nicolle LE,Bradley S,Colgan R ,Rice JC, Schaeffer A,Hotoon TM; Infectious Diseases Society of America guidelines for the diagnosis of and treatment of asymptomatic bacteriuria in adults. Clin Infect Dis 2005;40 :643-54

[21] Gleckman R, Esposito A, Crowley M, Natsios GA. Reliability of a single urine culture in establishing diagnosis of asymptomatic bacteriuria in adult males. J Clin Microbiol 1979;9 :596–7

[22] Hooton TM, Scholees D, Stapleton AE, et al. A prospective study of asymptomatic bacteriuria in sexually active young women. N Engl J Med 2000;343:992–97

[23] Risk factors for the development and persistence of asymptomatic bacteriuria in patients with type 2 diabetes .Ribera-Montes Mdel C, PascualPerez R, Orozco-Beltran D, Perez-Barba C, Pedrera-Carbonell V Med Clin (Barc). 2005 Nov 5;125 (16):606-10

[24] Boyko, EJ, Fihn, SD, Scholes, D, et al. Risk of urinary tract infection and asymptomatic bacteriuria among diabetic and nondiabetic postmenopausal women. Am J Epidemiol 2005; 161:557

[25] Zhanel, GG, Nicolle, LE, Harding, GKM, et al. Prevalence of asymptomatic bacteriuria and associated host factors in women with diabetes mellitus. Clin Infect Dis 1995; 21:316-22.

[26] Ribera MC.Pascual R,Orozio D,Perez Barba C,PedraV,Gil V. Incidence and risk factors associated with urinary tract infection in diabetic patients with and without asymptomatic bacteriuria. Eur J Clin Microbiol Infect Dis.2006June ;25 (6):389-93

[27] Although ASB is apparently a benign condition, prevention in elderly people is important.. Drugs Aging. 2005;22(10):801-7

[28] Harding, GK, Zhanel, GG, Nicolle, LE, Cheang, M. Antimicrobial treatment in diabetic women with asymptomatic bacteriuria. Manitoba Diabetes Urinary Tract Infection Study. N Engl J Med 2002; 347:1576-83

[29] Fant et al,1996.)-Fantl JA,Newman DK ,Colling J et al :Urinary incontinence in adults :1996 Update (AHCPR Publication No.96-0682)

[30] Frimodt- Moller, C.: Diabetic cystopathy. A review of the urodynamic and clinical features of neurogenic bladder dysfunction in diabetes mellitus. Dan. Med. Bull., 25: 49, 1978.

[31] Ewing, D. J., Campbell, I. W. and Clarke, B. F.: The natural history of diabetic autonomic neuropathy. Quart. J. Med., 185: 95, 1980.

[32] Kahan, M., Goldberg, P. D. and Mandel, E. E.: Neurogenic vesical dysfunction and diabetes mellitus. N.Y. J. Med., 70: 2448, 1970

[33] Lifford K, Curhan G, Grodstein F: Type 2 diabetes and risk of developing urinary incontinence in the Nurses' Health Study (Abstract). In Urologic Complications of Diabetes. Bethesda, MD, National Institutes of Health, 2003, p.

[34] Tomohiro ueda,Naoki yohimura,Osamu yoshida.Diabetic cystopathy: Relationship to autonomic neuropathy detected by sympathetic skin response. The Journal of Urology Vol 157.580-584.February 1997.

[35] Frimodt- Moller, C.: Diabetic cystopathy. A review of the urodynamic and clinical features of neurogenic bladder dysfunction in diabetes mellitus. Dan. Med. Bull., 25: 49, 1978

[36] Ueda R, Yoshimura N, Yoshida O. Diabetic cystopathy: relationship to autonomic neuropathy detected by skin response. J Urol 1997;157: 580-4

[37] Axelrod, S. L. and Blaivas, J. G.: Bladder neck obstruction in women. J. Urol., 137: 497, 1987.

[38] Sampselle CM, Harlow SD, Skurnick J, Brubaker L, Bondarenko I: Urinary incontinence predictors and life impact in ethnically diverse perimenopausal women. Obstet Gynecol 100:1230–1238, 2002

[39] Walter S. Autonomic neuropathy involving bladder and urethra function: autonomic cystopathy: facts or fiction—what do we know? Scand J Urol Nephrol 1996;30 (Suppl 179):67-8

[40] Diabetes Control and Complications Trial Research Group. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. N Engl J Med 1993;14:977- 86

[41] Wagg A, Malone-Lee J. The management of urinary incontinence in the elderly. Br J Urol 1998;82(Suppl 1):11-7.

[42] HARRISON'S PRINCIPLES OF INTERNAL MEDICINE 19th Edition page2399,2429,

[43] Stapleton A. Urinary tract infections in patients with diabetes. Am J Med 2002;113(suppl 1A):80S–4S

[44] . Valerius NH, Eff C, Hansen NE, et al. Neutrophil and lymphocyte function in patients with diabetes mellitus. Acta Med Scand 1982;211:463–7

[45] Hosking DJ, Bennett T, Hampton JR. Diabetic autonomic neuropathy. Diabetes Care 1978;27:1043–54

[46]. Stapleton A (2002) Urinary tract infections in patients with diabetes. Am J Med 113(Suppl 1A):80S–84S

[47] Osterby Hansen R: Bacteriuria in diabetic and non-diabetic out-patients. Acta Med Scand 176:721-730, 1964

[48]Schmitt JK, Fawcett CJ, Gullickson G: Asymptomatic bacteriuria and hemoglobin A1. Diabetes Care 9:518-520, 1986

[49]Keane EM, Boyko EJ, Barth Reller L, Hamman RF: Prevalence of asymptomatic bacteriuria in subjects with NIDDM in San Luis Valley of Colorado. Diabetes Care 11:708-712, 1988

[50]Perez Luque EL, de la Luz Villalpando M, Malacara JM: Association of sexual activity and bacteriuria in women with non-insulin-dependent diabetes mellitus. J Diabetes Complications 6:254-257, 1992

[51]Sawers JS, Todd WA, Kellett HA, Miles RS, Allan PL, Ewing DJ, Clarke BF: Bacteriuria and autonomic nerve function in diabetic women. Diabetes Care 9:460-464, 1986

[52]Vejlsgaard R: Studies on urinary infection in diabetics. II. Significant bacteriuria in relation to long-term diabetic manifestations. Acta Med Scand 179:183-188, 1966

[53]Zhanel GG, Nicolle LE, Harding GKM, Manitoba Diabetic Urinary Infection Study Group: Prevalence of asymptomatic bacteriuria and associated host factors in women with diabetes mellitus. Clin Infect Dis 21:316-322, 1995

[54]Joshi SR, Parikh RM. India - diabetes capital of the world: now heading towards hypertension. J Assoc Physicians India. 2007;55:323–4. [PubMed]

[55] Kumar A, Goel MK, Jain RB, Khanna P, Chaudhary V. India towards diabetes control: Key issues.Australas Med J. 2013;6(10):524–31.

[56] Wild S, Roglic G, Green A, Sicree R, King H. Global prevalence of diabetes-estimates for the year 2000 and projections for 2030. Diabetes Care. 2004;27(3):1047–53

[57]Whiting Dr, Guariguata L, Weil C, Shawj. IDF Diabetes atlas: Global estimates of the prevalence of diabetes for 2011 and 2030. Diabetes Res Clin Pract. 2011;94:311–21

[58] 7. Ramachandran A, Snehalatha C, Kapur A, Vijay V, Mohan V, Das AK, Rao PV, Yajnik CS, Prasanna Kumar KM, Nair JD. Diabetes Epidemiology Study Group in India (DESI). High prevalence of diabetes and impaired glucose tolerance in India: National Urban Diabetes
Survey. Diabetologia. 2001;44(9):1094–101

[59]. Mohan V, Shah S, Saboo B. Current glycemic status and diabetes related complications among type 2 diabetes patients in India: data from the A1chieve study. JAPI (Suppl) 201361:12–15. [PubMed]

[60] Mohan V, Seshiah V, Sahay BK, Shah SN, Rao PV, Banerjee S. Current status of management of diabetes and glycaemic control in India: Preliminary results from the DiabCare India 2011 Study. Diabetes.2012;61:a645–a677.

[61] Unnikrishnan RI, Rema M, Pradeep R, Deepa M, Shanthirani CS, Deepa R, Mohan V. Prevalence and risk factor of diabetic nephropathy in an urban south Indian population; The Chennai urban rural Epidemiology study (CurES-45) Diabetes Care. 2007;30:2019–2024

[62] . Rastogi A, Bhadada SK, Saikia UN, Bhansali A. Recurrent diabetic myonecrosis: a rare complication of a common disease. Indian J Med Sci. 2011;65(7):311–4. [PubMed]

[63]Iyer SN, Drake AJ 3rd, West RL, Tanenberg RJ. Diabetic muscle infarction: a rare complication of long-standing and poorly controlled diabetes mellitus. Case Rep Med. 2011;2011:407921

[64]Journal Of Uva clinical society, volume 04,october2009

[65] Calvet CM, Yoshikawa TT. Infections in diabetics. Infect Dis Clin North Am 2001; 15:407–21.

[66] Gleckman R, Esposito A, Crowley M, Natsios GA. Reliability of a single urine culture in establishing diagnosis of asymptomatic bacteriuria in adult males. J Clin Microbiol 1979;9:596–7.

[67] KASS EH. Asymptomatic infections of the urinary tract. Trans Assoc Am Physicians. 1956;69:56-64. PubMed PMID: 13380946.

[68]Geerlings SE, Stolk RP, Camps MJ, Netten PM, Collet TJ, Hoepelman AI; Risk factors for symptomatic urinary tract infection in women with diabetes. Diabetes Women Asymptomatic Bacteriuria Utrecht Study Group. Diabetes Care. 2000 Dec;23(12):1737-41.

[69] Patterson JE, Andriole VT. Bacterial urinary tract infections in diabetes. Infect Dis Clin North Am 1997; 11:735–50.

[70] Turan H, Serefhanoglu K, Torun AN, Kulaksizoglu S, Kulaksizoglu M, Pamuk B, Arslan H. Frequency, risk factors, and responsible pathogenic microorganisms of asymptomatic bacteriuria in patients with type 2 diabetes mellitus. Jpn J Infect Dis. 2008 May;61(3):236-8.

[71] Diseases of the kidney and urinary tract Eighth Edition Volume 1 Robert W.Schrier page 836-837

[72]Hoeprich P.Culture of the urine. J LAB Clin Med 1960;56;899

[73] Kaplan, S. A. and Blaivas, J. G.: Diabetic cystopathy. J. Diab. Complicat., 2: 133, 1988.

[74] Geerlings, SE, Stolk, RP, Camps, MJ, et al. Asymptomatic bacteriuria may be considered a complication in women with diabetes. Diabetes Care 2000; 23:744-49.

TEST

Sno	Age	Sex	BMI	Duration of Diabetes	OHA/Insulin	Peripheral Neuropathy	Autonomic Neuropathy	Diabetic Retinopathy	IHD	CVD	PVD	Echo	FBS	PPBS	HBA1C	Urea	Creatinine	Microalbuminuria	Urine culture	USG	Post void urine	PSA
1	38	F	23	3Y	OHA	-	-	+	+	-	-	N	136	166	7.1	33	1	+	Negative	N	>50 ml	NA
2	31	M	27	2Y	OHA	-	-	+	-	-	-	N	142	188	7.4	36	0.9	+	Negative	N	< 50 ml	NA
3	24	F	26	2Y	OHA	+	-	-	-	-	-	N	198	248	9.3	29	1.1	-	Negative	N	<50 ml	NA
4	41	F	23	5Y	OHA	-	-	+	-	-	-	N	220	297	10.5	33	0.9	-	Negative	N	>50 ml	NA
5	34	F	27	5Y	OHA	+	-	+	-	-	-	N	104	154	7.4	38	1.3	+	Negative	N	>50 ml	NA
6	40	M	19	5Y	OHA	-	-	+	+	-	-	N	201	226	8.3	29	1.1	-	Negative	N	<50 ml	NA
7	36	F	24	11Y	OHA/I	-	-	+	-	-	-	N	184	289	8.3	36	1.1	+	Positive	N	>50 ml	NA
8	37	F	31	1Y	OHA	-	-	-	-	-	-	N	168	220	8.8	35	1.2	+	Negative	N	< 50 ml	NA
9	36	M	26	6M	OHA	-	-	+	+	-	-	N	156	174	7.6	40	1.3	+	Negative	N	>50 ml	N
10	37	M	22	1Y	OHA	+	-	+	-	-	-	N	222	269	9.2	42	1.5	+	Negative	N	< 50 ml	NA
11	31	F	28	1Y	OHA	+	-	-	-	-	-	N	152	262	9.4	43	1.3	-	Negative	N	<50 ml	NA
12	26	F	23	2Y	OHA	-	-	-	-	-	-	N	110	144	7.2	44	1.2	-	Negative	N	<50 ml	NA
13	45	F	32	10Y	OHA	+	+	+	+	-	-	LVSD	192	276	9.2	38	0.9	-	Positive	N	<50 ml	NA
14	43	M	31	4Y	OHA	-	-	+	+	-	-	N	168	242	8.1	33	1	+	Negative	N	< 50 ml	NA
15	31	F	18	3Y	OHA/I	-	-	-	-	-	-	N	210	316	11.1	34	1.1	-	Negative	N	<50 ml	NA
16	32	M	18	4Y	OHA	-	-	-	-	-	-	N	228	294	10.1	38	1.3	-	Negative	N	<50 ml	NA
17	41	F	29	2Y	OHA	+	+	+	-	-	-	N	144	210	8.4	39	1.4	+	Negative	N	>50 ml	NA
18	36	F	32	6M	OHA	-	-	+	-	-	-	N	106	166	7.1	31	1.3	-	Negative	N	<50 ml	NA
19	44	M	23	8Y	OHA	+	-	+	+	-	-	N	98	188	7.5	38	1.4	-	Negative	N	<50 ml	NA
20	26	F	22	3Y	OHA	+	-	-	-	-	-	N	131	161	7.9	35	1.3	-	Negative	N	<50 ml	NA
21	39	F	28	8Y	OHA	-	-	+	-	-	-	N	202	320	10.4	42	1.6	+	Positive	N	<50 ml	NA
22	34	F	26	5Y	OHA	-	-	-	-	-	-	N	118	189	7.7	39	1.2	+	Negative	N	< 50 ml	NA
23	33	F	31	8Y	OHA	-	-	+	-	-	-	N	174	293	9.6	31	1	+	Positive	N	<50 ml	NA
24	39	M	28	2Y	OHA/I	-	-	-	-	-	-	N	104	204	7.8	36	1.1	-	Negative	N	<50 ml	NA
25	32	F	24	3Y	OHA/I	+	-	+	-	-	-	N	188	274	9.2	36	1.2	+	Positive	N	<50 ml	NA
26	42	M	32	1Y	OHA	-	-	+	-	-	-	N	154	257	8.8	34	1.4	+	Negative	N	>50 ml	N
27	41	F	28	8Y	OHA	+	-	+	-	-	-	N	122	152	7.6	36	1	-	Negative	N	>50 ml	NA
28	42	F	28	10Y	OHA	+	-	+	-	-	-	N	221	268	10.1	38	1.2	+	Negative	N	< 50 ml	NA
29	40	F	25	8Y	OHA	-	-	+	-	-	-	N	131	155	7.8	34	1	-	Negative	N	<50 ml	NA
30	35	M	25	3Y	OHA	-	-	+	-	-	-	N	212	276	9.4	37	1.1	+	Negative	N	>50 ml	N
31	43	M	30	5Y	OHA	-	-	+	+	-	-	N	112	168	7.3	28	1.4	+	Negative	N	< 50 ml	NA

32	41	F	30	5Y	OHA	-	-	-	+	-	-	N	136	192	7.6	38	1.1	-	Positive	N	<50 ml	NA
33	25	F	22	4M	OHA/I	+	-	-	-	-	-	N	254	362	11.3	42	1.3	-	Negative	N	<50 ml	NA
34	27	F	18	2Y	OHA	-	-	-	-	-	-	N	166	253	8.6	39	1.1	-	Negative	N	<50 ml	NA
35	29	M	23	1Y	OHA	-	-	-	-	-	-	N	131	287	8.7	38	1.2	-	Negative	N	<50 ml	NA
36	40	M	23	5Y	OHA/I	-	-	+	-	-	+	N	212	322	10.2	39	1.1	+	Negative	N	< 50 ml	NA
37	32	F	33	3Y	OHA	+	-	-	-	-	-	N	121	161	7.5	36	1.2	-	Negative	N	<50 ml	NA
38	45	F	27	5Y	OHA/I	-	-	+	-	-	-	N	184	336	9.9	34	0.9	+	Negative	N	< 50 ml	NA
39	27	F	28	6Y	OHA	-	-	-	-	-	-	N	174	272	9.4	47	1.3	+	Positive	N	<50 ml	NA
40	35	F	28	4Y	OHA	+	-	-	-	-	-	N	108	188	7.4	29	1.1	+	Negative	N	>50 ml	NA
41	32	M	32	4Y	OHA	-	-	+	-	-	-	N	142	184	7.8	29	0.9	+	Positive	N	>50 ml	N
42	38	F	30	10Y	OHA	-	-	+	-	-	-	N	148	202	7.9	34	1	+	Negative	N	< 50 ml	NA
43	44	F	22	4Y	OHA/I	+	-	+	-	-	-	N	122	166	7.5	38	1.2	+	Negative	N	< 50 ml	NA
44	28	F	27	3Y	OHA	-	-	-	-	-	-	N	174	201	8.7	42	1.3	-	Negative	N	<50 ml	NA
45	44	M	29	2Y	OHA	+	-	-	-	-	-	N	161	238	8.5	44	1.2	+	Negative	N	>50 ml	N
46	41	M	27	8Y	OHA	+	-	+	-	-	-	N	202	247	9.4	33	1	-	Positive	N	<50 ml	N
47	43	F	22	13Y	OHA/I	+	-	-	+	-	-	DD	192	254	9.8	36	1.4	+	Positive	N	>50 ml	NA
48	37	M	24	3Y	OHA	-	-	-	-	-	-	N	144	174	7.3	40	1	-	Negative	N	<50 ml	NA
49	27	M	33	2Y	OHA/I	-	-	-	-	-	-	N	206	316	10.4	44	1.5	-	Negative	N	<50 ml	NA
50	33	M	22	7Y	OHA	-	-	+	-	-	+	N	193	267	9.9	40	1.2	-	Positive	N	<50 ml	N
51	40	M	29	1Y	OHA	-	-	-	-	-	-	N	144	269	8.2	42	1.1	+	Negative	N	< 50 ml	NA
52	36	F	24	5Y	OHA/I	-	-	-	-	-	-	N	228	342	11.1	48	1.7	+	Positive	N	<50 ml	NA
53	44	M	22	4Y	OHA	-	-	+	-	-	-	N	188	302	9.3	40	1	+	Negative	N	< 50 ml	NA
54	36	M	32	7Y	OHA	-	-	+	-	-	-	N	110	185	7.2	36	1.1	-	Negative	N	>50 ml	N
55	45	M	23	3Y	OHA	+	-	-	-	-	-	N	114	210	7.9	35	1	+	Negative	N	< 50 ml	NA
56	37	F	27	2Y	OHA	-	-	-	-	-	-	N	225	302	10.6	42	1.3	-	Negative	N	<50 ml	NA
57	35	F	32	4Y	OHA	-	-	+	-	-	-	N	200	240	8.8	31	1.1	+	Positive	N	<50 ml	NA
58	29	F	22	2Y	OHA	-	-	-	-	-	-	N	280	368	11.4	43	1.3	-	Negative	N	<50 ml	NA
59	39	F	26	1Y	OHA	+	-	-	-	-	-	N	197	284	9.6	40	1.1	+	Negative	N	>50 ml	NA
60	34	F	24	5Y	OHA	-	-	-	-	-	-	N	133	192	7.8	39	1	-	Negative	N	<50 ml	NA
61	38	F	24	3Y	OHA	-	-	+	-	-	-	N	119	188	7.7	38	0.9	-	Negative	N	<50 ml	NA
62	40	F	29	9Y	OHA/I	+	-	+	+	-	-	N	186	282	9.9	34	1.2	-	Positive	N	<50 ml	NA
63	33	F	28	4Y	OHA	+	-	-	-	-	-	N	133	220	8.3	42	1.1	-	Negative	N	<50 ml	NA
64	38	M	27	2Y	OHA/I	-	-	-	-	-	-	N	202	298	9.8	39	1.3	+	Negative	N	< 50 ml	NA

65	40	M	31	1Y	OHA	-	-	-	-	-	-	N	122	188	7.2	38	1.1	+	Negative	N	>50 ml	N
66	35	M	24	5Y	OHA	-	-	-	-	-	-	N	161	222	8.9	42	1.2	-	Negative	N	<50 ml	NA
67	31	F	29	6Y	OHA/I	-	-	-	-	-	-	N	182	222	8.1	39	1.3	-	Negative	N	>50 ml	NA
68	43	F	27	2Y	OHA	+	-	-	-	-	-	DD	121	157	7.3	29	1	+	Negative	N	<50 ml	NA
69	42	F	24	1Y	OHA/I	-	-	-	-	-	-	N	191	278	10.8	31	1.1	-	Negative	N	<50 ml	NA
70	29	F	31	4Y	OHA	-	-	-	-	-	-	N	202	276	10.6	36	1.3	-	Positive	N	<50 ml	NA
71	45	M	26	2Y	OHA	+	-	-	-	-	-	N	126	202	8.1	33	1.2	-	Negative	N	<50 ml	NA
72	33	F	27	2Y	OHA/I	-	-	-	-	-	-	N	166	288	9.8	38	0.9	-	Negative	N	<50 ml	NA
73	38	F	23	11Y	OHA	-	-	+	-	-	-	N	141	221	8.3	41	1.1	+	Negative	N	<50 ml	NA
74	35	F	28	5Y	OHA	-	-	+	-	-	-	N	116	240	8.1	40	1.2	+	Negative	N	>50 ml	NA
75	43	F	33	7Y	OHA	-	-	-	-	-	-	N	131	244	9.2	36	0.9	+	Negative	N	<50 ml	NA
76	34	F	22	7Y	OHA	-	-	-	-	-	-	N	202	242	9.2	35	1.1	-	Negative	N	<50 ml	NA
77	43	F	31	2Y	OHA	+	-	-	-	-	-	N	108	178	7.8	36	1.2	-	Negative	N	<50 ml	NA
78	42	F	28	8Y	OHA	-	-	+	-	-	-	N	154	211	9.4	34	1.3	+	Negative	N	<50 ml	NA
79	30	F	29	2Y	OHA	-	-	-	-	-	-	N	176	254	10.2	39	1.6	-	Negative	N	<50 ml	NA
80	45	F	24	3Y	OHA	-	-	-	+	-	-	N	154	220	8.4	44	1.6	+	Positive	N	<50 ml	NA
81	39	M	28	3Y	OHA	-	-	-	-	-	-	N	218	329	10.6	40	1.1	-	Negative	N	<50 ml	NA
82	31	F	30	5Y	OHA	-	-	+	-	-	-	N	162	240	9.1	38	1.2	+	Positive	N	<50 ml	NA
83	34	M	24	3Y	OHA	-	-	-	-	-	-	N	132	206	7.6	36	1	-	Negative	N	<50 ml	NA
84	42	F	24	3Y	OHA	+	-	-	-	-	-	N	188	276	9.8	38	1.2	+	Negative	N	<50 ml	NA
85	45	F	26	11Y	OHA	+	-	+	-	-	-	N	211	344	11.3	44	1.8	+	Positive	MRD	<50 ml	NA
86	44	M	33	1Y	OHA	-	-	-	-	-	-	N	97	212	7.7	42	1.1	-	Negative	N	<50 ml	NA
87	34	M	22	8Y	OHA	-	-	-	-	-	-	N	164	311	9.4	36	1.3	-	Negative	N	>50 ml	N
88	40	F	23	10Y	OHA/I	-	-	+	+	-	-	LVSD	192	247	9.8	42	1.6	+	Positive	N	>50 ml	NA
89	44	F	31	4Y	OHA	-	-	+	-	-	-	N	111	162	7.2	33	1.2	+	Negative	NNA	<50 ml	NA
90	36	M	26	1Y	OHA	+	-	-	-	-	-	N	113	174	7.9	31	1.1	-	Negative	N	<50 ml	NA

CONTROL

S/N	Age	Sex	FBS	PPBS	HBA1C	Urea	Creatinine	Urine culture
1	21	M	103	113	6.5	31	1.1	NEGATIVE
2	26	F	98	122	6.8	33	1	NEGATIVE
3	31	M	88	131	6.9	38	1.1	NEGATIVE
4	35	F	96	111	7	29	0.9	NEGATIVE
5	33	F	108	124	7.1	32	1.2	NEGATIVE
6	26	M	101	128	6.7	34	1	NEGATIVE
7	34	F	86	99	6.6	30	1.1	NEGATIVE
8	41	M	98	116	7.2	35	1.3	NEGATIVE
9	42	F	104	106	7.3	36	1	POSITIVE
10	27	F	99	105	7	38	1.1	NEGATIVE
11	31	M	76	92	6.5	40	1.2	NEGATIVE
12	23	M	81	98	7.2	39	1.1	NEGATIVE
13	43	M	84	100	7.1	36	1	NEGATIVE
14	32	F	92	108	6.5	34	1.2	NEGATIVE
15	28	M	89	110	7.3	36	0.8	NEGATIVE
16	44	F	79	96	6.3	33	1	NEGATIVE
17	34	M	108	136	6.5	38	1.1	NEGATIVE
18	22	F	92	100	6.5	29	0.9	NEGATIVE
19	29	M	82	99	6.6	31	1.1	NEGATIVE
20	27	F	74	94	6.9	33	1	NEGATIVE
21	35	M	77	100	7.1	30	1.1	POSITIVE
22	30	M	86	101	7.2	31	1	NEGATIVE
23	35	F	76	101	6.6	33	1.2	NEGATIVE
24	36	M	84	112	6.5	30	1	NEGATIVE
25	24	F	90	110	6.9	40	1.3	NEGATIVE
26	30	F	85	105	6.8	35	1	NEGATIVE
27	31	F	88	106	6.7	33	0.9	NEGATIVE
28	34	F	94	126	7	32	1.1	POSITIVE
29	32	F	97	116	7	35	1	NEGATIVE
30	37	M	85	105	7	35	0.9	NEGATIVE
31	27	M	75	95	7.2	29	0.8	NEGATIVE
32	25	M	83	103	7.3	36	1.1	NEGATIVE
33	38	F	92	122	6.5	38	1.2	NEGATIVE
34	33	F	72	106	6.6	36	1.3	NEGATIVE
35	39	M	79	99	6.5	39	1.1	NEGATIVE
36	40	F	87	107	6.8	37	1.2	NEGATIVE
37	34	F	95	114	7	34	0.8	NEGATIVE
38	28	F	98	108	7	38	1	NEGATIVE
39	21	F	100	120	7.1	30	1.1	NEGATIVE
40	36	M	98	116	7.3	36	1	NEGATIVE

41	37	F	87	115	6.8	35	1.3	NEGATIVE
42	41	M	93	116	7	36	1	NEGATIVE
43	29	M	106	136	7.3	29	0.8	NEGATIVE
44	38	F	89	109	6.6	31	1	NEGATIVE
45	22	F	109	118	6.8	34	1.1	NEGATIVE
46	43	F	88	128	6.7	30	1.2	NEGATIVE
47	26	F	93	114	6.7	36	1.3	NEGATIVE
48	39	M	100	124	6.9	35	1	NEGATIVE
49	42	M	111	133	7	34	1.2	NEGATIVE
50	23	M	92	112	7.1	26	0.8	NEGATIVE
51	28	M	89	119	7.3	31	1.1	NEGATIVE
52	40	M	107	127	7	38	1.2	NEGATIVE
53	45	F	96	125	6.8	35	1	NEGATIVE
54	44	F	73	93	6.9	33	1.1	NEGATIVE
55	36	M	88	118	7	30	0.9	NEGATIVE
56	30	F	76	106	7.1	29	1	NEGATIVE
57	37	F	79	119	7.2	31	1.2	NEGATIVE
58	38	M	89	109	6.6	34	1.1	NEGATIVE
59	39	F	99	116	6.5	32	1	POSITIVE
60	42	F	85	109	6.8	39	1.3	NEGATIVE
61	43	F	91	111	6.9	36	1.1	NEGATIVE
62	30	F	99	116	7	30	0.8	NEGATIVE
63	40	F	100	120	6.8	28	1	NEGATIVE
64	45	F	94	118	6.7	30	0.8	NEGATIVE
65	36	F	99	129	6.5	35	1.2	NEGATIVE
66	27	M	102	125	6.6	30	1	NEGATIVE
67	42	F	108	128	6.6	38	1.1	NEGATIVE
68	22	F	99	118	7	33	1.3	NEGATIVE
69	39	F	104	124	7	35	1	NEGATIVE
70	28	M	96	136	7.1	31	1.2	NEGATIVE
71	37	F	99	116	6.8	36	1	NEGATIVE
72	29	M	89	105	6.5	34	1.3	NEGATIVE
73	38	F	98	104	6.9	38	1.4	NEGATIVE
74	37	F	108	128	7	38	1.1	NEGATIVE
75	26	F	98	124	7.1	35	1.2	POSITIVE
76	23	F	107	134	7.3	36	1	NEGATIVE
77	27	F	83	123	6.5	31	1.1	NEGATIVE
78	36	F	91	121	6.6	33	1.2	NEGATIVE
79	40	F	101	131	6.7	34	1.3	NEGATIVE
80	29	F	106	136	6.8	38	1	NEGATIVE
81	40	F	98	138	6.9	40	1.3	POSITIVE
82	24	F	106	125	7	37	1.1	NEGATIVE
83	39	F	74	96	7.1	38	1	NEGATIVE
84	30	F	81	101	7	39	1.3	NEGATIVE

85	38	F	92	124	7.3	32	0.8	NEGATIVE
86	28	F	74	125	6.5	36	1.1	NEGATIVE
87	37	F	76	126	6.8	38	1	NEGATIVE
88	27	F	88	135	6.9	35	1.1	NEGATIVE
89	25	F	89	108	7	34	1.1	NEGATIVE
90	30	F	98	111	7.1	33	1.1	NEGATIVE